

Albion Regulations:
Control of Spray Inoculation

R 71-16



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H.O.
Samuel T. Lawton, Jr., BMA

R71-16
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R-

R71-16

PROPOSAL

R 71-16
Asbestos and Spray Insulation

EXPLANATION OF PROPOSED REGULATIONS

Science is pointing its medical finger at asbestos and describing the "miracle fiber" as a mineral whose commercial blessings can exact a rather costly toll. Asbestos insulation and factory workers exposed to heavy concentrations of asbestos fiber over prolonged periods have suffered devastating consequences. They are visited with an alarmingly high incidence of asbestosis, which produces a diffuse fibrosis of the lower lung, of lung cancer and of an extremely rare cancer of the lining of the lung and abdomen, mesothelioma. The rates of lung cancer are extraordinarily high especially among asbestos workers who are smokers, indicating the synergistic potential of the mineral fiber.

The medical evidence is indefinite as to why asbestos is carcinogenic, with three theories in vogue: (1) The fibers once ingested or inhaled act as a continuous physical irritant, inducing a tumor after 20 to 30 years; (2) The fibers contain carcinogens such as benzo(a)pyrene; (3) Fibers accumulate in the lung and remain immobilized as "asbestos bodies" which cause cancer upon disintegration.

Similarly, while the adverse effect of prolonged exposure to heavy concentrations of asbestos is clearly documented, the medical data is disturbingly vague as to the results of long-term exposure to low and moderate concentrations. Nor is there a clear dose-time relationship for exposure to heavy concentrations. In short, as with many other environmental contaminants, the public has discovered too late that asbestos is potentially dangerous and too little of the extent of that threat.

But two depressing facts are agreed upon: That millions of Americans are likely to contain "asbestos bodies" in their lungs and that asbestos fiber once released is relatively indestructible. That is, the qualities of durability and of resistance to normal chemical and physical stress, which make asbestos such a commercial success, render it potentially dangerous to present and future generations because of the tendency of the fibers to accumulate in the atmosphere and in the lungs.

In light of these facts, the PCB has proposed the nation's most comprehensive set of asbestos controls. To ban all uses of asbestos would be "alarmism" at its most clamorous. But ignoring

the need to explore and, perhaps, to adopt controls for the significant contributors to a swelling volume of asbestos fiber in our air would constitute a foolish complacency.

To this end, the suggested regulations would identify these "significant contributors" as the commercial use of asbestos in construction work; the demolition of asbestos-containing structures; the manufacturing and processing of asbestos-containing products; the transportation of certain asbestos-containing products; the disposal of asbestos-containing waste; and the use of asbestos in the brakes of millions of vehicles.

Part II would impose several general requirements upon all commercial construction, repair, alteration, demolition, manufacturing or processing activity from which asbestos fiber is discharged into the environment. The persons responsible for such activity must obtain a permit from the EPA. The permit system will serve to produce the advanced assurance of compliance with the applicable control regulations and will thus reduce the cost and burden of in-the-field enforcement.

In addition these commercial activities would have to provide facilities to prevent the removal of asbestos fibers from the site of such activity on the clothing of employees. Indeed, this poses a rather new concept of air pollution control. Those who have died from lung ailments induced by long, close association to an asbestos worker may be powerful proof of the need for novelty.

Finally, Part II would provide for careful waste disposal procedures, as wind-blown refuse heaps and scrap piles contribute to the volume of asbestos fiber in our air. The simple steps for control eliminate any excuse for such dangerously sloppy housework.

Part III would prohibit the spraying of asbestos-containing insulation and fireproofing. Experience has indicated that procedural safeguards are inadequate and their enforcement is an overtaxing burden. Chicago has banned spray asbestos, and New York city and Philadelphia are considering doing so.

Procedural safeguards designed to reduce fiber emissions would be required to spray non-asbestos insulation. The biological effects of these fibers is unknown. Prudence would seem to dictate reasonable efforts to soften the potential future blow from spray fiber aspiring to asbestos' status.

Part IV suggests that various asbestos-related activities at construction sites be done within an enclosure. It would also require that asbestos construction products be so installed as to preclude the emission of fiber into the circulating air.

Part V expresses, ironically, what may be a key reason for banning spray asbestos. It would require that procedural safeguards be pursued in order to demolish a structure whose destruction would otherwise expose members of the public to asbestos fiber. Clouds of dust are often raised by demolition work. The facts that great numbers of persons may be exposed to this dust in urban areas and that the post-war generation of high-rises were the first buildings to use asbestos insulation extensively speak in favor of some such regulation.

Part VI suggests an emission standard for manufacturers of asbestos products. The standard is believed to be within the capture efficiency of baghouse filtration. The proposed boundary standard is thought to be a method for preventing dilution of exhaust gases.

The standard would measure only fibers longer than five microns. The justification for this seems to be the administrative convenience of avoiding difficulties in measuring fibers from .5 to five microns in length. Medical evidence has indicated that the very small fibers may be the most dangerous. And the Board will seek extensive evidence on this point.

Part VII would prohibit the use of brake lining in vehicles manufactured after January 1, 1975 and sold for use in Illinois.

If brake lining decomposition emits asbestos fiber, then the tremendous number of vehicles using asbestos, and the impracticability of controlling these numerous small emission sources would appear to speak to the need for a product ban. The prohibition is carefully worded to avoid the necessity of fitting vehicles produced prior to 1975 with non-asbestos brakes. It is contemplated that the ban will necessitate design changes, probably to disc brakes, and it is thought that requiring the individual vehicle owner to undergo this expense is unreasonable. The time lag of over three years is thought to be sufficient for vehicle manufacturers to accomplish any necessary design changes.

Proposed Regulation Only
June 9, 1971
CHAPTER V: TOXIC SUBSTANCES
ASBESTOS; SPRAY INSULATION

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PART I: INTRODUCTION

101. Authority

Pursuant to the authority in Sections 9, 10 and 13 of the Environmental Protection Act which empower the Board to adopt regulations forbidding the "sale, offer, or use for reasons of air pollution control" of any article, and to set "standards specifying the maximum amounts or concentrations of various contaminants that may be discharged into the atmosphere"; and to set standards for the issuance of permits for the operation of any equipment or facility capable of causing or contributing to air pollution; and to promulgate "conditions regarding the... use of any....article determined by the Board to constitute an air pollution hazard"; and to adopt effluent standards limiting the amounts of contaminants that may be discharged into the water of Illinois, the Board adopts the following rules and regulations:

102. Policy

It is the purpose of the General Assembly in adopting the Environmental Protection Act to maintain and enhance the purity of the air and water of Illinois in order to protect health, welfare and the quality of life. Accordingly, it is hereby determined that the uncontrolled discharge of asbestos fiber into the environment tends to severely endanger the public health and welfare and that the uncontrolled spraying of fiber-containing materials unreasonably interferes with the enjoyment of life and property.

It is the purpose of these regulations to control the amount of asbestos fiber released into the environment from the major sources of emission. Such control is necessary not only to protect those members of the public who are in proximity to heavy concentrations of asbestos fiber but also to safeguard the health of future generations endangered by the continuous discharge of asbestos fiber which can be toxic and which tends to be cumulative both in the atmosphere and in the human body.

Where health can be protected by the adoption of an emission standard or of procedural safeguards, such a course has been pursued. In those instances where restriction is unfeasible because of the tremendous number of small, uncontrollable emission sources (automotive) or because of the unusual nature of the emission source (spray asbestos at construction sites) a product ban has been resorted to. These prohibitions have been made with full consideration given to the available alternative materials.

It is also the purpose of these regulations to reduce the emission of non-asbestos particulate from spray-fireproofing and

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insulation. To protect against these emissions, the biological effects of which are unknown, procedural safeguards have been enacted.

103. Definitions

- Agency - The Illinois Environmental Protection Agency
- Asbestos - Any fiber or any mixture containing fiber of hydrated silicate mineral, which, on the basis of its crystalline structure, falls into one of two categories:
- (1) pyroxenes - chrysotile fiber;
 - (2) amphiboles - crocidolite, amosite, tremolite, actinolite or anthophyllite fiber.
- Board - The Pollution Control Board of Illinois.
- Commercial Activity - Any activity done for hire or having financial profit as a primary aim.
- Debris - Asbestos-containing waste produced by the demolition of a structure.
- Spraying - The pneumatic application of material used for fireproofing or insulation.
- Waste - Any asbestos-containing matter which has been or is intended to be discarded.

PART II: General Requirements

No commercial activity, not otherwise hereinafter prohibited, involving the discharge of asbestos fiber into the environment from the construction, alteration, repair or demolition of a structure or from the processing or manufacturing of asbestos-containing products, shall be conducted unless the person or entity in charge of such activity complies with the following regulations:

201. A permit shall be obtained from the Agency to engage in such activity. Before obtaining such permit, the applicant shall demonstrate compliance with the following safeguards:

(a) The designation of personnel to exercise full-time supervisory authority over all aspects of the activity from which the release of asbestos fiber into the environment could result, in such a manner as to insure compliance

with the pertinent asbestos control regulations.

(b) That each employee engaged in such activity has completed or will complete a satisfactory course of instruction on the potential hazards of exposure to asbestos fiber including the precautions that must be observed to prevent or restrict the dispersion of asbestos fiber into the environment.

(c) That such additional safeguards as are hereinafter specifically required and such precautions consistent with the purpose of these regulations as the Agency deems necessary under the circumstances shall be complied with.

202. Facilities shall be provided and procedures instituted and supervised that prevent the removal of asbestos-containing material from the site on the clothing of employees.

203. Asbestos-containing wastes shall be immediately vacuumed or otherwise collected where vacuuming is impossible, and shall be placed in a container resistant to tearing or breaking under normal handling conditions, which shall be tightly sealed and clearly marked as containing asbestos waste. Such containers shall be placed directly upon a vehicle for disposal by burial at an approved site.

Section 203 shall not apply to the demolition of a structure, except as provided in Section 501(a), (d) and (e) or to the disposal of sludge waste except as provided in Section 603.

PART III: SPRAY FIREPROOFING AND INSULATION

301. The spraying of asbestos-containing material is prohibited.

302. Non-asbestos particulate matter used for insulation or fireproofing shall not be sprayed unless the following procedures are taken:

(a) Before the start of spraying operations, all floor areas shall be swept clean and cleared of all objects, material and equipment other than that employed in the application of the fiber-containing material, or, in the alternative, all such objects, material and equipment shall be covered with plastic or plastic-coated tarpaulins.

(b) The entire floor or area to be sprayed shall be enclosed with plastic or plastic-coated tarpaulins in a manner which shall preclude the escape of fiber-containing material from the enclosure. All interior open areas such as elevator shafts and stairwells shall be enclosed in a manner which shall prevent the escape of fiber-containing

material from the working area. "Stack effect" of the shafts, stairwells and comparable spaces shall be considered in providing proper enclosures.

(c) Regardless of adherence to the stated control practices, any visible emission of fiber-containing materials beyond the building perimeter will be considered a violation.

(d) In case of visible emission of fibrous material from the construction site, immediate steps shall be taken to cause the cessation of such emissions by either effective control measures or work stoppage at the source of the emissions. There shall then be immediate and complete cleanup of all material that has escaped the construction site, and measures shall be taken that will insure that no further dispersal of any fibrous material into the atmosphere can occur.

(e) The entire sprayed area, all ledges and surfaces including tarpaulins within the enclosure shall be thoroughly vacuumed upon completion of the spraying operation and immediately before the enclosure is dismantled. The vacuum cleaner shall contain a strong, single-service, disposable inner bag of durable material which shall be removed from the vacuum cleaner and tightly sealed. The bag shall then be placed in a container and disposed of at an approved site in accordance with Section 203.

(f) All areas used for opening bags containing fibrous material or the changing of hoppers shall be enclosed so as to prevent the escape of such material from the immediate area in which such activity occurs.

(g) Any plenum or other structure coated with or containing fibrous insulation and used in the circulation of air in a building shall be thoroughly cleaned of all debris and waste insulation. All applied fiber-containing insulation within a plenum or duct shall be coated with a sealant which precludes exposure of the fiber-containing material to the circulating air.

PART IV: CONSTRUCTION, ALTERATION AND REPAIR OF STRUCTURE

401. (a) The cutting, trimming, fitting or stripping of asbestos-containing material in the construction, alteration or repair of a structure which is done at the site of such structure shall be conducted within a special enclosure designed to preclude the escape of asbestos fiber from the immediate area of such enclosure.

(b) The mechanical exhaustion of dust from such enclosure to the ambient air is prohibited unless such exhaust system is equipped with a fabric filter for dust collection.

402. Asbestos-containing material applied in the construction, alteration or repair of a structure shall be coated with a sealant, provided with a cover or installed in some other manner so as to preclude exposure of the asbestos fiber to the circulating air.

PART V: DEMOLITION

501. Where the risk of public exposure to asbestos fiber from the dislodging of asbestos-containing materials is present, no demolition of a structure shall be initiated unless all safeguards necessary and practicable to reduce the emission of dust are taken.

Such procedures shall include, but are not necessarily limited to:

(a) Boilers and pipes insulated with asbestos-containing material shall be wetted and stripped before toppling of walls is begun. This procedure shall be followed, where practicable, as to all other asbestos-lined surfaces. Such asbestos waste shall be immediately bagged and disposed of in accordance with Section 203.

(b) Demolition by toppling of walls is prohibited except within an enclosure approved by the Agency as part of the permit granted for demolition of that structure.

(c) Before the demolition of any section of the structure, adequate wetting to suppress the dust shall be employed.

(d) Asbestos-containing debris shall not be dropped or thrown from any floor but shall be transported by dust-tight chutes or buckets. Asbestos-containing debris in chutes or buckets shall be sufficiently wetted to preclude dust dispersion at the point of discharge.

(e) All asbestos-containing debris shall be thoroughly wetted before loading into trucks, other vehicles or containers. During transport such waste shall be enclosed or covered so as to prevent dust dispersion. Asbestos-containing debris shall be disposed by burial at an approved site.

502. In the event asbestos-containing matter becomes air-borne beyond the property boundaries or in the event freezing temperatures preclude the use of water for dust suppression, demolition work shall cease until adequate alternate measures can be taken.

PART VI: MANUFACTURING

601. (a) No factory, plant or enterprise which engages in the processing or manufacturing of any asbestos-containing product shall emit asbestos fiber into the ambient air in excess of .5 fiber per cubic centimeter of air. The ambient

air quality at the boundary of such factory, plant or enterprise shall not exceed .05 fiber per cubic centimeter of air.

(b) Only fibers of 5 microns or greater in length with a length to breadth ratio of 3 to 1 shall be counted.

(c) Sampling shall be determined by the membrane filter method with phase contrast microscopy at 430 X magnification.

602. No water used in processing or manufacturing which contains concentrations of asbestos fiber shall be discharged into sewage systems or to the waters of Illinois.

603. Waste sludge containing asbestos collected from settling ponds shall be enclosed during transport and shall be disposed by burial at an approved site.'

PART VII: ASBESTOS-CONTAINING PRODUCTS

701. No product which may emit asbestos-fiber during its transportation shall be transported unless such product is enclosed in such a manner as to preclude the emission of asbestos fiber into the ambient air.

702. The use of asbestos in the brake lining of vehicles manufactured after January 1, 1975, and sold for use in Illinois is prohibited.

R71-16 ASBESTOS REGULATIONS

PUBLIC COMMENT LISTING

Date Filed:

1. 10/14/71 City of Chicago, Dept. of Environmental Control
by H.W. Poston
2. 10/27/71 Rexene Polymers Company by M.G. Kaijala
3. 11/4/71 Armstrong Cork Co. by Jon W. Tryon
4. 11/18/71 Illinois Regional Insulation Contrators by
Jay C. Golinkin
5. 12/7/71 United States Mineral Products Co. by Harry S.
Gropp w/enclosures



CITY OF CHICAGO

department of environmental control

October 14, 1971

R71-16

RICHARD J. DALEY
MAYOR

H. W. POSTON
COMMISSIONER

P.C.# 1

Mr. David Currie
Chairman
Illinois Pollution Control Board
189 West Madison Street
Chicago, Illinois

Dear Mr. Currie:

I regret that I will not be able to give my statement in person at your hearing on the important topic of asbestos to be held on October 15th. However, please find attached a copy of my statement for entry into your record.

As you know, the City held extensive hearings on this topic last Spring and, should you wish, we will be pleased to make available material from that record. I believe that one of your associates has already perused the record and extracted certain portions of the testimony. We certainly are pleased to cooperate in this way with your efforts to enhance the quality of our environment.

Very truly yours,

H. W. Poston
g.g.x.

Commissioner

Enc.

STATEMENT ON ASBESTOS BY COMM. H. W. POSTON BEFORE THE
ILLINOIS POLLUTION CONTROL BOARD-OCTOBER 15, 1971

Asbestos is of critical importance not only because of its demonstrated relationship to industrial health problems but because of its larger role in the overall problem of particulate emissions to the ambient air.

We, in the City of Chicago, are concerned with finding ways and means of effectively controlling particulate emissions within the city. A recent effort toward this objective was the passage of an ordinance prohibiting use of asbestos-contained spray coatings in the City of Chicago after January 1, 1972.

Special public hearings were held in the spring of 1971 by the City Council Committee on Environmental Control. These hearings demonstrated the need for taking action to eliminate this source of hazard to the citizens of Chicago. Eminent scientists such as Dr. Irving Selikoff, Dr. George Wright and Dr. Louis Cralley presented documented evidence of the largest source of asbestos.

Perhaps the largest source of asbestos emissions to ambient air results from the activities of the construction industry. Seventy percent of the asbestos used in this country is used in the construction industry, and within that industry the most prevalent means by which asbestos fibers are introduced into the ambient air are the activities associated with the spray coating applications.

Therefore, after establishing the health implications of asbestos and determining that one of the major sources of asbestos in the ambient air resulted from spraying operations during building construction, the problem became one of control.

The most obvious method of control is containment, coupled with adequate cleanup of the work area; however, substantial evidence was presented to confirm that containment as a method of control was unsuccessful. Since no practical means are available for containment, the alternative was to consider eliminating the source of the problem.

Evidence was presented demonstrating that asbestos-free spray coatings are available and that these coatings are as effective and as economical as any on the market. This position was supported by business and labor. Based upon this evidence, the City Council passed an ordinance on May 21, 1971 banning the application of spray coatings containing asbestos fiber. The ordinance becomes effective January 1, 1972.

In conclusion let me simply say that we in Chicago are working diligently to reduce particulate emissions. The ordinance banning the use of asbestos-containing spray fibers was one portion of our overall program toward this objective.

REXENE POLYMERS COMPANY

(FORMERLY REXALL CHEMICAL)

LOCATION
INTERSTATE 55 AT JOLIET ARSENAL ROAD
JOLIET, ILLINOIS
(815) 422-5541

MAILING ADDRESS
P. O. BOX 550
JOLIET, ILLINOIS 60434

R71-~~14~~
16
October 25, 1971

RECEIVED

OCT 27 1971

Illinois Pollution Control
Board
129 West Madison
Chicago, Illinois 60602

POLLUTION CONTROL BOARD

p.c.# 2

Dear Sirs:

It has come to our attention that you are considering a proposal to limit treatment plant effluents to 750ppm dissolved solids irrespective of the solids level of the ground water being taken into the plant. Rexene Polymers Co., would like to voice its objection to this proposal, as being unrealistic.

As is true of most chemical plants, our cooling tower, which is our sole source of process cooling water, concentrates ground water solids by a factor of three to four. This means a substantial addition of completely innocuous, mineral solids (calcium and magnesium mainly) to our effluent stream. These are the same solids that are in our drinking water and it seems unrealistic to consider them pollutants. The concentration of these solids brings the cooling tower blowdown dissolved solids to twice your proposed level and the magnitude of the blowdown is sufficient to raise our total effluent well above your limit.

Our plant was designed to operate on well water exclusively and we have no facilities to use water from any other source. We initially operated a once through cooling water system, but in order to conserve water and help maintain the underground water table, we added a cooling tower.

Our polymerization process is very dependent upon high quality water, devoid of trace contaminants. We do not have the facilities nor the technology to clean up the water of the Des Plaines River sufficiently for use in our process. Your proposed regulations would force us to use river water and, as stated, this is not feasible.

The subject proposal is not consistent with your previously professed policy of demanding only that which is technically and economically attainable. In our view, your new

proposal is not meritorious and we respectfully request that you carefully consider our objection to it.

We will be happy to supply any additional information that you require relative to this objection.

Very truly yours,

REXENE POLYMERS COMPANY

A handwritten signature in dark ink, appearing to read 'M. G. Kajala', is written over the typed name.

M. G. Kajala, Technical
Manager

MGK/cg



AREA CODE TELEPHONE
717 397-0611

LANCASTER PA 17604

November 3, 1971

RECEIVED
NOV 4 1971

POLLUTION CONTROL BOARD

P.C.# 3

AIRMAIL

Pollution Control Board
State of Illinois
189 W. Madison Street
Suite 900
Chicago, IL 60602

Gentlemen:

Re: Asbestos Hearing - October 15, 1971 R71-16
Fielding Museum - Chicago, Illinois

As it was my opportunity to attend the above referenced hearing, I would like to make a few comments to clarify the position of Armstrong Cork Company. It is not our intent to weigh the serious economic impact of the suggested regulations with the health and welfare of the multitude of employees directly and indirectly involved within the asbestos industry, but rather we would like to reflect upon the importance of a tempered attitude in the implementation of any rules and regulations which may be promulgated.

Armstrong Cork Company is undertaking continuous and concentrated efforts to eliminate exposure of our personnel to any potential hazard from asbestos. Various collection techniques coupled with high grade housekeeping exemplify the double-barreled policy being pursued but the bulk of the regulations as applied to Armstrong Cork Company require time to ensure adequate implementation.

To be specific I refer to Part II, Section 201(a) "designation of personnel to exercise full-time supervisory authority over all aspects of the activity from which the release of asbestos fiber into the environment could result, in such manner as to ensure compliance with the pertinent asbestos regulations." Implementation of such a program demands schooling and extensive familiarity with regulations which have not as yet been put into complete effect. As the economic impact upon the final product is self-evident, but the recruitment and training of supervisory personnel is not that which can be done or even in a few short months, rather many months of time would be to create a staff as envisioned.

November 3, 1971

Subparagraph (b) mandates a "satisfactory course of instruction on the potential hazards of exposure to asbestos fiber" and again we are up against that which is at present unknown as even being a hazard when concerned with the small quantities of asbestos fiber which is currently utilized in our industry. Might we assume that the State is willing to bear the burden of compiling information which would better illustrate such hazards?

Section 202 mandates a dramatic expansion in plant facilities; the acquisition of land and construction of new facilities is not such which can be done immediately but a lag time of two to three years can be foreseen.

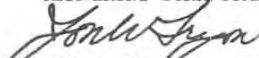
Section 203 refers to the disposition of "asbestos containing waste" at "an approved site." Solid waste landfills for the disposal of potential deleterious materials is becoming one of the most dramatic and insurmountable problems in waste control now within the United States, yet this regulation has blithely subsumed such difficulties. We have experienced that the acquisition and state approval for preparation of a solid waste landfill has taken two to four years in the two east coast states in which we have recently completed such action. The rules and regulations allowing for such landfills are being increasingly restrictive. Although the above problems may seem rather procedural in nature we submit that they are realistic, time-consuming and expensive and not capable of solution in less than three to four years.

Certain witnesses at the subject hearing alluded to the practicality of avoiding a scientific sampling technique to determine the presence or absence of asbestos fibers within an industrial atmosphere. We would suggest that the mere visual determination of dust existing within work areas represents a patently unfair and inaccurate determination of the actual presence of deleterious particulate. Manufacturing operations have heavy concentrations of nonhazardous dust which would distort the validity of macroscopic determinations. The Pollution Control Board must weigh the alleged potential hazards of asbestos with the hazard of depriving individuals and families of their livelihood. To suggest that the mere presence of dust, regardless of its composition, should be the substantive evidence to resolve the aforementioned dilemma represents a vacation from lucidity. Periodic grab samples scientifically analyzed for concentrations of deleterious fibrous material remain the fairest testing procedure for use within the vinyl asbestos tile industry.

We are most appreciative of the time and opportunity to present our position on a matter of such vital importance to our continued presence within the vinyl asbestos tile industry.

Respectfully submitted,

ARMSTRONG CORK COMPANY



Jon W. Tryon
Attorney

ILLINOIS REGIONAL INSULATION CONTRACTORS

SUITE 1533 • 220 N. LA SALLE STREET • CHICAGO, ILL 60601 • CODE 312-332-7946

November 18, 1971

Illinois Pollution Control Board
Suite 900, 189 West Madison St.,
Chicago Illinois 60602.

P.C. #4

Attention: Mr. Samuel Lawton

Re: PROPOSED REGULATION
CHAPTER V, DATED 6/9/71

Gentlemen:

In response to your invitation for comments relating to the proposed regulations for environment control, this letter is presented on behalf of the Illinois Regional Insulation Contractors. This organization is comprised of the heat and frost insulation contractors, operating in this territory (Chicago, Peoria, etc.). We install insulation materials on pipes, ducts, equipment, etc., in all types of industrial, commercial and institutional buildings and approximately 75% of all such work in the State of Illinois is performed by our members. Our employees constitute one of the recognized basic trades in the construction industry.

We share your concern with the potential health hazard of fibre containing insulation materials as covered in Part III, Paragraph 302 g, of the subject regulation, designed to "preclude" emission of fibre containing material into the circulating air of duct and plenums. However, the method designated (i.e. coating with a sealant) does not provide the procedural safeguard intended by this proposed regulation. It will not preclude the emission of such particulate into the circulating air.

It is only logical that use of any materials introduced on a smooth metal surface will create an airborne particle flight situation. Since metal surfaces create a clean, clear, and smooth flow condition, the use of fibrous materials does change the flow condition and must cause some entrainment of particulate material.

In support of this position we offer the following:

- 1) The common application of coated duct lining will not preclude entrainment of fibrous particles into the air. This is also true of ductwork fabricated from fibrous materials.
- 2) The integrity of coating is further diminished by mechanical factors not under the control or surveillance of the architect, engineer, or contractor. These factors include handling in the shop, in transit, in storage, and during installation.

- 3) The emissions are greatest at raw edges which occur at butt joints, elbows, access doors, etc. When installation procedures and conditions are less than optimal the incidence of entrainment is accelerated.
- 4) It must be remembered that any purification of the air (i.e. filtering) is done at the AH Unit prior to the introduction of the fibrous particles. Therefore, when duct systems have internal fibrous linings (or are made of fibrous materials), the most effective means of protection would be to insert an inner liner of metal mesh (or some similar materials). This liner should not be subject to erosion from the constant air stream or from mechanical damage and should mechanically prevent entrainment.

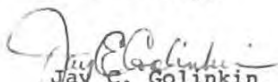
It could be argued that ideally the most effective solution to eliminate this potential hazard would be to prohibit the use of fibrous materials on the interior of air ducts and plenums. While it is not our position to invoke such a solution in our judgement the current extent of the use of these materials is excessive and unnecessary.

Presently entire duct distribution systems are now lined from start to finish, where all of the accoustic advantages could be accomplished with only a small amount of duct lining placed in the most judicious locations. The balance of the system is lined to provide thermal protection and advantages, which advantages could just as well be accomplished by external insulation. However, many architects find it simpler and more convenient to line the ducts throughout, although they have no way to effectively control the quality of the installation. Current practices admittedly are more economical, but the savings are not substantial. However, how significant are marginal economic considerations where the quality of the environment is under consideration?

We would strongly suggest that your committee take the precautionary measures we have outlined where lining for accoustic purposes is necessary, and that you also recommend that duct lining (and/or fibrous ducts) not be used beyond the point where the accoustic requirements are fulfilled.

We hope your attention in this matter will be of great benefit for this and future generations.

Sincerely,


Jay C. Golinkin
Chairman
Standard and Research
Committee.



UNITED STATES MINERAL PRODUCTS COMPANY

General Office and Plant
STANHOPE, NEW JERSEY 07874

Tel. 347-1200 Area Code 201
Cable: CAFCOINSUL New York

December 6, 1971

RECEIVED
DEC 7 1971

Illinois Pollution Control Board
189 W. Madison St.
Chicago, Illinois

POLLUTION CONTROL BOARD

Attn: Mr. David P. Currie, Chairman

Subject: Proposed Asbestos and Spray Insulation Regulations #871-16

Dear Mr. Currie:

p.c. # 5

After reviewing the proposed regulations, and basing our comments on two and a half years work in conjunction with Dr. Selikoff of Mt. Sinai Environmental Sciences Laboratory and with public agencies throughout the United States and Canada, we feel that in some sections the regulation is too loose, and can be easily circumvented, and in other areas is too restricted for products that have been thoroughly researched medically.

In the first paragraph of 102 - Policy and in paragraphs 302 (a); (b) twice; (c); (d); twice, (f) and (g) three times the words "fiber-containing" or "fibers" occur. We suggest the substitution or deletion of words as follows:

102 - delete "fiberecontaining"

302- (a) (b) (c) (d) (f) and (g) delete and substitute "spray fireproofing and insulation" as used in paragraph four of 102 - Policy.

In this way, you include all materials on the market now and those in the future. As the regulations now reads, two complete product groups are not covered. Also, in paragraph 103 - Spraying, we suggest the deletion of the word "pneumatic". Again, if a product is sprayed other than pneumatically, you are not covered.

We feel that in paragraphs 302 (f) and (g) the regulation is too restrictive if, and this is a big if, the product has been researched thoroughly, and classified. In the case of our CAFCO BLAZE-SHIELD TYPE D C/F material, we suggest either deletion of these two paragraphs or some qualification paragraph.

ISM

MINERAL FIBER AND FOAMED PLASTIC PRODUCTS FOR HIGH AND LOW TEMPERATURE
INSULATION, CEILING SYSTEMS, PACKAGING, FLUTATION, DISPLAY AND CONSUMER ITEMS.



CAFCO SPRAYED FIBER PRODUCTS FOR FIREPROOFING, INSULATION
OF BUILDINGS AND EQUIPMENT, AND ACOUSTICAL TREATMENT.

December 6, 1971

In justification of this suggestion, we have enclosed a booklet covering the medical ecology of this product. The Industrial Health Foundation classifies Type D C/F as "nuisance dust", and biologically inert as concrete dust, plasterboard dust, and acoustical tile dust etc.. We think it is important and significant to point out that Dr. Gross of IMF has published more books, papers and works on this subject than any other doctor in the world. You will note that our product has been analyzed by electron microscopy at Mt. Sinai and is 100% asbestos-free.

In addition, we have enclosed a dusting and flaking test report run on Type D C/F without any sealer. The test is in accordance with General Services Administration specifications and requirements for all Federal Buildings having air handling plenum chambers. Also, we have enclosed a list of jobs where Type D C/F has been used. This list represents over 40,000 square feet and covers such critical areas as New York, Philadelphia and Chicago. The non-crystalline refractory fibers have been manufactured at our plant for 96 years without any medical problems.

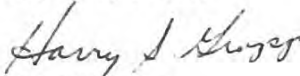
In summary, we have an excellent product that is safe for the public and the workers and is qualified for fireproofing and insulation. It is tested and listed by Underwriters' Labs for fire-resistance and has the approval of all agencies where it has been submitted.

Thank you, and the Illinois Pollution Control Board for your review and consideration of our requests.

HSG/eq

Very truly yours,

Encl. Medical Book on D C/F
C-270
C-636


Harry S. Gropp
Sales Manager
Architectural Products

cc: WDM
File

UNITED STATES MINERAL PRODUCTS COMPANY

STANHOPE, NEW JERSEY 07074

PHONE 201 - 347-1200

CAFCO BLAZE-SHIELD TYPE D C/F JOB LIST

<u>JOB NAME</u>	<u>LOCATION</u>	<u>CAFCO CONTRACTOR</u>
Disneyworld	Taft, Florida	American Bridge
Genesee County Community College	Datavia, New York	Arch. Mat. of Rochester, Inc.
State Office Building	Watertown, New York	"
Niagara Community College	Niagara Falls, N.Y.	"
Girard Trust Company	Philadelphia, Pa.	Wm. Armstrong & Sons
St. Ignatius Home	Philadelphia, Pa.	"
Philadelphia Electric	Philadelphia, Pa.	"
West Chester College	West Chester, Pa.	"
230 West Monroe Building	Chicago, Illinois	Asbestos & Magnesia Mat. Co.
West Side Junior College	Chicago, Illinois	"
Anaconda Copper	Anaconda, Montana	E.J. Bartells Co.
Sears	Columbus, Ohio	Buel Gatterdam Co., Inc.
State University College	Purchase, New York	Colletti & Laguidara, Inc.
Ardsley Shopping Center	Ardsley, New York	"
Howard County Community Hospital	Kokomo, Indiana	Commercial Insulation & Acoustics, Inc.
Wright Elementary School	Altoona, Pa.	A.J. Confer Co., Inc.
Worcester Center	Worcester, Mass.	Francis P. Connor, Inc.
New Hampshire Fire Insurance Bldg.	Manchester, N.H.	"
Washington Plaza	Stamford, Conn.	Engineered Systems, Inc.
Stamford Offices	Stamford, Conn.	"
Twin Lakes Shopping Center	Wichita, Kansas	Halsey-Tovis, Inc.
Eastern Hills Mall	Clarence, New York	Hudson Plastering Corp.
Cornell University	Ithaca, New York	Mancini & Klinchuck
Yellow Pages Directory Bldg.	Lynn, Mass.	Manganaro Bros., Inc.
Hanover Jr. High School	Hanover, Mass.	"
World Trade Center	New York City	Mario & DiBono Plastering Co.
One Penn Plaza	New York City	"
Ford Building	New York City	"
20 Cortland St.	New York City	"
New York Telephone	New York City	"

<u>JOB NAME</u>	<u>LOCATION</u>	<u>CAFCO CONTRACTOR</u>
Squibb Building	Lawrenceville, N.J.	T.J. McGlone & Co., Inc.
Playboy Club	Vernon, N.J.	"
Kresge International	Troy, Michigan	Service Art Plastering Co.
5 Story Office Building	Southfield, Michigan	"
Burroughs Corp.	Detroit, Michigan	"
Canton High School	Plymouth, Michigan	"
Down River Savings	Taylor, Michigan	"
Sterling Heights High School	Sterling Heights, Mich.	"
Ford Assembly Plant	Dearborn, Michigan	"
North Hospital	Baltimore, Maryland	Stebbing Company
Archdiocese Office Building	Philadelphia, Pa.	Whitman Bros., Inc.
J.C. Penny Company	Madison, Wisconsin	Wisconsin Acoustics & Fireproofing Co.
Hollywood Mall	Hollywood, Florida	Lotsaich Company
Eastern Airlines	Miami, Florida	"
Lehigh University	Bethlehem, Pa.	Duggan & Marcon, Inc.
Pennsylvania University	University Park, Pa.	"
New York Thruway Headquarters	Albany, New York	Davis Acoustical Company
John Hancock	Boston, Mass.	James H. Boyle & Son, Inc.
Mutual of New York	Syracuse, N.Y.	Colletti & Laguidara, Inc.
Post Office Facility	Merrifield, Va.	Krafft-Murphy Company
Maryland State Court & Office Center	Falls Church, Va.	C.J. Coakley Company
Lewis Gale Hospital	Salem, Va.	Shields, Inc.
E.T.S. Project	Princeton, N.J.	Wm. Armstrong & Sons, Inc.
Physical Education Bldg.	Harrisonburg, Va.	O'Ferrall, Inc.
Madison College	Philadelphia, Pa.	Wm. Armstrong & Sons, Inc.
KYW - TV Station	Philadelphia, Pa.	"
Ostophalic Hospital	Reading, Pa.	Whitman Bros., Inc.
St. Joseph's Hospital	West Haverstraw, N.Y.	Giamboi Fireproofing Corp.
Orange & Rockland Utilities, Inc.	Middletown, N.Y.	Right Finishing Co., Inc.
Orange Plaza		



KODARAS ACOUSTICAL LABORATORIES

DIVISION OF MICHAEL J. KODARAS, INC.

75-02 51st AVENUE/ELMHURST, NEW YORK 11373/TELEPHONE 212-446-1550

REPORT

RECEIVED
DEC 7 1971

PRODUCT: 7/8" THICK CAFCO BLAZE-SHIELD CF TYPE D

TESTED FOR: United States Mineral Products Company

TEST NO. KAL-855-1-69-R

DATE: December 17, 1969

TEST METHOD: The test method used in obtaining the data is in strict conformance with General Services Administration Public Buildings Service Interim Guide Specification No. 224-3A, Nov. 1964 entitled "Sprayed Fire Protection", Section (6) Dusting.

The test apparatus consisting of blower, system filter, test specimen dust collecting cloth and connecting ductwork was operated in a sealed test chamber until the air in the chamber was reasonably free of dust particles 5 microns and larger. The test specimen was then inserted in the test duct (downstream of the system filter) so that the face of the specimen and inside face of the duct were on the same plane with air velocity parallel to the face of the test specimen.

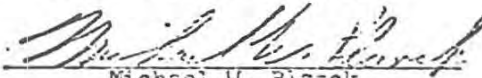
The specimen was purged for 6 hours at an impinging air velocity of 800 feet per minute and at an issuing velocity of 800 feet per minute. The dust collecting cloth was weighed and installed across the duct downstream of the specimen. After 24 hours at the 800 feet per minute impinging air flow, dust collecting cloth was weighed again. The difference in weight was noted and this represents total erosion from the test specimen.

DESCRIPTION OF SPECIMEN: The specimen consisted of 7/8" thick CAFCO BLAZE-SHIELD CF Type D fireproofing fiber, spray-applied to a sheet of 16" x 48" gypsum lath and left untamped. The exposed air flow surface area was 13" x 45" or 4.07 square feet. The density of the sprayed fireproofing was 15.2 lbs./cu. ft.

RESULTS OF TEST:

Total Erosion	.008 gr.
Total Erosion per square foot of specimen	.0020 gr.

KODARAS ACOUSTICAL LABORATORIES
MICHAEL J. KODARAS, INC.


Michael W. Blenck
Laboratory Director

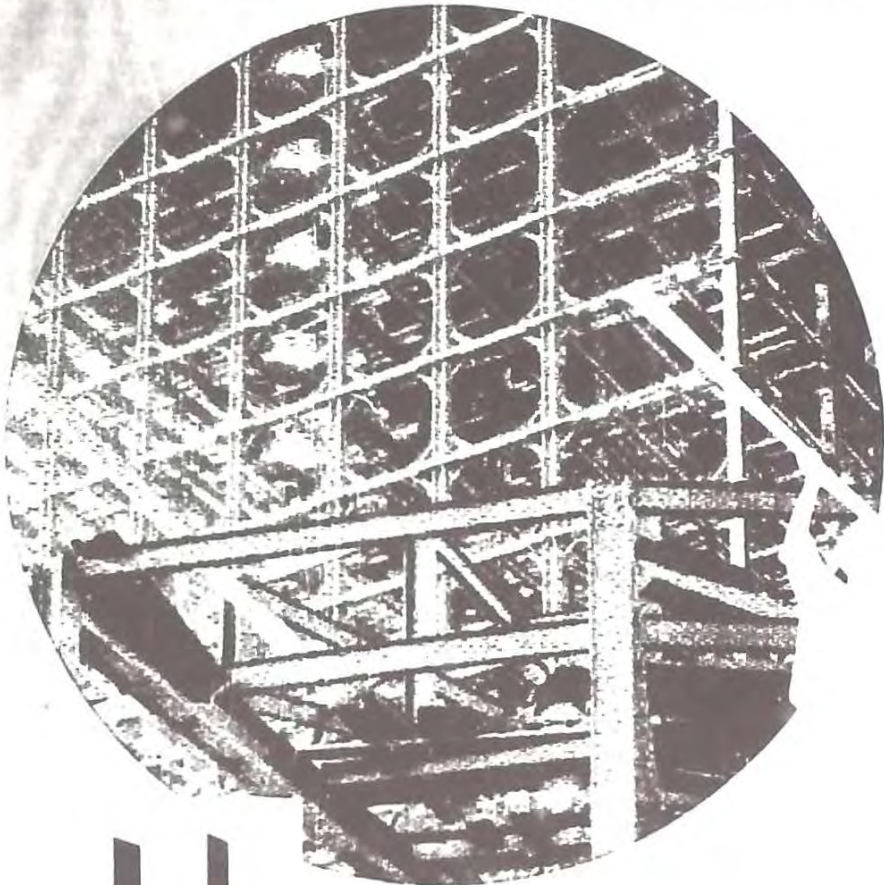
Cafco

7.14/UNI

TYPE D C/F

DIRECT-TO-STEEL SPRAY FIREPROOFING

CONTAINS NO ASBESTOS, NO FREE CRYSTALLINE SILICA



World Trade Center Twin Towers
Port of New York Authority

- permanent protection of structures
- protects against corrosion
- resists air erosion
- can be applied in adverse weather conditions

FOR USE ON:

Ceilings - reefs - floors - beams - girders - spandrels - columns - walls

OTHER CAFCO NON-ASBESTOS PRODUCTS

Power-Shield C/F
Deck-Shield C/F
Sound-Shield "85"





BLAZE-SHIELD

TYPE D C/F

CAFCO BLAZE-SHIELD Type D C/F is a fireproofing material that is applied in a thick, non-sagging, fireproofing coating for steel beams, joists, girders, and columns. It provides excellent thermal insulation and does not lose its insulating properties after exposure to fire. The material is applied in a thick, non-sagging, fireproofing coating for steel beams, joists, girders, and columns. It provides excellent thermal insulation and does not lose its insulating properties after exposure to fire. The material is applied in a thick, non-sagging, fireproofing coating for steel beams, joists, girders, and columns. It provides excellent thermal insulation and does not lose its insulating properties after exposure to fire.

BLAZE-SHIELD Type D C/F exceeds all 5-hour fireproofing requirements for use as a fireproofing material. It maintains high bond strength under normal structural movement and will not crack or spall off in flake. It provides deflection and bond impact characteristics for building up or.

It is economical to apply. BLAZE-SHIELD Type D C/F normally does not require a primary adhesive, tamping or a sealer coat, and can usually be applied from floor level, without scaffolding.

Environmental safety. Unlike asbestos, containing fireproofing, BLAZE-SHIELD Type D C/F qualifies for use in areas where asbestos fireproofing is unacceptable or where strict regulations make the use of asbestos, containing fireproofing economically infeasible. Also studies conducted by expert medical authorities have shown that there is no hazard to the health of workers or the public individually or collectively under normal conditions of use.

Fire rating by U.L. BLAZE-SHIELD Type D C/F is tested by U.L. for virtually any combination of column, beam or floor assembly (see fire rating table below). It can be applied to virtually any structural design.

Fast and permanent protection. BLAZE-SHIELD Type D C/F is applied in a thick, non-sagging, fireproofing coating for steel beams, joists, girders, and columns. It provides excellent thermal insulation and does not lose its insulating properties after exposure to fire. The material is applied in a thick, non-sagging, fireproofing coating for steel beams, joists, girders, and columns. It provides excellent thermal insulation and does not lose its insulating properties after exposure to fire.

Resistance to air erosion. BLAZE-SHIELD Type D C/F is applied in a thick, non-sagging, fireproofing coating for steel beams, joists, girders, and columns. It provides excellent thermal insulation and does not lose its insulating properties after exposure to fire. The material is applied in a thick, non-sagging, fireproofing coating for steel beams, joists, girders, and columns. It provides excellent thermal insulation and does not lose its insulating properties after exposure to fire.

Protection against corrosion. BLAZE-SHIELD Type D C/F is applied in a thick, non-sagging, fireproofing coating for steel beams, joists, girders, and columns. It provides excellent thermal insulation and does not lose its insulating properties after exposure to fire. The material is applied in a thick, non-sagging, fireproofing coating for steel beams, joists, girders, and columns. It provides excellent thermal insulation and does not lose its insulating properties after exposure to fire.

Ability to be applied in one coat. BLAZE-SHIELD Type D C/F is applied in a thick, non-sagging, fireproofing coating for steel beams, joists, girders, and columns. It provides excellent thermal insulation and does not lose its insulating properties after exposure to fire. The material is applied in a thick, non-sagging, fireproofing coating for steel beams, joists, girders, and columns. It provides excellent thermal insulation and does not lose its insulating properties after exposure to fire.

Excellent thermal insulation properties. BLAZE-SHIELD Type D C/F is applied in a thick, non-sagging, fireproofing coating for steel beams, joists, girders, and columns. It provides excellent thermal insulation and does not lose its insulating properties after exposure to fire. The material is applied in a thick, non-sagging, fireproofing coating for steel beams, joists, girders, and columns. It provides excellent thermal insulation and does not lose its insulating properties after exposure to fire.

Acoustical absorption. BLAZE-SHIELD Type D C/F is applied in a thick, non-sagging, fireproofing coating for steel beams, joists, girders, and columns. It provides excellent thermal insulation and does not lose its insulating properties after exposure to fire. The material is applied in a thick, non-sagging, fireproofing coating for steel beams, joists, girders, and columns. It provides excellent thermal insulation and does not lose its insulating properties after exposure to fire.

Light weight. BLAZE-SHIELD Type D C/F is applied in a thick, non-sagging, fireproofing coating for steel beams, joists, girders, and columns. It provides excellent thermal insulation and does not lose its insulating properties after exposure to fire. The material is applied in a thick, non-sagging, fireproofing coating for steel beams, joists, girders, and columns. It provides excellent thermal insulation and does not lose its insulating properties after exposure to fire.

FLOORS - ROOFS - CEILINGS

SYSTEM*	HOURS	THICKNESS	UL DESIGN NUMBER	CONSTRUCTION
FLUTED - FLAT FLOOR UNITS	3	1/2"	43-3 HR	
CELLULAR FLOOR OR ROOF UNITS	2 3	1/2" - 3/4" 3/4" - 3/4"	225-2 HR 86-3 HR	
FLUTED - FLAT FLOOR OR ROOF UNITS	3	3/4" - 3/4"	89-3 HR	
CORRUGATED FLOOR OR ROOF UNITS	2 3	3/4" - 3/4" 3/4" - 3/4"	269-2 HR 204-3 HR	

*French Header or standard header ducts - refer to U.L. test report for applicable thickness under ducts



DECK SHIELD C.F.

Spray fireproofing for large surface areas

DECK SHIELD is specifically formulated for spray application use on metal building walls, roof deck and ceiling design without concrete toppings and ship bulkheads.

FEATURES

- Metal Walls Listing** UL Design No. 13, 3 hr.
- Roof Deck Listing** UL Design No. RC 18, 1 hr., RC 7, 1½ hr. and RC 26, 2 hr.
- Quick Hardening** DECK SHIELD C.F. sets up hard in approximately ONE hour, eliminating delays so that other work may proceed immediately.
- Easy to Apply** DECK SHIELD C.F. is as easy to apply as BLAZE SHIELD Type D.C.F. and is applied with the same equipment.



POWER SHIELD C.F.

Thermal insulation for ambient to 1400 F.

Spray POWER SHIELD C.F. thermal insulation on power generating or process equipment such as boilers, turbines, tanks, ovens, dryers, even chimneys. It can be applied in any thickness to virtually any shape or contour.

FEATURES

- Efficient thermal insulation** Provides k factors of 0.26 and 0.53 at 75° and 500° F. mean test temperatures, respectively. Can be applied to equipment already in operation at temperatures to 600° F. Service temperature range is ambient to 1400° F.
- Economical installation** Spray application appreciably reduces installation costs. Eliminates cutting, fitting, laminating, staggering and securing separate slabs and pieces of insulating material.
- Joint-free coating** Monolithic coating has no seams at which heat losses can occur. There are no voids or crevices in which corrosive condensation can form.
- Permanence** POWER SHIELD C.F. is inorganic, chemically stable and flexible enough to expand and contract with the equipment it covers without cracking or spalling.

CAFCO FIELD ASSISTANCE SERVICE

Users and specifiers of CAFCO products can benefit from assistance from one of the largest contractor and service organizations in the world. Experienced, licensed CAFCO contractors and representatives are available to serve you throughout the United States and Canada and in 38 foreign countries. CAFCO service personnel will answer your questions concerning estimates, specifications and code approvals. A job inspection service is available to architects, general contractors and owners. Look to the CAFCO field service program for prompt, knowledgeable assistance designed to help you keep planning and construction schedules on target.



SOUND SHIELD-85

Sprayed acoustical fireproofing

Use SOUND SHIELD-85 for acoustical treatment of ceilings and walls of virtually any contour. It has a hard, white, attritious finish and UL fireproofing tested and listed for 1½ hr. provides thermal insulation as an extra benefit. k factor of 0.26 at 75° and 500° F.

FEATURES

- Can be applied to concrete, plaster, masonry or metal walls.
- Provides NCR rating as high as 0.85, 1½ inch thick.
- Surface that meets equivalent to plaster.
- Natural light reflectance rating of 76 percent.
- Fibrous core base composition prevents dust bleed through.



TABLE OF SOUND ABSORPTION

Frequency, Hz	0.125	0.25	0.5	1	2	4	8	16	32	64	128	256	512	1024	2048	4096	8192	16384	32768	65536	131072	262144	524288	1048576	2097152	4194304	8388608	16777216	33554432	67108864	134217728	268435456	536870912	1073741824	2147483648	4294967296	8589934592	17179869184	34359738368	68719476736	137438953472	274877906944	549755813888	1099511627776	2199023255552	4398046511104	8796093022208	17592186044416	35184372088832	70368744177664	140737488355328	281474976710656	562949953421312	1125899906842624	2251799813685248	4503599627370496	9007199254740992	18014398509481984	36028797018963968	72057594037927936	144115188075855872	288230376151711744	576460752303423488	1152921504606846976	2305843009213693952	4611686018427387904	9223372036854775808	18446744073709551616	36893488147419103232	73786976294838206464	147573952589676412928	295147905179352825856	590295810358705651712	1180591620717411303424	2361183241434822606848	4722366482869645213696	9444732965739290427392	18889465931478580854784	37778931862957161709568	75557863725914323419136	151115727451828646838272	302231454903657293676544	604462909807314587353088	1208925819614629174706176	2417851639229258349412352	4835703278458516698824704	9671406556917033397649408	19342813113834066795298816	38685626227668133590597632	77371252455336267181195264	154742504910672534362390528	309485009821345068724781056	618970019642690137449562112	1237940039285380274899124224	2475880078570760549798248448	4951760157141521099596496896	9903520314283042199192993792	19807040628566084398385987584	39614081257132168796771975168	79228162514264337593543950336	158456325028528675187087900672	316912650057057350374175801344	633825300114114700748351602688	1267650600228229401496703205376	2535301200456458802993406410752	5070602400912917605986812821504	10141204801825835211973625643008	20282409603651670423947251286016	40564819207303340847894502572032	81129638414606681695789005144064	162259276829213363391578010288128	324518553658426726783156020576256	649037107316853453566312041152512	1298074214633706907132624082305024	2596148429267413814265248164610048	5192296858534827628530496329220096	10384593717069655257060992658440192	20769187434139310514121985316880384	41538374868278621028243970633760768	83076749736557242056487941267521536	166153499473114484112975882535043072	332306998946228968225951765070086144	664613997892457936451903530140172288	1329227995784915872903807060280344576	2658455991569831745807614120560689152	5316911983139663491615228241121378304	10633823966279326983230456482242756608	21267647932558653966460912964485513216	42535295865117307932921825928971026432	85070591730234615865843651857942052864	170141183460469231731687303715884105728	340282366920938463463374607431768211456	680564733841876926926749214863536422912	1361129467683753853853498429727072845824	2722258935367507707706996859454145691648	5444517870735015415413993718908291383296	10889035741470030830827987437816582766592	21778071482940061661655974875633165533184	43556142965880123323311949751266331066368	87112285931760246646623899502532662132736	174224571863520493293247799005065324265472	348449143727040986586495598010130648530944	696898287454081973172991196020261297061888	1393796574908163946345982392040522594123776	2787593149816327892691964784081045188247552	5575186299632655785383929568162090376495104	11150372599265311570767859136324180752990208	22300745198530623141535718272648361505980416	44601490397061246283071436545296723011960832	89202980794122492566142873090593446023921664	178405961588244985132285746181186892047843328	356811923176489970264571492362373784095686656	713623846352979940529142984724747568191373312	1427247692705959881058285969449495136382746624	2854495385411919762116571938898990272765493248	5708990770823839524233143877797980545530986496	11417981541647679048466287755595961091061972992	22835963083295358096932575511191922182123945984	45671926166590716193865151022383844364247891968	91343852333181432387730302044767688728495783936	182687704666362864775460604089535377456991567872	365375409332725729550921208179070754913983135744	730750818665451459101842416358141509827966271488	1461501637330902918203684832716283019655932542976	2923003274661805836407369665432566039311865085952	5846006549323611672814739330865132078623730171904	11692013098647223345629478661730264157247460343808	23384026197294446691258957323460528314494920687616	46768052394588893382517914646921056628989841375232	93536104789177786765035829293842113257979682750464	187072209578355573530071658587684226515959365500928	374144419156711147060143317175368453031918731001856	748288838313422294120286634350736906063837462003712	1496577676626844588240573268701473812127674924007424	2993155353253689176481146537402947624255349848014848	5986310706507378352962293074805895248510699696029696	11972621413014756705924586149611790497021399392059392	23945242826029513411849172299223580994042798784118784	47890485652059026823698344598447161988085597568237568	95780971304118053647396689196894323976171195136475136	191561942608236107294793378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ESTABLISHED 1935

(412) 687-2100

INDUSTRIAL HYGIENE FOUNDATION OF AMERICA, INC.

5231 CENTRE AVENUE

PITTSBURGH, PA. 15232

Mr. Chairman, Honorable Members of the

Committee, I am Robert T. P. deTreville, M.D., Sc.D.,

Board Certified in Preventive Medicine (Occupational Medicine) 1957; Fellow of the American College of Physicians, American Academy of Occupational Medicine, Industrial Medical Association and American College of Preventive Medicine; member of the American Medical Association and American Industrial Hygiene Association; and since 1963, President of Industrial Hygiene Foundation of America, Inc. (IHF), a nonprofit, research association of industries for advancement of health protection and conservation and human relations in industry, i.e., in relation to industry's operations, products and wastes.

Originating in Mellon Institute in 1935 to help solve silicosis, the Foundation now occupies its own headquarters in Pittsburgh. Its members number about 400 industrial companies and associations. Its research is conducted in the field, in the Research Laboratory (located within the University of Pittsburgh, Graduate School of Public Health, Department of Occupational Health, as an Industrial Hygiene Research Unit) and the Mellon Institute of Carnegie-Mellon University, where out highly technical analytical studies are conducted. Our work is supported by member dues and by project support from government as well as industry.

Robert T. P. deTreville, M.D., Sc.D.

5/21/70



(412) 687-2100

INDUSTRIAL HYGIENE FOUNDATION OF AMERICA, INC.
5231 CENTRE AVENUE
PITTSBURGH, PA. 15232

June 5, 1970



Mr. R. L. Parks
Associate Managing Engineer
Fire Protection Department
Underwriters' Laboratories, Inc.
333 Pfingsten Road
Northbrook, Illinois 60062

Dear Mr. Parks:

In response to a request of one of our members (United States Mineral Products Company), we have reviewed in full the materials used in composition of CAFCO C/F products, with regard to their ability to produce adverse health effects; none of the materials used are considered by us to have pathogenic or toxicological bioeffects in man when used for their intended purpose—i. e., sprayed fiber fireproofing—provided that the degree of dustiness does not exceed the recommended safe threshold for biologically "inert," nuisance dusts in the List of Threshold Limit Values published annually by the American Conference of Governmental Industrial Hygienists (enclosures 1 and 2).

A more detailed report on this subject prepared by Industrial Hygiene Foundation and submitted to Mr. Verhalen, President of United States Mineral Products Company, earlier this week should be available to you upon request; it discusses the basis for considering mineral wool derived from blast furnace slag—a vitreous fiber—as being similar to other noncrystalline materials (e. g. fibrous glass and ceramic aluminum silicate) which have been examined by us and found to be biologically "inert."

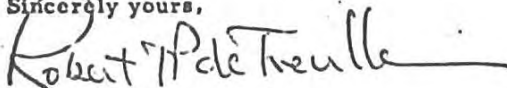
Industrial Hygiene Foundation, which has been studying the effects of such dusts on the lung for 35 years (enclosures 3 and 4) will continue its studies, and should any evidence of possible harm to employee or public health from these products be encountered we will notify you—as well as the company and the public—at once in order that timely controls may be instituted. To the best of our

Mr. R. L. Parks
June 5, 1970
Page 2

knowledge, however, controls other than for avoidance of nuisance dustiness, in or out of plant, are not required for CAFCO C/F products at this time.

Should questions arise, you may feel free to call Dr. Paul Gross, Director of Industrial Hygiene Foundation's Research Laboratories (412-621-3500, Extension 6111) or me (412-687-2100).

Sincerely yours,



Robert T. P. deTreville, M.D.
President

deT:mf

Enclosures

cc: Mr. J. P. Verhalen /



DEPARTMENT OF AIR RESOURCES
51 Astor Place, New York, N.Y. 10003

ROBERT N. RICKLES, Ph.D., P.E., COMMISSIONER

May 5, 1971

Mr. James P. Verhalen, President
U. S. Mineral Products Co.
Furnace Street
Stanhope, N.J. 07874

Dear Mr. Verhalen:

Please be advised that your sample of

CAFCO BLAZE-SHIELD TYPE D C/F,

a fire protection material, has been analyzed by
the Mount Sinai Environmental Science Laboratory
and has been found to be free of asbestos.

Yours truly,

Harold Romer, P. E.
Assistant Commissioner

HR/asb





ESTABLISHED 1929

(412) 687-2100

INDUSTRIAL HYGIENE FOUNDATION OF AMERICA, INC.
5231 CENTRE AVENUE
PITTSBURGH, PA. 15232

Mr. Chairman, Honorable Members of the

Committee, I am Paul Gross, M.D., Founding Fellow of the College of American Pathologists; Fellow of the American College of Chest Physicians, American College of Physicians, and American Association for the Advancement of Science; member of the American Association of Pathologists and Bacteriologists, American Industrial Hygiene Association, American Medical Association, American Society of Clinical Pathologists, American Society of Experimental Pathologists, American Thoracic Society, Industrial Medical Association, International Academy of Pathology, Society of Experimental Biology and Medicine, American Chemical Society, and American Conference of Governmental Industrial Hygienists (member, Threshold Limits Committee, and Chairman, Diagnostic Criteria and Disability Assessment for the Pneumoconioses Committee); and since 1948, Director of the Industrial Hygiene Foundation of American, Inc. Research Laboratory. Currently, I am Research Professor, Pathology of Industrial Diseases, University of Pittsburgh, Graduate School of Public Health, Department of Occupational Health, since 1968, and have authored over 160 publications in the field of pathological research.

IHF is a nonprofit, research association of industries for advancement of health protection and conservation and human relations in industry, i.e., in relation to industry's operations, products and wastes. Originating in

Mellon Institute in 1935 to help solve silicosis, the Foundation now occupies its own headquarters in Pittsburgh. Its members number about 400 industrial companies and associations. Its research is conducted in the field, in the Research Laboratory (located within the University of Pittsburgh, Graduate School of Public Health, Department of Occupational Health as an Industrial Hygiene Research Unit), and the Mellon Institute of Carnegie-Mellon University, where our highly technical analytical studies are conducted. Our work is supported by member dues and by project support from government as well as industry.

Paul Gross, M.D.

5/21/70

INDUSTRIAL HYGIENE FOUNDATION OF AMERICA, INC.

5231 CENTRE AVENUE

PITTSBURGH, PENNSYLVANIA 15232



Report on: HEALTH ASPECTS OF CAFCO C/F FIBERS

For: UNITED STATES MINERAL PRODUCTS COMPANY

Date: JUNE 4, 1970



(412) 687-2100

INDUSTRIAL HYGIENE FOUNDATION OF AMERICA, INC.

5231 CENTRE AVENUE

PITTSBURGH, PA. 15232

Report on

HEALTH ASPECTS OF CAFCO C/F FIBERS

for

UNITED STATES MINERAL PRODUCTS COMPANY

by

ROBERT T.P. DE TREVILLE, M.D., SC.D.
President

PAUL GROSS, M.D.
Director of Research Laboratory

June 4, 1970

Introduction

This report deals specifically with the health effects of the type of mineral wool used by United States Mineral Products Company in sprayed fiber applications for fireproofing, insulating and acoustical purposes, derived entirely from blast furnace slag. Plants manufacturing this type of mineral wool are constructed on or near a slag site for economic reasons, i. e., to minimize costs of moving raw materials. The composition of the blast furnace slags produced in the United States are relatively similar, as will be described later, and there is no likelihood of introduction of a major source of contamination such as has been reported in regard to high manganese or vanadium containing slag from German steel furnaces. It would not be economically feasible either to import German slag or to introduce into use in the United States a steelmaking process having high content of manganese or vanadium, both considered critically short materials in this country; furthermore, quality control procedures used in the industry would reveal promptly presence of any such contamination of raw materials used. For these reasons, consideration need not be given herein either to high manganese or high vanadium containing blast furnace slag.

In addition, while glass wool finds increasing use in many building material applications today, its relatively low melting characteristics make it less suitable for fireproofing purposes, and there are today no commercially available sprayed fiber products composed of glass fibers.

Rock wool has been found less economical to use because of high weight loss (from CO_2 liberation from the limestone) during processing, so that for reasons of safety and purposes, we may confine our attention herein to United States

blast furnace slag, the other components of the CAFCO C/F mixtures having been reviewed and found in our opinion to present no hazard to health of workers or the public individually or collectively under conditions of use.

What Is the Composition of Blast Furnace Slag?

The following analyses of cupola materials contained in an article by Carpenter and Spolyar¹ which appeared in 1945 are considered typical:

		<u>Blast Furnace Slag No. 1</u>	<u>Blast Furnace Slag No. 2</u>
Silica	SiO ₂	51.5%	35.0%
Alumina	Al ₂ O ₃	16.0	12.0
Ferric Oxide	Fe ₂ O ₃	6.5	1.0
Calcium Oxide	CaO	21.5	43.0
Magnesia	MgO	4.5	8.5

The silica reported is the total combined silica. Crystalline-free silica and chrysotile asbestos were looked for but not found in an x-ray diffraction analysis performed at the Mellon Institute of Carnegie-Mellon University (Annex No. 1).

How Is CAFCO C/F Used and What Types of Atmospheric Contamination Are Likely to be Found?

W. R. Bradley Associates performed an industrial hygiene survey on July 26, 1967 while an operator was spraying CAFCO BLAZE-SHIELD, an asbestos-containing product at a ratio of 10 pounds of fiber to 10 pounds of water, on walls and ceiling in a partially enclosed one story building, representing a large open space. A 12-hole spray nozzle was used. Air samples were

1: Carpenter, J. L. and Spolyar, L. W.: "Negative Chest Findings in a Mineral Wool Industry." Chest Studies in Mineral Wool Industry, October, 1945, pp. 389-390.

collected simultaneously at head-level ("breathing zone") and in a line perpendicular to the surface being sprayed at distances of 10, 20 and 40 feet behind the operator. Prior to sampling, the operator had been spraying for 20 minutes to simulate actual spray application conditions.

The purpose of the survey was to determine whether the American Conference of Governmental Industrial Hygienists (ACGIH) threshold limit value for asbestos-containing dust mixtures was being exceeded. Results were 4.4, 3.3, 2.7 and 2.1 mppcf, respectively, which were within then-existing limits (5 mppcf).

Bradley recommended that spray operators wear respiratory protection regardless, as conditions of spraying in confined quarters might cause the threshold limit value to be exceeded at times; however, he felt respiratory protection to be unnecessary for workmen located 10 or more feet away from such spraying operations using asbestos fibers when conducted in semi-open areas.

The fibrous particles other than asbestos in the CAFCO BLAZE-SHIELD product are composed of mineral wool from blast furnace slag and fibers less than 3.5μ in diameter are estimated to compose a small fraction of a percent by weight (0.06-0.3%).

Studies of environmental contamination were made by Bradley in July 1969 in three buildings in which asbestos-containing sprayed fireproofing had been used: The Newark, New Jersey Federal Building, the Newark Post Office Building and the John F. Kennedy Trans World Airlines Terminal. Impinger and membrane filter techniques of sampling were used concurrently. Outdoor air samples also were obtained in Newark and at the John F. Kennedy Airport.

for use as controls. In none of the samples or controls were fibers found which could be classified as asbestos; and in every case the fiber and/or particulate counts were higher out of doors than indoors. It is noted that the natures of identifiable fibers were: cotton lint, synthetic fibers, fibers of dead grass or plant life, and fibers from insect wings. A few fibers of mineral wool from acoustical tile, found in the Newark Federal Building, had diameters in excess of 50 μ . Results of the survey were considered negative; however, in order to document the absence of asbestos fiber contamination, it would be necessary to use more (and higher volume) samples than those reported by Bradley.

What Is Known of the Health Effects of
Mineral Wool Made From Blast Furnace Slag?

While studies of fibrous and nonfibrous mineral dusts of a wide variety of types have been conducted and reported by the Industrial Hygiene Foundation (IHF) since 1935, it must be stated at the outset that no studies have been performed specifically on mineral wool of blast furnace slag origin, alone or as marketed in the form of CAFCO C/F.

One reason for not performing such studies has been that the fiber diameters are quite large, as with glass wool insulation fibers. IHF first studied and reported on the lack of bioeffects of nonfibrous glass particles of respirable size. When it became possible to obtain an adequate supply of glass fibers having a mean diameter of about 1 μ , additional studies were performed which have confirmed that the fibrous shape of the glass dust did not alter its biological effect in the lungs, which was essentially inert or negative in nature. Based on this experimental evidence and the results of an epidemiological investigation on employees of the oldest, largest, fibrous glass

manufacturing plant, which demonstrated no adverse health effect of dust exposure in comparison with controls, in 1969 the ACGIH Threshold Limits Committee announced its intention to list fibrous glass as an inert or nuisance dust (i. e., to be controlled to 15 mg/m^3).

Discussion

Mineral wool from blast furnace slag, as a vitreous material in fibrous form, is comparable not only to fibrous glass but also to certain other non-crystalline dusts which have been investigated by us and found free of pathogenicity (i. e., "inert" biologically). Among these, ceramic aluminum silicate (Fiberfrax[®]), is fibrous in form.

From these studies we have become convinced and have published our conclusions that the shape of the fiber is not the cause of pathogenicity or adverse effect on the lungs in the case of asbestos. This view is now rather widely accepted and few still adhere to the "mechanical theory" of harm. When it is considered that fibers are, by definition, particles in which the length exceeds the diameter by three or more times, it can be appreciated that the vision of mechanical "pricking" of the tissues by needle-like filaments could certainly not apply in any event in the case of vitreous fibers such as mineral wool. Containment, good housekeeping practice and the wet spray technique itself characteristic of good spraying practices today will minimize nuisance contribution to air pollution by CAFCO C/F fibers.

While it was reported that an occasional fiber of asbestos may be found in CAFCO C/F by electron microscopy of individual fibers, there is no asbestos in the raw materials used and the only source of such fibers would appear to be

from contamination of manufacturing equipment used in making the CAFCO BLAZE-SHIELD product, which does contain chrysotile asbestos. X-ray diffraction fails to show presence of chrysotile asbestos, however, so it is apparent that the degree of hazard from asbestos fiber contamination of CAFCO C/F should be negligible.

The fact that fibrous glass has been studied epidemiologically by IHF has been mentioned above; it should be added, however, that the United States Public Health Service (USPHS) is performing studies of Social Security statistics in a cohort of workers. It will probably be a year or more before these studies are reported as considerable detailed work is involved. It is a time-consuming and costly procedure and there is question whether the USPHS would agree to perform a study of this type on workers in mineral wool production which appears to duplicate the glass fiber study, unless there is some evidence of possible public health hazard. (No good evidence has been presented to date to suggest such a hazard to health.) It would be difficult to justify expenditure of public funds simply to help a company or industry document absence of such a possibility for its own benefit in securing process and materials approval from governmental agencies.

Conclusions

Because of the lack of any evidence of pathogenesis (especially oncogenesis) in our studies with fiberglass and ceramic aluminum-silicate dusts, it would appear to us very unlikely that such pathogenic changes would be detected in the case of another vitreous fibrous material (i. e., the mineral wool used in CAFCO C/F fibers).

As a safeguard and as part of its ongoing Central Data Repository project (reporting industrial experience to help set and validate environmental and occupational health standards on materials produced or used in its membership), IHF is prepared to collect and evaluate data on the industry's employees in future years in two important categories:

- (1) Biostatistical studies of employees and retired workers; and
- (2) Pathological studies of lung tissues of employees and former employees of the industry, as these become available.

Aside from helping to validate our opinion regarding the inertness of mineral wool from blast furnace slag, it will be useful to IHF to include such data in its industry-wide pulmonary pathological investigations as a control. Costs incurred by IHF in this project will be borne by the industry as they occur. Findings of these studies will be reported by IHF periodically both to the ACGIH Threshold Limits Committee and in the scientific literature.

Recommendations

We see no objection to the utilization of CAFCO C/F as a substitute for asbestos-containing CAFCO BLAZE-SHIELD in fireproofing operations at this time. Should our further studies ever suggest presence of a health hazard, this information will be communicated at once to the company and the public in order to effect timely controls.

ANNEX 1

Carnegie-Mellon University

Mellon Institute
4400 Fifth Avenue
Pittsburgh, Pennsylvania 15213
[412] 621-1100

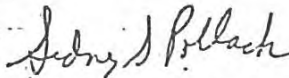
May 27, 1970

Dr. Robert DeTreville
Industrial Hygiene Foundation
of America, Inc.
5231 Centre Avenue
Pittsburgh, Pennsylvania

Dear Dr. DeTreville:

A sample of CAFCO Type D-C/F has been analyzed by means of the Debye-Scherrer x-ray diffraction technique and no chrysotile asbestos or crystalline forms of free silica were detected. Two patterns of the sample were prepared using nickel filtered copper x-radiation. One was made in a camera with a diameter of 11.46 centimeters and the other in a smaller camera having a diameter of 5.73 centimeters.

Sincerely,



Sidney S. Pollack
Fellow
X-Ray Diffraction Laboratory

SSP:vab

FIBROUS DUST PARTICLES AND FERRUGINOUS BODIES

Methods for Quantitating Them and
Some Results from the Lungs of City Dwellers

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ABSTRACT

Reproducible methods are described for quantitating inhaled fibrous dust particles and ferruginous bodies found in lungs. For isolating the inhaled fibers, the total dust is first recovered by digesting dried and defatted lung tissue with formamide. The carbonaceous material is then removed by a short treatment with perchloric acid. Ferruginous bodies are isolated by digesting lung tissue with sodium hypochlorite and removing the interfering carbonaceous dust by gradient centrifugation with sucrose.

The size of the fibers, their volume, and weight are estimated by first photographing an aliquot of the fibers and then tabulating their dimensions as obtained from scaled enlargements. These dimensions serve as the basis for estimating the volume and weights of all the fibrous dust isolated from a given pair of lungs.

The lungs of seven city dwellers, two long-time employees of a fiber glass factory, and one of an asbestos-products factory were studied. These lungs were found to contain 15,000 to 66,000 fibrous particles and 300 to 3,900 ferruginous bodies per gram of dry lung. The mean dimensions of the fibers in all lungs were within a surprisingly narrow range.

The recent demonstration of ferruginous bodies in 97% of the lungs of one hundred city dwellers by the digestion of lung tissue with sodium hypochlorite was associated with the finding of numerous naked fibers which were present in the ultimate sediment.¹ Some of these fibers could be identified as man-made by their curved ends, particularly when shaped like a shepherd's crook. Other fibers exhibited longitudinal splitting and were thereby classified as probably asbestos.

Because fibrous dust particles composed of materials other than asbestos, when introduced into lungs of animals, have been shown to result in the development of structures indistinguishable from asbestos bodies, the generic term, ferruginous bodies, had been proposed for them.² Nevertheless, electron diffraction analysis of the central cores of 28 randomly selected ferruginous bodies from city dwellers indicated a crystalline structure in all of them. This fact eliminated glass and ceramic fibers as being responsible for the formation of these particular bodies.³ By exclusion, and in the absence of other known possibilities, this left asbestos as the probable constituent of the central core of the ferruginous (asbestos) bodies. However, because chrysotile had been decisively excluded as being involved in the formation of the above 28 ferruginous bodies, and since more than 90% of the asbestos used in this country is chrysotile, an enigmatic situation obtains.

Although the occurrence of ferruginous bodies and of naked fibers in the lungs of city dwellers has not been associated with disease ascribable to the inhalation of fibrous dust particles, the suspicion has been publicized that pulmonary fibrosis and cancer may develop in the lungs of city dwellers from the inhalation of this fibrous dust presumed to be asbestos.^{4, 5}

While studying the sediment derived from the digestion of lung tissue, we were impressed with the large number of naked transparent fibers, most of which were less than three microns in diameter and less than 50 microns long. These had no distinguishing features that enabled us to differentiate asbestos from non-asbestos fibers at the magnifications employed (X 450). Subsequently, however, we have been able to do this readily with the electron microscope.⁶ Having shown that at least some of the fibers inhaled by urban dwellers are probably asbestos, we sought to determine how much fibrous dust was inhaled by these people during their life time and what was the size distribution of the fibers. We also wished to study the relationship of the fibrous dust (1) to the total dust inhaled, (2) to the number of ferruginous bodies found, and (3) to any possible pulmonary disease present.

To answer these questions, we first had to develop a method for quantitatively separating the transparent fibers from the carbonaceous material. We then needed to devise a procedure for counting the fibers so as to obtain reproducible results. We also found it necessary to modify our previously published method for isolating ferruginous bodies from the lungs¹ in order to obtain

quantitative yields. In this paper, we are reporting the details of these methods, as well as the results obtained on the lungs of a small number (seven) of urban dwellers (selected only in respect to sex), of a long-time employee of an asbestos-product factory, and of two men who had been exposed for a working lifetime to dust in a factory producing fibrous glass (from a factory which pioneered the production of fibrous glass, we have on hand for detailed study the lungs of 18 long-time employees who had died of natural causes. The above two workers are the first and second on which data have become available.)

Identification of both the naked fibers, as well as the cores of ferruginous bodies derived from lungs of these urban dwellers, will be studied and reported in a future paper.

MATERIALS AND METHODS

Preparation of Dry Lung Tissue

Formaldehyde-preserved lungs were washed with running tap water for 24 hours and then cut into thin slices by means of a meat slicer. The slices were extracted with three portions of acetone and three portions of dry ether for six to eight days, vacuum dried at 50° C for 48 hours, and then weighed.

Isolation of Dust from Lungs

The defatted, dried lung tissue was digested by heating with formamide according to the method of Thomas and Stegemann.⁶ However, in order to remove more of the associated impurities and to render the dust more readily dispersible, it was subjected to more vigorous washing. In addition to the solvents used by Thomas and Stegemann, methanol-chloroform mixture was used by us to wash the dust. This additional manipulation resulted in the removal of some unidentified brown colored impurities and made the dust more easily dispersible.

The procedure was carried out as follows: about 30 gm of dry tissue was placed into a 2,000 ml flask containing 1,000 ml of dry formamide (Fisher, reagent grade) and the mixture was kept under continuous stirring at $130 \pm 5^\circ \text{C}$ for 72 hours. The resulting liquid digest was centrifuged in 200 ml flasks at 2,000 G for one hour. The sediment, consisting of crude impure dust, was collected into a single flask and washed three to four times with 150 ml portions of warm formamide. The sediment was submitted to further digestion with 200 ml of formamide for 24 hours, and it was then washed with 150 ml portions of following warm solvents: formamide; formamide-water (1:1); water; methanol; methanol-chloroform (3:2); chloroform-hexane (2:3); and hexane. The sediment was kept in contact with each of the solvents successively under continuous stirring at least for two hours and was repeatedly washed with several portions of each solvent until the washings were clear and colorless. It was finally dispersed in a small volume of hexane, vacuum dried at 50°C for 24 hours, and weighed.

Separation of Naked Fibers from Dust

Ten milligrams of the isolated dust were suspended in 5 ml of 70% perchloric acid and heated in a sand bath at 160°-180° C until the carbonaceous material disappeared (about thirty minutes). The residue, consisting of mineral particles and fibers, was repeatedly washed with water and finally dispersed, by vigorous shaking, in 2 ml of water containing 0.01% Tween 60. A portion of this suspension was used for the preparation of slides for counting and photographing the fibers.

Separation of Ferruginous Bodies from Lung Tissue

Two grams of dry lung tissue were digested with three portions of 5% sodium hypochlorite for two to three days until the tissue dissolved. During the last 24 hours, the mixture was kept at 50° C and occasionally stirred. The undissolved residue was washed four times with 150 ml portions of warm water for at least four hours. Then it was quickly washed with 1% acetic acid and again with water and dispersed in 50 ml of water containing 0.01% of Tween 60 by stirring or agitating. The suspension was gently poured over an equal volume of 65% sucrose in a 200 ml centrifugation flask so that it overlay the sucrose. This was centrifuged at 400 G for 15 minutes. Under these conditions carbonaceous material and remaining microscopic tissue residues collected on the top of the sucrose layer but mineral particles, naked fibers, and ferruginous bodies in particular penetrated the sucrose layer. The top layer was then discarded and the sucrose diluted with the same volume of

water and centrifuged at 1,000 G for 15 minutes. After washing the sediment with water, resuspending it in 50 ml of water containing 0.01% of Tween, and again gently pouring it over the surface of 65% sucrose, the separation procedure was repeated. The sediment was again washed with water and suspended in 2 ml water containing 0.01% of Tween 60. Slides were prepared for counting from this suspension.

Preparation of Slides for Counting

Clean coverslips (22 x 22 mm) were thinly coated on one surface with a glycerine-albumin mixture such as is used for fixing paraffin sections to slides. These coverslips (coated side up, and two for each suspension) were then placed upon the head of small nails protruding about 3 cm from a suitable board. The heads should be about 4 mm in diameter. A small drop of balsam such as is used between a tissue section and its coverslip, had previously been placed upon the head of the nails as an adhesive. The orientation of the nails was such that the coverslips lay level in all directions.

After thorough mixing, 0.1 ml of the dust-, or ferruginous body-suspension was removed by means of an ultramicro syringe and gently delivered upon the horizontally positioned coverslips. Surface tension kept the fluid from escaping the limits of the coverslip. The fluid was allowed to dry in the air. The board with its coverslips fastened to the nail heads was then placed in an oven at 60° C for two hours. This coagulated the albumin and fixed the originally suspended particles to the glass. The concentration of the suspension

used for such a preparation was adjusted so that the number of objects to be counted was neither excessively large nor small. For counting naked fibers, we have found a satisfactory concentration of the suspension to be such that 1 ml contained the equivalent of 1 mg of dust; for counting ferruginous bodies, 1 ml of the suspension contained the equivalent of 0.2 gm of dried lung. With these dilutions, we generally counted in the neighborhood of 500 fibers and 30 to 50 ferruginous bodies per coverslip.

The coverslip method for counting fibers and ferruginous bodies was selected primarily because it circumscribes and minimizes the area which needs to be examined microscopically. Attempting to count fibers or ferruginous bodies as a suspension under a coverslip proved difficult because during the time required for counting, the suspensions tended to dry out and this rendered identification of the particles difficult. On the other hand, the addition of fluid during the counting caused extensive and explosive displacement of suspended particles, thereby making continuation of the count useless. Fixation of the particles to the coverslip seemed to solve this problem.

After removing the tacky balsam with xylol, the coverslip for counting naked fibers was placed, coated side down, upon a clean glass slide and distilled water was introduced along one edge of the coverslip. When too many bubbles resulted, the coverslip was raised at one edge to allow them to escape, and it was then gently lowered. The reason for the use of water is better visibility. With balsam, the fibers are nearly invisible. The fixation by means of the albumin was such that the water beyond the edge of the coverslip

did not contain fibers that had become detached from the coverslip. During the counting it became necessary to add water to prevent drying. In doing so, care was taken not to touch the coverslip and thereby change its position, nor was the water so excessive that the coverslip floated. The coverslip for counting ferruginous bodies was mounted by means of balsam, coated side down, upon a glass slide. Balsam is used because it renders naked fibers and mineral particles less visible, thereby allowing the ferruginous bodies to stand out more prominently.

We used an Orthomat microscope camera (Leitz) for counting, employing X 10 oculars and a X 12.5 objective. One of the eye pieces contained the outline of a rectangle, representing the frame of a 35 mm film. This was positioned so that its long axis was parallel to the viewers line of sight. After counting the contents of this rectangle during a vertical traverse (mechanical stage), the slide was moved exactly the width of the rectangle for each successive vertical traverse. Some of the fibers were found to be very small and indistinct because their index of refraction was near that of glass. Frequent use of the high dry (X 45) objective helped to dispell uncertainties.

Method of Studying Naked Fibers

It is assumed that the naked fibers are randomly distributed upon the coverslip and that a random selection of these fibers will result in a representative sample from which valid conclusions can be drawn in regard to the fibrous particles in the lungs as a whole. Beginning at any convenient place

on the coverslip and using the X 12.5 objective, a fiber was located and then photographed with the X 45 objective. Between 150 and 200 fibers were generally photographed but care was taken so that all fibers that came successively into view were photographed in order to preserve their randomness. We generally used three rolls of Kodak Panatomic X (36 exposure) film per suspension. At times, it was possible to photograph two or more fibers in the same field.

The next step was to project the negatives by means of an enlarger upon a sheet of white paper at an approximate magnification of X 10, giving a total magnification of X 1500 ($45 \times 10 \times 1/3 \times 10$). Outline drawings were made of the projected fibers (Fig. 1). All measurements were made from the outline drawings so made. Each fiber was given a number and tabulated with its dimensions. From this tabulation, the mean diameter and mean length of the fibers were calculated. Other data were also derived, such as the percentage of fibers whose diameter is equal to or less than 2 and 3 μ , respectively; and the percentage of fibers whose length is within the range of 5 to 25 μ and those longer than 50 μ (Table 1). The assumption was made that all fibers are cylindrical in shape and that they have an approximate density of 2.5 gm per cubic centimeter. By means of these assumptions, the approximate total volume of all fibers photographed was calculated as well as their approximate weight.

RESULTS

The results obtained by the methods described above are tabulated in Table 1. The amount of dust per gram of dried lung varied considerably, the greatest difference being four fold. The number of naked fibers per mg of dust varied from about 500 to 9,000; per gram of dry lung, they ranged from 15,100 to 66,400. This was also reflected in the percentage of the total weight of the naked fibers in terms of the total weight of the dust. This percentage ranged from 0.01% to 0.47%.

The mean diameter of the naked fibers was remarkably constant, ranging between 1.9 and 2.2 μ , with only one deviation to 2.8 μ . Very few fibers (generally 3 to 9%) were more than 3 μ in diameter. In one lung, 30% of the fibers were thicker than 3 μ . In most of the lungs, about two-thirds of the fibers were between 5 and 25 μ long and only 3 to 9% were longer than 50 μ . Rare gigantic fibers, such as the three pictured in Fig. 1, were seen in two lungs. The formation of a closed loop, and the presence of conchoidal fractures marked these as probably vitreous and man-made. Their size precluded sojourn within the air spaces and suggested lodgement in the smaller bronchi. These fibers were, therefore, not included in the tabulation.

The ferruginous bodies, while much more numerous than could be suspected from an examination of either the lung juice or lung sections, varied quantitatively as much as the naked fibers. The number of such bodies per mg of dust varied from 11 to 440. The number of naked fibers per ferruginous body varied from 5 to 211. Conversely, the percentage of fibers represented as ferruginous bodies varied from 0.7 to 16%.

Some of the ferruginous bodies were very irregular in appearance and deviated markedly from those usually found in sections of asbestotic lungs (Fig. 2). Some of the bodies had apparently been formed by encrustations developing upon the ends of fractured crystalline plates (Fig. 3). Such crystalline plates were suggestive of, and consistent with, talc crystals.

Differences in duplicate counts ranged from 1% to 15%, with a mean difference of 9%.

Accuracy Check

An aqueous suspension of respirable fibrous glass dust (approximately 60% fibers with a mean diameter of $1.9 \mu\text{m}$ and 40% non-fibrous particulates) was prepared so that 1 ml contained $0.33 \mu\text{g}$ glass. The number of fibers per ml was determined as outlined above. In addition, an aliquot of 261 fibers was photographed, measured, and their estimated weight calculated from their dimensions. The total number of fibers per ml was found to be 9480 and their estimated weight m/ml was calculated to be $0.21 \mu\text{g}$. When the weight of the non-fibrous particulates (40% of $0.33 \mu\text{g}$ or $0.13 \mu\text{g}$) was added, the sum, or $0.34 \mu\text{g}$ proved to be a very close approximation of the actual weight. However, inasmuch as the percentage of fibers in the glass is only a rough estimate, the closeness of the check must be considered as due to chance. On the basis of this estimate, $1 \mu\text{g}$ of this glass dust contains 28,000 fibers of various size.

As a further check of the accuracy of the method, a known amount of fibrous glass dust and of carbon were added to two-gram portions of rat liver

and thoroughly mixed (Table 2). These were then digested with formamide and further processed as outlined in the text. Based on the above estimate that 1 μ g glass contained 28,000 fibers, the error in estimating the weight of the added glass was as high as $\pm 27\%$ (Table 2).

COMMENTS

The method used for quantitating naked fibers in lungs has deficiencies, most of which operate to yield results that are less than the probable actual values. Chrysotile fibers, for example, are known to be more soluble than other forms of asbestos. It is not known to what extent such fibers may tend to disappear during the prolonged process of digesting the lung with formamide. Also, there is little doubt but that some of the thinnest fibers (less than 1 μ m in diameter) may be overlooked at the scanning magnification employed and there is certainty that fibers substantially thinner than one micron are not counted. Inasmuch as a fiber is defined as a particle whose length is at least three times its diameter, some of the shorter fibers may be overlooked because during the scanning, careful measurements are not done. Inaccuracies may also develop because the sample from which counts are made is such a small aliquot of the whole (1/300 to 1/2500 in the present series) and possibilities of error exist due to loss of material during centrifugation and washing of the sediment containing the fibers.

Attempts to separate fibrous mineral particles from carbonaceous material based on gradient centrifugation with sucrose solution gave

inconsistent and variable results and this method was, therefore, abandoned. Although we recognized the solubility of chrysotile in strong acids, we felt that the destruction of carbonaceous material with perchloric acid for thirty minutes would probably have little demonstrable effect on the fibers present. This question was investigated by photographing a field of chrysotile fibers immediately after contact with perchloric acid and again (the same field) after thirty minutes of heating the preparation to 160° C. No detectable change in the number or size of the fibers in the photographs were observed.

Some of the fibers photographed (Fig. 1) have ends with sharp, angulations and some have the step-like indentations suggestive of asbestos fibers. An occasional fiber shows peculiar branching, more like that of a plant than the longitudinal splitting of asbestos fibers. Yet, fibers of vegetable or other organic composition can be ruled out since these could not have survived the digestion with perchloric acid.

When we attempted to use the sediment obtained by the formamide method of digesting the lungs to determine the number of ferruginous bodies, we found that none could be found. It appears that during the process of digestion, formic acid is liberated and this acid destroys the ferruginous encrustations. In returning to the method of isolating ferruginous bodies by means of sodium hypochlorite,² we found that in separating the carbonaceous material from the bodies with chloroform, many of the ferruginous bodies remain suspended and embedded in the carbonaceous material and quantitative recovery

of these structures is difficult to obtain with this method. For this reason, we turned to gradient centrifugation with sucrose solution and were able to obtain reproducible results.

The often bizarre shape of the ferruginous bodies encountered in many of the control lungs, examined suggests exposure to fibrous dust different from the usual type of asbestos. In this regard, the development of ferruginous bodies from fractured crystalline plates (Fig. 3) is particularly interesting and it suggests that some of the city dwellers may have had exposures to talc dust. As a matter of fact, such fractured crystalline plates were seen not infrequently while counting naked fibers.

The early investigations into the prevalence of lungs in which one or more ferruginous bodies were to be found employed smears of lung fluid or thick sections. From these studies it was learned that in most cities in which these investigations were made, only 20 to 40 odd percent of the lungs examined contained what were then called "asbestos" bodies; and in more than 70% of these lungs, only one or two ferruginous bodies were found. The large number of these structures and a still larger number of naked fibers (up to 200 times larger) discovered in the present series of lungs comes as a surprise because, even though in a previous study we found ferruginous bodies in 97% of the lungs examined,¹ in 75% of the latter, only five or fewer ferruginous bodies could be counted. The present findings seem to confirm the conclusions of Cralley, et al,⁷ regarding the ubiquity of respirable fibrous dust particles.

It is unfortunate that we do not know how much of the lungs had been removed by the prosectors, so that we cannot determine exactly how much total dust and how many fibers were present in each pair of lungs. It appears, however, that some lungs contained (estimated) at least three to four million fibers.

As pointed out previously, the method employed in this investigation suffers from the fact that it ignores fibers which may be present but which are substantially less than one micron in diameter. However, it is likely that a few of such fibers may be represented as the central cores of some of the ferruginous bodies.

ACKNOWLEDGMENT

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Dr. Werner Stober and Mr. Hermann Flachsbart of the University of Rochester kindly gave us technical advice.

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LEGENDS OF ILLUSTRATIONS

- Fig. 1** Outlines of fibers as traced on a sheet of white paper from images projected from three rolls of 35 mm negative film by means of an enlarger. Most of the fibers have diameters in the neighborhood of $2\ \mu$ and lengths less than $25\ \mu$. A rare fiber is gigantic (up to 1 mm in length) and too large to be accommodated in alveolar tissue. These were probably lodged within the smaller airways and, therefore, were not included in the quantitations.
- Fig. 2** This is a collection of individually photographed ferruginous bodies isolated from the lungs of a 74 year-old Pittsburgh woman (A69-80) who died of *E. coli* septicemia. She had no pulmonary disease other than edema. The tremendous variation of these bodies in size and shape make a mockery of the classical description, "a symmetrical, segmented, bipolar-clubbed structure about $50\ \mu$ long," etc.
- Fig. 3** This is a small collection of "atypical" ferruginous bodies which developed from transparent thin crystalline plates rather than from fibers. Although several have smooth edges, others show sharply angulated steps and recesses suggestive of fractures. These resemble talc crystals with clubs of ferruginous material forming on narrow projections of the plates.

TABLE 1

Data Involved in Determining Accuracy of Methodology

Actual Composition prior to Formamide Digestion			Theoret. (a) Wt. of Glass in Suspension $\mu\text{g/ml}$	Findings			
Rat Liver g	Carbon mg	Fibrous Glass mg		No. Fibers per ml	Wt. of Glass $\mu\text{g/ml}$	Error	
						$\mu\text{g/ml}$	%
2	100.5	0.20	0.63	2120	0.76	+0.13	21
2	50.0	0.20	0.79	1630	0.58	-0.21	27
2	50.0	0.20	0.79	2800	1.00	+0.21	27

(a) Obtained following digestion with formamide, further digestion of an aliquot with perchloric acid, washing, and suspending sediment in distilled water

TABLE 2.

Quantitation of Fibrous Dust Particles and
Ferruginous Bodies in Human Lungs

No. of Fibers		Mean Vol. of Fibers μ^3	Percent Fiber Wt. Dust Wt.	Mean Diameter of Fibers μ	Percent of Fibers with Diameters		Mean Length of Fibers μ	Percent of Fibers with Lengths		Ferruginous Bodies			
per gm. Dry Weight of Lungs $\times 10^3$	per mg. Dust				$\leq 2\mu \leq 3\mu$			$5-25 \mu$	$>50 \mu$	No. per mg. Dust	No. per gm. Dry Lung	No. Fibers per Body	Percent Fibers as Bodies
27.9	1,840	153	0.09	2.2	69	91	28	64	8	*	*	*	*
23.9	1,850	133	0.06	2.3	58	87	25	68	5	12	160	149	0.7
84.8	26,400	44	0.28	1.4	95	98	17	90	2	2,280	73,000	12	8
15.4	564	84	0.01	2.0	74	97	25	67	8	11	300	51	2
66.4	2,845	88	0.06	2.0	70	95	25	68	8	108	2,450	26	4
15.1	783	143	0.02	1.9	61	93	20	75	4	*	*	*	*
20.8	1,150	54	0.02	2.0	76	97	18	80	3	153	2,700	7.5	13
21.4	1,560	88	0.04	2.2	71	97	22	70	5	292	3,927	5	18
28.8	4,700	121	0.14	2.2	57	92	27	63	8	440	2,700	11	9
63.3	9,070	210	0.47	2.8	33	70	28	58	9	43	300	211	0.5

g tissue was digested with formamide, which destroyed the ferruginous bodies
 glass factory
 products plant

ASBESTOS REGULATIONS:
CONTROL OF SPRAY INSULATION

PREAMBLE

It is hereby determined that the uncontrolled discharge of asbestos fiber into the environment tends to severely endanger the public health and welfare and that the uncontrolled spraying of other fiber-containing material unreasonably interferes with the enjoyment of life and property.

1. Definitions

- | | | |
|---------------------|---|--|
| Agency | - | The Illinois Environmental Protection Agency. |
| Asbestos | - | Any fiber or any mixture containing fiber of hydrated silicate mineral, which, on the basis of its crystalline structure, falls into one of two categories:

(1) pyroxenes - chrysotile fiber;
(2) amphiboles - crocidolite, amosite, tremolite, actinolite or anthophyllite fiber. |
| Commercial Activity | - | Any activity done for hire or having financial profit as a primary aim. |
| Debris | - | Asbestos-containing waste produced by the demolition of a structure. |
| Spraying | - | The pneumatic application of material used for fireproofing or insulation. |
| Waste | - | Any asbestos-containing matter which has been or is intended to be discarded. |

2. General Requirements

No commercial activity, not otherwise hereinafter prohibited, involving the discharge of asbestos fiber into the environment from the construction, alteration, repair or demolition of a structure or from the processing or manufacturing of asbestos-containing products, shall be conducted unless the person or entity in charge of such activity complies with the following regulations:

A. A permit shall be obtained from the Environmental Protection Agency to engage in such activity. Before obtaining such permit, the applicant shall demonstrate compliance with the following safeguards:

(1) The designation of personnel to exercise full-time supervisory authority over all aspects of the activity from which the release of asbestos fiber into the environment could result, in such a manner as to insure compliance with the pertinent asbestos control regulations.

(2) That each employee engaged in such activity has completed or will complete a satisfactory course of instruction on the potential hazards of exposure to asbestos fiber including the precautions that must be observed to prevent or restrict the dispersion of asbestos fiber into the environment.

(3) That such additional safeguards as are hereinafter specifically required and such precautions consistent with the purpose of these regulations as the Environmental Protection Agency deems necessary under the circumstances shall be complied with.

B. Facilities shall be provided and procedures instituted and supervised that prevent the removal of asbestos-containing material from the site on the clothing of employees.

C. Asbestos-containing wastes shall be immediately vacuumed or otherwise collected where vacuuming is impossible, and shall be placed in a container resistant to tearing or breaking under normal handling conditions, which shall be tightly sealed and clearly marked as containing asbestos waste. Such containers shall be placed directly upon a vehicle for disposal by burial at an approved site.

This subsection (C) shall not apply to the demolition of a structure, except as provided in Section 4(A)(1) and (6) or to the disposal of sludge waste except as provided in Section 5(C).

3.. SPRAY FIREPROOFING AND INSULATION

A. The spraying of asbestos-containing material is prohibited.

B. Non-asbestos particulate matter used for insulation or fireproofing shall not be sprayed unless the following procedures are taken:

(1) Before the start of spraying operations, all floor areas shall be swept clean and cleared of all objects, material and equipment other than that employed in the application of the fiber-containing material, or, in the alternative, all such objects, material and equipment shall be covered with plastic or plastic-coated tarpaulins.

(2) The entire floor or area to be sprayed shall be enclosed with plastic or plastic-coated tarpaulins in a manner which shall preclude the escape of fiber-containing material from the enclosure. All interior open areas such as elevator shafts and stairwells shall be enclosed in a manner which shall prevent the escape of fiber-containing material from the working area. "Stack effect" of the shafts, stairwells and comparable spaces shall be considered in providing proper enclosures.

(3) Regardless of adherence to the stated control practices, any visible emission of fiber-containing materials beyond the building perimeter will be considered a violation.

(4) In case of visible emission of fibrous material from the construction site, immediate steps shall be taken to cause the cessation of such emissions by either effective control measures or work stoppage at the source of the emissions. There shall then be immediate and complete cleanup of all material that has escaped the construction site, and measures shall be taken that will insure that no further dispersal of any fibrous material into the atmosphere can occur.

(5) The entire sprayed area, all ledges and surfaces including tarpaulins within the enclosure shall be thoroughly vacuumed upon completion of the spraying operation and immediately before the enclosure is dismantled. The vacuum cleaner shall contain a strong, single-service, disposable inner bag of durable material which shall be removed from the vacuum cleaner and tightly sealed. The bag shall then be placed in a container and disposed of at an approved site in accordance with Section 1(C).

(6) All areas used for opening bags containing fibrous material or the changing of hoppers shall be enclosed so as to prevent the escape of such material from the immediate area in which such activity occurs.

(7) Any plenum or other structure coated with or containing fibrous insulation and used in the circulation of air in a building

shall be thoroughly cleaned of all debris and waste insulation. All applied fiber-containing insulation within a plenum or duct shall be coated with a sealant which precludes exposure of the fiber-containing material to the circulating air.

4. CONSTRUCTION, ALTERATION AND REPAIR OF STRUCTURE

A. (1) The cutting, trimming, fitting or stripping of asbestos-containing material in the construction, alteration or repair of a structure which is done at the site of such structure shall be conducted within a special enclosure designed to preclude the escape of asbestos fiber from the immediate area of such enclosure.

(2) The mechanical exhaustion of dust from such enclosure to the ambient air is prohibited unless such exhaust system is equipped with a fabric filter for dust collection.

B. Asbestos-containing material applied in the construction, alteration or repair of a structure shall be coated with a sealant, provided with a cover or installed in some other manner so as to preclude exposure of the asbestos fiber to the circulating air.

5. DEMOLITION

A. Where the risk of public exposure to asbestos fiber from the dislodging of asbestos-containing materials is present, no demolition of a structure shall be initiated unless all safeguards necessary and practicable to reduce the emission of dust are taken.

Such procedures shall include, but are not necessarily limited to:

(1) Boilers and pipes insulated with asbestos-containing material shall be wetted and stripped before toppling of walls is begun. This procedure shall be followed, where practicable, as to all other asbestos-lined surfaces. Such asbestos waste shall be immediately bagged and disposed of in accordance with Sec. I(C).

(2) Demolition by toppling of walls is prohibited except within an enclosure approved by the Environmental Protection Agency as part of the permit granted for demolition of that structure.

(3) Before the demolition of any section of the structure, adequate wetting to suppress the dust shall be employed.

(4) Asbestos-containing debris shall not be dropped or thrown from any floor but shall be transported by dust-tight chutes or buckets. Asbestos-containing debris in chutes or buckets shall be sufficiently wetted to preclude dust dispersion at the point of discharge.

(5) All asbestos-containing debris shall be thoroughly wetted before loading into trucks, other vehicles or containers. During transport such waste shall be enclosed or covered so as to prevent dust dispersion. Asbestos-containing debris shall be disposed by burial at an approved site.

B. In the event asbestos-containing matter becomes airborne beyond the property boundaries or in the event freezing temperatures preclude the use of water for dust suppression, demolition work shall cease until adequate alternate measures can be taken.

6. MANUFACTURING

A. (1) No factory, plant or enterprise which engages in the processing or manufacturing of any asbestos-containing product shall emit asbestos fiber into the ambient air in excess of .5 fiber per cubic centimeter of air. The ambient air quality at the boundary of such factory, plant or enterprise shall not exceed .05 fiber per cubic centimeter of air.

(2) Only fibers of 5 microns or greater in length with a length to breadth ratio of 3 to 1 shall be counted..

(3) Sampling shall be determined by the membrane filter method with phase contrast microscopy at 430 X magnification.

B. No water used in processing or manufacturing which contains concentrations of asbestos fiber shall be discharged into sewage systems or to the waters of Illinois.

C. Waste sludge containing asbestos collected from settling ponds shall be enclosed during transport and shall be disposed by burial at an approved site.

7. ASBESTOS-CONTAINING PRODUCTS

It is hereby determined that the continuous emission of asbestos fiber from asbestos-containing products contributes sig-

nificantly to the increasing volume of asbestos-fiber in the ambient air and endangers the health of future generations.

A. No product which may emit asbestos-fiber during its transportation shall be transported unless such product is enclosed in such a manner as to preclude the emission of asbestos fiber into the ambient air.

B. The use of asbestos in the brake lining of vehicles manufactured after January 1, 1975, and sold for use in Illinois is prohibited.

R71-16 PUBLIC COMMENT LIST

R71-16 PUBLIC COMMENTS

Advances
(Exhibits)

ASSETS
Exhibits

R 71-16
(Exhibits)



R71-16

EXHIBIT LISTING

STATE OF ILLINOIS
POLLUTION CONTROL BOARD

DATE: October 21, 1971
MEMO TO: Clerk's Office
FROM: Mr. Lawton
SUBJECT: Asbestos Exhibits #R71-16

I hand you herewith Exhibits 1 through 26 inclusive,
in accordance with the attached 1 st.

STL:sh

R71-16 Exhibits

1. Newsletter #24
2. (a) Granite City Star Newspaper notice
(b) Certificate of Publication from Keystone Printing Service, Inc.
C, Ch. T-16
3. Litton Asbestos Report
4. "Asbestos", I. J. Selikoff;
Environment, 11(2): 3-7, March, 1969.
5. "Asbestos as an Urban Air Contaminant", Thomson;
Archives of Pathology 81(5) 458-464, May, 1966.
6. "The Interrelationship of Selected Asbestos Exposure Indices",
Lynch, Ayer & Johnson;
American Industrial Hygiene Association Journal; Sept-Oct., 1970;
pp. 598-604.
7. "Asbestos Air Pollution in New York City", Nicholson;
Presented at the 2nd International Air Pollution Conference,
Washington, D. C.; Dec. 1970.
8. "Brake Lining Decomposition Products", Lynch;
Journal of the Air Pollution Control Association,
Vol. 18, No. 12, December, 1968.
9. "Identification & Control of Asbestos Exposures", Cralley;
American Industrial Hygiene Association Journal; February, 1971,
pp. 82-85.
10. Report of the Philadelphia Department of Public Health, Ad Hoc
Committee on Environmental and Occupational Hazards of Spray
Asbestos Fibers";
Committee; August 31, 1970.
11. (a) Testimony before Chicago City Council Environmental Control
Committee of Irving J. Selikoff, March 3, 1971;
(b) Testimony before Chicago City Council Environmental Control
Committee of Lewis Cralley, March 3, 1971;
(c) Testimony before Chicago City Council Environmental Control
Committee of George Wright, March 23, 1971;
(d) Testimony before Chicago City Council Environmental Control
Committee of J. Munroe McNulty, May 11, 1971.

12. Chapter 17 of Chicago Municipal Code amended Defining Asbestos Matter and Banning Usage Thereof;
13. Recommended Health Safety Practices, Nima (National Insulation Manufacturers Association, Inc.
14. "Fibrous and Mineral Content of Cosmetic Talcum Products:, Cralley, Key, Groth, Lainhart and Ligo; American Industrial Hygeine Association Journal, pp. 350-354.
15. "Cigarette Smoking, Asbestos, and Pulmonary Fibrosis", Weiss; American Review of Respiratory Disease, Vol. 104, 1971.
16. "Asbestos Fibres in Beverages and Drinking Water," Nature, Vol. 232, July 30, 1971, pp. 332-333.
17. "Talc-Treated Rice and Japanese Stomach Cancer", Science, Vol. 173, pp. 1141-42.
18. "Recommendations for Handling Asbestos," Engineering Equipment Users Association, E.E.U.A. Handbook No. 33:1969, pp. iii-45.
19. "Airborne Asbestos", Report Prepared by the Committee on Biologic Effects of Atmospheric Pollutants of the Division of Medical Sciences, National Research Council; National Academy of Sciences, National Academy of Engineering; 1971.
20. No. IITRI-C8216 Supplement A, "Asbestos in Brake Linings" Harwood. (enc)
21. Report by Dr. William J. Nicholson.
22. "Sprayed Fireproofing"; Mr. Harold Romer.
23. Statement by F. L. Pundsack.
24. Statement by Glifford L. Sheckler, Chairman, National Insulation Manufacturers Association Health and Safety Committee.
25. Report of Caterpillar Tractor Company.
26. Air and Water News. (enc)

27. Edwards + Lynch "The Method Used by the US Public Health Service for Enumeration of Asbestos Dust on Membrane Filter"; Ann. Occup. Hygiene (Oxford), 11 (1): 1-6, Jan. '68.

28. "Effects of Low Concentrations of Asbestos"; N Eng J med. Vol. 285, # 43; Dec. 2, 1971; Murphy et al.

R71-16

EXHIBIT 1



STATE OF ILLINOIS
POLLUTION CONTROL BOARD

189 WEST MADISON STREET SUITE 900

CHICAGO, ILLINOIS 60602

DAVID P. CURRIE, CHAIRMAN
SAMUEL R. ALDRICH
JACOB D. DUMELLE
RICHARD J. KISSEL
SAMUEL T. LAWTON, JR.

TELEPHONE
312-793-3620

NEWSLETTER #24
June 14, 1971

DUPAGE COUNTY REGIONALIZATION PLAN HEARINGS AUTHORIZED

At its Danville meeting on June 9, the Board authorized hearings on a proposal which would require the development of a regional wastewater treatment system in DuPage County. The proposal, if adopted, would be a county-wide system of nine sewage treatment plants in accordance with the Regional Wastewater Plan approved by the Northeastern Illinois Planning Commission. Presently, over 90 sewage treatment plants are operating in the county. The county and local governmental units involved would have until January 1, 1972, to submit a plan for accomplishing the regionalization scheme and final compliance would be required by January 1, 1975.

In his explanation of the proposal, Chairman Currie stated that the proliferation of small sewage plants in densely populated and rapidly developing DuPage County makes impossible the correction of existing pollution and constitutes a threat of further pollution in the future. Furthermore, small plants have higher per capita construction and operating costs. County taxpayers, therefore, have to pay a higher price for scattered small treatment plants than they would for larger and more efficient treatment facilities.

Hearings are scheduled for July, August, and September. The hearings will provide an opportunity for the people of DuPage County and Illinois to provide factual information and to express their views on the concept of regionalization. A copy of the proposal is appended to this Newsletter.

ASBESTOS HEARINGS SCHEDULED

Finding that the uncontrolled spraying of asbestos fiber and other fiber-containing material unreasonably interferes with the enjoyment of life and property and presents a severe threat to the public health

June 14, 1971

and welfare, the Board authorized in Danville that hearings be held on detailed proposed regulations which would restrict the commercial use of products containing asbestos. The set of regulations, if adopted, would be the most comprehensive set of controls for the toxic substance ever enacted.

The proposed regulations would, among other things, require a permit for the manufacturing of asbestos products, for the commercial use of asbestos in construction work and for the demolition of asbestos-containing buildings. Demolition of buildings containing asbestos would have to be conducted in accord with strict controls to reduce dust emission. Plants manufacturing asbestos products would be required to meet an emission standard. The spraying of asbestos fiber, done in high-rise construction, would be unlawful. And the use of asbestos in the brake lining of vehicles manufactured after January 1, 1975 and sold for use in Illinois would be prohibited. A copy of the proposed regulation is appended to this Newsletter.

Asbestos-containing products have, in recent years, been widely used, especially in the fireproofing of buildings and in the lining of automobile brakes. These uses and others contribute annually to the discharge of asbestos fiber into the atmosphere. The fiber, once released, is thought to be virtually indestructible, resulting in a gradual, irreversible increase in the amount of asbestos fiber present in the ambient air. Medical evidence has shown that heavy, prolonged exposure to asbestos fiber can cause a lung disease known as asbestosis and a very rare cancer of the lining of the lungs, mesothelioma.

NSSD PHOSPHORUS VARIANCE DENIED

At the Danville meeting, the Board voted to deny a variance request in North Shore Sanitary District v. EPA, PCB 71-36. The District requested that six of its seven lakeside plants (North Chicago, Lake Bluff, Lake Forest, Park Avenue, Ravine Drive, and Carey Avenue) be exempted from the phosphorus effluent standards until December 31, 1972. The one year variance was not requested for its Waukegan plant.

The Board's opinion, written by Mr. Dumelle, points out that the District's cost estimates for compliance were not realistic. The district maintained that temporary phosphorus treatment facilities would cost \$654,000 the first year and \$292,000 annually, thereafter. This sum includes costs for tanks, hardware, chemicals, additional sludge handling, additional labor, and a minimal salvage value. But, at a subsequent hearing relating to the Districts compliance to the Board's order in League of Women Voters et al v. North Shore Sanitary District, PCB 70-7, 12,13, 14, the District made a statement which is in effect an amendment to this variance request, thus substantially lowering the estimated cost. It withdrew its request for a variance as to the North Chicago plant. This removes almost half of the

hydraulic capacity from the request. The Board stated that other factors also reduce the estimated cost: The District could easily use a cheaper method of removal than the alum precipitation method it had chosen, such as the free waste pickle liquor method it uses at its Waukegan plant; as phosphorus removal becomes a more widespread requirement in Illinois, nearby Districts will be willing buyers of used equipment, the salvage value will be more than minimal. The Act admonishes the Board to grant variances only where cases of arbitrary or unreasonable hardship are shown. But the Board ruled that "[m]ere imposition of a money cost standing alone cannot be considered an unreasonable hardship."

The Board stated that since many of the District's recent activities are stymied by litigation it can have no real confidence that if this variance is granted, an extension of the variance will not be asked for a year from now.

LAKE MICHIGAN THERMAL STANDARD ADOPTED

The Board adopted the proposed draft of Thermal Standards for Lake Michigan, R70-2. These standards permit the continued existence of heat sources now in operation which have not been shown to cause harm, so long as they conform to the modified water quality standard which consists of a 3° F. rise limitation and a series of monthly temperatures which are never to be exceeded. The maximum permissible temperature is 80° F. Sources under construction as of January 1, 1971 must meet the new standard, and there are a number of additional conditions designed to minimize any adverse effect and to require modification if significant harm results. Finally, the potential damage inherent in the proliferation of large heat sources is prevented by prohibiting significant new heat sources not under construction as of January 1, 1971. A copy of the proposal is continued in the addenda section of Newsletter #21.

OTHER ACTIONS

At its June 9 meeting in Danville the Board announced the following actions:

In League of Women Voters et. al. v. North Shore Sanitary District, PCB 70-7, 12, 13, & 14, the Board ordered several control programs to be started immediately. These interim measures will help abate pollution violations until District completes its expansion program ordered previously by the Board (March 31, 1971). This order in no way affects the Board's order for the District's phosphorus variance request (PCB 71-36).

In Allied Mills, Inc. v. EPA, PCB 71-16, the Board granted a water pollution variance until July 1, 1972, at which time its Taylorville waste treatment facilities must be in compliance with SWB-14. The company must post a performance bond to insure complete treatment of its storm and process waste waters.

In EPA v. C.E. Koons, PCB 71-30, the Board issued a cease-and-desist order restraining Respondent from depositing refuse on his Flora property. He is also to remove or cover the refuse and to pay a \$100 penalty. Due to financial difficulty, he claimed he couldn't operate a landfill in accord with the law. The Board finds that "[p]overty is no excuse; people who haven't money to do business as the law requires shouldn't do business."

In EPA v. Southern Illinois Asphalt Co. Inc., PCB 71-31, the Board issued a cease-and-desist order until Respondent obtain the required permit and also issued a \$5,000 penalty for its failure to do so. The Board also recommended the Agency consider the effect the plant has on the surrounding residential area in McLeansboro before granting the pending permit application. Mr. Lawton's concurring opinion points out that "...compliance with the regulations, which are designed for general application, does not justify the causing of a nuisance."

In Northern Petrochemical Co. v. EPA, PCB 71-47, the Board granted an air pollution variance so that the company may conduct a fire-fighting training course on its Morris property. This case is similar to Deere & Co. v. EPA, PCB 70-20, where the Board noted that "[t]he importance of instructing employees in fire-fighting techniques is clear ... and that ... there is no substitute, in learning how to fight fires, for actually fighting fires."

In EPA v. Truax-Traer Coal Co. and Consolidation Coal Co., PCB 71-54, the Board dismissed a purported variance petition because the petitioner failed to comply with statutory and regulatory provisions for the filing of a variance petition. In effect the petitioner sought a declaratory judgement that the Act does not apply to its Jackson County operations. The Board also directed the Agency to file within twenty days a detailed report for abatement proposals for the petitioner's operations.

In Electric Wheel Co. v. EPA, PCB 71-55, the Board granted a variance until December 31, 1971, so that the petitioner may complete its ACERP with minor modifications at its Quincy foundry. The Board felt that the Agency's delay in filing recommendations and the company's good faith in pursuing the program were sufficient reasons to grant the variance.

In City of Lincoln v. EPA, PCB 71-56, the Board denied a variance request to burn allegedly diseased elm trees. Upon the City's own admission, the trees weren't infectious but only dead and rotten. The Board stressed that the diseased exception is not to be abused by attempting, under cover of the exception, to burn vegetation blown down by a storm.

In Citizens Utilities Company of Illinois v. EPA, PCB 71-125, the Board granted the DuPage County company's petition to expedite proceedings. The variance request is for temporarily bypassing a stabilization lagoon so it may be cleaned as hot weather has made it substantially non-functional. In this exceptional situation, the Board has waived the 21-day notice rule. But sufficient notice is given by this newsletter to inform interested parties. The hearing will be held in the early part of the week of June 21st.

MISSISSIPPI RIVER THERMAL STANDARDS

The record in Mississippi River Thermal Standard, R 70-16, a citizen initiated proposal to change the Mississippi River thermal discharge regulations, will remain open until June 21. In addition to the testimony and exhibits received in the public hearings at Rock Island and Alton the record has been open to receive communications from interested persons and the testimony regarding thermal discharges in the hearing on the Quad-Cities nuclear electric generating plant permit application, PCB 71-20.

CORRECTIONS

Newsletter #22 contained two typographical errors as follows:

- I. Explanation of Proposed Water Quality Standards Revisions
R 71-14; Part IX: Permits page 15, second full paragraph -
The date in the first line should be December 31, 1973
not December 31, 1971.
- II. Proposed Water Quality Standards Revisions - # R 71-14;
Part II: Water Quality Criteria
Section 203 (b) - The range of pH should be 6.0 to 9.0
not 6.5 to 9.0.

NEW CASES FILED AND HEARINGS AUTHORIZED

PCB 71-127 Illinois State Fair Agency.

Springfield petitioner seeks a variance from open burning regulations to conduct interim fire-fighting training and to dispose of single story building. No hearing was authorized pending Agency recommendation.

PCB 71-128 City of Zion.

Petitioner requests an open burning variance to burn diseased trees in Lake County. No hearing was authorized pending Agency recommendation.

PCB 71-129 Commonwealth Edison Company.

Petitioner seeks a variance from air pollution regulations at three stations in Winnebago, Will and Tazewell counties. A hearing was authorized.

June 14, 1971

PCB 71-130 Chicago-Dubuque Foundry Corporation.

East Dubuque petitioner seeks a variance from air pollution regulations. The company wants to wait until the United States EPA adopts National Ambient Air Quality Standards before installing control equipment. No hearing was authorized pending Agency recommendation.

PCB 71-131 Willow Creek Drainage District.

Morgan County petitioner seeks a variance from open burning regulations so it may burn refuse from clean-up operations. No hearing was authorized pending Agency recommendation.

PCB 71-132 American National Bank & Trust Co.

Petitioner seeks a variance from the prohibition of new sewer connections to the North Shore Sanitary District in order to connect a new sixty-five unit complex. A hearing was authorized.

PCB 71-133 Thomas and Sandra Keading.

Lake County petitioners seek variance to connect a one family residence to the NSSD. A hearing was authorized.

PCB 71-134 Union Oil Co. of California.

Chicago Refinery of Union 76 Division seeks open burning variance to conduct fire-fighting training classes. No hearing was authorized pending Agency recommendation.

PCB 71-135 Ravinia Festival.

Petitioner seeks open burning variance so it may burn diseased elm trees at its Highland Park location. No hearing was authorized pending Agency recommendation.

PCB 71-136 A.H. Zamost.

Petitioner requests a variance to connect a new single family residence to the NSSD. A hearing was authorized.

ILLINOIS POLLUTION CONTROL BOARD

Notice is hereby given that the Illinois Pollution Control Board has scheduled the following hearings in addition to those previously announced along with the changes of scheduled meetings as described. Public comments are invited.

June 21	1:00 p.m.	<u>EPA v. City of Champaign v. The Board of Trustees of the University of Illinois and Alpha Material and Fuel Company, PCB 71-51C, Champaign City Council Chambers, Champaign, Illinois.</u>
June 22&23	10:00 a.m.	Hearing continued from June 8, 1971.
June 23	10:00 a.m.	<u>Elgin Jaycees v. Tri-County Landfill Co. et. al., PCB 71-59, Pre-hearing conference, City Council Chambers, Elgin Municipal Bldg., 150 Dexter Ct., Elgin, Illinois.</u>
June 26	10:00 a.m.	<u>Citizens Utilities Co. of Ill. v. EPA PCB 71-125, DuPage County Sheriff's Office, Courtroom #7, 205 Reber, Wheaton, Illinois. (Accelerated Date).</u>
June 29	10:00 a.m.	<u>Proposed Water Quality Standards Revisions, R 71-14, Central Junior High School, Auditorium, 21st & Delmore, Granite City, Illinois.</u>
June 24	10:30 a.m.	<u>Employees of Holmes Brothers v. Merlan, Inc. and L. Mervis, PCB 71-39, City Council Chambers, City Hall, Danville, Illinois.</u>
June 30	10:30 a.m.	<u>Proposed Water Quality Standards Revisions, R 71-14, Southern Illinois University, Neckers Hall, B-440, Carbondale, Illinois.</u>
June 30 & July 7	10:00 a.m.	<u>EPA v. Incinerator, Inc. PCB 71-69, Cicero Public Library, 5221 W. Cermak Rd., Cicero, Illinois</u>
July 1	10:00 a.m.	<u>National Gypsum Co. v. EPA, PCB 71-99, Council Chambers, City Hall, 106 N. Utica St., Waukegan, Illinois.</u>

June 14, 1971

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NEWSLETTER #24

July 7, 8, 10:00 a.m.
& 9

July 8 10:00 a.m.

July 8 10:00 a.m.

July 13 9:15 a.m.
10:30 a.m.
11:45 a.m.
2:00 p.m.
3:15 p.m.

July 14 10:00 a.m.

July 14 & 15 10:00 a.m.

July 21 & 22 10:00 a.m.

July 29 10:00 a.m.

July 23 10:00 a.m.

LATE NOTICES

July 17, 24, 25
thru July 2.

EPA v. Modern Foundry & Manufacturing Co., PCB 71-108, City Council Chambers, Belleville, Illinois.

Board Meeting, Belleville Court House, Belleville, Illinois.

Scott Volkswagen, Inc. v. EPA, PCB 71-111; Waukegan Public Library, 128 North County, Waukegan, Illinois.

Ray Wickstrom, PCB 71-110; Howard & Barbara Weinstein, PCB 71-113; David S. McAdams, PCB 71-122; Robert D. Charles, PCB 71-126; Dole Construction Co., PCB 71-128; Waukegan Public Library, 128 North County, Waukegan, Illinois.

City of Monmouth, Ill. v. EPA, PCB 71-121; City Hall Council Chambers, 112 N. Main St., Monmouth, Illinois. (Corrected Date).

Sherwin-Williams Co., PCB 71-111; Graham Paint & Chemical Co., Inc., PCB 71-115; General Paint & Chemical Co., PCB 71-116; Enterprise Paint Mfg. Co., PCB 71-117; Armstrong Chemcon, Inc., PCB 71-118; Jewel Paint & Varnish Co., PCB 71-119; The Valspar Corp., PCB 71-120; H.L. Industries, Inc., PCB 71-120; Illinois Liquor Control Commission Hearings Room, Room 419, 180 W. Randolph St., Chicago, Illinois.

Ralston Purina v. EPA, PCB 71-88, University Union, 3rd floor lounge on July 21, Ballroom on July 22, Illinois State University, Normal, Illinois.

EPA v. Eli Amizoni, PCB 71-89, Supervisors Office, Court House, Eureka, Illinois. (Corrected Date).

Henry Hannah v. Minnesota Paints, Inc., PCB 71-123, City Hall Council Chambers, 915-16th Ave., East Moline, Illinois.

EPA v. Granite City Steel Co., PCB 70-34, Circuit Court Courtroom, City Hall, Granite City, Illinois.

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ILLINOIS POLLUTION CONTROL BOARD
COMING FORMAL MEETINGS AND HEARINGS

June 17 & 18	10:00 a.m.	<u>Lake Michigan Waste Water Discharge Prohibition R 71-5, and Proposed Water Quality Standards Revisions, R 71-14, Auditorium, Technological Institute, Northwestern University, Evanston, Illinois.</u>
June 17	9:30 a.m.	<u>EPA v. J.C. Dill, PCB 71-42, Danville City Hall, Danville, Illinois.</u>
June 18	10:00 a.m.	<u>Robert H. Monyek v. EPA, PCB 71-80, Waukegan City Hall, 106 N. Utica, Waukegan, Illinois.</u>
June 18	1:00 p.m.	<u>National Starch & Chemical Corp. v. EPA, PCB 71-83, Village Hall, Meridosia, Ill.</u>
June 19	10:00 a.m.	<u>EPA v. Claremont Hills Water & Sewer Co., Inc. PCB 71-87, McHenry County Court House Annex, 121 W. Calhoun, Woodstock, Illinois.</u>
June 21	10:00 a.m.	<u>Robert C. Wagnon v. EPA, PCB 71-85, 10th Floor, Lake County Court House, Waukegan, Illinois.</u>
June 21 22 & 23	1:00 p.m. 10:00 a.m.	<u>EPA v. City of Champaign v. The Board of Trustees of the University of Illinois and Alpha Material and Fuel Company, PCB 71-51C, Champaign City Council Chambers, Champaign, Illinois. Hearing continued from June 8, 1971.</u>
June 23	10:00 a.m.	<u>Elgin Jaycees v. Tri-County Landfill Co. et. al., PCB 71-59, Pre-hearing conference, City Council Chambers, Elgin Municipal Bldg., 150 Dexter Ct., Elgin, Illinois.</u>
June 23	10:00 a.m.	<u>Board Meeting, Chicago Circle Campus, 750 S. Halsted St., Chicago, Illinois.</u>
June 24	10:00 a.m.	<u>Chambers, Bering, Quinlan Co. v. EPA, PCB 71-102, Supervisors Room, Macon County Building, Decatur, Illinois.</u>
June 24	10:00 a.m.	<u>Proposed Water Quality Standards Revisions, R 71-14, State of Illinois Regional Office Building, 4302 N. Main St., Rockford, Illinois.</u>
June 24	10:30 a.m.	<u>Employees of Holmes Brothers v. Merlan, Inc. and L. Mervis, PCB 71-39, City Council Chambers, City Hall, Danville, Illinois.</u>

June 25	10:00 a.m.	<u>Proposed Water Quality Standards Revisions</u> , R 71-14, Galena City Hall, Galena, Illinois.
June 25	10:30 a.m.	<u>John Ciancio v. EPA</u> , PCB 71-100, Waukegan Public Library, 128 N. County, Waukegan, Ill.
June 25	1:30 p.m.	<u>Mr. and Mrs. Wallace W. Piroyan v. EPA</u> , PCB 71-103, Waukegan Public Library, Waukegan, Ill.
June 25	10:30 a.m.	<u>EPA v. Charles Rhodes</u> , PCB 71-53, Room 502, Macon County Courthouse, 253 E. Wood Street, Decatur, Illinois.
June 26	10:00 a.m.	<u>Citizens Utilities Co. of Ill. v. EPA</u> , PCB 71-125, DuPage County Sheriff's Office, Courtroom #7, 205 Reber, Wheaton, Illinois. (Accelerated Date).
June 28	10:00 a.m.	<u>Quad City Area Regional Air Pollution Control Board v. Village of Cordova</u> , PCB 71-97, County Building, 1504 Third Ave., Rock Island, Illinois.
June 29	10:00 a.m.	<u>Proposed Water Quality Standards Revisions</u> , R 71-14, Central Jr. High School, Auditorium, Granite City, Illinois.
June 29	10:00 a.m.	<u>EPA v. Williamson County Housing Authority</u> , PCB 71-109, City Hall Herrin, Illinois.
June 29 & 30	10:00 a.m.	<u>EPA v. City of Urbana v. Urbana & Champaign Sanitary District, Saline Branch Drainage District, and the Board of Trustees of the University of Ill.</u> , PCB 71-51U, Urbana City Council Chambers, Urbana, Illinois.
June 29 - July 2	10:00 a.m.	<u>U.S. Industrial Chemical Co. v. EPA</u> , PCB 71-44, Forty-Martyrs School Hall, Tuscola, Illinois.
June 30	10:00 a.m.	<u>Proposed Water Quality Standards Revisions</u> , R 71-14, Southern Illinois University, Neckers Hall, B-440, Carbondale, Illinois.
June 30	9:30 a.m.	<u>Flintkote v. EPA</u> , PCB 71-68, Wabash County Courthouse, 4th & Market, Mount Carmel, Illinois.

June 30 & July 7	10:00 a.m.	<u>EPA v. Incinerator, Inc.</u> , PCB 71-69, Cicero Public Library, 5221 W. Cermak Rd., Cicero, Illinois.
June 30	10:00 a.m.	<u>EPA v. City of Marion</u> , PCB 71-25, 2nd Floor Courtroom, City Hall, Marion, Illinois.
July 1	10:00 a.m.	<u>Standard Brands v. EPA</u> , PCB 71-98, City Hall, Pekin, Illinois
July 1	10:00 a.m.	<u>National Gypsum Co. v. EPA</u> , PCB 71-99, Council Chambers, City Hall, 106 N. Utica St., Waukegan, Illinois.
July 7, 8, & 9	10:00 a.m.	<u>EPA v. Modern Foundry & Manufacturing Co.</u> , PCB 71-108, City Council Chambers, Belleville, Illinois.
July 8	10:00 a.m.	<u>Freeman Coal Mining Corp. v. EPA</u> , PCB 71-78, City Council Chambers, 1100 Main St., Mt Vernon, Illinois.
July 8	10:00 a.m.	<u>Scott Volkswagon, Inc. v. EPA</u> , PCB 71-112; Waukegan Public Library, 128 North County, Waukegan, Illinois.
July 8	10:00 a.m.	Board Meeting, Belleville Court House, Belleville, Illinois.
July 12	9:30 a.m.	<u>EPA v. Bath, Inc. and John C. Walker</u> , PCB 71-52, Room 502, Macon County Court- house, 253 Eastwood, Decatur, Illinois.
July 13	9:15 a.m. 10:30 a.m. 11:45 a.m. 2:00 p.m. 3:15 p.m.	<u>Ray Wickstrom</u> , PCB 71-105 <u>Howard & Barbara Weinstein</u> , PCB 71-107 <u>David S. McAdams</u> , PCB 71-113 <u>Robert D. Charles</u> , PCB 71-122 <u>Dole Construction Co.</u> , PCB 71-126; Waukegan Public Library, 128 North County, Waukegan, Illinois.
July 14	10:00 a.m.	<u>City of Monmouth, Ill. v. EPA</u> , PCB 71-121 City Hall Council Chambers, 112 N. Main St., Monmouth, Illinois. (Corrected Date).

June 14, 1971

July 14 & 15	10:00 a.m.	<u>Sherwin-Williams Co., PCB 71-111;</u> <u>Graham Paint & Varnish Co., Inc., PCB 71-114;</u> <u>General Paint & Chemical Co., PCB 71-115;</u> <u>Enterprise Paint Mfg. Co., PCB 71-116;</u> <u>Armstrong Chemcon, Inc. PCB 71-117;</u> <u>Jewel Paint & Varnish Co., PCB 71-118;</u> <u>The Valspar Corp., PCB 71-119;</u> <u>N.L. Industries, Inc., PCB 71-120;</u> <u>Illinois Liquor Control Commission Hearing</u> <u>Room, Room 419, 188 W. Randolph St.,</u> <u>Chicago, Illinois.</u>
July 21 & 22	10:00 a.m.	<u>Ralston Purina v. EPA, PCB 71-88,</u> <u>University Union, 3rd floor lounge on</u> <u>July 21, Ballroom on July 22, Illinois</u> <u>State University, Normal, Illinois.</u>
July 29	10:00 a.m.	<u>EPA v. Eli Amigoni, PCB 71-89, Supervisors</u> <u>Office, Court House, Eureka, Illinois.</u> <u>(Corrected Date).</u>
July 23	10:00 a.m.	<u>Henry Hannah v. Minnesota Paints, Inc.,</u> <u>PCB 71-123, City Hall Council Chambers,</u> <u>915-16th Ave., East Moline, Illinois.</u>

LATE NOTICES

June 17, 18, 24, 25, & 28 thru July 2.	9:30 a.m.	<u>EPA v. Granite City Steel Co., PCB 70-34</u> <u>Curcuit Court Courtroom, City Hall,</u> <u>Granite City, Illinois.</u>
June 17 & 18	10:00 a.m.	<u>Lloyd A. Fry Roofing Co. v. EPA, PCB 71-4,</u> <u>71-33, One North LaSalle St., Suite 3700,</u> <u>Chicago, Illinois (Corrected Date).</u>
July 23 & 30	10:00 a.m.	<u>DuPage County Regionalization, R 70-17</u> <u>location to be announced.</u>
July 26	10:00 a.m.	<u>Monsanto Co. v. EPA, PCB 71-110, Sauget</u> <u>City Hall, 2897 Monsanto Avenue, Suaget,</u> <u>Illinois.</u>

R 70-17
DuPage County Regional Sewage Treatment

EXPLANATION OF PROPOSED REGULATIONS
(by Mr. Currie)

DuPage County, bedroom to Chicago, is experiencing the familiar growing pains of a rapidly developing suburban area. Beset by a typical multiplicity of local governments and with its county government hampered by a legal incapacity to supplant local sewage treatment facilities without local consent, DuPage has sprouted an inordinate number of separate treatment plants (83 in 1970 (R. 319)), many of them quite small indeed, and in many cases quite close to one another.¹ Small plants are a necessity in remote areas sparsely populated, but not in DuPage County, which has a high population density and a considerable total population. In DuPage County the costs of transporting sewage to central locations for treatment are low.

Inquiry hearings held in February 1971 have established the disadvantages of unnecessarily small plants, including their threat of pollution, as set forth in the proposed Rule 1001. The testimony of Dr. John Pfeffer of the Institute for Environmental Quality (R. 176-94) is a concise summary of this proof. Operation costs for a plant treating one million gallons per day, he testified, are three times those of a plant treating 100 million gallons, on a per-gallon basis; construction costs are considerably higher as well. As an example of the savings of regional sewage treatment, Dr. Pfeffer cited costs of alternative treatment systems to serve Albany County, New York: For each of the eight affected municipalities to treat its own sewage would have cost \$78,500,000, required 110 operating personnel, and cost \$29.00 per capita for the first year. A regional two-plant system cost \$53,100,000 and required only 35 operators, at half the per capita cost.

On the question of the unsatisfactory effluent from small plants Dr. Pfeffer referred to the use of less effective trickling filters for secondary treatment in such plants, because of the necessity for expert operators, beyond the means of small plants, to run the more acceptable activated sludge facilities. This fact we have recognized in prescribing more lenient effluent standards for small plants (e.g., SWB-12, SWB-13, as amended by # R 70-3, Jan. , 1971); it is an additional reason why we cannot allow unnecessarily small plants in areas that can support large ones. Dr. Pfeffer pointed also to the probability that sophisticated advanced treatment methods required now or in the future will likewise be beyond the effective competence of small plants. Moreover, he testified, larger plants are more reliable, both because

1. For excruciating details see Northeastern Illinois Planning Commission, Preliminary Wastewater Plan for the Inner Area (1969). pp. 5-4 - 5-10.

of better maintenance and because of their backup capacity to avoid pollution during malfunctions. In a larger plant, he said,

You have generally multiple units, which, if one of the particular units becomes inactivated for some reason, you can increase the load slightly to the remaining unit without markedly affecting the efficiency.

If you are sitting here with a small plant that has one primary clarifier and you have to shut that clarifier down to replace the mechanism then all the water is bypassed, and in the past it has been letting raw sewage into the stream

So far as our hearings have disclosed, everybody agrees, and has agreed for years, that the present proliferation of small plants is intolerable and that they should be promptly replaced by eight to a dozen relatively large plants each serving a relatively large area. The County has been pressing for regionalization since 1956 (R. 27). The DuPage County Association of Sanitary Districts endorsed a plan for regional treatment in 1968 (R. 20). The DuPage County Mayors and Managers Conference in February of this year adopted a plan for regional treatment (R. 9).

Thus the various governments responsible for sewage treatment in DuPage County-- the municipalities through their Mayors and Managers, the Sanitary Districts, and the County itself--all agree as to what should be done. But they are unable to decide who is to do it or how it is to be paid for. A 1970 bond issue to establish a county-operated regional system was roundly defeated, apparently chiefly because it was thought to impose an excessive proportion of the financial burden on areas of the county that already had provided for their own sewage treatment (R. 9, 22, 23). Even today there remains disagreement. The County apparently expects to run the regional plants (R. 27-31); the municipalities want to control a county-wide waste water board operating several subregional plants (R. 16-17); the sanitary districts want the county divided into several independent sanitary districts (R. 202, 235-36).

In the meantime it is alleged that cities sometimes refuse to accept sewage from outside their boundaries, necessitating the construction of inefficient small plants or the undesirable use of septic tanks in densely populated areas (R. 251, 263) and that the county has blocked the expansion of existing sanitary districts (R. 229-30). At the same time the cities and sanitary

districts do not seem eager to ask the county to take over their functions. Federal construction grants are unavailable for DuPage County until a regional plan is established (R. 121, 125). Bills have been introduced in the General Assembly to set up various regional treatment authorities with the requisite financial powers (R. 16, 234-35). They languish for want of consensus over who is to be abolished and who expanded. We have even been asked by a respectable quasi-public agency to allow inadequate facilities to continue violating the pollution standards while the debate goes on (R. 61). The situation is exquisitely Byzantine.

Legislation to clarify the administrative responsibility and financial authority for carrying out the agreed program is obviously desirable. The General Assembly should create, without referendum, a county-wide sanitary district with exclusive authority to construct and operate sewage treatment facilities throughout the county, and with adequate authority to raise money by taxes, general obligation and revenue bonds, special assessments, user and connection charges, and other means. The County, or the General Assembly, should also enact a subdivision ordinance, if one is not now in effect, requiring any new land developer to pay the cost of additional facilities required to collect and treat its anticipated sewage before permission is given to begin development. We trust some such legislation will be enacted this autumn.²

We cannot, however, count on it, nor afford to wring our hands while we wait. It has taken fifteen years already, and nothing has been done. We have no assurance that legislation will be forthcoming. The existing governments in DuPage County have authority to do what is necessary even under the present patchwork laws; and what they have power to do we can and should order them to do in order to remove the present threat of pollution. See Environmental Protection Act, section 13 and 13 (c), authorizing the Board to adopt regulations to prevent water pollution, specifically including standards relating to the construction and operation of facilities designed to prevent pollution.

2. The bill proposed by DuPage and Lake Counties to allow for county-run subregions each bearing its own costs would be satisfactory if it did not leave the creation of subregions up to a referendum and allow deannexation by local option, and if section 46 of the Environmental Protection Act were simultaneously amended to include counties, as discussed below.

The County and all municipalities and Sanitary districts have authority to construct, acquire, and operate sewage treatment facilities (Ill. Rev. Stat., ch. 24, § 11-141-2 (Municipalities); ch. 34, § 3111 (counties); ch. 42, §§ 306, 306.1 (sanitary districts)). All have the power of condemnation without regard for municipal or sanitary district boundaries (ch. 24, § 11-141-2; ch. 34, § 3110; ch. 42, § 307). Municipalities and sanitary districts may accept and treat sewage from outside their boundaries (ch. 24, § 11-146-1; ch. 42, § 316); may contract with one another, if adjacent, for the common construction or use of facilities (ch. 24, § 11-147-1); and may contract for the county to provide sewage treatment, the local government paying its share of costs (ch. 24, § 11-147-4; ch. 34, § 3131). All these governments have authority to raise funds by revenue and general obligation bonds, by user and connection charges, by general taxation, and by special assessment under the new Constitution effective July 1 (R. 168).

The Northeastern Illinois Planning Commission, after extensive study, has adopted a Regional Wastewater Plan for the Chicago metropolitan area. This plan, endorsed by the Environmental Protection Agency at our hearings (R. 149), contemplates a nine-plant system consisting of the existing plants operated by Glen Ellyn and West Chicago and by the Wheaton and Hinsdale Sanitary Districts; a consolidation of the existing Elmhurst and Salt Creek Sanitary District plants; and new plants at Naperville, Woodridge, and (two) in the Addison-Woodridge area (See NIPC, Regional Wastewater Plan, adopted March, 1971; R. 59-61, 63-64). This plan appears, on the basis of the evidence so far before us, to be an entirely reasonable means of eliminating the present problem. In the absence of contrary evidence, we think it should be carried out at once.

We could draft regulations prescribing exactly which government is to build which plant. It would appear sensible, if nothing better can be worked out in a reasonable time, that the Wheaton and Hinsdale Sanitary Districts and Elmhurst, Glen Ellyn, and West Chicago each take responsibility for treatment in its region, since they are operators of existing plants that are to be expanded to become parts of the new plan, and that the County build the new plants. Or we could order everyone else to contract for the County to supply service everywhere. We shall not hesitate to order just this if the parties and the legislature cannot agree on a better institutional framework such as a single county-wide district as suggested above. But we propose to allow six months for such a framework to be created before doing so. Consequently the present proposal is to require the implementation of the NIPC plan by January, 1975, allowing six months for a resolution of the administrative

questions, which will be settled in an adjudicative Board hearing that will approve a final detailed program, and three years for the construction of facilities. The proposal makes clear that future plants must conform to the regional principle; that no new development will be permitted after 1975 until regionalization is accomplished; and that regionalization will not be permitted to postpone compliance with existing or future water quality standards or requirements for advanced waste treatment.

Mayor Nottke of Itasca put the matter clearly and succinctly:

There has been much talk, much dialog, much discussion going on, and nothing has been done I think we are going to lean on the Pollution Control Board We need you here to say, "This must be done." We can't sit back and wait on the county or the mayors and managers to come up with a solution, because in my opinion . . . it will never happen. We are not going to get an agreement because we have constant bickering between the sanitary districts and the communities. They are all looking for perpetuation of their system, for autonomy, for control. I think it has got to be taken away and I say the Pollution Control Board has to be the controlling agency and you have to come up with a definite situation and say, "This is going to be it." You have a mandate from the people of DuPage County, and that is to form a nine-plant district (R. 275, 280, 289).

We agree that it is for us to unravel the present jam by prescribing what shall be done. We cannot create sanitary districts, but we can order plants built and intend to do so. The streams cannot wait for consensus among those who will not come to terms. We add that it is not only the people of DuPage County who suffer from the inability of their governments to get together on this issue; those downstream of DuPage's inadequate plants are forced to accept whatever DuPage puts into its revolting streams. It is therefore imperative that the people of the County not be permitted a veto power over the construction of necessary treatment works, and it will become even more essential, if the Legislature chooses to entrust the job of carrying out NIPC's plan to the County itself, that section 46 of the Environmental Protection Act be amended, as the Board has already requested, to permit non-referendum bonds by counties as well as by other local governments when ordered by this Board. The omission of counties from section 46 was an oversight that has no justification in policy; we urge once again that it be quickly rectified.

Public comment on the proposed regulations is invited for the hearings to be held in July, August, and September.

PROPOSED REGULATIONS
(Authorized for hearing only, June 9, 1971.
Not yet adopted by the Board.)

CHAPTER IV: WATER POLLUTION
PART 11: Regional Sewage Treatment

- 1101 Preamble. The proliferation of numerous small sewage treatment plants in densely populated and rapidly developing Du Page County constitutes a severe and intolerable impediment to the correction of present water pollution and a continuing threat of additional pollution in the future. Not only do the higher unit costs of constructing and operating small plants waste finite dollars and therefore contribute to pollution; small plants cannot produce as satisfactory an effluent as can larger plants, because they cannot provide certain types of sophisticated treatment, because they cannot practicably be adequately supervised or maintained, and because they cannot provide adequate standby treatment capacity to prevent pollution in the event of a malfunction. The establishment or continued operation of sewage treatment plants so small as to exhibit these deficiencies, in areas such as DuPage County where the population density is high enough to make larger plants economically feasible and indeed economically far more desirable, is contrary to the anti-pollution policy of the Environmental Protection Act.
- 1102 Regional Plants to Be Constructed. On or before January 1, 1975, all sewage treatment plants now or then existing in DuPage County shall be replaced by nine plants designed and constructed in substantial accordance with the Regional Wastewater Plan approved by the Northeastern Illinois Planning Commission March 3, 1971. Governmental agencies operating these nine plants shall thereafter accept and treat all sewage from tributary areas as designated by that Plan, and all other sewage treatment plants in DuPage County shall cease operation, except as provided in Rule 1005 of this Chapter.
- 1103 Programs to Be Submitted. On or before January 1, 1972, the County of DuPage and every municipality or sanitary district with current responsibility for the treatment of sewage in DuPage County shall file with this Board and with the Environmental Protection Agency a detailed and firm program for compliance with the requirements of Rule 1102 of this Chapter. Such programs, preferably submitted on a joint basis, shall include among other things the following:

a. A description of the location, capacity, treatment facilities, cost, and service area for the proposed plant that is to accept and treat the sewage from the area under the applicant's jurisdiction;

b. A firm commitment by one or more responsible governmental agencies to construct and operate the proposed plant, together with a firm schedule for design, award of contracts, commencement of construction, full operation, and abandonment of obsolete plants:

c. A firm commitment by the affected governmental agencies for the financing of the necessary facilities, through taxation, user charges, revenue and general obligation bonds, special assessments, connection charges, loans, grants, contracts, or otherwise;

d. An attorney's opinion affirming and explaining the authority of the responsible governmental agencies to carry out their undertaking as described;

e. Assurance that the program conforms with Rule 1002 of this Chapter and with all applicable regulations respecting air and water pollution and the disposal of solid waste;

f. Assurances as to what interim measures will be provided to secure compliance with applicable regulations regarding sewage treatment, including water quality standards, before January 1, 1975.

1104 Compliance Hearing. (a) Upon receipt of the programs required by Rule 1103 of this Chapter, or in default of their timely receipt; the Board shall schedule a hearing or hearings in accordance with Chapter I of these Rules and Regulations, in which to determine whether the programs submitted will achieve compliance with this Part. The governments specified in Rule 1103 of this Chapter shall be parties petitioner in such hearings, and the Environmental Protection Agency shall be respondent. The Agency may file a countercomplaint against any person allegedly responsible for any failure to conform with the requirements in this Part.

b. After hearings as specified in paragraph (a) of this Rule, the Board shall determine whether the programs submitted are adequate to achieve compliance with this Part. The Board may modify the submitted programs in any manner conducive to the achievement of the policies or requirements of this Part, and shall order the parties to do whatever is necessary or proper to achieve those ends.

- 1105 Future Construction. No permit shall be granted for the construction or operation of any additional sewage treatment plant in DuPage County, except for interim facilities in the event of a demonstrated emergency, that does not conform with the principles of the Regional Wastewater Plan described in Rule 1102 of this Chapter, or that is of capacity so small as to produce significant diseconomies of scale or to create a significant risk of inadequate treatment, according to the policies expressed in Rule 1101 of this Chapter.
- 1106 Limitation on New Connections. After January 1, 1975, no source of wastes may be connected, and no existing source may be increased in the strength or volume of discharge, to any sewer in DuPage County not tributary to a sewage treatment plant that meets the requirements of this Part.
- 1107 Other Regulations Preserved. Nothing in this Part shall relieve any person from the obligation to comply in all respects with any existing or future regulations, including but not limited to the water quality standards and implementation plan for streams in DuPage County.

§ R 71-16
Asbestos and Spray Insulation

EXPLANATION OF PROPOSED REGULATIONS

Science is pointing its medical finger at asbestos and describing the "miracle fiber" as a mineral whose commercial blessings can exact a rather costly toll. Asbestos insulation and factory workers exposed to heavy concentrations of asbestos fiber over prolonged periods have suffered devastating consequences. They are visited with an alarmingly high incidence of asbestosis, which produces a diffuse fibrosis of the lower lung, of lung cancer and of an extremely rare cancer of the lining of the lung and abdomen, mesothelioma. The rates of lung cancer are extraordinarily high especially among asbestos workers who are smokers, indicating the synergistic potential of the mineral fiber.

The medical evidence is indefinite as to why asbestos is carcinogenic, with three theories in vogue: (1) The fibers once ingested or inhaled act as a continuous physical irritant, inducing a tumor after 20 to 30 years; (2) The fibers contain carcinogens such as benzo(a)pyrene; (3) Fibers accumulate in the lung and remain immobilized as "asbestos bodies" which cause cancer upon disintegration.

Similarly, while the adverse effect of prolonged exposure to heavy concentrations of asbestos is clearly documented, the medical data is disturbingly vague as to the results of long-term exposure to low and moderate concentrations. Nor is there a clear dose-time relationship for exposure to heavy concentrations. In short, as with many other environmental contaminants, the public has discovered too late that asbestos is potentially dangerous and too little of the extent of that threat.

But two depressing facts are agreed upon: That millions of Americans are likely to contain "asbestos bodies" in their lungs and that asbestos fiber once released is relatively indestructible. That is, the qualities of durability and of resistance to normal chemical and physical stress, which make asbestos such a commercial success, render it potentially dangerous to present and future generations because of the tendency of the fibers to accumulate in the atmosphere and in the lungs.

In light of these facts, the PCB has proposed the nation's most comprehensive set of asbestos controls. To ban all uses of asbestos would be "alarmism" at its most clamorous. But ignoring

the need to explore and, perhaps, to adopt controls for the significant contributors to a swelling volume of asbestos fiber in our air would constitute a foolish complacency.

To this end, the suggested regulations would identify these "significant contributors" as the commercial use of asbestos in construction work; the demolition of asbestos-containing structures; the manufacturing and processing of asbestos-containing products; the transportation of certain asbestos-containing products; the disposal of asbestos-containing waste; and the use of asbestos in the brakes of millions of vehicles.

Part II would impose several general requirements upon all commercial construction, repair, alteration, demolition, manufacturing or processing activity from which asbestos fiber is discharged into the environment. The persons responsible for such activity must obtain a permit from the EPA. The permit system will serve to produce the advanced assurance of compliance with the applicable control regulations and will thus reduce the cost and burden of in-the-field enforcement.

In addition these commercial activities would have to provide facilities to prevent the removal of asbestos fibers from the site of such activity on the clothing of employees. Indeed, this poses a rather new concept of air pollution control. Those who have died from lung ailments induced by long, close association to an asbestos worker may be powerful proof of the need for novelty.

Finally, Part II would provide for careful waste disposal procedures, as wind-blown refuse heaps and scrap piles contribute to the volume of asbestos fiber in our air. The simple steps for control eliminate any excuse for such dangerously sloppy housework.

Part III would prohibit the spraying of asbestos-containing insulation and fireproofing. Experience has indicated that procedural safeguards are inadequate and their enforcement is an overtaxing burden. Chicago has banned spray asbestos, and New York city and Philadelphia are considering doing so.

Procedural safeguards designed to reduce fiber emissions would be required to spray non-asbestos insulation. The biological effects of these fibers is unknown. Prudence would seem to dictate reasonable efforts to soften the potential future blow from spray fiber aspiring to asbestos' status.

Part IV suggests that various asbestos-related activities at construction sites be done within an enclosure. It would also require that asbestos construction products be so installed as to preclude the emission of fiber into the circulating air.

Part V expresses, ironically, what may be a key reason for banning spray asbestos. It would require that procedural safeguards be pursued in order to demolish a structure whose destruction would otherwise expose members of the public to asbestos fiber. Clouds of dust are often raised by demolition work. The facts that great numbers of persons may be exposed to this dust in urban areas and that the post-war generation of high-rises were the first buildings to use asbestos insulation extensively speak in favor of some such regulation.

Part VI suggests an emission standard for manufacturers of asbestos products. The standard is believed to be within the capture efficiency of baghouse filtration. The proposed boundary standard is thought to be a method for preventing dilution of exhaust gases.

The standard would measure only fibers longer than five microns. The justification for this seems to be the administrative convenience of avoiding difficulties in measuring fibers from .5 to five microns in length. Medical evidence has indicated that the very small fibers may be the most dangerous. And the Board will seek extensive evidence on this point.

Part VII would prohibit the use of brake lining in vehicles manufactured after January 1, 1975 and sold for use in Illinois.

If brake lining decomposition emits asbestos fiber, then the tremendous number of vehicles using asbestos, and the impracticality of controlling these numerous small emission sources would appear to speak to the need for a product ban. The prohibition is carefully worded to avoid the necessity of fitting vehicles produced prior to 1975 with non-asbestos brakes. It is contemplated that the ban will necessitate design changes, probably to disc brakes, and it is thought that requiring the individual vehicle owner to undergo this expense is unreasonable. The time lag of over three years is thought to be sufficient for vehicle manufacturers to accomplish any necessary design changes.

Proposed Regulation Only
June 9, 1971
CHAPTER V: TOXIC SUBSTANCES
ASBESTOS; SPRAY INSULATION

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PART I: INTRODUCTION

101. Authority

Pursuant to the authority in Sections 9, 10 and 13 of the Environmental Protection Act which empower the Board to adopt regulations forbidding the "sale, offer, or use for reasons of air pollution control" of any article, and to set "standards specifying the maximum amounts or concentrations of various contaminants that may be discharged into the atmosphere"; and to set standards for the issuance of permits for the operation of any equipment or facility capable of causing or contributing to air pollution; and to promulgate "conditions regarding the... use of any....article determined by the Board to constitute an air pollution hazard"; and to adopt effluent standards limiting the amounts of contaminants that may be discharged into the water of Illinois, the Board adopts the following rules and regulations:

102. Policy

It is the purpose of the General Assembly in adopting the Environmental Protection Act to maintain and enhance the purity of the air and water of Illinois in order to protect health, welfare and the quality of life. Accordingly, it is hereby determined that the uncontrolled discharge of asbestos fiber into the environment tends to severely endanger the public health and welfare and that the uncontrolled spraying of fiber-containing materials unreasonably interferes with the enjoyment of life and property.

It is the purpose of these regulations to control the amount of asbestos fiber released into the environment from the major sources of emission. Such control is necessary not only to protect those members of the public who are in proximity to heavy concentrations of asbestos fiber but also to safeguard the health of future generations endangered by the continuous discharge of asbestos fiber which can be toxic and which tends to be cumulative both in the atmosphere and in the human body.

Where health can be protected by the adoption of an emission standard or of procedural safeguards, such a course has been pursued. In those instances where restriction is unfeasible because of the tremendous number of small, uncontrollable emission sources (automotive) or because of the unusual nature of the emission source (spray asbestos at construction sites) a product ban has been resorted to. These prohibitions have been made with full consideration given to the available alternative materials.

It is also the purpose of these regulations to reduce the emission of non-asbestos particulate from spray-fireproofing and

insulation. To protect against these emissions, the biological effects of which are unknown, procedural safeguards have been enacted.

103. Definitions

- Agency - The Illinois Environmental Protection Agency
- Asbestos - Any fiber or any mixture containing fiber of hydrated silicate mineral, which, on the basis of its crystalline structure, falls into one of two categories:
- (1) pyroxenes - chrysotile fiber;
 - (2) amphiboles - crocidolite, amosite, tremolite, actinolite or anthophyllite fiber.
- Board - The Pollution Control Board of Illinois.
- Commercial Activity - Any activity done for hire or having financial profit as a primary aim.
- Debris - Asbestos-containing waste produced by the demolition of a structure.
- Spraying - The pneumatic application of material used for fireproofing or insulation.
- Waste - Any asbestos-containing matter which has been or is intended to be discarded.

PART II: General Requirements

No commercial activity, not otherwise hereinafter prohibited, involving the discharge of asbestos fiber into the environment from the construction, alteration, repair or demolition of a structure or from the processing or manufacturing of asbestos-containing products, shall be conducted unless the person or entity in charge of such activity complies with the following regulations:

201. A permit shall be obtained from the Agency to engage in such activity. Before obtaining such permit, the applicant shall demonstrate compliance with the following safeguards:

(a) The designation of personnel to exercise full-time supervisory authority over all aspects of the activity from which the release of asbestos fiber into the environment could result, in such a manner as to insure compliance

with the pertinent asbestos control regulations.

(b) That each employee engaged in such activity has completed or will complete a satisfactory course of instruction on the potential hazards of exposure to asbestos fiber including the precautions that must be observed to prevent or restrict the dispersion of asbestos fiber into the environment.

(c) That such additional safeguards as are hereinafter specifically required and such precautions consistent with the purpose of these regulations as the Agency deems necessary under the circumstances shall be complied with.

202. Facilities shall be provided and procedures instituted and supervised that prevent the removal of asbestos-containing material from the site on the clothing of employees.

203. Asbestos-containing wastes shall be immediately vacuumed or otherwise collected where vacuuming is impossible, and shall be placed in a container resistant to tearing or breaking under normal handling conditions, which shall be tightly sealed and clearly marked as containing asbestos waste. Such containers shall be placed directly upon a vehicle for disposal by burial at an approved site.

Section 203 shall not apply to the demolition of a structure, except as provided in Section 501(a), (d) and (e) or to the disposal of sludge waste except as provided in Section 603.

PART III: SPRAY FIREPROOFING AND INSULATION

301. The spraying of asbestos-containing material is prohibited.

302. Non-asbestos particulate matter used for insulation or fireproofing shall not be sprayed unless the following procedures are taken:

(a) Before the start of spraying operations, all floor areas shall be swept clean and cleared of all objects, material and equipment other than that employed in the application of the fiber-containing material, or, in the alternative, all such objects, material and equipment shall be covered with plastic or plastic-coated tarpaulins.

(b) The entire floor or area to be sprayed shall be enclosed with plastic or plastic-coated tarpaulins in a manner which shall preclude the escape of fiber-containing material from the enclosure. All interior open areas such as elevator shafts and stairwells shall be enclosed in a manner which shall prevent the escape of fiber-containing

material from the working area. "Stack effect" of the shafts, stairwells and comparable spaces shall be considered in providing proper enclosures.

(c) Regardless of adherence to the stated control practices, any visible emission of fiber-containing materials beyond the building perimeter will be considered a violation.

(d) In case of visible emission of fibrous material from the construction site, immediate steps shall be taken to cause the cessation of such emissions by either effective control measures or work stoppage at the source of the emissions. There shall then be immediate and complete cleanup of all material that has escaped the construction site, and measures shall be taken that will insure that no further dispersal of any fibrous material into the atmosphere can occur.

(e) The entire sprayed area, all ledges and surfaces including tarpaulins within the enclosure shall be thoroughly vacuumed upon completion of the spraying operation and immediately before the enclosure is dismantled. The vacuum cleaner shall contain a strong, single-service, disposable inner bag of durable material which shall be removed from the vacuum cleaner and tightly sealed. The bag shall then be placed in a container and disposed of at an approved site in accordance with Section 203.

(f) All areas used for opening bags containing fibrous material or the changing of hoppers shall be enclosed so as to prevent the escape of such material from the immediate area in which such activity occurs.

(g) Any plenum or other structure coated with or containing fibrous insulation and used in the circulation of air in a building shall be thoroughly cleaned of all debris and waste insulation. All applied fiber-containing insulation within a plenum or duct shall be coated with a sealant which precludes exposure of the fiber-containing material to the circulating air.

PART IV: CONSTRUCTION, ALTERATION AND REPAIR OF STRUCTURE

401. (a) The cutting, trimming, fitting or stripping of asbestos-containing material in the construction, alteration or repair of a structure which is done at the site of such structure shall be conducted within a special enclosure designed to preclude the escape of asbestos fiber from the immediate area of such enclosure.

(b) The mechanical exhaustion of dust from such enclosure to the ambient air is prohibited unless such exhaust system is equipped with a fabric filter for dust collection.

402. Asbestos-containing material applied in the construction, alteration or repair of a structure shall be coated with a sealant, provided with a cover or installed in some other manner so as to preclude exposure of the asbestos fiber to the circulating air.

PART V: DEMOLITION

501. Where the risk of public exposure to asbestos fiber from the dislodging of asbestos-containing materials is present, no demolition of a structure shall be initiated unless all safeguards necessary and practicable to reduce the emission of dust are taken.

Such procedures shall include, but are not necessarily limited to:

(a) Boilers and pipes insulated with asbestos-containing material shall be wetted and stripped before toppling of walls is begun. This procedure shall be followed, where practicable, as to all other asbestos-lined surfaces. Such asbestos waste shall be immediately bagged and disposed of in accordance with Section 203.

(b) Demolition by toppling of walls is prohibited except within an enclosure approved by the Agency as part of the permit granted for demolition of that structure.

(c) Before the demolition of any section of the structure, adequate wetting to suppress the dust shall be employed.

(d) Asbestos-containing debris shall not be dropped or thrown from any floor but shall be transported by dust-tight chutes or buckets. Asbestos-containing debris in chutes or buckets shall be sufficiently wetted to preclude dust dispersion at the point of discharge.

(e) All asbestos-containing debris shall be thoroughly wetted before loading into trucks, other vehicles or containers. During transport such waste shall be enclosed or covered so as to prevent dust dispersion. Asbestos-containing debris shall be disposed by burial at an approved site.

502. In the event asbestos-containing matter becomes air-borne beyond the property boundaries or in the event freezing temperatures preclude the use of water for dust suppression, demolition work shall cease until adequate alternate measures can be taken.

PART VI: MANUFACTURING

601. (a) No factory, plant or enterprise which engages in the processing or manufacturing of any asbestos-containing product shall emit asbestos fiber into the ambient air in excess of .5 fiber per cubic centimeter of air. The ambient

air quality at the boundary of such factory, plant or enterprise shall not exceed .05 fiber per cubic centimeter of air.

(b) Only fibers of 5 microns or greater in length with a length to breadth ratio of 3 to 1 shall be counted.

(c) Sampling shall be determined by the membrane filter method with phase contrast microscopy at 430 X magnification.

602. No water used in processing or manufacturing which contains concentrations of asbestos fiber shall be discharged into sewage systems or to the waters of Illinois.
603. Waste sludge containing asbestos collected from settling ponds shall be enclosed during transport and shall be disposed by burial at an approved site.'

PART VII: ASBESTOS-CONTAINING PRODUCTS

701. No product which may emit asbestos-fiber during its transportation shall be transported unless such product is enclosed in such a manner as to preclude the emission of asbestos fiber into the ambient air.
702. The use of asbestos in the brake lining of vehicles manufactured after January 1, 1975, and sold for use in Illinois is prohibited.

Annexed Notice

ILLINOIS POLLUTION CONTROL BOARD
NOTICE OF HEARING

Please take notice that the Pollution Control Board of the State of Illinois will hold public hearings with regard to Asbestos Regulations, No. R77-16, on the following dates:

9:30 A.M., October 6, 1971, Granite City Courthouse, Granite City, Illinois
10:00 A.M., October 13, 1971, Lecture Room, Field Museum, Chicago, Illinois
10:00 A.M., October 19, 1971, Public Library, Waukegan, Illinois
David P. Currie,
Chairman
No. 1743 — Sept. 18

W

Publisher's Certificate

THIS IS TO CERTIFY that the notice, a true copy of which is hereto annexed, was published in the Waukegan News-Sun a secular newspaper of general circulation in the County of Lake and State of Illinois, published daily except Sunday in the City of Waukegan, Lake County and State of Illinois by the Keystone Printing Service, Inc., a corporation organized and existing under the laws of the State of Illinois, _____ times, once each week, for _____ weeks successively; that the date of the first publication was on the _____ day of _____

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IN WITNESS WHEREOF said Keystone Printing Service, Inc., publisher aforesaid, has hereto caused its corporate name to be hereto affixed on this the _____ day of _____, A.D. 19____, by its duly authorized officer or agent pursuant to a resolution heretofore adopted at a regular meeting of the Board of Directors of said Keystone Printing Service, Inc., as follows:

RESOLVED, that a certificate of publication of legal notices may be signed on behalf of this corporation and in its corporate name with or without its corporate seal by any or either of the following officers or agents: President, Vice-president, Secretary, Treasurer, or Cashier.

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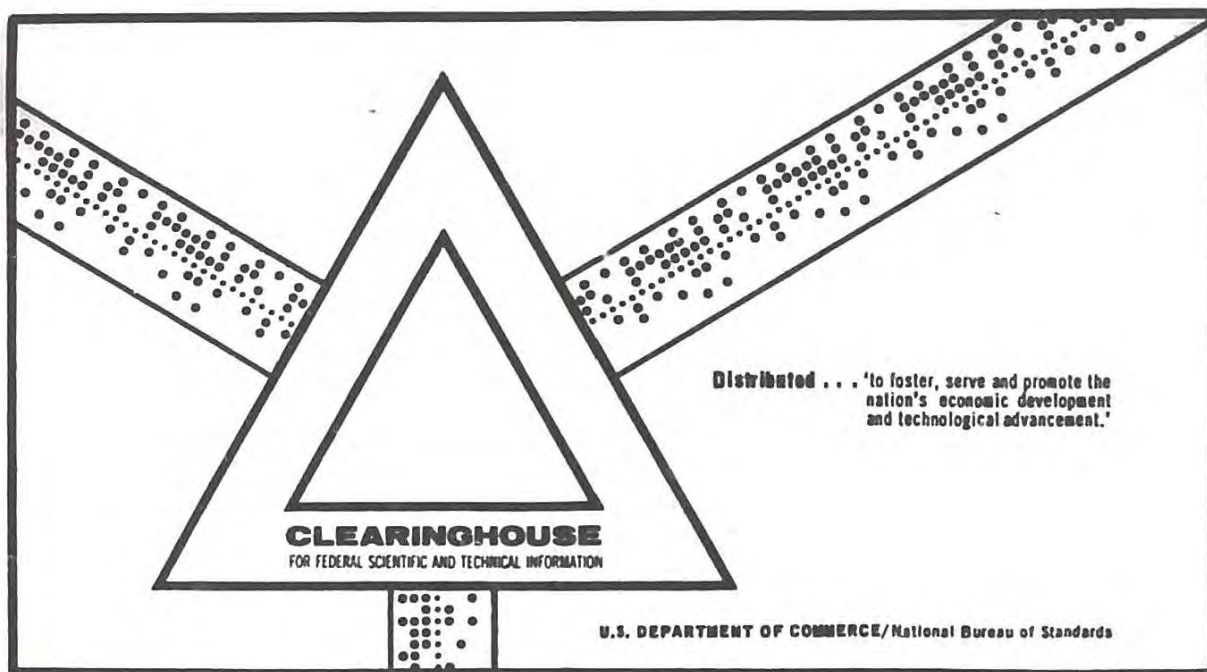
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AIR POLLUTION ASPECTS OF ASBESTOS

Ralph J. Sullivan, et al.

Litton System , Incorporated
Bethesda, Maryland

September 1969



This document has been approved for public release and sale.

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FOREWORD

As the concern for air quality grows, so does the concern over the less ubiquitous but potentially harmful contaminants that are in our atmosphere. Thirty such pollutants have been identified, and available information has been summarized in a series of reports describing their sources, distribution, effects, and control technology for their abatement.

A total of 27 reports have been prepared covering the 30 pollutants. These reports were developed under contract for the National Air Pollution Control Administration (NAPCA) by Litton Systems, Inc. The complete listing is as follows:

Aeroallergens (pollens)	Ethylene
Aldehydes (includes acrolein and formaldehyde)	Hydrochloric Acid
Ammonia	Hydrogen Sulfide
Arsenic and Its Compounds	Iron and Its Compounds
Asbestos	Manganese and Its Compounds
Barium and Its Compounds	Mercury and Its Compounds
Beryllium and Its Compounds	Nickel and Its Compounds
Biological Aerosols (microorganisms)	Odorless Compounds
Boron and Its Compounds	Organic Carcinogens
Cadmium and Its Compounds	Pesticides
Chlorine Gas	Phosphorus and Its Compounds
Chromium and Its Compounds (includes chromic acid)	Radioactive Substances
	Selenium and Its Compounds
	Vanadium and Its Compounds
	Zinc and Its Compounds

These reports represent current state-of-the-art literature reviews supplemented by discussions with selected knowledgeable individuals both within and outside the Federal Government. They do not however presume to be a synthesis of available information but rather a summary without an attempt to interpret or reconcile conflicting data. The reports are

necessarily limited in their discussion of health effects for some pollutants to descriptions of occupational health exposures and animal laboratory studies since only a few epidemiologic studies were available.

Initially these reports were generally intended as internal documents within NAPCA to provide a basis for sound decision-making on program guidance for future research activities and to allow ranking of future activities relating to the development of criteria and control technology documents. However, it is apparent that these reports may also be of significant value to many others in air pollution control, such as State or local air pollution control officials, as a library of information on which to base informed decisions on pollutants to be controlled in their geographic areas. Additionally, these reports may stimulate scientific investigators to pursue research in needed areas. They also provide for the interested citizen readily available information about a given pollutant. Therefore, they are being given wide distribution with the assumption that they will be used with full knowledge of their value and limitations.

This series of reports was compiled and prepared by the Litton personnel listed below:

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Douglas A. Olsen, Ph.D.
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The NAPCA project officer for the contract was Ronald C. Campbell, assisted by Dr. Emanuel Landau and Gerald Chapman.

Appreciation is expressed to the many individuals both outside and within NAPCA who provided information and reviewed draft copies of these reports. Appreciation is also expressed to the NAPCA Office of Technical Information and Publications for their support in providing a significant portion of the technical literature.

ABSTRACT

Inhalation of asbestos may cause asbestosis, pleural or peritoneal mesothelioma, or lung cancer. Mesothelioma is a rare form of cancer which occurs frequently in asbestos workers. All three of these diseases are fatal once they become established. The dose necessary to produce asbestosis has been estimated to be 50 to 60 million particles per cubic foot-years. No information is available on the dose necessary to induce cancer. Random autopsies of lungs have shown "asbestos bodies" in the lungs of one-fourth to one-half of samples from urban populations. Thus, the apparent air pollution by asbestos reaches a large number of people.

Animals have been shown to develop asbestosis and cancer after exposure to asbestos.

No information has been found on the effects of asbestos air pollution on plants or materials.

The likely sources of asbestos air pollution are uses of the asbestos products in the construction industry and asbestos mines and factories. Observations in Finland and Russia indicate that asbestos does pollute air near mines and factories. However, no measurements were reported of the concentration of asbestos near likely sources in the United States. A concentration in urban air of 600 to 6,000 particles per cubic meter has been estimated.

Bag filters have been used in factories to control

asbestos emissions: the cost of this type of control in a British factory was approximately 27.5 percent of the total capital cost and about 7 percent of the operating cost. No information has been found on the costs of damage resulting from asbestos air pollution.

No satisfactory analytical method is available to determine asbestos in the atmosphere.

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1. INTRODUCTION

Asbestos is a general name given to a variety of useful fibrous minerals. The value of asbestos ensues from the indestructible nature of products fabricated from the various grades of mineral fibers. The major asbestos minerals are chrysotile, crocidolite, amosite, and anthophyllite, while tremolite and actinolite are considerably less important. Over 90 percent of the asbestos is chrysotile. The United States uses about one-fourth of the world production of this substance, practically all imported from Canada and Africa.

Inhalation of asbestos dust has long been recognized as an industrial hazard.^{161,165} Early in this century, exposure to high concentrations of the fibrous dust was causally associated with asbestosis. In 1935, evidence began to accumulate that cancer of the lung is also associated with inhalation of asbestos.⁵⁵ More recently, certain rare cancers, pleural mesotheliomas and peritoneal mesotheliomas, have been associated with inhalation of asbestos fibers by asbestos workers.¹⁷⁶ Helmann⁹² states that "The finding of several such rare tumors in any given group makes that group suspect of having special and distinct environmental characteristics, in this case, exposure to asbestos dust."

Nonoccupational environmental exposure to asbestos was found as early as 1927 when Haddow⁸¹ reported finding so-called "asbestos bodies" in the lungs of a person living near

an asbestos factory. Since then, several investigators^{11,175,186,202,226} have reported finding neighborhood cases. The subject of nonoccupational environmental exposure to asbestos assumed a new dimension beginning with the reports of Thomson and his colleagues.²¹³⁻²¹⁶ After examining the lungs in consecutive autopsies, they found that approximately one-fourth of the populations in both Capetown, South Africa, and Miami, Fla., have "asbestos bodies" in their lungs. Other investigators have confirmed that one-fourth to one-half of the population in Pittsburgh,⁴² San Francisco,⁴³ Milan,⁶⁷ Glasgow,¹⁸⁸ New York,¹⁹⁵ Montreal,⁷ Jerusalem,¹⁸⁴ Finland,¹⁶⁷ and Sweden⁸² have "asbestos bodies" in their lungs. These findings indicate that either these asbestos particles or other particles that resemble asbestos in many ways, including the way in which the body reacts to them, are being inhaled either with the ambient urban air or through direct exposure to asbestos.

2. EFFECTS

2.1 Effects on Humans

Asbestosis (a diffuse pulmonary fibrosis), pleural calcification, pleural plaques, lung cancer, and pleural and peritoneal mesotheliomas can result from exposure to asbestos. Asbestos bodies are commonly found in the lungs of persons exhibiting these complications. Diagnosis of any of these or finding "asbestos bodies" in the lungs signifies the need to review the case history for previous asbestos exposure. Surveys of people living or working near asbestos mines and factories have revealed that many nonoccupational cases of asbestosis and mesothelioma have occurred either from the polluted air or from asbestos carried home on the workers' clothing.^{175,176,178} However, in many cases no exposure to asbestos can be established.

The fate of the asbestos fiber once it is inhaled* and deposited in the lung is still questionable. The short fibers, <0.5 μ in length, have been pathologically ignored, probably because they are much too narrow to be visible under a light microscope. The longer fibers which are encrusted in an iron-bearing protein (asbestos bodies) become easily visible. Wagner and Skidmore²²⁸ and Morris *et al.*¹⁷² have shown that rats which have inhaled asbestos lose the asbestos (probably the short fibers) from their lungs. The biological half-life

*A discussion on respirable fibers is presented in Appendix B (page 92).

for asbestos appears to be 20 to 90 days, depending on the mineral type. Some of the fibers are removed by phagocytosis to the lymph nodes.

2.1.1 Asbestosis

Among asbestos workers, evidence of pulmonary asbestosis is common. This condition results in a diffuse fibrosis, usually in the lower lobes of the lung. Pulmonary asbestosis has been called a monosymptomatic disease, with dyspnea as the main complaint.¹⁹⁷ The British Occupational Hygiene Society¹³⁶ has reported that basal rales are the first symptoms of asbestosis.

Asbestosis usually develops after long exposure to high concentrations of asbestos dust. The risk varies directly with the length of exposure and the dust concentration. Following continued exposure to high concentrations of dust, asbestosis may develop fully in 7 to 9 years and may cause death as early as 13 years from onset of exposure. The common exposure period before recognition of asbestosis (as observed among asbestos workers) is 20 to 40 years, with death following about 2 to 10 years later. Once established, asbestosis progresses even after the exposure to dust ceases; illness or death can occur long after exposure to concentrations not producing immediate effects.¹⁹⁷

The prolonged latency period between exposure and the first signs of asbestosis makes it difficult to establish dose-time relationships. Cooper⁴³ suggested that a time-weighted

average concentration of asbestos fibers of 5 mppcf* is too high. He cites Wells²³⁴ idea that multiplying average counts by years of exposure provides a rough guide to the total dose allowable. After 50 to 60-mppcf-years, workers began to show evidence of asbestosis. At an average concentration of 5 mppcf, this total allowable dose would be reached in 10 to 12 years. Unfortunately, dust concentrations have been infrequently reported and measurements have been hampered by the varied nature of the sources. Marr¹⁵³ and Selikoff *et al.* have reported that insulation workers are exposed to dust concentrations below 5 mppcf, yet have exhibited a high prevalence of asbestosis. Thus, Cooper's argument is strengthened.

In 1946, there were about 700 cases of asbestosis in Germany among a total of approximately 8,000 employees in the asbestos industry.¹⁰⁸ Megelius²³⁵ found 125 cases of asbestosis of the lung in X-ray examinations of 476 asbestos workers in one company in Finland. Of 132 asbestos workers examined by Bohme,²⁶ 29 percent showed X-ray evidence of asbestosis. The occurrence of asbestosis in members of this worker group rose with the duration of the employment: 5 percent in workers exposed to asbestos for less than 3 years; 56 percent for those employed for 5 to 10 years, and 79 percent for those with over 10 years' exposure. A similar morbidity of 80 percent among

*5 million particles per cubic foot based on total dust count and 8-hour-day, 40-hour-week exposure.

English asbestos workers with over 20 years of employment was reported by Marewether and Price¹⁶⁵ in 1930. Moro¹⁷⁷ noted that the incidence of asbestosis was 65 percent in 167 asbestos workers studied by X-ray. Selikoff *et al.*¹⁹⁷ investigated 1,522 asbestos insulation workers in the New York-New Jersey metropolitan area. Among 392 individuals examined more than 20 years from the onset of exposure, X-ray evidence of asbestosis was found in 339. In half of these, the asbestosis was moderate or extensive. In individuals with less than 20 years of exposure, radiological evidence of asbestosis was less frequent and when present, was much less likely to be extensive.

2.1.2 Pleural Calcification and Plaques

Pleural calcification resulting from exposure to asbestos is usually bilateral and affects the parietal pleura.¹²⁰ Selikoff¹⁹³ stated that bilateral pleural calcification involving the diaphragm is diagnostic of asbestosis. This pleural calcification can be readily identified by X-ray.¹¹⁷

Kiviluoto¹²⁰ discovered 499 cases of pleural calcification during a community X-ray survey of 6,312 adults in the Kuusjari commune in Finland. In Ilomantsi commune he found no such calcification among 7,101 adults. He observed that the Kuusjari commune contained an asbestos mine and suggested that these people had been subject to a localized environmental asbestos exposure. This investigator¹²¹ also observed 77 cases of pleural plaques out of 35,000 routine chest X-rays. The case histories of these 77 revealed that 52 had previous

exposure to asbestos either in the home or in their occupations, where they handled asbestos products. Of the other 25 persons, 16 were questioned and no previous asbestos exposure could be ascertained. Raunio¹⁸⁶ continued the study and found 1,516 adult cases of pleural calcification from 633,201 X-rays taken in 13 Finnish towns and 106 rural communes. In Tuusniemi commune, where an asbestos quarry is located, pleural calcification was found in 9 percent of the population; in urban populations it was found in 0.7 percent of the people; and in rural areas calcification was found in only 7 out of 265,273 people examined (0.002 percent). However, Meurman¹⁶⁷ found the pleural plaques were common (39.3 percent of his cases) in all Finland.

Approximately 2.8 percent of the agricultural workers in Czechoslovakia¹⁰⁵ and Bulgaria²³⁸ have also been shown to have appreciable pleural calcification. In Bulgaria the soils worked by the farmers contained asbestos; even stone fences were made of outcrops of anthophyllite mineral. However, in Czechoslovakia no asbestos or known exposure to asbestos was found. After examining children living in the city of Asbest in the Soviet Union, Bobyleva *et al.*^{24,25} concluded that they were suffering from impaired health caused by air pollution from asbestos plants.

2.1.3 Cancer

2.1.3.1 Cancer of the Lung

The most common complication of asbestosis is cancer of the lung. However, cancer of the lung apparently induced by

asbestos may appear unaccompanied by asbestosis.

The association of lung cancer with exposure to asbestos dust has been the subject of many investigations in the second quarter of this century. In 1935, Lynch and Smith¹⁴⁹ in the United States described lung cancer found during autopsy of a patient with asbestosis. According to Homburger's data,¹⁰¹ over a 20-year period eight cases of asbestosis were found in 4,137 autopsies at the medical school of Yale University. Of these eight asbestosis cases, four were associated with lung cancer (50 percent). In contrast, lung cancer was found in only two (12 percent) of 17 cases of silicosis.

Lynch and Cannon¹⁴⁷ in 1949 found lung cancer in only three cases (7.5 percent) of 40 patients with asbestosis. Gloyne⁷¹ in 1951 reported that according to autopsy data for the London Hospital of Chest Diseases, lung cancer was observed in 14 percent of the 121 patients with asbestosis, but in only 6.9 percent of those with silicosis.

Usually, the lung cancer incidence in men is four to six times higher than in women, but among patients with asbestosis it is only twice as high for men. This has been confirmed by Bohlig, Jacob, and Kallias.²⁹ They note that among women working in the asbestos industry, lung cancer is observed at an earlier age than among the rest of the population. In his review, Behrens¹⁹ reported 44 cases of lung cancer (14 percent) in 309 autopsies of patients with asbestosis. After examining

the data of various authors, Isselbacher *et al.*¹¹¹ reported that of 603 persons with asbestosis, lung cancer was found in 83 (13.6 percent) at autopsy. Hueper¹⁰⁸ and Doll⁵⁵ record an even higher percentage (15 percent). Doll emphasizes that the majority of those who died had worked under conditions of high dust content in the air.

Braun and Truan³³ found lung cancer in 12 (three cases were not conclusive) of 187 workers in the Canadian asbestos industry who had died. Boehme²⁶ reported 74 patients with asbestosis; lung cancer was found in six of these. The average age of the patients was 53; the average period from the beginning of work to death was 28 years. Keal¹¹⁴ points out that of 30 patients with asbestosis, 14 (47 percent) died of lung cancer and 10 from carcinoma of the peritoneum or ovaries.

Some additional statistics on the association of lung cancer with asbestos workers are listed in Table 5 in the Appendix.

Cancer of the lung produced by asbestos needs further study. The latent period between exposure and evidence of carcinoma may be even longer than for asbestosis. Nothing is known about the dose-time relationship. Cases of lung cancer have been observed when only a very short exposure or no exposure to asbestos was known. Furthermore, the low number of "asbestos bodies" observed in one-fourth to one-half of the urban population may be sufficient to cause cancer. Because

the long "asbestos bodies" remain in the lungs, a person who has inhaled asbestos may carry the potential (for the rest of his life) to develop carcinoma of the lung. Moreover, it has not been determined whether more than one fiber is necessary to induce a malignant tumor. Cox⁴² has suggested that the probability of cancer induction is proportional to the number of asbestos fibers, number of susceptible cells, the concentration of carcinogens on the fibers, and the time from exposure.

Why asbestos is carcinogenic is not clearly understood. At least three hypotheses have been advanced:

- (1) That the fibers act as a physical irritant which after 20 to 30 years of constant irritation induces a tumor.
- (2) That the fibers contain small amounts of carcinogens--such as benzo(a)pyrene, nickel, and chromium--which are eluted from the fibers by the serum in the lungs. These carcinogens then produce the cancer. Harington and Roe⁸⁸ have shown that (a) chrysotile contains little or no benzo(a)pyrene, but about 100 μ g of chromium per g of fiber and 5,000 μ g of nickel per g of fiber; (b) crocidolite contains 0.2 to 24 μ g of benzo(a)pyrene per 100 g of fiber and negligible amounts of nickel and chromium and (c) amosite contains 0.2 to 2.4 μ g of benzo(a)pyrene per 100 g of fiber, 100 μ g of nickel per g of fiber and 15 μ g of chromium per g of fiber (see Appendix B, page 92). The authors have not only shown that these agents can be dissolved in the lung serum, but also that airborne fibers can adsorb

carcinogens present in the air. In studies of the adsorption of benzo(a)pyrene by asbestos, chrysotile adsorbed 100 percent of benzo(a)pyrene from solution after 48 hours at 37°C, compared with 40 percent for crocidolite and 10 percent for amosite. Harington and Roe suggest that these carcinogens can be adsorbed on airborne fibers. Thus, the fibers become a transporting vehicle to carry a concentrated quantity of carcinogens into the lung.

- (3) That the fibers accumulate in the lung and are immobilized as "asbestos bodies" which disintegrate after 20 to 40 years. The resulting free particles cause asbestosis or carcinoma of the lung.

In addition, Hammond⁸³ has suggested that asbestos is a cocarcinogen; i.e., it increases the cancer-producing potential of small amounts of some other agent. In a study of asbestos workers, he and his colleagues¹⁹⁹ found cancer of the lung only in cigarette smokers. The number of deaths attributed to cancer of the lung was eight times higher in asbestos workers who smoked cigarettes than in smokers who were not exposed to asbestos (see Section 2.1.3.4).

2.1.3.2 Mesothelioma of the Pleura and Peritoneum

Primary tumors of the pleura and peritoneum are so rare that for years they were considered to be pathologic curiosities. In 1960 the first large series of cases of diffuse mesothelioma were reported by Wagner *et al.*²²⁹ in South Africa.

In trying to explain this mysterious epidemic, Wagner,²²⁶ noting that "asbestos bodies" were found in the lungs of some of their patients, obtained detailed life histories of these patients. By 1960, he was able to establish an association with exposure to the Cape of Good Hope asbestos fields, or the industrial use of asbestos, in 32 of 33 patients with histologically proved pleural mesothelioma. The majority of these patients had not actually worked with asbestos but had lived in the vicinity of the mines and mills, and some had left these areas of exposure as young children. The average period between exposure and development of the tumor was 20 to 40 years. By 1962, Wagner²²⁶ had diagnosed a total of 87 pleural and two peritoneal mesotheliomas. In only two cases was it impossible to establish a history of exposure to asbestos dust. Of these 87 cases, 12 had been industrially exposed and the remainder had been environmentally exposed from living in the vicinity of the mills and dumps. This association between mesothelioma and asbestosis became even more intriguing when in 1955, Bonser *et al.*³⁰ described a series of 72 autopsies on patients with asbestosis in which four cases of peritoneal mesothelioma were found. Subsequently, Mancuso and Coulter¹⁵² found five peritoneal mesotheliomas in 1,495 asbestos workers, and Hourihane,¹⁰² upon reviewing the necropsy files of the London Hospital from 1917 to 1962, found 34 cases of mesothelioma, half of the pleura and the other half of the peritoneum. All

if these patients had pulmonary asbestosis, even though in a few there was no history of exposure to the asbestos dust. In a subsequent study¹⁰³ Hourihane found 74 cases of mesothelioma in a London hospital over a 10-year period.

Borow *et al.*³¹ report that in two years they observed 11 cases of mesothelioma during surgery in New Jersey. These cases, added to six others previously diagnosed by them, totaled 17 cases of mesothelioma, eight of which were peritoneal and nine pleural. They suggest that the high prevalence of these rare tumors in New Jersey can be explained by its close proximity to a major asbestos mill, where a large percentage of all the asbestos fiber mined in North America is converted to commercial use.

In an attempt to determine whether mesothelioma of the serosal surfaces was related in any way to asbestos exposure in the United States, Selikoff *et al.*¹⁹⁸ studied 307 consecutive deaths among asbestos insulation workers in the Northeastern United States. They found 10 deaths caused by four pleural and six peritoneal mesotheliomas. In addition, the workers had a high death rate attributed to cancer of the stomach, colon, and rectum. Of the 307 deaths, 40.4 percent were attributed to cancer, 5.5 percent to asbestosis, and 54.1 percent to other causes. In a second study, the investigators reviewed 26 consecutive autopsies of patients with asbestosis, and found four mesotheliomas of the pleura and three of the peritoneum.

A series of 83 patients from the London Hospital with a diagnosis of mesothelioma (confirmed by necropsy or biopsy) were studied by Newhouse and Thompson¹⁷⁶ for possible exposure to asbestos. The series consisted of 41 men and 42 women; 27 of the patients had peritoneal and 56 pleural tumors. Although the earliest death recorded from this group was in 1917, only 10 patients died before 1950, while 40 (48 percent) died between 1960 and 1964. In 76 of the 83 cases, full occupational and residential histories were obtained. Forty patients (52.6 percent) had a history of occupational exposure to asbestos or of domestic exposure (living in the house with an asbestos worker). In comparison, only 11.8 percent (9 of 76) of the patients from the same hospital suffering from other diseases had previous exposure to asbestos. There was also evidence that neighborhood exposures may be important. Among those in this study with no history of occupational or domestic exposures to asbestos, 30.6 percent of the mesothelioma patients and 7.6 percent of the inpatients with other diseases lived within half a mile of an asbestos factory. Of the 31 patients with occupational exposures to asbestos, only 10 held jobs scheduled under the British Asbestos Regulations of 1931. The interval between first exposure and the development of the terminal illness from mesothelioma ranged between 16 and 55 years. The duration of exposure varied widely, ranging from two months to over 50 years. In 47 patients in this mesothelioma

series, lung tissue or sputum was available for examination. In 30 (62.5 percent), either asbestosis or the presence of asbestos bodies was noted.

Mesothelioma is now considered a frequent cause of death among asbestos workers. No attempt has been made to summarize the reports of mesothelioma, since they appear almost weekly in the current literature. So far, however, there appear to be few cases among the general population. Selikoff¹⁹⁸ reviewed 31,652 deaths among the general population of over 1,048,183 in the United States and found only three cases of mesothelioma. Moreover, he¹⁹⁵ points out that asbestos is not the only cause of mesothelioma; it has also been produced by silica²²⁴ and polyurethane.¹⁰⁷

2.1.3.3 Other Cancers

Extrapulmonary cancer has also been noted as a cause of death among asbestos workers. Kogan *et al.*¹³² in 1966 reported 14 cases (31.1 percent), 11 women and 3 men. Four of the women died of uterine cancer, two of intestinal cancer, two of breast cancer, and one of liver cancer. Among the men, one died of stomach cancer, another of cancer of the urinary bladder, and a third of cancer of the prostate. Other cases of extrapulmonary cancer have been cited in Section 2.1.3.2.

2.1.3.4 Synergism

While the exact cause of lung cancer or pleural peritoneal mesothelioma induced by asbestos is not known, air pollution by

other pollutants may accelerate the morbidity. One form of air pollution which is easily studied in individuals is smoking. Selikoff *et al.*¹⁹⁹ recently studied the mortality of 370 asbestos insulation workers. In this group 24 men died of lung cancer and all had a history of smoking. (See Table 1 below.) This rate was eight times greater than the expected mortality rate, with age and smoking habits taken into account.

TABLE 1
DEATHS OF ASBESTOS INSULATION
WORKERS IN NEW YORK, 1963-67¹⁹⁹
(By Smoking Habits)

Smoking Habits	No. of Cases ^a	Observed Deaths	Expected Deaths
Never smoked regularly	48	0	0.05
History of pipe, cigarette smoking only	39	0	0.13
History of regular cigarette smoking ^b	283	24	2.98
Total	370	24	3.16

^aAll with more than 20 years from onset of exposure.

^bIncludes cigarette smokers who also smoked pipes or cigars.

The blue asbestos, crocidolite, from South Africa is believed by many^{53,79,136,140,209} to be much more carcinogenic than the other minerals of asbestos. Studies in Finland¹⁶⁷ indicate that anthophyllite also produces cancer. Wagner,²²⁶

Smith *et al.*,²⁰⁶ and recently Godwin and Jagatic,⁷³ reported that they had induced mesothelioma in mice using chrysotile. Moreover, animal experiments^{76,78,98-100,113,172,225,228} have demonstrated that pulmonary complications occur with crocidolite, chrysotile, amosite, and anthophyllite. Some investigators believe that the fiber is not the carcinogenic agent, but rather the vehicle on which the carcinogens are carried to the target tissue. As stated earlier, chrysotile contains the most nickel and chromium of all asbestos minerals, while crocidolite contains the most benzo(a)pyrene.^{87-89,159} All three of these impurities in asbestos are suspected of being carcinogenic. This knowledge, together with results which indicate carcinogens can be adsorbed from urban air or tobacco smoke, indicate that there may be no necessity to distinguish urban asbestos dust by mineral types.

2.1.4 "Asbestos Bodies"

As stated earlier, the recent finding of "asbestos bodies" in one-fourth to one-half of the urban population (see Table 6 in the Appendix) has added new impetus to the examination of asbestos as a general air pollutant.

An "asbestos body" has been defined as "an elongated golden or reddish-brown structure usually with clubbed ends; the shaft, which often shows a segmented or beaded appearance, is usually straight, but sometimes curvilinear with a tendency toward symmetry; usually it is from 3 to 5 μ in diameter and

20 to 100 μ in length. The coating contains iron demonstrable by Perle's stain (Prussian blue reaction), and probably composed of ferritin or ferritin-like material; it may cover the structure completely, masking the central fiber from direct view, or may be incomplete in the central portion of the shaft or in the interstices of the body, revealing an expanse of naked fiber.²²⁰

There is no doubt that the "asbestos bodies" formed in the lungs of the asbestos workers contain asbestos. Stumphiuss and Meyer²¹⁰ have investigated the composition of the "kernel" in the "asbestos bodies" removed from deceased shipyard workers (an occupational group with only indirect exposure). They found by electron microscopy and X-ray microanalyses that the "asbestos bodies" did indeed contain some minerals of asbestos. Out of 27 fibers, 17 were classified as serpentine (possibly chrysotile) and 10 as amphibole (possibly crocidolite). But what about the so-called "asbestos bodies" in the lungs of the general population? While these "asbestos bodies" probably contain some asbestos, there is no experimental evidence to date which shows what fractions contain asbestos or whether they contain any asbestos at all.^{79,194} This subject is currently being debated by several investigators.^{53,79,209} In fact, some object to the use of the term "asbestos bodies" and prefer to call them "ferruginous bodies." Moreover, Gross *et al.*⁷⁹ have shown that "ferruginous bodies"—which appear identical under the microscope to those formed from asbestos—

can be produced from ceramic aluminium silicate, glass fibers, and silicon carbide fibers. However, Thomann²¹⁵ claims that a skilled pathologist can tell the difference. This controversy should soon be resolved, since both Gross⁵³ and Selikoff¹⁹⁴ are investigating the composition of the central fiber with the electron microprobe.

Generalized contamination with fibrous material is evidenced by the figures in Table 6 in the Appendix. Two other obvious conclusions are that "asbestos bodies" are found more frequently in older people than in younger, and more frequently in men than in women.

In none of the aforementioned studies has there been any quantitative count of "asbestos bodies" in the lungs of the general public. In most studies, "asbestos bodies" found were scanty, although in some instances the bodies were numerous.²¹⁴ In most of the investigations, the method of Thomson²¹⁴ was used (smears taken from basal lobes of the lung were examined and the asbestos bodies counted). Since only about one-half-millionth of a lung is examined, the finding of only one asbestos particle may be extrapolated to mean that perhaps a half-million fibers are present in the lung.²¹⁴ In one recent study in Pittsburgh, Utidjian *et al.*²²⁰ made an attempt to quantify their results. In this study 98 percent of the 100 lungs examined contained "asbestos bodies" ("ferruginous bodies"). The results are given in Table 2. For comparison they suggested that if those cases with only one "asbestos body" were ignored, then

47 percent of the lungs examined contained two or more bodies, (20.5 percent of the women and 60.7 percent of the men). This 47 percent is in substantial agreement with the comparable 41 percent reported by Cauna⁴⁰ (see Table 6 in the Appendix) for residents of Pittsburgh.

TABLE 2
DISTRIBUTION OF "ASBESTOS (FERRUGINOUS) BODIES"
IN LUNGS IN PITTSBURGH²²⁰

Sex	No. of Cases	Mean Age	Fibers/Unit of Lung	Distribution (Percent)
Men	1	89	0	2
	21	62	1	37
	15	64	2-5	27
	19	70	>5	34
Total	56	65		100
Women	2	29	0	5
	29	57	1	66
	8	68	2-5	18
	5	64	>5	11
Total	44	60		100

2.2 Effects on Animals

2.2.1 Commercial and Domestic Animals

Kiviluoto¹²¹ reported finding some asbestos bodies in a cow near an asbestos mine. In 1931, Shuster²⁰¹ reported finding extensive pulmonary fibrosis in a dog kept for ratting in an asbestos factory. The lungs of the dog also contained some asbestos fibers but no asbestos bodies were found.

Peacock and Peacock¹⁸² have studied the effect of asbestos on white leghorn fowl. They tried dusting the birds with asbestos but found that the fibers did not penetrate far into the lung. When the fibers were injected into the lumen of the air sac, an immediate inflammatory reaction occurred, macrophages appeared and engulfed the fibers, and giant cell formation was observed. Four of the 17 chickens examined developed tumors; of the six injected with crocidolite, two developed tumors; of the 10 injected with amosite, only one developed a tumor; and one chicken injected with an unidentified variety of asbestos also developed a tumor.

2.2.2 Experimental Animals

Studies with experimental animals have shown that asbestos can induce fibrosis (asbestosis), cancer of the lung, and mesothelioma and can form "asbestos bodies."

Wagner²³⁴ described experiments with rats in which 600 animals were exposed to various minerals of asbestos. The results are given in Table 3.

Gross and De Treville⁷⁶ made the following observations in studies on rats, hamsters, and guinea pigs. In rats that have inhaled high concentrations (86,000 $\mu\text{g}/\text{m}^3$) of chrysotile asbestos fibers for only a few months, minimal fibrotic lesions can be observed in the lungs of all animals. However, this form of asbestosis in rats is nonprogressive. In hamsters that have inhaled chrysotile dust, a fibrous envelope which is

were observed along with asbestos fibers. After 226 days the lungs of animals dusted for more than 1,000 hours over a 78-day period developed a wide-spread, progressive fibrosis with only a few asbestos fibers and "asbestos bodies" present in the lung tissue. The experimenters concluded that asbestos fibers too small to be seen under the microscope will produce asbestosis.

In order to evaluate the possible distribution of asbestos within New York City, Selikoff^{194,195} is now examining the lungs of rats found in the city for asbestos fibers.

In a similar investigation carried out in South Africa, wild animals captured near an asbestos mine were examined. However, the small number of "asbestos bodies" found in them precluded any conclusions.²³⁴

2.3 Effects on Plants

No information has been found in the literature on the effects of asbestos air pollution on plants.

2.4 Effects on Materials

No information has been found in the literature on the effects of asbestos air pollution on materials.

2.5 Environmental Standards

Both the American Conference of Governmental Industrial Hygienists²¹⁸ and the American Industrial Hygiene Association¹¹⁰ have recommended an industrial threshold limit value for asbestos dust of 5,000,000 particles per cubic foot

(5 mppcf), based on total dust count and on an 8-hour day, 40-hour week. This value was recommended by Dreesen *et al.*⁵⁶ after a study of 541 employees in three textile plants using chrysotile. Only three doubtful cases of asbestosis were found in those exposed to dust concentrations of less than 5 mppcf, whereas numerous cases were found above 5 mppcf.

Recently, the British Occupational Hygiene Society¹³⁶ published its standards for chrysotile. The Society has recommended a maximum accumulated exposure of 2.8 mppcf-years (10^8 particle-years per m^3). For example, maximum doses of 0.056 mppcf-years (2×10^6 particle-years/ m^3) for 50 years, 0.112 mppcf-years (4×10^6 particle-years/ m^3) for 25 years, or 0.28 mppcf-years (10^7 particle-years/ m^3) for 10 years are recommended. They have also recommended that dustiness be designated by categories according to the following scheme:

Dust Category	Concentration Averaged Over 3 Months (Million Particles/ m^3)
Negligible	0-0.4
Low	.5-1.9
Medium	2.0-10
High	Over 10.0

Only fibers longer than 5.0 μ in length with a 3:1 length-to-breadth ratio are counted.

With these standards the risk of asbestosis may be reduced to 1 percent; that is, 1 percent of the workers exposed to a dose of 10^8 particle-years/ m^3 would contract asbestosis.

3. SOURCES

3.1 Natural Occurrence

Asbestos is a broad term embracing several fibrous minerals. The minerals are divided into two groups: (1) Pyroxenes—chrysotile; (2) Amphiboles—crocidolite, amosite, tremolite, actinolite, and anthophyllite. Properties of these minerals are listed in Table 7 of the Appendix.

Asbestos probably occurs in nearly every country in the world, but only a few of the deposits are commercially valuable. Over 90 percent of the world asbestos production is chrysotile, and Canada is the major source of this mineral for the United States.⁹³ Table 8 in the Appendix lists the world production. From these figures an estimate of known free-world deposits is possible.

It is noteworthy that some soils near asbestos mines contain considerable quantities of asbestos. In Finland, farmers working these high-asbestos-content soils have been observed to suffer from asbestosis.¹²⁰

3.1.1 Mining

The mining of asbestos in the United States has increased 180 percent in the last 10 years. (See Table 9 in the Appendix.) This mining may constitute a source of air pollution. A high percentage of the increase in domestic production has been credited to California producers, who accounted for 65 percent of the total output in 1966. Four

companies produced chrysotile asbestos fiber: Atlas Minerals Corp. and Coalinga Asbestos Co., Fresno County; Pacific Asbestos Corp., Calaveras County; and Union Carbide Corp., San Benito County. The latter company processed the crude material in a plant at King City, Monterey County, whereas the other producers operated plants near the mine sites.¹⁷¹

Amphibole asbestos was mined by Powhatan Mining Co. near Burnsville, Yancey County, N.C. Their output increased 66 percent during 1966.¹⁷¹

There are four chrysotile mines in Arizona in the Salt River Valley near Globe. Since these mines are underground, only the waste needs to be considered in connection with air pollution (other than the possible pollution from transporting the mineral). Nearly all of the output from these mines was used in the cement industry to manufacture asbestos cement and building products: 28 percent was classified as filter fiber and 2 percent as spinning grade; the rest consisted of sand and waste, floats, or other short fibers. Jacquays Mining Corp. operated the Regal and Chrysotile Mines and shipped the ore to a company mill at Globe after hand-sorting the chrysotile. Western Asbestos Manufacturing Co. operated the Phillips Mine, and the Metal Asbestos Corp. the Lucky Seven Mine.¹⁷¹

In Vermont, the Vermont Asbestos Mines Division of the Ruberoid Co. quarried and processed chrysotile near Lowell in Orleans County. Twenty-four grades were produced for spinning, cement stock, paper stock, and other uses. Some waste rock

was used for roadstone.¹⁷¹

Data on these mines are summarized in Tables 10 and 11 in the Appendix.

Other deposits of asbestos have been found in Georgia and Maryland.

Although no measurements have been made of the asbestos air pollution from mining in the United States, some evidence of the extent of pollution can be drawn from measurements and observations in foreign countries. The extent of air pollution from an asbestos mine in Finland was studied by Laamanen, Noro, and Maunio.¹³³ They found asbestos dust at distances up to 50 km from the mines, including dust-fall rates ranging from 1.52 g/100 m²/month at 4 km to 34.6 g/100 m²/month at 0.5 km. They concluded that asbestos dust is disseminated from mining and milling areas rather extensively and that the degree of pollution varies according to the distance from the mine or mill and the prevailing winds.

Schepers²³⁴ described the dust from asbestos mines and mills in South Africa as dust which "rolled through like a morning mist," producing "itching skins caused by asbestos adhering to our clothes. Even the food at the local hotel was gritty with dust."

Sluis-Cremer²⁰² reports dust counts in asbestos mines and mills of South Africa as listed in Table 4. He pointed out that living quarters near the mines were polluted with asbestos and that the main source of pollution was airborne asbestos

blown off dumps and roads made from the mine tailings.

TABLE 4
DUST COUNTS IN ASBESTOS MINES AND
MILLS IN SOUTH AFRICA, 1947²⁰²

Location	Dust Count, mpcf (mgm ³)	
	Mines	Mill
Northwest Cape Province	2.8-24 (100-840)	10-55 (360-1920)
Transvaal	2.3-6.5 (80-228)	4.6-20 (162-720)

3.2 Production Sources

World production of asbestos during the period 1956 to 1967 increased at the average rate of approximately 13 percent per year. Figure 1 shows that the world production nearly tripled during the period 1945 to 1965, while United States consumption only doubled during the same period. However, during the period 1956 to 1967, domestic apparent consumption fluctuated between 665,000 and 813,000 short tons per year and may be leveling off as substitute materials (such as fiberglass and plastics) provide competition.

The relative importance of the various industrial uses of asbestos is given in Table 12 in the Appendix. It is seen that the highest input of asbestos occurs in the asbestos cement, floor tile, asbestos paper products, and asbestos textile industries. The proportions of asbestos used in various products are shown in Tables 13 and 14 in the Appendix.

Thousands of Short Tons
U.S. CONSUMPTION
WORLD PRODUCTION

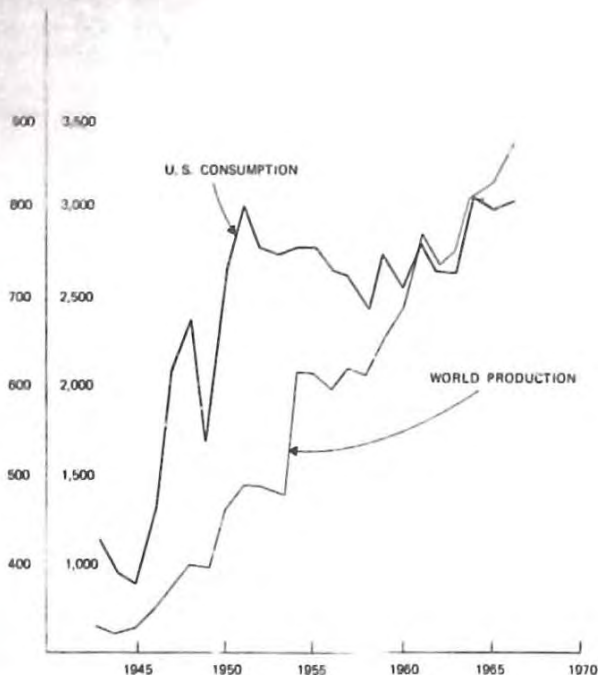


FIGURE 1

Comparison of Trends in World Production and U.S. Consumption of Unmanufactured Asbestos^{170,171}

Of the 78,056 short tons of domestic production in 1965, 66 percent were produced in California and 31 percent* in Vermont, amounting to 97 percent of the total production. This 97 percent was produced and processed in five counties.¹⁷⁰ More than 50 percent of the 124 plants comprising the asbestos products industry were located (in 1963) in the States of California, New Jersey, Illinois, Pennsylvania, and Texas, in decreasing order.⁴¹

Bobylova *et al.*^{24,25} have shown that the air can be polluted by asbestos from plants manufacturing asbestos products. This asbestos may be carried in the air for distances of 25 to 50 miles. In a study of asbestos air pollution from three plants in the U.S.S.R., they found that at a distance of 3 km from the plant, the dust concentration ranged from 0 to 6,000 $\mu\text{g}/\text{m}^3$; at 1.0-1.5 km it was 3,000-33,000 $\mu\text{g}/\text{m}^3$; and at 0.5 km it was 6,000 to 34,000 $\mu\text{g}/\text{m}^3$.

In the United States some attempts²⁰⁶ have been made to determine the concentration of asbestos near asbestos factories, but the asbestos content of the atmosphere was masked by the other dusts. Asbestos fibers were detected, but a quantitative count was not possible.

1. Product Sources

The uses of asbestos are numerous. Some products which use asbestos are the following: asbestos cement which may be

*Extrapolated.

applied as mortar or plaster, or sprayed on walls; insulating materials for the covering of pipes, ducts, boilers, cables, and conduits; siding shingles, roofing shingles, tiles, flat and corrugated sheets, wallboard, clapboard, and automobile undercoating; threads, yarns, wicks, cords, tapes, cloths, sheets, and blankets; friction materials, brake linings, clutch facings, gaskets, and lagging cloths; and asphalt tiles, plastics, and similar materials.¹⁰⁸

Abrasion of brake linings and clutch facings has been suggested as a primary source of asbestos air pollution. Ayer¹¹³ and Lynch¹⁴⁵ have examined the emissions from brakes on automobiles and found that the fiber is destroyed by the heat of friction. Asbestos crystalline structures are also destroyed and are recognizable only by the chemical composition. Newhouse and Thompson¹⁷⁵ have reported one case of mesothelioma in a mechanic.

The existence of a wide potential for direct or indirect occupational exposure has been cited^{32,34,108,120} as a possible explanation for the frequent occurrence of "asbestos bodies" in the general public. Asbestos is now used in more than 3,000 products. Most people working in the construction and demolition of buildings come into contact with asbestos. Electricians and homeowners strip asbestos insulation off wires; the carpenter saws asbestos boards and often pounds the asbestos-insulated furnace ducts to make them fit. As a result, the air around a construction site is contaminated with asbestos fibers,

and the foreman, carpenter, painter, plumber, or new occupants all breathe this dust. In most homes, the owner will at some time handle these asbestos products during normal home maintenance. A large number of workers in other industries are similarly exposed. Some of these are listed in Table 15 in the Appendix.

Although only a few of these people work in the asbestos industry, all may have inhaled sufficient asbestos to show "asbestos bodies" upon autopsy. The hazard is there, but how great is the hazard? It will be necessary to obtain quantitative concentration data to delineate its seriousness.

From the above discussion it may be concluded that approximately 100,000 asbestos workers in the United States have a high exposure to asbestos.¹⁹⁵ An additional 3.5 million construction workers—carpenters, welders, electricians, masons, plumbers, steamfitters, tile setters, etc.—are indirectly exposed, either by themselves handling asbestos products or by working on the job with people handling asbestos.¹⁹⁵

3.4 Environmental Air Concentrations

Only one estimate of the environmental air concentrations of asbestos in the United States was found. Smith and Tabor²⁰⁶ have roughly estimated that urban air in the vicinity of heavily traveled streets contains 600 to 6,000 particles/m³. They indicate that the validity of these values is highly suspect because the methods available for the determination of asbestos

are inadequate at the concentrations found in the urban air.

4. ABATEMENT

The asbestos industries in the United States have developed elaborate ventilation systems to prevent high dust concentrations which might be inhaled by the workers.^{13, 15} This dusty air is passed from the ventilators through fabric sleeve filters and then discharged to the atmosphere. The asbestos fibers are easily filtered out since the fibers form a mat which becomes an absolute filter.¹³ In addition to the ventilation system, it has been necessary to carry out some operations (such as spinning and weaving) as wet processes to eliminate dust. As a result, the pollution from factories is minimal. Attempts to measure concentrations in the vicinity of an asbestos plant have proved futile with present analytical methods.

Pollution during the transportation of asbestos has been controlled by enclosing the material in plastic-coated bags.

Although the most common procedure used to suppress dust emission is wetting of the material, it is unfortunately not desirable to wet a large number of asbestos products.

In New York, insulators are required to enclose the area when asbestos fireproofing is blown onto steel frames, but even this does not prevent pollution. Asbestos fibers are reported to be a common occurrence in the air around construction sites.

No information has been found on the abatement methods used in United States asbestos mines and in asbestos mills near the mines.

5. ECONOMICS

No information has been found in the literature on the damage costs or economic losses due to the effects of asbestos air pollution on humans, animals, plants, or materials. However, a large fraction of the people in the United States have been exposed to asbestos, including the following:

- (1) approximately 100,000 workers using asbestos in their occupations,
- (2) approximately 3,500,000 construction workers exposed indirectly to asbestos as they work with asbestos products or near those who handle asbestos products,
- (3) approximately 50,000,000 Americans who possibly have "asbestos bodies" in their lungs.

No attempt has been made to assess the cost of health impairment for these people. Workmen's compensation laws for dust diseases are in effect in most States.²³⁴

No information has been found on the cost of the present and future abatement of air pollution by asbestos in this country. The data in Table 16 in the Appendix, which refer to the asbestos industry in Britain,⁹⁵ show that the dust extraction equipment cost alone, for a given size and type of plant, amounts to 27.5 percent of the total capital cost and approximately 7 percent of the operating cost of that plant. The type of control equipment used is primarily designed to meet government specifications relating to occupational health standards.

Data on the production and consumption of asbestos are presented in Section 3.

6. METHODS OF ANALYSIS

Of the methods presently being used^{1,2,206} to count dust samples in the asbestos industry,* none is applicable to atmospheric asbestos air pollution. There are at present no proven satisfactory methods for the collection, detection, and identification of asbestos fibers in the 0.1 to 5.0 μ range in ambient air. Satisfactory sampling can probably be accomplished by use of a membrane filter-pump system. The major difficulty lies in the problem of identifying a very few asbestos fibers in the presence of relatively large numbers of a wide variety of other inorganic particulate matter found in the same air. Attempts to determine the asbestos content of urban air have revealed the need for development of new methods. Battelle Memorial Institute is currently developing one such method for the National Air Pollution Control Administration.²⁰⁵

*In all the asbestos monitoring methods used, microscopic counting of the fibrous particles is necessary to determine the proportion of fibrous material, and even then it is not known what fraction of the fibers are asbestos. Counting of fibers by eye under the microscope is tedious and difficult. If the number of fibers is less than 1 percent (<5 wt%) of total dust, the other dust masks the fibers, and quantitative results cannot be obtained.

In parts of the asbestos industry where the asbestos-to-dust ratio is high (>5 wt%), it is often possible to determine the asbestos content indirectly.¹³⁶ For example, if the proportion of asbestos in the airborne dust was known by microscopic count for a given sampling location, the concentration (at least the order of magnitude) could then be inferred from a simple measurement of the concentration of the total dust.

Modern analytical methods and instrumentation used in the asbestos industry are listed below:

Microscopic particle counting of samples on membrane filters^{1,2,14,15,57,97,136,187}

Thermal precipitators^{1,2,97,136,187}

Impingers^{1,2,14,15,136,187}

Rayco particle counter^{1,2,136,187}

Mass concentration methods^{1,14,136,187}

Microsieving¹¹⁶

Digestion¹¹⁶

Column chromatography of organics adsorbed on the surface¹¹⁶

X-ray diffraction^{14,46,47,116}

Low-temperature ashing¹¹⁶

Atomic adsorption spectrophotometry^{14,116}

Electron microprobe¹¹⁶

Neutron activation¹¹⁶

Owens jet counter^{1,2}

Konimeter^{1,2}

7. SUMMARY AND CONCLUSIONS

Asbestos is an air pollutant which carries with it the potential for a national or worldwide epidemic of lung cancer or mesothelioma of the pleura or peritoneum. Asbestos bodies have been observed in random autopsies of one-fourth to one-half of the population of Pittsburgh, Miami, and San Francisco and will probably be found in the people of every large city. Although asbestos has been shown to produce asbestosis, lung cancer, and mesothelioma in asbestos workers, the relationship between "asbestos bodies" and cancer or asbestosis has not been determined.

The latent period required to develop asbestosis, lung cancer, or mesothelioma is 20 to 40 years, and the exposure required to cause asbestosis has been estimated to be 50 to 60 mppcf-years. No such exposure relationship has been established between asbestos and lung cancer or mesothelioma. Asbestosis, lung cancer, and mesothelioma are all diseases which, once established, progress even after exposure to dust ceases.

Experiments with animals have shown that animals may develop asbestosis or cancer after inhaling asbestos.

No information has been found on the effects of asbestos air pollution on either plants or materials.

The likely sources of air pollution appear to be the vast number of asbestos products used in our modern society, particularly in the building industry. Mines, factories, and

shipping yards may also constitute pollution sources.

The world production of asbestos has approximately doubled over the past 10 to 12 years, whereas the domestic consumption has apparently remained relatively constant. These results indicate that while the asbestos air pollution problem of the world may be increasing, the air pollution potential in the United States has remained relatively unchanged. The increase in the number of lung cancer or mesothelioma cases reported in the current literature may be due to increases in asbestos use that occurred 20 to 40 years ago. Moreover, the effects of the asbestos being inhaled today may not be reflected in the general health of the population until the 1990's or the next century.

The number of people exposed to asbestos has been estimated to be 100,000 asbestos workers, 3.5 million people working in areas where asbestos is handled in ways which emit small quantities of dust, and 50 to 100 million people who have breathed or will breathe enough fibers to show positive "asbestos bodies" at autopsy.

No measurements have been made of the concentration of asbestos in urban air. A single estimate of 400 to 6,000 particles per cubic meter has been reported.

Bag filters are used to control asbestos in the exhaust gases from asbestos factories. Wetting the asbestos or its products has also been used to keep the dust from becoming airborne. The cost of abatement equipment in a British factory

amounted to 27.5 percent of the total factory cost and approximately 7 percent of the operating cost. Similar data on the costs of abatement or economic losses due to asbestos air pollution in the United States were not found.

No satisfactory method is available to determine the asbestos content at the concentration found in the ambient air. One method is presently under development.

Based on the material presented in this report, further studies are suggested in the following areas:

- (1) Identification of the fibers in the lungs of deceased members of the general public should be completed. (Two studies are in progress.)
- (2) The relationship between these "asbestos bodies" and lung cancer or mesothelioma should be determined.
- (3) The time-concentration relationship between inhalation of asbestos fibers and lung cancer and mesothelioma should be determined.
- (4) Methods must be developed to determine the asbestos concentration in air. (One method is under development.)
- (5) The concentrations of asbestos fibers in urban air, near mines and factories, and in the vicinity of building construction and demolition should be measured.
- (6) The methods and cost of control of asbestos emission from mines, construction or demolition sites, and other normal uses should be determined.

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APPENDIX

TABLE 5
CANCER OF THE LUNG AMONG ASBESTOS WORKERS (Continued)

Place	Population Studied	No. of Workers	Years Followed	No. With Lung Cancer	Comparison Groups
California	Insulating workers, mixed, 15 years in trade, aged 35-64	529	1954-1957	10/41 deaths	2.8 Expected
Dresden	All asbestos trade mixed exposures	2,636	1924-1963	34/150 deaths	11.4 Expected
United States	Asbestos textile workers employed in 1948-1951, aged 15-64	2,833	1951-1963	24/285 deaths	11.9 Expected
United Kingdom	Reported deaths from asbestosis	584	1924-1963	146/584 (25%)	
Bulgaria	Agricultural workers near an asbestos mine	3,325	1962	155/3325 asbestosis	
New York	Asbestos insulators employed more than 15 years, aged 40-60	152	1945-1965	18/46 deaths	3.1 Expected
Pennsylvania	Asbestos textile and friction workers	68	1957-1962	13/68 deaths	

APPENDIX

TABLE 6
"ASBESTOS BODIES" IN CONSECUTIVE OR RANDOM AUTOPSIES

Location	Total Cases	% Positive	% Positive by Age*							Sex % Positive*		Year of Study	Reference
			<24	25-34	35-44	45-54	55-64	65-74	>75	Female	Male		
Capetown	500	26.4	.5 (4.3)	2.4 (25.6)	4.4 (28.4)	4.6 (22.4)	7.4 (31.6)	7.4 (28.0)		7.8 (20)	18.6 (30.4)	1960	214, 216
Miami	500	27.2	0	0	1 (16.1)	3.4 (23)	5.6 (27.2)	9.2 (30.6)	9.1 (31.9)	8 (20.4)	19.2 (31.6)	1961	214, 216
Pittsburgh	100	41	0 (0)	5 (93)	5 (46)	3 (38)	13 (54)	8 (40)	6 (30)	16 (34)	25 (47)	1964	40
Milan	100	51		(←	3 (14)	→)	30 (60)		18 (66)	16 (44)	35 (54)	1966	67
Tyneside	311	20.3	0 (0)	0.3 (25)	1.6 (19.2)	2.9 (17.3)	4.8 (18.5)	7.7 (28.9)	2.9 (15)	4.2 (13)	16.1 (25.5)	1967	11
Jerusalem	100	26	←	1 (14)	→)	←	7 (21)	18 (30)	→	10 (29.1)	16 (22.2)	1967	184
Glasgow	100	23	0	0	0	1 (12)	4 (22)	11 (32)	7 (19)	0 (0)	23 (37)	1967	188
Finland	264	57.6	1.5 (57)	3.0 (57)	3.8 (71)	8.7 (55)	20.0 (64)	16.7 (58.7)	3.8 (34.5)	23.9 (54.3)	33.7 (60)	1966	167
Sweden	34	35.3	0	0	0	0	11.7 (80)	14.7 (25)	18.8 (43)	14.7 (38)	20.6 (33)	1966	82

(continued)

TABLE 6

"ASBESTOS BODIES" IN CONSECUTIVE OR RANDOM AUTOPSIES (Continued)

Location	Total Cases	% Positive	% Positive by Age*							% Positive*		Year of Study	Reference
			<24	25-34	35-44	45-54	55-64	65-74	>75	Female	Male		
Johannesburg		39.2										1965	234
San Francisco		42										1966	43
Belfast	200	20										1965	60
London	50	6										1964	102
Montreal	100	48		3 (30)	48 (50)			27 (50)		16 (36)	32 (57)	1966	7
New York	355	50.5								3.9 (29.8)	46.5 (53.7)	1966	195

*Numbers in parentheses represent percentage of group-age or sex.

APPENDIX

TABLE 7

COMPOSITION AND PROPERTIES OF ASBESTOS MINERALS⁶⁶

Approximate Formula	Percentage of Major Components		
	Chrysotile 3MgO·2SiO ₂ ·2H ₂ O	Crocidolite Na ₂ O·3FeO·Fe ₂ O ₃ · 8SiO ₂ ·H ₂ O	Amosite 1.5 MgO·3.5FeO· 8SiO ₂ ·H ₂ O
Silica SiO ₂	40.3	51.4	49.3
Alumina Al ₂ O ₃	0.7		
Ferrous Oxide FeO	1.0	20.3	40.9
Ferric Oxide Fe ₂ O ₃	1.5	17.5	0.4
Manganous Oxide MnO		0.1	0.7
Calcium Oxide CaO	0.2	0.8	0.4
Magnesium Oxide MgO	42.4	1.4	5.7
Sodium Oxide Na ₂ O		6.2	0.2
Potassium Oxide K ₂ O			0.3
Carbon Dioxide CO ₂	0.2	0.4	0.2
Water of Crystallisation H ₂ O	13.7	1.9	1.9

(continued)

TABLE 7

COMPOSITION AND PROPERTIES OF ASBESTOS MINERALS (Continued)

Approximate Formula	Chrysotile $3\text{MgO} \cdot 2\text{SiO}_2 \cdot 2\text{H}_2\text{O}$	Crocidolite $\text{Na}_2\text{O} \cdot 3\text{FeO} \cdot 2\text{SiO}_2 \cdot 8\text{SiO}_2 \cdot \text{H}_2\text{O}$	Amosite $1.5 \text{MgO} \cdot 0.5 \text{FeO} \cdot 8\text{SiO}_2 \cdot \text{H}_2\text{O}$
<u>Trace Organic Impurities</u>			
Oil-wax (mg/100 g fiber)	4-7.6	4-200	4-20
Benzo(a)pyrene (ug/100 g fiber)	none detected	0.2-24	0.2-2.4
<u>Trace Inorganic Impurities (ug/g fiber)</u>			
Pb	3	5	20
Sn	<5	<5	<5
Ga	<2	<2	2
Bi	<5	<5	<5
V	50	<2	<2
Mn	<2	<2	<2
Cu	35	7	7
Ti	50	50	300
Ag	<0.2	0.2	0.2
Ni	5,000 (1,000-14,000)	<10 (<100)	1,000 (<100)
Zn	<200	700	1,000
Co	<5 (<100)	<5 (<100)	<5 (<100)
Mn	130 (400-500)	160 (200)	7,000 (7,900)
Cr	1,000 (400-900)	20 (<100)	150 (<100)

(continued)

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TABLE 7

COMPOSITION AND PROPERTIES OF ASBESTOS MINERALS (Continued)

Approximate Formula	Chrysotile	Crocidolite	Amosite
<u>Radioactive Contaminants (uuc/g fiber)</u>			
K ⁴⁰	0.14	0.02	0.55
Th ²³⁸		<0.01	0.05
Ra ²²⁶	0.07		0.15
<u>Physical Properties</u>			
Flexibility	Very flexible	Fair to good	Good
Length	Short to 3"	Short to 3"	1/4" to 6"
Texture	Harsh to silky	Harsh to soft	Coarse but pliable
Tensile strength	Very high	Very high	Fair
Acid resistance	Fairly soluble	Very good	Good
Heat resistance	Good	Poor	Good
Spinnability	Very good	Fair	Fair

APPENDIX

TABLE 8
WORLD PRODUCTION OF ASBESTOS¹⁷⁰
(Short Tons)

Location	1962	1963	1964	1965	1966 ^{b, c}
North America					
Canada (sales)	1,215,814	1,275,530	1,420,769	1,387,555	1,479,281
United States (shipments)	33,190	66,396	101,092	118,275	125,928
South America					
Argentina	203	365	542	243 ^c	240 ^a
Bolivia (exports)	56	10	7	3	4
Brazil	4,900 ^a	1,440 ^e	1,430 ^{a, c, f}	1,204	1,820
Europe					
Austria	503	638			
Bulgaria	1,323 ^c	1,323 ^c	1,433 ^c	1,433 ^c	1,430 ^a
Finland ^g	10,869	10,201	11,611	13,307	13,250
France	28,034 ^c	26,094 ^c	24,289 ^c	25,506 ^c	7,720 ^a
Greece		74	65 ^a	85 ^a	85 ^a
Italy	60,860	63,016	75,573	79,214	90,464
Portugal		29		53 ^c	10
U.S.S.R.	710,000 ^{a, c}	755,000 ^{a, c}	810,000 ^{a, c}	865,000	925,000 ^a
Yugoslavia	7,401	9,074	9,280	10,585	8,411
Africa					
Botswana	2,375	2,368	2,161	888	880 ^a
Kenya	212	78	204	136	73
Mozambique	370				
N Rhodesia, Southern	142,195 ^c	142,254 ^c	153,450	176,149 ^c	175,000 ^a
South Africa	221,302	205,744	215,592	240,752	276,597
Swaziland	32,830	33,350	39,862	40,884	36,142
United Arab Republic	606	192	1,739	3,225	2,057

(continued)

APPENDIX

TABLE 8
WORLD PRODUCTION OF ASBESTOS (Continued)
(Short Tons)

Location	1962	1963	1964	1965	1966 ^{b, c}
Asia					
China	100,000	110,000	130,000	140,000	140,000
Cyprus	22,391	19,962	13,755	17,622 ^c	24,449
India	1,865	3,038 ^c	3,710 ^c	4,989	7,646
Japan	15,407	18,210	17,979	16,451 ^c	17,067
Korea, South	1,333	2,120	1,402	1,710 ^c	687
Philippines	1,037	421	586		
Taiwan	525	604	526	883	721
Turkey	709	408	1,291	1,376	1,258
Oceania					
Australia	18,416	13,374	13,545	11,647	13,472
New Zealand	457	439			
World Total ^a	2,655,000 ^c	2,760,000 ^c	3,050,000 ^c	3,140,000 ^c	3,350,000

^aEstimated.^bPreliminary.^cRevised.

^dAsbestos also is produced in Czechoslovakia, Eritrea, Malagasy, North Korea, and Rumania. No estimates for these countries are included in the total because production is believed to be negligible.

^eCompiled from data available May 1967.^fBahia only.^gIncludes asbestos flour.

TABLE 9

THE PRODUCTION AND APPARENT CONSUMPTION
OF ASBESTOS IN THE UNITED STATES¹ 190,171

Quantities in Short Tons				
Year	Production	Imports	Exports	Consumption
1967	123,190	645,110	47,710	720,580
1966	125,928	726,459	46,996	805,391
1965	118,275	719,559	43,126	794,708
1964	101,092	739,361	27,147	813,306
1963	66,396	667,800	10,044	724,212
1962	53,190	675,953	2,949	726,194
1961	52,014	616,529	3,799	(665,440)
1960	45,223	659,945	5,525	709,193
1959	45,459	713,047	4,461	754,045
1958	43,979	644,331	3,026	685,284
1957	43,653	682,732	2,893	723,492
1956	41,312	689,910	2,950	782,272
1955	50,431	716,480	7,001	759,910
1950				727,002
1945				378,030
1940				270,000
1935				170,000

Values (X 1,000)				
Year	Production	Imports	Exports	
			Asbestos	Asbestos Products
1967	\$11,100	\$66,000	\$6,030	
1966	11,056	73,100	5,763	\$21,963
1965	10,162	70,457	5,294	19,139
1964	8,143	72,973	3,199	16,288
1963	5,108	61,739	1,304	16,267
1962	4,677	64,112	598	14,274
1961	4,347	(63,000)	759	
1960	4,231	63,345	857	
1959	4,391	65,006	793	
1958	5,127	58,314	424	13,233
1957	4,918	60,104	350	15,223
1956	4,742	61,939	375	14,181
	4,534	59,339	1,497	12,464

APPENDIX

TABLE 10
REGIONAL DISTRIBUTION OF ASBESTOS MINING AND PROCESSING¹ 190,171
(Ranking and Production)

Rank	State		Quantity (Short Tons)			Value (X 1,000)	
	1960	1964	1964	1965	1966	1965	1966
1	Ut.	Calif.	55,041	74,587	81,671	\$6,177	\$6,945
2	Ariz.	Vt.	"	"	"	"	"
3	N.C.	Ariz.	"	3,469	"	441	"
4	Calif.	N.C.	"	(78,056)	(81,671)	(6,177)	(6,945)
			(55,041)				

¹Data withheld to avoid disclosure of producer's confidential information to the Bureau of Mines.

TABLE 11
ASBESTOS MINES IN THE UNITED STATES, 1966¹⁷⁰

State	Mineral	Production Rank ^a	Name of Producer	Location—County or City of Mine	Processing Plant
California	Chrysotile	1	Atlas Minerals Corp.	Fresno County	Near Mine
	Chrysotile	2	Coalinga Asbestos Co.	Fresno County	Near Mine
	Chrysotile	3	Pacific Asbestos Corp.	Calaveras County	Near Mine
	Chrysotile	4	Union Carbide Corp.	San Benito County	Monterey County (King City)
Vermont	Chrysotile	1	Fish Bonding Co. ^b	Napa	
			Vermont Asbestos Mines Div. of Ruberoid Co.	Orleans County (Lowell)	
Arizona	Chrysotile	1	Jacquays Mining Corp.	Salt River Valley (North of Globe)	
	Chrysotile	2	Western Asbestos Mfg. Co.		
	Chrysotile	3	Metate Asbestos Corp.		
North Carolina	Crocidolite	1	Kyle Asbestos Mines of Ariz. ^a		
			LeTourneau Asbestos Corp. ^a	Yancey County (Burnsville)	

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^aRanked by production only within the State.
^bNot in operation during previous years.

APPENDIX

TABLE 12
APPARENT ASBESTOS CONSUMPTION, 1945-170
(In thousands of short tons)

Use	World ⁹⁷	United States ^a
Asbestos Industry Production		76
Mining and Processing		
Asbestos in Products		
Textiles	46	17
Cement	2,190	548
Friction Materials	111	28
Asbestos Paper	220	55
Floor Tile	307	77
Plaster, Wood Coating, Gypsum	85	21
Plastics	21	5
Miscellaneous	221	35
Total Products	3,221	806

^aBased on 1959 world consumption.

TABLE 13

PRODUCTION OF ASBESTOS IN VARIOUS ASBESTOS PRODUCTS¹⁷⁰

Product	Percent Asbestos
Asbestos textiles	80-100
Asbestos cement	15-90 ^b
Friction materials and gaskets	30-80
Asbestos paper and products	80-90
Floor tile	10-30
Other asbestos products	U.D. ^c

^aChrysotile asbestos is used unless otherwise stated.^b25 to 90% chrysotile, with some materials containing as much as 85% amosite and small amounts of crocidolite and anthophyllite.^cThese products contain undetermined quantities of chrysotile, tremolite, actinolite, and anthophyllite.

TABLE 14

QUANTITY AND VALUE OF ASBESTOS INPUT BY INDUSTRY 1963¹⁷⁰

Product	Quantity		Value	
	Short Tons(000)	Fraction of Total	\$ (000,000)	Fraction of Total
Asbestos textiles	66	0.02	26.4	0.06
Asbestos cement	2,190	0.68	328.5	0.78
Friction materials and gaskets	111	0.04	11.1	0.03
Paper and products	220	0.07	19.8	0.05
Floor tile	367	0.09	13.2	0.03
Paint and coating				
Paints	85	0.02	3.7	0.01
Plastics	21	0.01	0.2	
Other	220.7	0.07	19.7	0.04
Total	3,220.7	1.00	423.3	1.00

TABLE 15

POPULATION GROUPS WITH OCCUPATIONAL AND ENVIRONMENTAL EXPOSURE TO ASBESTOS¹⁰⁸

Occupational Groups	Nonoccupational Groups
Asbestos rock miners, loaders, truckers, crushers, millers, asbestos spinners, weavers, electrical appliance and wire manufacturers, masons, carpenters, heating equipment workers, rubber workers, shingle and tile manufacturers, building material manufacturers, filtering material manufacturers, molders of asbestos products, asbestos-asphalt makers, putty manufacturers, asbestos cement makers, asbestos paper, cardboard and brake-lining producers, asbestos felt insulation workers, asbestos sound insulation workers, asbestos insulators, pipe coverers, asbestos tube wrappers, asbestos cork insulation workers, construction workers, automobile makers, garage attendants	Residents in vicinity of asbestos processing and textile mills inhaling plant effluents polluted with asbestos dust, and individuals living and working along roads on which asbestos is trucked; residents in the vicinity of asbestos mines; residents in vicinity of building construction and demolition, inhabitants of homes or offices with asbestos acoustical tile

TABLE 16
ASBESTOS CONTROL EQUIPMENT⁹⁵

Cost Data	
Asbestos Textile Industry ^a	
Dust extraction equipment	
Total capital cost (fixed)	\$1,500,000
Operating cost per year	\$ 250,000
Operating cost/labor cost	7. %
Operating cost/total cost conversion	2. %
Asbestos Mines ^b	
Dust extraction equipment	
Total capital cost (TCC)	\$ 360,000
(TCC/TCC of plant) x 100	27.9%
Operating cost (per year)	\$ 195,000
Specifications	
Asbestos Textile Industry ^a	
Volume of dust-containing air extracted from textile machines	1,000,000 ft ³ /min or 700 ft ³ /min/operative
Quantity of asbestos dust filtered per year (at above rate)	700 tons or 2.8 tons/working day
Asbestos Mines	
Total installed horsepower	796
Part used to generate air for dust removal	230 (29%)
Total air needed (for aspiration and dust removal) per pound of fiber produced	1,350 ft ³

^aFigures apply to the Turner Bros. Asbestos Co. plant at Rochdale, England.

^bFigures apply to the Cape Asbestos Co. at Pange in the Transvaal. The most modern mill (in Canada), which is 10 times larger, needs only about half the quantity of air stated (using gravity instead of air-swept mills and horizontal transportation of ore).

TABLE 17
ANALYSIS OF ASBESTOS AND ASBESTOS PRODUCTS EXPORTS AND IMPORTS¹⁷⁰

	Exports		Imports		Re-exports	
	Short Tons	Dollars (000)	Short Tons	Dollars (000)	Short Tons	Dollars (000)
	1965	1966	1965	1966	1965	1966
Crude and spinning fibers	1,251	1,455	326	325	50	176
Scraping fibers	24,221	28,017	3,622	3,973	81	130
Waste and refuse	17,523	17,218	1,323	1,414	130	13
Total	42,995	46,690	5,271	5,712	131	306
Gaskets and packing	1,732	2,678	4,528	5,261	1	1
Brake lining	3,065	4,728	5,236	1	1	1
Clutch lining (number)	2,020,864	2,246,700	1,697	5,000	5,000	4
Textiles and yarn	784	1,052	1,326	1,297	113	231
Shingles and claspboard	5,465	4,742	1,588	1,332	44	45
Asbestos-cement	6,101	14,698	16,649	5,114	5,232	44
Sub total						
Other products			4,389	5,058		8
Total			19,087	21,907		52
						56
	Imports		Exports		Re-exports	
	Short Tons	Dollars (000)	Short Tons	Dollars (000)	Short Tons	Dollars (000)
	1965	1966	1965	1966	1965	1966
Chrysotile						
Crude	12,496	6,596				
Spinning/						
Textiles	17,339	16,839				
All other	643,149	642,894				
Total	672,984	666,329				
Groedolite	21,165	26,995				
Amosite	17,042	23,834				
Total	711,191	716,258				

*Not available.

APPENDIX

TABLE 18

SELECTED STATISTICS FOR THE ASBESTOS MANUFACTURING INDUSTRY⁴¹
(Employment Size)

Number of Employees*	Number of Companies	Number of Plants	Number of Production Workers	Value of Shipments \$ (000)
1-49	39	39	308	8,264
50-99	6	6	231	4,827
100-2,499	10	17	2,445	89,131
over 2,500	18	62	12,754	407,014
Total	73	124	15,738	509,236

*The employment size class is determined by the total company employment in all manufacturing activities in the U.S., including central offices and auxiliaries serving manufacturing establishments. All establishments of a company are therefore included in the same employment size column regardless of establishment size.

APPENDIX

TABLE 19

SELECTED STATISTICS FOR THE ASBESTOS PRODUCTS INDUSTRY⁴¹

Expenditures (in \$000)	1958	1963
New plant and equipment		
New structures	2,419	2,613
New machinery and equipment	10,418	9,768
Total	12,837	12,381
Used plant and equipment	428	1,289
Total	13,265	13,670

TABLE 20
ASBESTOS USES⁸⁵

Textiles:

Varieties used: Chrysotile, crocidolite, and in part amosite

Yarns and Cords:

Processes: Weaving of yarns and cords
Braiding (interlacing)

Classification of chrysotile fabrics:

Class	Quality Code	Asbestos Content (%)
1	AAAA	75-79.9
2	AAA	80-84.9
3	AA	85-89.9
4	A	90-94.9
5	Underwriters Commercial	95-100

Sealing and Packing Materials:

Packing (woven fabrics)
stuffing for boxes and sleeves
manhole rings, boiler covers

Flat Packing:

Gaskets, flanges (on pipes) and containers

1. Without metal: high pressure gasket sheets (rubber)
2. With metal: material for sealing cylinder heads and exhausts in motors and combustion engines, and for sealing compressors and turbines

Asbestos Boards and Papers:Boards:

Filtering and clarifying
Coverings, coatings, casings, and jacketings for all kinds of surfaces
Manufacturing of welders' and melters' shields
Slideways in the glass industry
Handles and fire-doors
Auto Parts
Safes
Protective walls
Curtains, etc.

(continued)

TABLE 20 (Continued)
ASBESTOS USES⁸⁵

Sheets:

Inner/outer linings of furnaces and heating vessels, drying ovens, incubators, heaters, climate-controlled spaces, etc.

Plates:

Insulating buildings against vibrations (aluminum-asbestos)
Solar-heat reflecting surfaces (70% of solar heat)

Special Asbestos Papers:

Filters

Asbestos Cement (10 to 25% asbestos):

Slabs
Corrugated sheets
Pipes
Corrugated tiles for roofs in industry, agriculture, and dwellings
Planks for platforms in buildings under construction
Balcony canopies
Rain gutters
Interior walls
Ventilating shafts
Air conditioning assemblies
Pressure piping (for underground drinking water distribution systems, fuel gas, and sewage)
Cooling towers (electricity-generating stations)

Thermal Insulants and Fire-Proofing:

Sprayed asbestos (insulant in both heating and refrigeration), ~~sound absorbent~~ (eliminates booming and improves acoustical properties of walls and ceilings)
Magnesia asbestos (80% magnesia, 15% asbestos) as thermal insulant for covering pipes

Friction Material:

Woven: Brake lining
Nonwoven: Clutch lining
Transmission lining

(continued)

TABLE 20 (Continued)

ASBESTOS USES⁸⁵Asbestos Plastics:

Flooring tiles (asbestos-asphalt tiles and, increasingly, asbestos-polymers of vinyl)
 Pressed or molded (thermal insulation and in electrical machinery)
 Resinated asbestos felt (manufacturing of wings and firing of missiles and expansion cones for nozzles of boost motors). Other uses in aircraft industry: nozzles for motor tubes, missile tailpipes, and missile-heat barriers; fuselages for guided missiles, fuel tanks for fighter bombers, cabin floors, etc.
 Radar (large molded reflectors and scanners)

Asbestos Acid-Resistant Compositions:

Used mostly in chemical industry

TABLE 21

1967 LIST OF MANUFACTURED ASBESTOS PRODUCTS⁸⁵

Industry and Product Description	Quantity Measure
Miscellaneous Nonmetallic Mineral Products	
Asbestos Products	
Asbestos Friction Materials	
Brake Linings	
Woven, containing asbestos yarn, tape, or cloth	Linear feet
Molded, including all nonwoven types	Cubic feet
Clutch facing	
Woven, containing asbestos yarn, tape, or cloth	Thousand pieces
Molded, including all nonwoven types	Thousand pieces
Asbestos-Cement Shingles and Clapboard	
Siding shingles and clapboard, including accessories	Squares
Roofing shingles	Squares
Asphalt Floor Tile	
Asphalt floor tile	Thousand square yards
Vinyl Asbestos Floor Tile	
Vinyl asbestos floor tile	Thousand square yards
Asbestos Textiles and Other Asbestos-Cement Products	
Asbestos textiles	
Yarn, cord, and thread	Pounds
Cloth	Pounds
Other asbestos textiles, including roving, lap, wick, rope, tape, carded fibers, etc.	Pounds
Asbestos-cement products	
Flat sheets and wallboard, all thicknesses converted to 4" basis	100 square feet
Corrugated sheets	100 square feet
Pipe, conduits, and ducts, including pressure pipe	Short tons

(continued)

TABLE 21 (Continued)

1967 LIST OF MANUFACTURED ASBESTOS PRODUCTS⁸⁵

Industry and Product Description	Quantity Measure
Asbestos felts	Short Tons
Roofing-asphalt or tar saturated	Short Tons
Other	Short Tons
Other asbestos and asbestos-cement products, including millboard and prefabricated housing components	
Gaskets and Insulation	
Gaskets, All Types	
Gaskets (for sealing nonmoving parts)	
Asbestos, asbestos-metallic, and asbestos-rubber	
Packing (except leather, rubber, and metal) and Asbestos Insulations	Pounds
Asbestos compressed sheet	
Packing (for sealing moving parts)	
Asbestos, asbestos-metallic, and asbestos-rubber	Thousand pounds
Insulation materials containing asbestos pipe insulation	
Cellular and laminated	Linear feet
85 percent magnesia	Linear feet
Diatomaceous silica, calcium, silicate, expanded silica, and asbestos fiber	Linear feet
Other pipe insulation	Linear feet
Block insulation, including sheet and lagging	Thousand board feet
85 percent magnesia	board feet
Diatomaceous silica, calcium silicate, expanded silica, and asbestos fiber	Thousand board feet
Other block insulation, including cellular and laminated	Thousand board feet
All other asbestos insulation	

TABLE 22

ASBESTOS PRODUCT MANUFACTURING PLANTS, 1963⁸¹

Location	Total Plants	No. of Plants with Employment of						
		1-19	20-49	50-99	100-249	250-499	500-999	1,000 or more
New Hampshire								
Belknap	1				1			
Hillsborough	1					1		
Total	2				1	1		
Massachusetts								
Essex	1			1				
Franklin	1	1						
Middlesex	2	1		1				
Suffolk	2	2						
Worcester	1			1				
Total	7	4		3				
Connecticut								
Fairfield	3		1	1				1
Hartford	1	1						
Middlesex	1				1			
Total	5	1	1	1	1			1
New York								
Albany	1						1	
Kings	3	1	1			1		
Orange	1					1		
Suffolk	1	1						
Total	6	2	1			2	1	
New Jersey								
Bergen	1		1					
Essex	3	2	1					
Hudson	1					1		
Mercer	2					2		
Morris	1				1			
Passaic	3	2				1		
Somerset	4				2	1	1	
Union	1	1						
Total	16	5	2		3	5	1	

(continued)

TABLE 22 (Continued)

ASBESTOS PRODUCT MANUFACTURING PLANTS, 1963⁴¹

Location	Total Plants	No. of Plants with Employment of						
		1- 19	20- 49	50- 99	100- 249	250- 499	500- 999	1,000 or more
Pennsylvania								
Elk	1				1			
Lancaster	1							1
Montgomery	2				2			
Northampton	1			1				
Philadelphia City	3					1		
Potter	1	2						
Total	9	3		1	3	1		1
Ohio								
Cuyahoga	1	1						
Paulding	1				1			
Portage	1				1			
Ross	1				1			
Total	4	1			3			
Indiana								
Henry	1					1		
Huntington	1					1		
Kosciusko	1				1			
Lagrange	1	1						
Lake	1				1			
Rush	1		1					
Total	6	1	1		2	2		
Illinois								
Cook	8	5	1			2		
Kankakee	1					1		
Lake	4				2	1	1	
Will	1				1			
Total	14	5	1		3	4	1	
Michigan								
Wayne	1	1						
Total	1	1						

(continued)

TABLE 22 (Continued)

ASBESTOS PRODUCT MANUFACTURING PLANTS, 1963⁴¹

Location	Total Plants	No. of Plants with Employment of						
		1- 19	20- 49	50- 99	100- 249	250- 499	500- 999	1,000 or more
Wisconsin								
Milwaukee	1	1						
Total	1	1						
Missouri								
St. Louis	2			2				
St. Louis City	3	1			2			
Total	5	1		2	2			
Kansas								
Barton	1	1						
Total	1	1						
Virginia								
Essex	1	1						
Frederick	1						1	
Norfolk City	1	1						
Total	3	2					1	
North Carolina								
Mecklenburg	2			1		1		
Union	1				1			
Total	3			1	1	1		
South Carolina								
Charleston	1						1	
Marlboro	1			1				
Total	2			1			1	
Georgia								
DeKalb	1	1						
Talbot	1	1						
Troup	1						1	
Total	3	2					1	

(continued)

TABLE 22 (Continued)
 ASBESTOS PRODUCT MANUFACTURING PLANTS, 1963⁴¹

Location	Total Plants	No. of Plants with Employment of						
		1- 19	20- 49	50- 99	100- 249	250- 499	500- 999	1,000 or more
Florida								
Dade	1	1						
Total	1	1						
Alabama								
Mobile	1			1				
Total	1			1				
Mississippi								
Hinds	1				1			
Union	1	1						
Total	2	1			1			
Louisiana								
Jefferson	3				2	1		
Orleans	3		1		1	1		
Total	6		1		3	2		
Texas								
Dallas	2	2						
Ector	1	1						
Grayson	1				1			
Harris	3				2	1		
Hill	1				1			
Total	8	3			4	1		
California								
Alameda	2		1	1				
Contra Costa	1			1				
Los Angeles	9	1	2	1	4	1		
Orange	1	1						
Sacramento	1	1						
San Benito	1	1						
San Joaquin	1				1			
San Mateo	1	1						
Santa Clara	1				1			
Total	18	5	3	3	6	1		
UNITED STATES								
TOTAL	124	40	10	13	34	21	4	2

APPENDIX B

RESPIRABLE ASBESTOS FIBERS

A number of questions arise regarding respirable asbestos fibers. What length of fiber is respirable? What is the particle-to-mass ratio? Should all fibers, whatever their length or diameter, be counted? If not, can any instrument be designed to select the right size distribution in the atmosphere?

Timbrell²¹⁸ has studied the deposition of fibrous material in the respiratory system. Fibers 50 or even 200 μ long are found in the lungs because the free-falling speed depends largely on the diameter. Thus, particles less than 3.5 μ (most asbestos particles are less than 0.5 μ) in diameter can possibly penetrate deeply into the lung. The more symmetrical a fiber is, the greater its chance of penetrating. The largest compact particles normally found in the lung are about 10 μ in diameter. Limitation on the lengths of the fibers which reach pulmonary air spaces is imposed by the nasal hairs and the small diameters of the respiratory bronchioles. These limitations are summarized in Table 23.

Respirable fibers have been defined by the British Occupational Hygiene Society²⁰⁴ as fibers less than 200 μ long, less than 3.5 μ in diameter, and having a length-to-breadth ratio of 3:1. Only the fibers longer than 5 μ in length are counted.

TABLE 23

PENETRATION OF FIBERS THROUGH NASAL HAIRS²¹⁸

Length of Fiber (microns)	% Penetration through Nasal Hairs		
	1st Stage	2nd Stage	3rd Stage
0.5	100	100	100
50	75	57	42
100	53	24	11
150	31	10	3
200	26	5	1
250	20	3	
300	17	2	
350	14	1	

Walter²²⁹ has investigated the mass of average particles in the asbestos textile industry (see Table 24). He found that respirable dust contains approximately 50 percent asbestos and that 10 particles of dust per μg contain 5×10^5 particles of asbestos per μg . From this conversion factor the threshold limit value for asbestos can be calculated* as approximately $350 \mu\text{g}/\text{m}^3$ (5 mppcf).

TABLE 24

PARTICLE-MASS RELATIONSHIP OF ASBESTOS AS
A FUNCTION OF FIBER LENGTH

Total Concentration Particles/ cm^3	$\mu\text{g}/\text{m}^3$ (approx)	Incinerating Residue $\mu\text{g}/\text{m}^3$ (approx)	Fiber Length in Microns (approx)
100	10	50-60	150
200	40	200-300	500
400	1,300	700-800	700
600	1,500	1,800-2,000	900,000

*Particle counted as $\pi \times 1 \times 100 \text{ cm}$.

*1 mppcf = $1 \times 10^6 \text{ p}/\text{ft}^3 = 150 \mu\text{g}/\text{m}^3$. The concentration estimated in air is $500 \times 1,000 \text{ m}^3/\text{m}^3 \times 1.2 = 12 \times 10^6 \mu\text{g}/\text{m}^3$.

Finally, there remains the problem of counting respirable fibers in ambient air. It appears that a fairly sophisticated instrument will be required which can (1) separate the other particles from fibers, (2) identify the asbestos fibers in a host of other fibers, and (3) count only those fibers longer than 5 μ and shorter than 200 μ with diameters less than 3.5 μ .

— R71-16

EXHIBIT 4

ASBESTOS

By I. J. Selikoff

I DO NOT KNOW if it is entirely appropriate to consider our current problems with asbestos an "unanticipated environmental hazard." As I look back, it would seem that we should have had some premonition.

For example, 70 years ago H. Montague Murray, then Senior Physician at the Charing Cross Hospital in London, examined a man of 33 suffering with pulmonary fibrosis. He had been working in the carding room of an asbestos textile factory. Of the ten men working in that room thirteen years before, he was the sole survivor. He, too, died a year later; at post mortem it was found that he had severely scarred lungs containing numerous asbestos fibers. Dr. Murray described this case to the Departmental Committee on Compensation for Industrial Disease which, after thanking Dr. Murray, noted that "... considerable trouble is now [in 1907] taken to prevent the inhalation of the dust, so that the disease is not so likely to occur as heretofore."

The disease which was referred to then had no name, nor did it when Auribault described numerous deaths among French workers in an asbestos textile factory in 1906, nor did it when Pancoast found abnormal X-rays of the chest in 1919 in a group of American asbestos workers. Surely, taxonomy has a particular virtue in science; individuals dying of this non-disease became much more evident when Cooke described another case in 1924 and 1927 and gave the illness its current name—*asbestosis*. We can now accurately record, for example, that ten percent of the deaths among asbestos insulation workers in New York in the past 25 years were due to *asbestosis*.

The new category was soon replete with cases.

However, comfort was provided, this time in the Home Office Report put before the English Parliament in 1930. While pulmonary disease among asbestos workers in the past was acknowledged, it was simultaneously concluded that the outlook was good. "In the space of a decade, or thereabouts, the effect of energetic application of preventive measures should be apparent in a great reduction in the incidence of fibrosis."

Such optimism persisted for the next 30 years; *asbestosis* was acknowledged as a rather rare disease, occasionally occurring among a small group of workmen employed in an isolated, esoteric industry.

There were a few rumblings during this time whose echoes should perhaps embarrass us now. Thus, Kenneth Lynch reported a death from lung cancer in association with *asbestosis* in 1935, and several similar reports of this association between two then uncommon diseases followed. For the most part, however, these reports were categorized as bibliographical curiosities. Even the careful studies of Richard Doll reported in 1955 failed to disturb our calm, perhaps because of the reticence with which the statistician summarized his findings. Listing eighteen deaths due to lung cancer in 105 consecutive post mortem examinations at one asbestos works in England, he concluded that "... lung cancer was a specific industrial hazard of certain asbestos workers ...". Even those who accepted Doll's conclusions—and there were some who did not—could point to a problem with a rather narrow spectrum. A single neoplasm (lung cancer) was (past tense) a specific industrial hazard! (no hint that it could be more than this) of certain asbestos workers (the heavily exposed individuals in this factory).

In the past several years, the spectrum has widened considerably. First, it has been recognized that the hazard associated with direct occupational exposure to asbestos is by no means a narrow or infrequent one. The experiences of insulation workers in this country are instructive. These men, employed in the construction industry and often working out-of-doors and with materials containing only limited amounts of asbestos, are much less exposed than many factory workers had been. Yet their health experiences have been disastrous.

We have studied all members of the Insulation Workers Union in the New York metropolitan area. On December 31, 1942 there were 632 members of this Union. Calculations in studies by E. Cuyler Hammond, J. Churg and myself indicated that by December 31, 1962, according to life expectancy in the general population of U.S. white males, there should have been 203 deaths among these men. Instead, there were 255—an excess of twenty percent. Using age, year and sex-specific rates, there should have been six or seven deaths due to cancer of the lung or pleura. Instead, there were 45. There should have been nine or ten deaths due to cancer of the stomach or colon; there were 29. There should, of course, have been no deaths due to asbestosis; there were twelve.

Similar calculations to mid-1967 gave similar results. Whereas 47 deaths were expected, there were 94. Where eight or nine deaths due to cancer were predicted, there were 49. Again, there was a significant excess of specific types of cancer. While there should have been two or three deaths due to cancer of the lung or pleura, there were 27, and there were eight deaths due to gastrointestinal cancer against two expected. Once more, asbestosis claimed fifteen lives against none expected.

Studies are now under way in our laboratory of insulation workers in other parts of the country. Mortality data indicate that the New York findings are unfortunately true elsewhere, as well. It is not known exactly how many asbestos insulation workers are employed in the United States; estimates range from 50,000 to 100,000. In the face of the mortality information, one hopes the lower figure is more accurate.

Information is incomplete concerning the number of workmen in other trades in the United States

with direct occupational asbestos exposure. Total of 100,000 to 125,000 are often proposed. Again, the lower figure is hoped for, especially in view of the absence of satisfactory mortality information concerning such asbestos factory workers, and since Mancuso's findings are disquieting.

Analysis of these mortality experiences is disturbing from another point of view, in addition to its magnitude. I refer to the occurrence of pleural

TABLE I
Observed and Expected Number of Deaths
among 632 Asbestos Workers Exposed to
Asbestos Dust Twenty Years or Longer, 1943-1962

Cause of Death	1943- 1947	1948- 1952	1953- 1957	1958- 1962	Total 1943- 1962
Total cancer, all sites					
Observed	13	17	25	39	95
Expected	5.7	8.1	13.0	9.7	36.5
Cancer of lung and pleura					
Observed	6	8	13	18	45
Expected	0.8	1.4	2.0	2.4	6.6
Cancer of stomach, colon, and rectum					
Observed	4	4	7	14	29
Expected	2.0	2.5	2.6	2.3	9.4
Cancer of all other sites combined					
Observed	3	5	6	7	21
Expected	2.9	4.2	8.4	5.0	20.5
Asbestosis					
Observed	0	1	4	7	12
Expected					less than 1
Total, all causes					
Observed	28	54	85	88	255
Expected	39.7	50.8	56.6	54.4	203.5

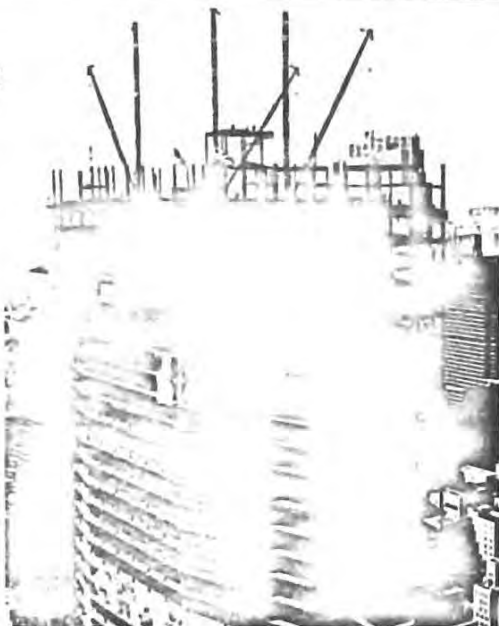
and peritoneal mesothelioma (tumors of the lining of the lung and abdominal cavities) among these workmen with direct occupational exposure. During the 1950's, there were several reports of deaths in asbestos workers caused by these diffuse tumors of the mesothelial surfaces. Such isolated cases would have received little notice had it not been for the fact that the tumor has always been considered extraordinarily rare. It is no longer rare among asbestos workers. Indeed, it is so common a cause of deaths among them now—while still rare among individuals not known to be exposed to asbestos—as to almost constitute a tumor specific to asbestos exposure. Several reviews of autopsy files suggest that this rapid increase, with hundreds of cases now known in Great Britain, South Africa, the United States and Canada, is not due to refinement in diagnosis. Thus, review of all possible cases in several major pathology departments in Scotland has demonstrated an increase from one or two cases a year in the 1950's to some twenty cases a year or more at the present time. Fourteen of the last 120 deaths in New York among asbestos insulation

A pioneer in modern asbestos epidemiology, Dr. Irving J. Selikoff is Professor of Environmental Medicine at New York City University's Mount Sinai School of Medicine, Director of its Environmental Sciences Laboratory, and is also President of the New York Academy of Sciences. He was awarded the American Public Health Association's Albert Lasker Award in Medicine for his research on isoniazid, an antibiotic drug widely used in the treatment of tuberculosis.

workers have been due to mesothelioma. The problem has assumed sufficient importance to have warranted the establishment of Mesothelioma Reference Panels in a number of countries, by the International Union Against Cancer, and the recent establishment of a Mesothelioma Registry in this country by the National Institute of Environmental Health Sciences of the U.S. Public Health Service.

[These experiences highlight one portion of the current spectrum of asbestos disease. It is now recognized that *direct occupational exposure* to asbestos results in a hazard much more significant than formerly appreciated. In the last several years, however, additional problems have been recognized which add another dimension; the *possibility that the utilization of asbestos is associated with a much wider risk, perhaps one of the community at large.* This is less well defined, much less well documented, but because of its potential importance, of considerable concern.]

The recognition of this potentially wider spectrum of asbestos disease has been based upon three germinal observations. The first was the publication in 1960 by Wagner and his colleagues of 47 cases of mesothelioma in South Africa, all from the northwest Cape Province, an asbestos-rich area of the country. Analysis of the cases showed that few had worked directly with the mineral but that 45 of the 47 may have had contact with it, often indirect. They had lived in an area in which asbestos was mined, or had worked in an environment in which indirect, haphazard exposure was conceivable. In striking contrast, there were no cases of mesothelioma in the rest of South Africa reported in the



Insulation workers employed in the construction industry have had disastrous health experiences even though their work is often outdoors and involves only limited contact with asbestos. Up until the past several years, it was thought that lung cancer from asbestos was an industrial hazard limited to the heavily exposed individuals in asbestos factories.

TABLE 2

Observed and Expected Number of Deaths among
370 Asbestos Workers from January 1, 1963
to April 30, 1967

Cause of Death	Observed Deaths	Expected Deaths*
Total, cancer (all sites)	49	86
Cancer of lung, pleura, bronchus, and trachea	27	23
Bronchogenic carcinoma	24	..
Pleural mesothelioma	3	...
Peritoneal mesothelioma	7	...
Cancer of stomach	3	0.6
Cancer of colon and rectum	5	1.2
Cancer of all other sites combined	7	4.5
Asbestosis	15	
Heart and circulatory disease including stroke	22	28.5
All other causes of death	8	10.4
Total, all causes	94	47.5

*Based upon U.S. mortality data disregarding smoking habits.

**United States data not available, but figure should be only slightly less than 2.3.

***United States data not available, but these are rare causes of death in general population.

same period, and none in 10,000 consecutive autopsies of individuals with silicosis. This report unequivocally raised the question of whether asbestos-associated mesothelioma was limited to direct occupational exposure, or whether much less intimate contact could produce the same result.

In the same year, 1960, Kiviluoto raised an analogous question. Pleural calcification had been known to occur frequently in characteristic form among asbestos workers. In our experience, indeed, the majority of older asbestos workers may be expected to show these changes when X-rayed. It was, therefore, striking to find typical "asbestotic" pleural calcification in 499 of 6,312 individuals X-rayed in one county in Finland while the neighboring county had none in 7,101. Kiviluoto astutely noted that the first county contained an asbestos mine and while none of the individuals with pleural calcification had worked in this mine, they had lived nearby. His countrymen, Laamanen and his colleagues, later demonstrated the presence of asbestos fibers in the air in the vicinity of the mine and while this may not reflect the true explanation for Kiviluoto's findings, the question of environmental contamination was strongly suggested.

The third observation was equally sobering. It had long been known that asbestos fibers, when

Inhaled into the human lung, may become coated with an iron-rich characteristic golden brown material, forming an "asbestos body." These are found, for example, in very large numbers in the lungs of asbestos workers. In 1963, Thomson reported that he found bodies with the same appearance in the lungs of people who were not asbestos workers. Smears of the lung bases allowed him to demonstrate these bodies in 26 percent of 500 consecutive autopsies in Capetown. His conclusion was that asbestos had indeed become a modern urban hazard, and he predicted that asbestos-induced mesothelioma might become as frequent as cigarette-associated lung cancer is at the present time.

Thomson's observations have been widely confirmed, and particles having the appearance of asbestos bodies have been reported in similar percentages in Montreal, Milan, London, Newcastle, Glasgow, Belfast, Dresden, Pittsburgh and Miami. In New York City, using a somewhat more sensitive technique, we find that approximately half of the lungs in 1,975 consecutive autopsies examined by us show these particles. When one considers that we examine only one square centimeter of lung tissue 0.0175 centimeter in thickness, it is obvious that these particles are now very common among city-dwellers at this time.

Opportunity for non-occupational contact with asbestos at the present time can easily be discovered. There are, for example, some three thousand products which currently utilize this valuable material. To the present, lack of recognition of the potential of non-occupational exposure has led to the absence of appropriate safeguards during industrial use. Demolition of buildings, often rich in asbestos-containing materials, is suspect, while sheer carelessness and ignorance may explain its

TABLE 4
Asbestos Bodies in Shipyard
and Construction Workers: Quantitative Results

	Blue Collar (No Shipyard or Construction)	Shipyard and Construction	White Collar Males
Total Number of Workers	246	129	206
Workers Having Asbestos Bodies	121 (49%)	90 (70%)	87 (42%)
Workers Having No Asbestos Bodies	125	39	119
Workers Having Five or More Asbestos Bodies	20 (8%)	29 (23%)	10 (5%)

recommendation and use as playroom material in kindergartens or in toy cosmetics.

Indirect occupational exposure is suggested by recent observations in our laboratory. We find, for example, that while approximately 30 percent of housewives examined at post mortem had asbestos bodies demonstrated, comparable figures are 40 percent for white collar males and 50 percent for blue collar males. This rises to over 70 percent for men with any history whatsoever of having worked either in the construction industry or in shipyards. The latter, moreover, are much more likely to have numerous asbestos bodies than any of the other groups.

A special hazard may be present as the result of an interaction recently described. We have found that there is a special risk of cigarette smoking among asbestos workers. Since January 1, 1963 we have observed 87 experienced insulation workers who were not regular smokers or who were pipe or cigar smokers; we have yet to see a death from bronchogenic carcinoma among these men. On the other hand, of 283 equally experienced workmen who smoked cigarettes and who were watched for the same period of time, we have seen 24 such cancers, although only three were expected. We have calculated that the risk of dying of lung cancer is approximately 92 times greater for an asbestos worker who smokes cigarettes than a man of the same age who neither works with asbestos nor smokes cigarettes. *J. G. Cunningham*

These observations naturally raise the question whether the inhalation of asbestos fibers among individuals in the general population might carry a special risk among the cigarette smokers in that population.

Wagner's observations have been extended by two recent studies. Muriel L. Newhouse investigated 76 consecutive cases of mesothelioma examined at the London Hospital, the majority within the last several years. She found, to no one's surprise, that

TABLE 3

Asbestos Bodies in New York City
Dwellers Without Occupational Exposure—1,975 Cases

Male				Female			
Age	Total cases	Total with asbestos bodies	Percent with asbestos bodies	Age	Total cases	Total with asbestos bodies	Percent with asbestos bodies
0-1	40	0	—	0-1	34	0	—
2-9	2	1	—	2-9	2	0	—
10-19	4	0	—	10-19	8	3	36%
20-29	22	4	18%	20-29	12	4	33%
30-39	50	14	28%	30-39	18	7	39%
40-49	178	83	47%	40-49	58	18	31%
50-59	280	149	53%	50-59	85	38	45%
60-69	359	205	57%	60-69	147	57	39%
70-79	321	180	56%	70-79	151	65	43%
80-89	105	62	59%	80-89	80	40	50%
90+	7	4	57%	90+	12	8	66%
Total	1368	702	51%	Total	607	240	39%

51 had been asbestos workers. Much more unexpected, however, was her discovery that nine had merely lived in the household of an asbestos worker, raising the serious question of a special hazard of family exposure. Then, of the 36 who neither worked with asbestos nor had relatives who worked with asbestos, eleven had lived within a half mile of an asbestos plant. The latter cases, of course, suggested a special risk associated with neighborhood contact with asbestos.

Similar findings were reported by Jan Lieben last year in Pennsylvania. Of 42 cases of mesothelioma reviewed by the Pennsylvania State Department of Health, twenty had identifiable occupational exposure. In addition, eight individuals had lived within the vicinity of an asbestos plant and three had had family contact.

We have confirmed that family contact is a distinct possibility. Sampling of both ambient air and settled dust in the homes of New York insulation workers has shown characteristic asbestos particles to be present, presumably brought home on the clothes of the workmen.

It is obvious that the spectrum of asbestos disease has considerably widened in the last several years. In addition to lung cancer and asbestosis, a number of other tumors are now recognized as constituting a hazard. Rather than a small number of heavily exposed workmen, a large number are involved in less intense direct occupational exposure or even in indirect occupational exposure. The latter may represent a real problem; there are, for example, 3,500,000 men employed in the construction and shipbuilding industries, or five percent of the total employed population in the United States. Significant exposure may even extend to the families of these men and perhaps to residents in the neighborhoods surrounding their places of employment. Finally, this ubiquitous material may well be found to permeate the community at large. What such exposures may bring is not now known. Disease associated with asbestos is rarely seen sooner than 20 to 30 years from the onset of exposure. Thus, among our insulation workers in New York, I have yet to see a mesothelioma in a man who began work after 1930 or a case of lung cancer in an asbestos worker who had worked in that industry less than twenty years. Our concern is with the future.

At the outset of this presentation, I questioned whether asbestos might properly be included as an unanticipated environmental hazard. Yet the less iconoclastic among us might have countered with: it must have been—we could not have knowingly allowed this to come to pass! Surely the explanation lies in the length of the period that elapses between exposure and disease which kept us from really knowing the problem until now.

Perhaps so. Indeed, I do not share the demonological theory of industrial hygiene history, in which industry is seen as callously exchanging human disease for profits. Rather, I would say industry shared the ignorance and lethargy of us all.

The answer will come in these next several years. We have some reason for optimism. First, we have the law of averages on our side—sooner or later, some important environmental hazard is bound to be controlled. I suspect asbestos will be one. We have reason to believe that the dose-disease relationship is on our side—to the present, disease has been uncommon except with significant exposure. And our data suggest further that such significant exposure can be prevented, that we can learn to live and work with asbestos. All it will take is the doing—but herein lies another tale. □

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The Interrelationships of Selected Asbestos Exposure Indices

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Based on the assumption that the biological effect of asbestos is related to the concentration of respirable fibers, an index was developed which yields the concentration in numbers of asbestos fibers visible under 430 $\times$ phase-contrast illumination. Data are presented relating this index to overall dustiness as measured by impinger counts and to absolute fiber concentration and distribution as measured by electron microscopy. The relation between count estimates and gravimetric measures of total dust and clay-silicate asbestos deduced from magnesium analyses are given for gross and respirable samples. These relations are developed for use in operations in the manufacture of asbestos textile, friction, cement, and insulation products. The most convenient, practical, and direct index of asbestos fiber exposure proved to be the concentration of fibers longer than 5 microns counted on membrane filters at 430 $\times$ phase-contrast illumination.

Introduction

AT THE NEW YORK ACADEMY OF SCIENCES Conference on the Biological Effects of Asbestos in 1965, Gibson¹ noted the proliferation of dust sampling methods and appealed for international comparability of dust measurement data to be related to biological data. He also observed that, as dust levels are lowered, it becomes more important to use the index that is biologically most appropriate.

At the present time the goal of international comparability has been partly achieved by the adoption of the concentration in numbers of fibers longer than 5 microns counted on membrane filter samples at 430 $\times$ magnification with phase-contrast (PC) illumination as the standard method in both Britain and the United States.^{2,3} However, information that would permit evaluation of the biological ap-

propriateness of this index is still not complete.

It is rarely possible to make an absolute measurement of that characteristic of an airborne dust which is directly responsible for its biological effect. To be practical, measurement for the purpose of assessing hazard and evaluating control must not only be convenient and relevant but also permit the use of statistically adequate sampling strategies. To evaluate indices obtained from varying exposure situations, an examination of the consistency of their relation to other and absolute measures was undertaken.

The large number of methods which might be used to measure exposure to airborne dust containing fibers results from what might be called the "degrees of freedom" of the dust cloud. In measuring the concentration of a gas in air, there is only one attribute to be measured, and one accurate method gives the same result as another. With granular dust, however, the attribute could be either number or weight, and with fibrous dust, length gives another degree of freedom; furthermore,

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a dust of mixed fibers and grains gives yet another.

Because of this variety of attributes, there could be as many as 100 indices of exposure to the asbestos in a dust cloud. In this United States Public Health Service epidemiological study of the asbestos products industry, the choice was narrowed down to the count and weight methods shown in Table I. Based on the results of analysis of almost 10,000 samples, this paper presents summaries of the relationships between these methods in order to (1) exhibit the effect of product and operation, (2) reveal the bias of the methods, and (3) evaluate the usefulness of the indices derived.

Impinger Relationships

The early epidemiologic data on asbestos were based on the impinger method and, until the beginning of the present study, most sampling in the United States was done by this method, since only in this way could the exposure be evaluated against the threshold limit value (TLV) of 5 million particles per cubic foot (mppcf)^{1,2} Consequently, it is necessary to develop ratios between present methods of measurement and impinger counts performed in the traditional manner so that the results that would have been obtained years ago had present methods been used might be predicted.

The ratios shown in Table II were developed from simultaneous pairs of impinger-membrane filter samples and are expressed as the fiber counts equivalent to 1 mppcf by impinger. Equivalence means the fiber concentration that would be found, on the average, when the impinger samples were taken, on the average, a concentration of 1 mppcf in the same product area. The product areas are textile for asbestos spun and woven products, friction for brake linings and clutch facings, and asbestos cement pipe.

In all cases the correlation coefficients from the large numbers of pairs of simultaneous samples are not over 0.6, as would be expected from pairs of measurements of a real environment, each having its own intrinsic error and not necessarily measuring the same thing. The impinger results are counts of all visible

TABLE I
Selected Measures of Fiber Exposure

Count	Weight
Total dust on impinger or filter	Total dust
3.0 μ fibers on MF or 4.0 μ fibers	Respirable dust
Fibers ≥ 5 microns on MF or 4.0 μ fibers	Chrysotile by magnesian
Total fibers on MF by electron microscope	Respirable chrysotile by magnesian

TABLE II
Impinger/Membrane Filter Count Ratios (fibers/ml for 1 mppcf)

Product	Fibers			Number of Pairs
	Total	$\geq 5 \mu$	$\geq 10 \mu$	
Textile	10.3	5.9	2.7	500
Friction	3.7	2.2	1.0	200
Pipe	4.8*	1.9*	0.7	100

*Correlation coefficient not calculated at 95%.

particles, mostly grains with generally less than 20% fiber. These few fibers seen in the impinger sample are a small fraction of the fibers counted on the membrane filters, since the membrane filter is a more efficient collector of fibers and the magnification/illumination used results in improved visibility. In the case of the impinger versus total and ≥ 5 -micron fiber ratios in the pipe plants, the correlation coefficient was not significantly different from zero at the 95% confidence level, and the hypothesis that the measures of exposure are unrelated cannot be rejected. In this case a usable prediction ratio will probably need to be based on ratios calculated for subsets within pipe manufacturing operations.

Size and Visibility

To appraise the value of an exposure index based on measurement of only a part of the things which may have biological significance, it is necessary to examine how that part relates to the whole. A count of fibers longer than a stated length as seen through a precisely defined optical system may be chosen as an index in order to eliminate as a variable the microscopic system's resolving power. It follows, however, that some fibers that are seen will be below the stated length and others will be below the limit of visibility and will not be seen at all.

TABLE III
Fiber Size Distribution

Operation	430X Phase Contrast % of Total Fibers	
	Longer than 5 μ	Longer than 10 μ
Textile		
Fiber preparation and carding	50	25
Spinning, twisting, and weaving	61	36
Friction		
Mixing	68	34
Grinding, cutting, and drilling	63	31
Pipe		
Mixing	57	28
Finishing	56	27
Insulation		
Mixing	55	27
Finishing	50	26

The proportions of >5 -micron and >10 -micron fibers of all the fibers visible at 430X PC on membrane filters are shown in Table III, broken down by operation within product area. Despite the fact that the textile operations and the three mixing operations are all cases of secondary dust dispersion (that is, the small dust particles already exist in the bulk material and are being shaken loose) whereas grinding and finishing are primary

TABLE IV
Ratio of 5.0- to 10.0- μ Fiber Concentrations
(phase contrast)

Operation	Fibers	
	Total	$>5 \mu$
Textile		
Fiber preparation and carding	2.0	2.1
Spinning, twisting, and weaving	1.8	1.9
Friction		
Mixing	1.1	1.1
Grinding, cutting, and drilling	1.0	1.1
Pipe		
Mixing	1.2	1.2
Finishing	1.6	1.8
Insulation		
Mixing	1.0	1.1
Finishing	1.8	2.0

dispersion, that is, the dust is being produced and dispersed simultaneously by mechanical abrasion, the proportions are quite consistent. Although mode of dispersion undoubtedly has an effect on the proportion of large particles, this effect apparently has a negligible influence on the number distribution of particles in the size range of biological significance. Similarly, the variation in bulk asbestos counts from the long fiber used in textile to the short fiber used in pipe has no apparent effect on the count proportions of fibers by length.



FIGURE 1. Glass-cloth asbestos dust viewed at 430X phase contrast.



FIGURE 2. Glass-cloth asbestos dust viewed at 970X phase contrast.



FIGURE 1. Electron micrograph of chrysotile asbestos dust.

TABLE VI
Count/Weight Ratios

Product	Type of Fiber Count	Geometric Mean (fibers per gram)	Geometric Standard Deviation
Textile	Total	11,500	2.5
	>5 μ	6,700	3.3
	>10 μ	3,100	1.5
Fraction	Total	26,700	3.4
	>5 μ	13,900	3.6
	>10 μ	5,800	1.4
Pipe	Total	46,500	2.8
	>5 μ	22,900	2.9
	>10 μ	8,800	1.4

pear to be greater than those obtained by optical microscopy. The technical difficulties involved in the preparation and counting of asbestos samples make quantitative comparisons impossible from the data obtained, but it appeared that the major difference between electron microscope counts and optical counts was in those fibers shorter than 5 μ in length. These small fibers, when they are present, dominate the electron microscope count. Even if the optical technical difficulties mentioned could be overcome, what would result from an electron microscope count method, would be fiber concentration strongly biased by these very small fibers. Present evidence does not indicate that they should be given such a dominant role.

Weight Relationships

Differences between counters and certain

statistical considerations make it difficult to obtain close agreement between counts even within a laboratory, and the differences are even greater between laboratories. These factors, plus the reluctance of counting, led industrial hygienists to seek weight methods of analysis wherever they are appropriate. Recent examples of this trend are the new weight TNA's for quartz and bituminous coal. This reasoning applied to asbestos led to the collection of many gross and respectable mass samples, analysis of which yielded some relationships between counts and weight and among weight methods.

Chrysotile asbestos and its parent rock, serpentinite, are hydrated magnesium silicates containing a relatively constant 25% magnesium. Owing to the extremely high sensitivity of the atomic absorption spectrometer for magnesium (0.01 μ g/ml for 1% absorption), it is possible to count asbestos fibers on one-half of a membrane filter at 50% PC and to analyze the other half for magnesium. In this way, the direct count-weight ratios shown in Table VI were obtained. Several difficulties with this method are readily apparent. Only chrysotile asbestos is determined; thus, the area is the area in which type of asbestos concentration pipes is not included in the weight of a sample. Further, all other sources of magnesium, especially the nonfibrous parent rock, associated with the chrysotile asbestos, interfere directly.

Although count data did not exhibit any consistent shift in fiber length distribution with product, operation, or mode of dispersion, the weight of asbestos, calculated from the magnesium content, gave decreasing fiber weight in this order: textile, fraction, pipe (Table VI). When the count-weight ratios were used to convert the tentative TNA of 12 fibers per milliliter to the equivalent gross weight concentrations of asbestos (based on magnesium determinations), the gross weight concentrations were 2 mg/m³ for textile, 1 mg/m³ for fraction, and 0.5 mg/m³ for pipe. A single value for the weight TNA for all product areas would be equivalent to a fourfold difference in fiber threshold between textile and pipe plants.

Since only the dust capable of penetrating into the pulmonary air spaces is biologically significant in pneumoconiosis-producing diseases, a number of samples were collected by means of respirable-mass size-selective sampling devices. A horizontal elutriator having characteristics similar to those proposed by the British Medical Research Council¹⁰ for respirable dust sampling was used ahead of membrane filters on which fibers were counted at 430X PC. A membrane filter sample without the elutriator was taken simultaneously, and the counts from the pair of samples were used to calculate the data in Table VII. The finding that 60% of the fibers over 10 microns were able to penetrate the elutriator reveals that many of the longer fibers are thin enough to have falling speeds in the respirable range. Other simultaneous sample pairs, using a Henschel and a gross dust filter side by side, were analyzed for asbestos by magnesium, giving the results shown in Table VIII. The 95% confidence intervals of the mean percent passing are typical of the range of most of this data. Use of the gross weight of dust found on these same samples, together with the asbestos by magnesium analyses, resulted in Table IX. The percent asbestos in the product, total dust, and respirable dust does not vary greatly for the same product. However, the percent of asbestos to be expected in either the total or the respirable dust will vary from 5% to 65% from product to product.

Summary

The impinger measures all types of dust and as the TLV for asbestos decreases, the non-asbestos dust present becomes a major source of error in the estimation of exposure to asbestos. Other count methods, however, are available to count asbestos fibers, with varying degrees of convenience. Weight methods that cannot differentiate asbestos from other dust suffer from the same disadvantage as the impinger in that the asbestos fiber concentration could differ by a factor of 10 for the same total or respirable dust concentration, depending on the product area. The method for chrysotile weight by magnesium

TABLE VII
Responsibility of Textile Fibers by Count

Textile Count	% Passes Elutriator
Fiber	81
Non	80
Total	60

TABLE VIII
Asbestos Dust Passing an Elutriator

Product	Mean Weight % Passing	95% Confidence Interval
Textile	31	24-38
Textile	29	21-37
Gips	11	9-49
Textile	22	19-25

TABLE IX
Asbestos in Product and Dust

Product	% Asbestos Content		
	Product	Total Dust	Respirable Dust
Textile	55.15	68	61
Textile	55.00	22	36
Plaster	18.35	15	18
Shale	19.31	25	28
Insulation*	5.15	1	6

* Not included in respirable.

Not included in counts.

analysis is not specific or general enough to form the basis for a threshold for asbestos.

Conclusions

Certain general statements are possible, based on the data presented:

1. There is, at present, no practical alternate method for the routine measurement of exposure to airborne asbestos fibers. The technical difficulty of the electron microscope for fiber counting made its routine use unfeasible when it is used, the results are heavily biased in favor of very small fibers.

2. Each of the methods considered measure something different and, therefore, they do not correlate well. Ratios between results of different methods should be used for qualitative prediction only.

3. Each method is strongly influenced by one attribute of the dust cloud. The biological optimum method cannot be selected un-

less it is known which attribute is biologically most appropriate.

4. The preferred index of asbestos exposure is fibers longer than 5 microns counted on membrane filters at 430 $\times$ phase contrast. The method of counting is convenient and practical, and fibers >5 microns constitute a direct index of asbestos fiber exposure.

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Radiation Publications

The International Commission on Radiation Units and Measurements has recently issued two new reports which are of interest to industrial hygienists dealing with radiation problems. These reports are:

ICRU Report 16: *Linear Energy Transfer*. This report defines the concepts of linear energy transfer and the methods of calculations of distribution of absorbed dose. The applications and limitations of the linear energy transfer concept are also covered. In a series of nine appendices, measurement, range, distribution, dosimetry, and symbols are presented. An extensive bibliography is included. The publication may be purchased for \$3.00 per copy.

ICRU Report 17: *Radiation Dosimetry: X-Rays Generated at Potentials of 5 to 150 kV*. In this report there are discussions of measurement of energy fluence, spectral distribution, measurements and absorbed dose. This publication may be obtained for \$2.50 per copy.

Address orders or inquiries to ICRU Publications, P.O. Box 4369, Washington, D. C. 20003.

Proposed Revision

The American National Standards Institute is proposing to revise the following standard:

Z9.1-1951 *Safety Standard for Ventilation and Operation of Open-Surface Tanks*. This standard covers the protection of operators from contact with gases, vapors, mists, or liquids used or created, related, released or disseminated by open-surface tank operations and the design of ventilating systems for controlling and removing said gas, vapors, or mist. This guide does not cover protection from fire.

Comments relative to the proposed revision of this guide will be welcome at the Board of Standards Review and the comments should be received by November 24, 1970. Copies of the standard may be purchased from the American National Standards Institute, Department BSR, 1100 Broadway, New York, New York, 10003.

— R71-16

EXHIBIT 5

Asbestos as an Urban Air Contaminant

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THE USE of asbestos has increased in amount and variety in the past 30 years to a degree unprecedented in any other common mineral. Asbestos-containing products are today ubiquitous in the home, office or factory, and the street. Few are aware of the extent to which there has been created in recent years in towns and cities what could be called an asbestos environment.

The world's production of asbestos is now over 3,000,000 tons per year, five times that of 20 years ago and equal in amount to the total world production of the ten years from 1920 to 1929. In 1962, 726,000 tons of asbestos were consumed in the United States and more than half of this was used in flat or corrugated roofing materials, pipes, and asphalt or vinyl floor tiles. In contrast, what was the first large-scale industrial use of asbestos, the production of asbestos-containing textiles, utilized only 10,000 tons, while it is estimated that 20,000 tons were incorporated into brake and clutch linings and packings. The great bulk of asbestos used in the United States today as in the world generally, over 90%, is chrysotile, which differs from other varieties of asbestos, among other things, in the greater ease with which it splits into fibers of excessive fineness, down to those which can be measured only in Angström units (Å).

Pulmonary asbestosis was first noticed in workers in asbestos factories mainly concerned in the production of textiles before the introduction of measures to control dust. Since then the condition has been noted in miners of asbestos. Consequent upon the

widespread use of asbestos in a large and increasing variety of manufactured goods, asbestos is now a potential or accepted hazard in an enormous range of occupations related to the manufacture or use of asbestos-containing products, such as workers in asbestos cement factories, the insulating industries, paint and plastic factories, ship building and ship breaking, in the building industry, etc.

Because asbestos is virtually indestructible and so many of the asbestos-containing products are used in towns, the average urban dweller is theoretically exposed to the inhalation of asbestos fibers on a residential basis. The first investigation of the extent to which an ordinary urban dweller is exposed to asbestos fibers was reported by Thomson et al in 1963, who found that 30% of the males and 20% of the females of 500 consecutive autopsies over the age of 15 in Cape

Fig 1—Pseudoasbestos body of linear type. Note the black center. Basal lung smear (unstained, ×900).



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Reprint requests to Medical School, Observatory, Cape Town, Republic of South Africa (Dr. Thomson).

town showed asbestos bodies.

All dusts in finely divided form reaching the lungs are cumulative. High concentrations exist as usual in the atmosphere transport and deposition with carbon in the order of 100 mg/lung. In pulmonary asbestosis, much less is transported but the fine of the short particles is recognizable histologically. The longer asbestos fibers are carried into asbestos bodies, which are yellow globules or a collection of protein and iron, producing a characteristic appearance. Before this happens many sharp asbestos fibers are found towards the lung bases own. The cardiac action, and the forward movement is less rapid and more rapid in the active young. Once a pseudoasbestos body it is usually either, as it is usually too small to be sized or transported by the sharp points are now protein and iron encrusted. It is possible that the inhalation of asbestos fibers over a long period of a lifetime, may result in a concentration at the lung bases. In a pig seem to be the basal asbestos fibers are gradually converted into asbestos bodies. The evidence is that asbestos bodies takes place. No doubt there are variations in the efficiency of this conversion. A careful examination of the lungs in asbestosis can show the oblique illumination of the asbestos fibers that are in the last known exposure. It is estimated that over 100 million years before the first asbestos fibers in the lungs. Asbestos bodies are rare. To evaluate exposure to asbestos, recognition of asbestos bodies at post mortem in the lungs should be both

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of asbestos in a large number of manufactured goods. The potential or accepted hazard range of occupations related to the use of asbestos products, such as workers in asbestos factories, the insulating industry, and plastic factories, ship building, in the building

asbestos is virtually indestructible. In asbestos-containing products in towns, the average urban population is exposed to the asbestos fibers on a residential basis. Investigation of the extent to which an urban dweller is exposed to asbestos was reported by Thomson and Graves, who found that 30% of the females of 500 examined over the age of 15 in Cape

Town showed asbestos bodies in the lung bases.¹

All dusts in finely divided form inhaled into the lungs are cumulative and what focal concentrations exist are usually the result of lymphatic transport and lymphatic blockage, as with carbon in the ordinary urban dweller's lung. In pulmonary asbestosis, however, much less is transported by lymphatics, only some of the short particles, and relatively little is recognizable histologically in the regional lymph nodes, unlike other inhaled dusts. The longer asbestos fibers are converted into asbestos bodies by the deposition of a yellow globules or a complete casing of protein and iron, producing an easily recognized and quite characteristic structure. But before this happens many of the very thin and sharp asbestos fibers move downwards towards the lung bases owing to lung movements, cardiac action, and gravity. This downward movement is likely to be more efficient and more rapid in the healthy lungs of the active young. Once a fiber has become an asbestos body it is unlikely to move further, as it is usually too long to be phagocitized or transported by lymphatics and its sharp points are now blunted by the protein and iron encrustations. It is thus possible that the inhalation of a small number of asbestos fibers over a long period, or indeed a lifetime, may result in a focal concentration at the lung bases. Man and the guinea pig seem to be the only animals in which asbestos fibers are regularly and rapidly converted into asbestos bodies, and all the evidence is that the formation of asbestos bodies takes place only in the lungs.

No doubt there are variations in the speed and efficiency of this conversion. Certainly careful examination of the lungs of cases of severe asbestosis can show by phase-contrast or oblique illumination far more very short bare asbestos fibers than asbestos bodies, even if the last known exposure to asbestos occurred many years before death, though longer asbestos fibers in the length-range of asbestos bodies are rare.

To evaluate exposure to asbestos by the recognition of asbestos bodies in the lung bases at post mortem implies that asbestos bodies should be both characteristic and

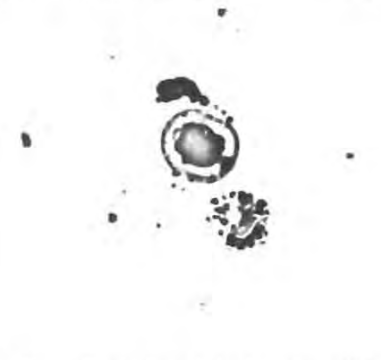


Fig 2—Pseudoasbestos body of round type. Note the black center. Basal lung smear (unstained, $\times 900$).

specific. Four terms are employed in the literature on this subject: asbestos bodies or asbestosis bodies, and "curious bodies" or pseudoasbestos bodies. These four names are used to varying degrees, sometimes misinterpreted, and sometimes misused. It is clear from the findings of Thomson et al that the term "asbestos body" is unjustified and should be discarded, as most lungs containing these bodies, albeit in very small numbers, show no pulmonary asbestosis. To pathologists experienced in this field, the asbestos body is a highly characteristic structure and is not imitated by curious bodies or pseudoasbestos bodies to a degree that leads to any confusion, but some, mainly physicians, radiologists, or chemists writing on asbestosis appear to interpret the term pseudo as approximating to identical. Inasmuch as the formation of the asbestos body results from the deposition of a yellow coating of iron and protein on a very thin asbestos fiber it is not surprising that other inhaled material may on occasions show a similar deposition. What is more surprising is that this deposition seems to occur relatively infrequently around substances other than asbestos fibers. This deposition has been described as occurring around many inhaled materials, but of these only coal dust or a black carbonaceous material is commonly seen with a yellow coating, but it should be stressed that the use of the word commonly is determined by the almost universal fre-



Fig 3—True asbestos body. The fine colorless central asbestos fiber can be seen in places between the segments. Basal lung smear (unstained, $\times 900$).

quency of carbon in the adult human lung, and not by the frequency with which encrustations occur round such carbon-like material. It is mainly in deeply anthracotic lungs that this sort of curious body or pseudoasbestos body is occasionally encountered, but neither in its form nor its color can it possibly be confused with a genuine asbestos body. The center of these curious bodies is of course black. It is usually punctate or else irregularly shaped, and when linear, which it rarely is, it is much thicker than the fine colorless asbestos fiber which lies in the center of a true asbestos body and is visualized with the light microscope only with difficulty. The main exception is that in talc granuloma of the lung small asbestos bodies can be seen, but these are in all probability not pseudo, but genuine asbestos bodies, due to the inhalation of small quantities of tremolite, a true asbestos sometimes associated with talc. In any case if enough talc has been inhaled to show these small asbestos bodies, the lungs are the seat of a pneumoconiosis in which talc particles and the response they evoke dominate the histological picture.

There are statements in the literature on asbestos mainly by people other than pathologists, that the asbestos body is nonspecific. These opinions are not held by histopathologists today. These differences are illustrated in Fig 1, 2, and 3.

It should be stressed that the findings we now report do not indicate the extent of the inhalation of asbestos dust but only indicate the inhalation of asbestos fibers long enough to be converted into recognizable asbestos

bodies and indeed only those fibers which are so converted. The smear method does not permit the detection of bare asbestos fibers. It may well be that the asbestos fiber, when surrounded by a casing of protein and iron as in an asbestos body is converted to a relatively innocuous structure, as in state both the traumatic and the chemical explanations of its mode of action would be inhibited. The fibrogenic and carcinogenic properties may be due to those fibers not converted, or more probably to the very fine fibers visible only in the electron microscope. Whether that view be correct or not, it is at least reasonable to regard the asbestos body as a marker of asbestos inhalation.

The investigation we now report was started in Miami, Fla. in 1961, using the same technique as in the Cape Town series.

Material and Methods

From each lung a smear was made on a 3 x 5 inch (7.5 x 2.5 cm) microscope slide, usually consisting mainly of blood and quite thick. This was air dried, flooded with xylene, and mounted with balsam or balsam substitute in a large cover glass. All smears were made from cut surfaces of the bases of the lower lobes of fresh unfixed lungs. Smears from lungs fixed in formaldehyde are unsuitable for this purpose. The golden-yellow asbestos bodies were readily seen with the low power of the microscope (16 mm objective) as confirmed by the 4 mm objective. Counting was done by low-power fields (LPF) and in those recorded as negative, 300 LPF were examined.

Smears were taken from consecutive autopsies from the Jackson Memorial Hospital, Miami, Fla. on subjects aged 15 years and over to a total of 500. In the autopsy service of the Jackson Memorial Hospital not all the subjects were urban dwellers. Some came from outlying villages and remote districts. Others of course, were retired people coming from more industrialized cities and towns where opportunities of inhalation of asbestos might well be greater than in Miami. We have no figures for these two groups but feel that the positive findings recorded are nearer minimal ones than average ones for urban dwellers in the United States.

We can but reiterate the opinion already expressed¹ that the method used is subject to certain variables, mainly the amount of fluid or blood exudate present in the lung bases. If any of these are in excess, and pulmonary edema and congestion are the more important, fluid from a small number of air vesicles or capillaries will be sufficient to cover the slide and a smaller area of lung will be examined for asbestos bodies. The main constituents in these smears are red blood cells

asbestos bodies showed the same morphology and length as seen in ordinary asbestososis. More were seen in the off-end of an asbestos body was and was ignored in counting, and raises complete asbestos bodies. Exceptionally in lungs showing bacterial growth, the bodies were darker in color, apparently "shining" as seen at autopsy on the internal organs in contact with them. They were otherwise quite typical and the normal yellow color was present.

Results

The Miami series was planned on the Cape Town figures and the findings seem almost identical. The proportion of male to females was of 500 each is the same. The figures being given first: males 196, 194. In Miami, the younger age groups are better represented than half, 275, are aged only 189 in the Cape Town series. In the younger age groups we have a reverse and there are only 189 in the age of 34 or under in the Cape Town series. The findings on the Negro and the findings on the white are discussed separately from the findings in general the occupations are the same. The only variation between the two groups is that Negroes have only 10% more positive cases than whites. The results are given in Table 1. The overall positive findings are similar to those in Cape Town: 29.4% in males and 20.4% in females. The presence of asbestos bodies was by the method used. It is expected there is an increased frequency in the males is no age group. The peak incidence is in the 45-54 age group, slightly less in the next age group, but drops to 29.4% in those over 65. The largest numerical group is the 45-54 age group. The suspicion that asbestos is a hazard of urban living is amply than expected. An estimate of the relative number of asbestos

only those fibers which are detected by the smear method. The detection of bare asbestos fibers by a casing of proteinaceous structure, as in the traumatic and the chemically induced action would be due to those fibers which are probably the very best in the electron microscope. It would be correct or not, it is to regard the asbestos bodies as asbestos inhalation.

ation we now report was in Miami, Fla., in 1961, using the same as in the Cape Town series.

Material and Methods

A smear was made on a 3 x 5 in. microscope slide, usually of blood and quite thick. This was washed with xylene, and mounted on a substitute in a large cover glass. The smears were made from cut surfaces of the lower lobes of fresh unfixed lungs, fixed in formaldehyde for this purpose. The golden-yellow asbestos bodies were readily seen with the low power microscope (16 mm objective) and the 4 mm objective. Counting was over fields (LPF) and in the positive, 300 LPF were examined. The smears were taken from consecutive autopsies at the Memorial Hospital, Miami, Fla., 15 years and over to a total of 500. The subjects were urban dwellers from outlying villages and countries of course, were retired people from industrialized cities and countries of inhalation of asbestos much less than in Miami. We have no field groups but feel that the positive rate is nearer minimal ones than average dwellers in the United States.

We reiterate the opinion already expressed that the method used is subject to criticism. The amount of fluid or blood in the lung bases. If any of these and pulmonary edema and congested, fluid from a small number of capillaries will be sufficient to wash a smaller area of lung will be asbestos bodies. The main constituents are red blood cells.

The asbestos bodies showed the same range of morphology and length as seen in sections of pulmonary asbestosis. More were segmented than unsegmented. What was probably the swollen broken-off end of an asbestos body was occasionally seen and was ignored in counting, and usually in such cases complete asbestos bodies were also present. Exceptionally in lungs showing postmortem analysis and bacterial growth, the asbestos bodies were darker in color, apparently the result of "sloughing" as seen at autopsy on the surfaces of abdominal organs in contact with the colon, but they were otherwise quite typical and bodies retaining the normal yellow color were usually also present.

Results

The Miami series was planned to compare with the Cape Town figures and the material and findings seem almost identical. The proportion of male to females in the two series of 500 each is the same, the Miami figures being given first: males 304, 306, females 196, 194. In Miami, however, the older age groups are better represented and more than half, 275, are aged 65 or over, but only 189 in the Cape Town series. In the younger age groups we have of necessity the reverse and there are only 17 cases in Miami under the age of 34 compared with 70 in the Cape Town series. One quarter were Negro and the findings will not be discussed separately from the Caucasians, in general the occupations are very similar in both. The only variation between the two racial groups is that Negroes have approximately 10% more positive cases than Caucasians. The results are given in Table 1. The overall positive findings are remarkably similar to those in Cape Town, 31.6% of the males and 20.4% of the females showing the presence of asbestos bodies in the lung bases by the method used. As one would expect there is an increased frequency with age, manifest in both sexes but the maximal frequency in the males is not in the oldest age group. The peak incidence of 36.5% positive is in the 45-54 age group and is only slightly less in the next two decades, but drops to 29.4% in those aged 75 and over, the largest numerical group of all.

The suspicion that asbestos fibers are now a hazard of urban living is confirmed more simply than expected. An assessment of the relative number of asbestos bodies is given

in Table 2. It should be stressed that these are quantitative only to the bases of the lungs, and not to the lungs as a whole. In the great majority of the positive cases the bodies were scanty or very scanty. In 18 cases, all males, just under 6% of the males examined, the asbestos bodies were numerous, from more than 1 in 2 LPF to 1 in 2 to 9 LPF. These are likely to be cases in which industrial or occupational exposure to asbestos has been present, and the one example of very frequent asbestos bodies was in a mining engineer who died from bronchial carcinoma and has an asbestosis of classical type. This is supported by the fact that of the 40 positive females, 37 show very scanty asbestos bodies, three show scanty, and in none were they frequent or very frequent. In the 17 males with frequent asbestos bodies no significant pulmonary asbestosis was evident at autopsy, although sections from the lung bases are not available to diagnose or exclude a limited or minimal basal asbestosis.

We have no details as to the occupations of this group but it would seem unlikely that the source of their asbestos was other than occupational in one or other of the many occupations in which asbestos-containing products are made or used. It should be stressed that in spite of frequent asbestos bodies in the lung bases none of this group

TABLE 1—The Frequency of Asbestos Bodies in Basal Lung Smears in Age Groups

Age Groups, Yr	Males	Females	Total
15-24			
Pos	0% (0)	0% (0)	0% (0)
Neg	100% (5)	100% (4)	100% (9)
25-34			
Pos	0% (0)	0% (0)	0% (0)
Neg	100% (3)	100% (5)	100% (8)
35-44			
Pos	14.3% (4)	7.8% (1)	16.1% (5)
Neg	85.7% (24)	92.2% (12)	89.9% (36)
45-54			
Pos	36.6% (15)	6.7% (1)	23.0% (16)
Neg	63.4% (26)	93.3% (12)	77.0% (38)
55-64			
Pos	35.3% (24)	11.4% (4)	27.2% (28)
Neg	64.7% (44)	88.6% (12)	72.8% (56)
65-74			
Pos	33.3% (29)	26.0% (13)	30.6% (42)
Neg	66.7% (56)	74.0% (37)	69.4% (93)
75 and over			
Pos	29.4% (25)	35.7% (20)	31.9% (45)
Neg	70.6% (60)	64.3% (36)	68.1% (96)
Cases examined	304	196	500
Cases positive	31.6% (96)	20.4% (40)	27.2% (136)

Concentration of Asbestos Bodies in Basal Lung Smears

	Male	Female	Total
Only in 81-200 LFF*	64	37	101
Only in 10-30 LFF	14	8	22
Only in 2-9 LFF	17	0	17
Subtotal			
LFF	1	0	1

*rates low-power fields.

primary disability and all died from cancer. The finding of 1 in 17 of the male hospital cases of Miami having frequent asbestos bodies in the lung bases is identical with the findings in the Cape Town males and would seem novel and alarming. No older data are available to determine whether this is a new development or merely the result of the first investigations in this field. It seems reasonable to regard it as a new development, determined by increasing use of asbestos in a wide variety of industries and occupations in the past years, but it must be admitted that the abundant asbestos bodies confined to the lung bases could as readily have been noted at autopsy in the past as they are today. Unless sections for histological examination are taken from the lung bases, and routine ones rarely are, both asbestos fibrosis and the asbestos bodies can easily be missed. In both the Cape Town and Miami series the ages of those with abundant asbestos bodies were similar to those with scanty asbestos bodies. Perhaps equally significant is the large proportion of cases in which fewer asbestos bodies were demonstrated, approximately one-fourth of all the remaining autopsies. Most of these the bodies were scanty or absent. No doubt some of these inhaled asbestos fibers in their occupations but this situation is unlikely in the case of the young, and it would seem reasonable to assume that for the group as a whole occupations were not responsible, leaving us to look to the home and the air of the city as the most possible sources for the asbestos fibers. There is no lack of theoretical possibilities for the modern urban home which usually has asbestos-containing products from the roof to the basement. Asbestos roofing or roof gutters, rainwater pipes, ceilings, and

floor tiles either plastic or asphalt are common enough. No doubt asbestos ceiling tiles, painted and properly maintained are unlikely to discharge asbestos fibers, but asbestos floor tiles even if polished are subject to wear and presumably to the liberation of asbestos. Central heating furnaces and pipes insulated by asbestos are very common in the basements of American homes but are absent in Miami as they are in Cape Town. Asbestos insulation of electrical equipment in the home such as electric irons and stoves is almost universal. We cannot even make a guess as to how many asbestos fibers are inhaled in the average home.

The air of the street would seem a more probable source for the inhalation of these scanty asbestos fibers. The modern automobile is accepted as an important factor in urban air pollution and in addition to its contribution of carbon monoxide and benzopyrene, asbestos fibers may well be an added factor. The average private motor vehicle wears out three or four sets of brake linings and one or two clutch linings in its lifetime, and commercial and public transport vehicles wear out many more. These linings consist largely of asbestos which is ground to dust as the linings wear, and most wear occurs in built-up areas. This alone in most cities would involve the discharge of tons of asbestos dust and fibers in the streets each year. No doubt it could be shown under favorable experimental conditions that very high temperatures can be reached on the surface of brake drums, but we doubt very much if the very high temperatures necessary to fuse asbestos, 1,160°C—1,230°C are ever attained in the ordinary wear of brake or clutch linings in the case of the average driver. The increasing use of brakes of the disc type with their more efficient dispersal of heat are even less likely to attain temperatures of this height in ordinary use. A further contribution is from those car mufflers (silencers) which contain asbestos packing, and from car underbody coating which may contain up to 50% asbestos. The disintegration by natural weathering of asbestos-containing products used in buildings such as roofing materials must result in some asbestos fibers getting into the air

of towns and cities, but at present we have no means of knowing how much are significantly contributors.

It is probable however that the amount available for city air contamination at the moment very small and that the amount of asbestos dust had transferred to human life, but unfortunately, virtually indestructible. Alkalies, acids, oxidation and other means by which materials are removed chemically or physically, and the integration of asbestos-containing materials merely results in the liberation of dust and fibers liable to be inhaled. One assesses the urban hazard from asbestos fibers as a trifling one, but potentially significant one, inevitable that it must become a major hazard in the years. The 3,000,000 tons used annually are added to the 3,000,000 tons used in previous years, and the infinitesimal portion becomes a significant air contaminant in towns, and will increase as more asbestos is used on the surface of the earth or removed by freshwater. It is to be the only effective means of removing this hazard, but whatever is removed in this way, it is likely to be less fixed one and an increasing amount of airborne asbestos would seem inevitable.

The greatest potential hazard is not asbestos fibers of the type seen with the light microscope, but fine that they can be visualized with electron microscope. The asbestos that accounts for over 90% of today's asbestos is chrysotile, and it excels all others in its capacity to produce fibers of excessive fineness, fibers of 200-250 Å. Asbestos of this diameter as an air contaminant is a significant degree now or hereafter constitute a menace again, but it is relatively powerless. Removal by wind or water, or by which dust of ordinary size is removed from the air or by which the particle inhaled the

plastic or asphalt are doubt asbestos ceiling. ily maintained are unlik- estos fibers, but asbest- if polished are subject- nably to the liberation- heating furnaces and pig- estos are very common- American homes but- i as they are in Cape Town- on of electrical equipm- as electric irons and sto- al. We cannot even man- w many asbestos fibers at- erage home.

street would seem a mea- for the inhalation of the- fibers. The modern auto- d as an important factor- ion and in addition to- carbon monoxide and ben- fibers may well be an addi- rage private motor vehi- or four sets of brake lining- clutch linings in its lifetime- and public transport vehic- more. These linings const- as which is ground to dust- ear, and most wear occur- . This alone in most citie- the discharge of tons of- fibers in the streets each- it could be shown under- mental conditions that ver- es can be reached on the- drums, but we doubt ver- y high temperatures neces- estos, 1,160 C—1,230 C are- the ordinary wear of brake- in the case of the average- easing use of brakes of the- their more efficient disper- n less likely to attain the- height in ordinary use. A- tion is from those car mu-) which contain asbestos- on car underbody coating- stain up to 50% asbestos- on by natural weathering- ing products used in build- ofing materials must rest- e fibers getting into the

of towns and cities, but at the moment we have no means of knowing whether these are significantly contributory or not.

It is probable however that the amounts available for city air contamination are at the moment very small and could be ignored if asbestos dust had transient effects or a limited life, but unfortunately asbestos is virtually indestructible. It resists heat, alkalis, acids, oxidation and reduction, and other means by which materials are altered chemically or physically, and mechanical disintegration of asbestos-containing products merely results in the liberation of asbestos dust and fibers liable to be inhaled. Whether one assesses the urban hazard of inhaling asbestos fibers as a trifling matter or as a potentially significant one, it would seem inevitable that it must become greater with the years. The 3,000,000 tons of asbestos used annually are added to the millions of tons used in previous years and even if an infinitesimal portion becomes available as an air contaminant in towns, the actual amount will increase as more asbestos accumulates on the surface of the earth. Wind dispersal or removal by freshwater or saltwater seem to be the only effective means of reducing this hazard, but whatever proportion is removed in this way, it is likely to be a more or less fixed one and an increase in the total amount of airborne asbestos fibers in cities would seem inevitable.

The greatest potential danger of all may be not asbestos fibers of sizes that can be seen with the light microscope, but those so fine that they can be visualized only by the electron microscope. The variety of asbestos that accounts for over 90% of that used today is chrysotile, and this is the type that excels all others in its capacity to split into fibers of excessive fineness, down to a diameter of 200-250 Å. Asbestos fibers of this diameter as an air contaminant in cities of significant degree now or in the future, would constitute a menace against which man would be relatively powerless. Sedimentation, removal by wind or water, or any other means by which dust of ordinary particle size is removed from the air of cities are likely to be relatively ineffective. In general, the finer the particle inhaled the more liable a pneu-

moconiosis is to result. We could even suggest that these very fine particles might adsorb from the air of cities among other things, carcinogens of the benzpyrene type which are known to be present in small quantities in the air of cities.

Miami is a city with a high rainfall yet it is clear that the inhabitants are inhaling asbestos fibers. To give their present significance offers little difficulty. Those that have more than scanty asbestos bodies in the lung bases are the exceptions and presumably their occupations are responsible. In all the other positive cases the bodies are scanty or very scanty and of no significance as a cause of pulmonary fibrosis or disability. If asbestos bodies in these cases are mainly the results of the contamination of the air of cities then such contamination is of no significance at the moment to the middle-aged and elderly, but it may be otherwise to the young with a lifetime's exposure in front of them.

There is today an increasing amount of evidence to suggest that the less intense grades of pulmonary asbestosis and minor—even trivial ones—are more likely to be associated with pleural than with pulmonary changes, with pleural thickenings usually calcified, and with malignant mesothelioma of the pleura or peritoneum.² In the asbestos area of the Northern Cape in South Africa, the main indication of pulmonary disease determined by asbestos in miners or in people living in the vicinity of the asbestos mines, was recognition of the undue frequency of pleural mesothelioma rather than involvement of the lung parenchyma by fibrosis or carcinoma.³ In the same way pleural thickenings evident radiologically because of calcification were reported by Kiviluoto from Finland in people living in the vicinity of asbestos mines, though only 12%-24% of those with pleural plaques had pulmonary fibrosis recognized radiologically.⁴ Similarly, Selikoff et al found in 307 consecutive deaths of asbestos insulation workers four examples of mesothelioma of the pleura and six of the peritoneum, although only 17 cases died from asbestosis.⁵ The fact that only one of the 18 cases with abundant asbestos bodies at autopsy in Miami

had an associated carcinoma of the bronchus and none had mesothelioma is not evidence against that view, as we do not know how long the asbestos fibers had been there. An exposure to asbestos of 20 years or more seems necessary for the production of malignant mesothelioma. If we are correct in assuming that these men inhaled asbestos as a result of its recent widespread use, we can presume that in most of them the asbestos fibers have not been there for a long time. On the basis of industrial exposure in industries using asbestos-containing products as it occurs today, one can foresee both an increase in minimal or basal pulmonary asbestosis and of malignant mesothelioma of the pleura, and with a full autopsy and with a pathologist with an open mind, an even greater increase in frequency of mesothelioma of the peritoneum. Even if there is no increase in the air contamination of cities by asbestos, which would seem unlikely, a young urban dweller will have the exposure of a lifetime available for concentration at the lung bases, unlike most of those over 50 years of age today, who in their earlier years had little opportunity of inhaling asbestos fibers. But to convert the scanty or very scanty bodies which we have demonstrated to be present in so many urban dwellers to the frequency present in a minimal basal asbestosis, would require an increase by hundredfolds, and to get a more diffuse classical asbestosis with pulmonary disability the multiplying factor might well be in many millions. Clearly the latter is well outside the most pessimistic evaluation of the future and only the former is possible. But if to an increasing and cumulative concentration at the lung bases from urban air contamination is added the hazard of small amounts of asbestos inhaled in an increasing number of occupations using asbestos-containing products, then minimal or basal asbestosis and mesothelioma of pleura or peritoneum may become much commoner conditions than they are today, or are accepted to be today. The main unknown is what proportion of cases with a small amount of asbestos in the lung bases or in the adjacent pleura or peritoneum will develop

mesotheliomas if the asbestos is present there for 20 years or more?

The object of this work is to draw attention to some of the results of a relatively new development, the widespread use of asbestos in cities and to stimulate the obtaining of comparable figures from other parts of the world, to establish base lines of urban asbestos inhalation of today and thus enable us to detect significant changes in the future.

Summary

Asbestos bodies were demonstrated in smears from the lung bases in 20% of the females and 30% of the males of 500 consecutive autopsies in subjects of 15 years or over in Miami, Fla. In the great majority of these cases, including all the females, the asbestos bodies were scanty, were not associated with any pulmonary changes, and were regarded as the result of contamination of the urban atmosphere by asbestos.

In 6% of all males examined the asbestos bodies were numerous and were presumably of occupational origin.

It is suggested that both urban air contamination and industrial exposure to asbestos are likely to increase in the future, in view of the increasing consumption and diversity of uses of asbestos and asbestos-containing products and of its virtual indestructibility.

An increase in the frequency of malignant mesothelioma of pleura and peritoneum is forecast rather than pulmonary disease or disability.

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Accepted for publication Jan 31, 196
From the Pathology Institute, Depar
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University, Halifax, Nova Scotia, Can
Reprint requests to 5800 University
Nova Scotia, Canada.

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EXHIBIT 7

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ASBESTOS AIR POLLUTION IN NEW YORK CITY

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Report received 11/3/70

Presented at the Second International Air
Pollution Conference, Washington, D.C.

December 1970

ASBESTOS AIR POLLUTION IN NEW YORK CITY

Abstract

Data will be presented on the levels of asbestos in ambient air in New York City and other nearby areas. This information has relevance to the establishment of air quality criteria for asbestos air pollution.

This report reviews experiences in several major areas:

1. Sampling locations: to provide both background data and surveillance of known or suspect emission sources.
2. Sample collection: instrumentation and materials.
3. Analytical procedures: sample processing including beneficiation, sources of error and range of variation, applicability of optical and electron microscopy.

The reliability and applicability of quantitative results are considered in terms of unique identification of asbestos fiber variety, fiber-fibril size, number and mass of asbestos content, and relation to potential biological significance.

Introduction

Asbestos is the name applied to a group of fibrous minerals able to withstand high temperatures, and relatively resistant to acids or alkalines. The major commercial forms of asbestos are chrysotile with extensive commercial deposits in Canada and the USSR and lesser quantities in the U.S., amosite and crocidolite, each mined in the Union of South Africa, and anthophyllite, mined in many countries including Finland. In the United States, as well as the world, over 90 per cent of commercially used asbestos is chrysotile, the fibrous form of the mineral serpentine.

Over three thousand uses of asbestos have been documented⁽¹⁾ and the list grows yearly. The total U.S. use of asbestos has risen from 156,000 short tons in 1920 to 720,000 short tons in 1965.⁽²⁾ [However, the largest growth in the use of asbestos has occurred in those uses unlikely to greatly contribute to atmospheric pollution. Such "locked-in" uses of asbestos include vinyl floor tile, siding and roof shingles, wallboard, and asbestos cement products. Of course, the emanation into the ambient air could occur during the mining of asbestos, the transportation of the raw fiber, and the various manufacturing operations utilizing asbestos if adequate dust collection is not accomplished.]

Uses of asbestos in friable form with greater potential for environmental pollution include the application to and removal of insulation from pipes and boilers, the mixing and application of asbestos-containing dry cements, erosion from brake linings, and, in recent years, the fireproofing of high rise buildings by a fibrous spray mixture containing from 12 to 30 per cent asbestos. It is this latter use, particularly, that has given rise to extensive dissemination of asbestos into the business and residential community surrounding a construction site. For the past 10 years, during which spray fireproofing came to be extensively used, it was not uncommon to see extensive snowfalls of asbestos-containing material over widespread areas of New York and other metropolitan centers. In some cases, the fireproofing was even done with no attempt to enclose the spray area by tarpaulins or other means.

Asbestos Associated Disease

In addition to the nuisance caused by the spray material, its asbestos content gives rise to serious concern for the health of the people exposed to the airborne fibers. For the past decade, evidence has accumulated indicating that a risk of pleural and peritoneal mesothelioma may exist at levels of asbestos exposure lower than that found in direct occupational exposure. Mesothelioma, a diffuse neoplasm of the lining of the chest or abdomen, is so rare in the general population that it has yet to be separately

classified in the International Classification of Causes of Death. Its occurrence in the general population may be so infrequent as to cause only one death in 10,000.⁽³⁾ However, after occupational exposures to asbestos, it can be the cause of death in one of 10 individuals.⁽⁴⁾ Cases of mesothelioma, in numbers significantly higher than expected, have now been found in workers only indirectly exposed to asbestos;^(5,6) for example, shipyard personnel, and in members of the families of asbestos workers.^(7,8) But of greatest concern for our purposes here is the evidence of risk to people who simply lived in the vicinity of an asbestos mine or factory.^(7,8,9)

Mesothelioma has a long lapsed period between onset of exposure to asbestos and the appearance of disease, in most cases over 30 years.⁽¹⁰⁾ [Since no measurements were made 30 or 40 years ago of ambient air asbestos levels in those circumstances now known to cause disease, we have today no real knowledge of a dose-response relationship for this asbestos-produced disease. Moreover, since the extensive spraying of asbestos-containing fireproofing material has only existed in the construction industry for approximately 10 years, disease caused by the exposure resulting from this practice is 20 years away.] However, continuing the uncontrolled spraying practices of the past decade in the face of mounting evidence of risk to all metropolitan residents would be playing asbestos roulette with the lives of millions of people.

Control of Asbestos Emissions in Spraying of Insulation

New York City and subsequently other municipal and state governments did not wish to wait until the definitive dose-response data were obtained related to the hazard of spray fireproofing. On May 13, 1970, the Department of Air Resources of New York City promulgated stringent regulations ⁽¹¹⁾ controlling the use of all sprayed mineral fibers. Prior to the implementation of required control procedures and continuing thereafter, the Department of Air Resources, in cooperation with the Environmental Sciences Laboratory of Mount Sinai School of Medicine, instituted a program for the sampling and analysis of asbestos in the ambient air of New York City.

[These data are of importance in providing a measure of existing levels of asbestos at sites distant from sources, the burden on the ambient air from known asbestos emission sources, and the effectiveness of the spray fireproofing control procedures instituted by New York City.] Moreover, they will be useful in the establishment of air pollution control criteria by the federal government.

Characteristics of Chrysotile Asbestos

The measurement of the quantity of asbestos in ambient air presents unique problems not faced in the quantitation of other pollutants. Chrysotile in ambient air can range in size from fibers having a diameter of 250 Å and a length of 1000 Å to fibers having diameters up to 10,000 Å and lengths of tenths of millimeters. They are present in a

background of large amounts of fly ash, soot, cellulose fibers, and additional fibrous and particulate mineral matter. The ratio of the mass of this other material to asbestos can be as great as a million to one. Moreover, we would ideally wish to know both the quantity of asbestos present and its size distribution.

Since chrysotile is the major asbestos component of the spray fireproofing mixes used at this time in Manhattan and since it is the dominant asbestos mineral used in most other potential sources, it was the only form determined in these analyses. Moreover, its unique morphology allows its identification by appearance alone by electron microscopy. To ascertain the presence of other asbestos minerals (without unique morphology) would require electron diffraction measurements on each separate fiber present, a laborious task indeed for extensive ambient air measurements.

Chrysotile asbestos is a hydrated sheet silicate having an idealized chemical composition of $\text{Mg}_3(\text{OH})_4(\text{Si}_2\text{O}_5)_{12}$. Alternate layers of magnesium hydroxide (brucite) octahedra and silica tetrahedra form a spiral structure with a hollow central capillary, an outer diameter of from 200 Å to 400 Å, and an arbitrary length. The spiral structure results from a slight mismatch in the dimensions of the structural units of the brucite and silica layers. Excessive curvature would also result in a mismatch. Hence there is a minimum radius of curvature which defines the inner capillary.

Since some curvature is necessary, there is a maximum outer radius for the structure. The unit "fibrils", so formed, loosely bond together into fiber bundles having arbitrary diameters, and lengths up to several inches.

Chrysotile may present a spectrum of appearances in the electron microscope depending on previous mechanical treatment and conditions within the electron microscope. Chrysotile, as received from the mine, with no milling or physical treatment clearly shows the tubular structure of the unit fibrils. After milling, the fibers are shorter, many are broken into fibrils. The outer surface may lose definition and appear amorphous. Greater changes are found in samples having extensive physical and chemical damage or in samples subjected to extensive bombardment in the beam of the electron microscope. In all cases, however, the appearance of chrysotile is unique and can be identified with confidence.

Sampling Procedures

[The Department of Air Resources has established sampling stations for measuring asbestos levels in lower Manhattan, in the vicinity of the construction of high rise office buildings. In addition, periodic samples were taken at 11 of the 38 permanent monitoring stations in all five boroughs of New York. These samples have been supplemented by sampling at other urban, suburban, and rural sites by the staff of Mount Sinai's Environmental Sciences Laboratory.]

Most samples were collected on 8" x 10" Millipore RA membrane filters having an effective pore size of 1.2 microns. The remainder were obtained on 37 mm diameter Millipore AA personal monitors with a pore size of 0.8 microns. While the effective pore size of these filters is larger than the size of many of the asbestos fibers of interest, it has been shown that the surface charge properties of the filter and the asbestos fibers as well as the circuitous path through the filter allow virtually complete collection of all asbestos material.

The collected samples were analyzed by electron microscopy at Mount Sinai. Prior to scanning at 42,000 X magnification, the collected samples were ashed in a low temperature activated oxygen asher for 20 to 30 minutes. This served to remove the membrane filter material, all organic material in the collected sample, soot, and other carbonaceous material. The residue, consisting mostly of fly ash and mineral matter, was dispersed on a microscope slide by grinding in a solution of 1% nitrocellulose in amyl acetate for 2 to 5 minutes. Upon evaporation of the amyl acetate, the dispersal was scanned for uniformity by optical microscopy and a representative area chosen for transfer to an electron microscope grid for scanning.

[During this procedure, chrysotile fiber bundles present are broken into their elementary fibril form. This alteration makes it impossible to accurately record the fiber-fibril ratio in any sample and our results only give in-

formation on the mass of chrysotile present in ambient air. Efforts so far to avoid these alterations have not been very successful. Procedures such as direct transfer of the collected material onto an electron microscope grid by dissolving the membrane filter have been found to often significantly distort the fiber distribution. Moreover, the presence of extraneous matter in urban air severely restricts the ability to scan the grids so prepared. Other methods of direct deposition onto grids, such as electrostatic or thermal precipitation have also been found to give distorted fiber distributions. While some of the above problems can be overcome in the analysis of selected samples, the time required to obtain an accurate size distribution in each sample precludes such determination in routine ambient air scanning.

Typically, six grids were prepared from several ashed samples and two 100 micron X 100 micron squares of each grid scanned and the quantity of chrysotile asbestos determined. The fibers observed per square ranged from a few fibrils to over 100. Background contamination levels in all cases were less than 2. This background level was maintained, however, only by adherence to strict clean room procedures as normal room air provided a ready source of contamination. While random statistical variations occurred in the number of fibers counted per square, greater variations arose from the nonuniformity of prepared films, both between squares of the same grid and squares of grids produced from different portions of the original sample. One

additional source of variation between measurements on samples prepared from different portions of the same filter is the random presence of a few large fiber bundles. These, of course, increase in number in samples taken near construction sites during spray fireproofing operations.

Results of Sampling

Table I shows the distribution of chrysotile asbestos in different locations in New York. The mass values quoted were determined knowing the fraction of the sample dispersed on the electron microscope grid, measuring the volume of each asbestos fibril observed, and multiplying by the appropriate chrysotile density (2.56). While these data are preliminary, they suggest that the chrysotile asbestos levels in Manhattan are greater than elsewhere in New York. This might be expected from the extensive use of asbestos in construction in Manhattan.

To verify that construction sites were indeed a significant source of asbestos fiber, sampling was conducted in lower Manhattan about construction sites where extensive spraying of asbestos-containing fireproofing was taking place. [Table II shows the results of this sampling and demonstrates that spray fireproofing can contribute significantly to asbestos air pollution. In some instances, chrysotile asbestos levels approximately 10 times "background" are observed.]

Sampling was undertaken at several other urban and more rural areas. Results are shown in Table III. The samples in Philadelphia were taken near a construction site during spray operations, stringently controlled. The samples from Ridgewood and Port Allegany were near no obvious source of asbestos emission. While limited, these data do suggest that asbestos air pollution may be found to be ubiquitous, at least in the Northeast United States.

While values ranging from approximately 10 to 200×10^{-9} grams per cubic meter of sampled air may appear to be an exceedingly small quantity of asbestos, it is well to recall that chrysotile easily fragments into ultimate fibrils 200 Å to 400 Å in diameter and often 2000 Å or smaller in length. Thus, 10^{-9} grams of chrysotile asbestos could represent a million fibrils.

The data presented here are only preliminary. The analytic procedures used were semi-quantitative; any given measurement may be accurate only within a factor of 2 or 3. The data do serve to establish, however, the existence of a widespread background contamination of chrysotile asbestos in the ambient air and that chrysotile levels rise to considerably higher values about construction sites in urban areas. Much more information, however, is required before complete definition of asbestos air pollution is obtained. More quantitative data are needed on mass levels in both urban and rural locations. Moreover, fiber size

distributions, both near known emission sources and in the ambient air, will be required.

Table I

Chrysotile Content of Ambient Air in N Y C
Preliminary Results

<u>Sampling Locations</u>	<u>Asbestos air level in 10^{-9} grams/m³</u>
Manhattan	25-60
Bronx	25-28
Brooklyn	19-22
Queens	18-29
Staten Island	11-21

Table II

Chrysotile content of N.Y.C. air in vicinity of spray
fireproofing with asbestos-containing materials

<u>Site</u>	<u>Asbestos level in 10^{-9} grams/m³</u>
1 (downwind from source)	45-180
2 (45° from source)	15-30
3 (upwind from source)	20
4 (downwind from source)	45
5 (upwind from source)	20

Table III

Chrysotile content of air in three selected locations

<u>Location</u>	<u>Asbestos level in 10^{-9} grams/m³</u>
Philadelphia, Pa.	45-100
Ridgewood, N.J. (suburban)	20
Port Allegany, Pa.*	10-30

* We have also found amosite fibers in the air of this community;
a factory using this material is present.

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EXHIBIT 11 (a)

March 7, 1971

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Control who is here; Alderman Shannon, who is immediately to my right.

The first witness that we will call will be Dr. Irving J. Selikoff.

IRVING J. SELIKOFF,
called as a witness herein, testified as follows:

DIRECT EXAMINATION

BY

CHAIRMAN BILANDIC:

Q Will you please state your name and address for the record?

A Yes. My name is Irving J. Selikoff.

I am Professor of Medicine and Professor of Environmental Medicine at the Mount Sinai School of Medicine of the City of New York.

I am Director of its Environmental Sciences Laboratory.

I am past President of the New York Academy of Sciences.

I am Editor and Chief of the Journal on Environmental Research and hold similar positions.

I recount these as an index of my interests and the particular interest related to

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the subject that you are discussing this morning.

Q Doctor, may I pursue your background and qualifications a little further?

Are you a Doctor of Medicine?

A Yes, I am a Doctor of Medicine.

For many years following graduation from medical school, I was interested in chest diseases.

Q Will you tell us about your training and education in the field of medicine?

A Yes. I graduated from Royal Colleges in Scotland. I interned and had residencies at the Seaview Hospital in New York, Mount Sinai Hospital in New York and Beth Israel Hospital in Newark.

I am Visiting Physician at the Seaview Hospital. I am Attending Physician at Mount Sinai Hospital.

I am Consulting Physician at Elmhurst Hospital. I am Consulting Physician at Barnett Memorial Hospital and my interest, before being concerned with environmental diseases, was largely in the field of tuberculosis, where I received, the last year, an award from the American Public Health

Association for research in chemotherapy and the treatment of tuberculosis.

So, my concerns are combined, both of my medical perspective and those of an environmental scientist.

Q Now, I see that you are a fellow of the American College of Chest Physicians.

A Yes.

Q Will you comment on that briefly, Doctor?

A Yes, I am a fellow of the American College of Chest Physicians, which incidentally has its national headquarters here in Chicago.

I am also a fellow of the American Public Health Association.

I am a fellow of the New York Academy of Sciences. I am a fellow of the New Jersey Academy of Medicine. I am currently Governor of the New York Academy of Sciences and currently Trustee of the New Jersey Academy of Medicine.

Q And had you not also been a past President of the American College of Chest Physicians?

A Yes. There are a number of such additional kudos that we pick up during life as we go along.

Q All right.

Now, for the benefit of myself and other members of the Committee and the public that may be attending this meeting, is the admission as a fellow in the American College of Chest Physicians somewhat similar to the admission to say, becoming a fellow in the American College of Surgeons?

A Yes, this is similar.

There are other medical attributes that can be utilized. For example, I am a diplomate of the American Board of Preventive Medicine, but I think perhaps enough has been said on that.

Q The significant point that I am trying to make is that these fellowships in these organizations are by professional achievement, examination and contribution to that particular field of medicine rather than like some other organizations where it is merely a matter of paying dues.

A Yes, sir.

Q That is the point that we wanted to establish.

We know that anybody could become a member of the American Medical Association merely

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by having a license and sending the annual dues in and some other ones, but all of these that you have mentioned where you are a fellow in these various professional associations, these are by contribution to the field of learning in that subject and professional achievement?

A Yes, sir.

Q All right.

Now, Doctor, the next question I want to ask you is relative to any writings that you have done in the field of medicine and particularly writing that you have done on the subject of asbestos in its medical and in its environmental aspects.

A I have written some 60 scientific articles.

I have offered two books. These have been in the areas of chest diseases, cancer of the lungs and asbestos problems.

With regard to the latter, I have edited a monograph entitled "Biological Effects of Asbestos."

I have written some 25 articles on various aspects of health hazards associated with asbestos.

Q Now, are the books that you have written on this subject textbooks which are used in the Mount Sinai School of Medicine or other schools of medicine?

A Yes. The monograph, I think it is fair to say is probably the standard work in this field.

Q That is used in the medical schools throughout the country, generally?

A Throughout the world.

Q Doctor, will you tell us something about asbestos? What is it?

A Asbestos really doesn't exist. It is a general term, a generic word which includes a variety of fibrous minerals. These are substances which in geological time, as the molten rock solidified, had their molecules line up in linear fashion so that they resulted in fibers which were mineral in constitution.

A variety of such minerals exist, the most important of which are known as chrysotile. This constitutes 95 percent of all of the asbestos types that are mined.

Others include amosite, crocidolite,

anthophyllite, tremolite and actinolite.

Q So that I won't have any difficulty with my reporter, who will probably call me later for those names, will you spell those for us, please?

A Chrysotile is spelled c-h-r-y-s-o-t-i-l-e.

Amosite is spelled a-m-o-s-i-t-e.

Crocidolite is spelled c-r-o-c-i-d-o-l-i-t-e. Anthophyllite is spelled a-n-t-h-o-p-h-y-l-l-i-t-e.

Tremolite is spelled t-r-e-m-o-l-i-t-e and actinolite is spelled a-c-t-i-n-o-l-i-t-e.

All of these with their various spellings and unusual names have the common property of being fibrous and mineral and if you can imagine cotton or wool or linen made of mineral, you could almost make a list of the various uses for which these are unique.

They are almost wearproof. They are largely chemicalproof. They are waterproof. They are rockproof.

They have great tensile strength and indeed I suspect that if they did not exist, much of the world's industry would have to be radically changed.

We are accustomed to thinking, for example, that oil is the 20th Century material. It has increased in use some 60 times from the beginning of the century to the present.

It is not commonly realized that the use of asbestos has increased about 1000 times from 1900 to the present, showing how important and how valuable a material this is.

For example, as I walked into this room, going through the halls with tile floors, you look down and you know that you are walking on tiles that are 50 percent asbestos.

The steampipes above us in the ceiling are generally or may be enclosed in insulation composed of about 10 to 15 percent asbestos.

The television cameras probably have the wire in their innards coated with asbestos to be fireproof, et cetera.

So that it is --

Q How about the acoustical ceilings?

A They generally are not asbestos. They are other inorganic materials because not only can we mine inorganic fibers, but we can manufacture them and in

some respects the manufactured ones are even better than those that are mined and in other respects they are not as good, but it is a material which is all about us and essential to many things that we now do.

Q Now, is one of the significant and important uses of asbestos its application to steel beams in the high rise buildings that we have in our urban communities?

A Yes. Because it is a mineral, it doesn't burn and it will not -- not only not burn itself, but it will not support combustion and will indeed fireproof steel structures in our high rise buildings.

This use, although it can be traced back to the early 1930's, really began in the United States in 1958 when the first high rise building was so fireproofed in New York.

Prior to that, other methods of fireproofing were used including encasing the steel beams in concrete, for example, but it was found that the use of sprayed insulation could be done much more rapidly and much more cheaply and in the next 12 years this use of fireproofing materials, many of which contain asbestos, rapidly increased and it is now an important construction technique.

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Q All right.

I think we have now touched upon one of the real significant reasons why the use of asbestos has grown so rapidly.

As you just mentioned, steel beams of the high rise buildings were fireproofed before by encasing them in concrete or otherwise by surrounding them with bricks?

A Yes.

Q Or cement?

A Yes.

Q And some other materials?

A Yes.

Q Now, the builders who are building these buildings higher and higher, and so these other materials were so heavy that you couldn't build a high building structurally with this heavy material?

A Yes.

Q So that they had to find a material that had certain qualities that was light in weight and it was fireproof and it would do this job adequately?

A Yes.

Q And asbestos was the answer to the industry's

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need to be able to build these high buildings, is that a fair statement, Doctor?

A Yes, sir, that is a very fair statement, very accurate and to the point.

Q Now, could you tell us the reason for having to encase these steel members?

Is it because the heat that may be generated because of a fire would cause the steel members to buckle and twist and the building would then collapse?

Would that be the reason?

A Yes, sir, and even if a fire were quite localized, if the heat generated by such a localized fire penetrated the steel to an extent that it would cause it to melt and buckle, the entire building could perhaps come down.

So, fireproofing in every city is of prime concern to the building department, and indeed I presume to the Fire Department.

Q All right.

So would we have this use of asbestos growing to this extent -- could you tell us now, is the United States the single largest consumer of

asbestos?

A Yes, the United States uses approximately 300,000 tons of asbestos each year which is about one-quarter of the entire world's production.

Q Could you tell us something about the amount that may be used in an area somewhat like the City of Chicago?

A I think that knowledgeable representatives of the asbestos industry might perhaps do that better than me.

Q In the field of asbestos use, we have it used in various factories for making, like you indicated, insulation for wire.

We have it used in the manufacture of tile, the manufacture of a lot of other products.

A Yes.

Q Plasterboard?

A Yes.

Q Or if that is the proper term.

A Yes.

Q That has asbestos in it?

A Yes.

Q Now, we do have problems with it at these

asbestos?

A Yes, the United States uses approximately 300,000 tons of asbestos each year which is about one-quarter of the entire world's production.

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Q Or if that is the proper term.

A Yes.

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A Yes.

Q Now, we do have problems with it at these

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least 20 years following the onset of exposure; generally even more, 25, 30 or 35 years. It is a slow process.

In that year, this woman was found to have died of badly scarred lungs, and the question was raised whether her work in an atmosphere heavily laden with asbestos fibers might not have resulted in this death.

In subsequent years, particularly from 1927 to 1931, many more cases of a similar type were found.

Q That was all within the factories where these people worked?

A All within the textile factories and primarily in Great Britain.

We did such surveys here in the United States in Pennsylvania and elsewhere in 1934, 1935 and 1936, and we found exactly the same thing.

We blamed ourselves that we did not or we had not looked at this possibility during the 1920's, for example, but that was water under the bridge and it was rapidly found then that if people were to be exposed in factories to such high

concentrations of dust, that there was a strong likelihood that they would develop very serious and even fatal lung scarring.

This would have been bad enough, but in 1935, a case was reported in South Carolina of a man in one such textile factory who, when he died with asbestosis, scarred lungs -- asbestosis, also had a cancer of the lung.

Now, we might say at this point in 1971: "What was so exciting about reporting a case of cancer of the lung? We see it all about us. We know about it in our families and among our friends, et cetera."

In 1935, however, cancer of the lung was rare and the reason is that it also requires 20, 25 or 30 years from the onset of smoking cigarettes, to the development of lung cancer, and since there were few cigarette smokers in 1910, there weren't many people dying in 1935 of lung cancer.

This has been an epidemic of the '40's, '50's and '60's and the chance of a rare disease such as asbestosis, and a rare disease such

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as cancer of the lung occurring in the same person was considered sufficiently unusual to warrant reporting.

From 1935 to 1955 and 1960, additional such cases were reported and again, almost all in factories of this type. So that by the 1960's, it became fairly well recognized that such occupational exposure to asbestos carried a very significant risk of dying of lung cancer in addition to asbestosis.

Q Now, Doctor, will you please tell us what asbestosis is?

A Right. Asbestosis is the term for scarring of the lungs caused by the inhalation of asbestos.

I might add that in general it takes a fair amount of inhaled asbestos to cause any sufficient scarring to result in disability or death. Small amounts of inhaled asbestos will not generally do this.

The problem of lung cancer may be perhaps summarized by telling you of what is now the case in New York.

I have been studying the Insulation Workers Union members in New York City and at the

N21
present time among the members of this union, one out of every four or five deaths that occurs is due to lung cancer -- it is a disaster!

One out of every four or five deaths in this union is due to lung cancer. It must be --

Q Are these people who work in factories?

A No.

Q Or in other applications?

A No, they work in the construction industry only. Their union is one of the unions in the Building Trades Council and I am sure you have such a Council here in Chicago and indeed the union in New York, Local 12, has a sister union here in Chicago, Local No. 17 of the union, and the proper title of which is the International Association of Heat and Frost Insulators and Asbestos Workers.

However, they are like the factory workers in that over the years, over the many years, they have had rather regular, rather intense exposure to the materials containing asbestos which they use.

In those days, when they began to work in this trade, scientists were not smart enough to have told them that the material that they are using

and the way they are using these materials constituted a possible hazard.

We now know differently and we now tell them differently and we have devised many methods by which they are no longer exposed to such concentrations of dust. There are many new trade practices so we will solve this problem.

Q Doctor, so that we could try to isolate the problem and direct our attention to what may or may not develop to be the more important aspects of the study that we are undertaking here, could you summarize for us the present status of asbestos control within factories?

Is it within reasonable limits? Is it basically corrected?

A No.

Q Or is there something further that we can do?

A There is much more that has to be done, but the responsible segments of the industry are busily engaged in doing it and I am confident that the engineering principles, now being known, I am confident, as I say, that they will be applied and that this problem

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will be solved as an industrial hazard.

There is much more still to be done, especially in some segments of the industry which are still lagging in their efforts, and I think they should be prodded.

Now, I think that such prodding is essential to the health of the people working in these factories, but this problem is solvable and I am confident that more or less rapidly it is going to be solved.

However, this I don't think is the critical area of our discussion.

Q Exactly, and this is what I want to get to.

A Right.

Q So that we have pretty well made great strides and a considerable improvement insofar as, I say, the manufacture of floor tile or the making of plasterboard, which is done within a confined space, so the only problem there is to protect the worker and generally as much as could be done up to this point is being done and a lot more remains to be done.

Insofar as harm to the general public

from those factories, as far as that is concerned, is that reasonably well contained?

A I don't know if it is contained.

There has been very little done to measure whether or not these factories are emitting some of their waste products into the air, but if they are, this too can be readily controlled.

There are measures of appropriate containment which can be applied and with proper stimulation, I am sure will be applied.

Q Then of course, we would have another aspect, and that would be in the mining of asbestos, and that is an entirely different field and an entirely different area?

A And a large part in entirely different countries.

Q And that is being contained in another area?

A Yes.

Q Now, what do you consider the critical area as you were starting to tell us?

A Right. Well, may I have a moment or two to develop this?

Q Certainly. Take all the time you want.

A Because it is a little unusual.

During the 1950's, a second tumor came to be identified with occupational exposure to asbestos.

Let me tell you about the other tumor and spell it because it is an unusual name. It is spelled m-e-s-o-t-h-e-l-i-o-m-a, mesothelioma.

It is a very rare tumor.

You ask how rare? Well, the first modern paper on the subject was written at our hospital, Mount Sinai Hospital in New York in 1931 and two cases were reported.

From 1930 to 1960, we saw three more cases and this is in a 1400-bed hospital doing around 400 autopsies a year.

So as you can appreciate, it has been a very rare tumor. Fortunately, it is rare because it is invariably fatal.

The lining of the chest is called the mesothelium. The lining of the abdomen is called the mesothelium and when a tumor occurs in this lining, it is called mesothelioma and obviously it can't be

localized. It spreads all over, and as I say, it invariably is fatal.

Fortunately, it has also been rare. In the 1950's, a few cases were reported that occurred in men who were working with asbestos.

We now know that men who work with asbestos have an extraordinary risk of getting this fatal tumor.

Again, among the members of the union in Chicago, about one out of every ten deaths is due to this rare tumor.

In the general population, it is one out of 10,000. In New York City, it is around one out of ten also, and again in general, around one out of 10,000, so that we know that among those regularly exposed at work to asbestos, it is a serious risk.

What bothered us, however, was that in 1960, Wagner, a pathologist in South Africa, reported this very unusual tumor in 47 people in five years in South Africa, in one part of South Africa that was noted for its chrysotile mines.

This type of asbestos --

27 Q So that the South African studies have been mine connected?

A No, this has been the problem. Some of the cases did not work in the mines. They were in people who simply lived in the towns in which these mines were existent. They were people whose relatives had worked in the mines, but they themselves had not, but the relatives had come home with this material on their clothes.

There have been young people who, as children, 30 years before, had played on the slag heaps of these mines.

One was a youngster, one as a youngster had ridden to town in a donkey cart in which the bags of material were being brought, et cetera.

This raised the question then whether exposure, other than occupational, might have a similar effect.

It raised a whole new picture. It was or it should not have been a surprise to us because we knew in the United States in the late 1940's that women who washed their clothes, the clothes of their husbands from beryllium factories might get diseases associated

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with the inhalation of beryllium.

Beryllium disease also occurred in people, for example, living within a quarter of a mile of a beryllium plant in Ohio.

So we perhaps should not have been surprised, but we were and our surprise was increased when Newhouse in London, Dr. Newhouse studied 76 cases of mesothelioma at the London Hospital.

She found, to no one's surprise any more, that 31 had worked with asbestos.

Well, this is nothing unusual now because when you work with asbestos, you have a risk of dying of mesothelioma but of the 46 that did not work with asbestos, nine had simply lived in a household of an asbestos worker who came home with dust on his clothes and eleven had simply lived within a half-mile of one of the asbestos plants in London.

This then further strengthened the possibility that environmental contamination with asbestos might be associated with such diseases and there have been similar reports since. Now, such reports in fact were strengthened and then asbestos diseases were found not only in men directly working

with the material, but other people in the same industries as, for example, not only the insulation workers in a shipyard, but the electrician in the shipyard, the steamfitter, the shipfitter, the plumber, et cetera, because they were in the same atmosphere, on the same ship with the man actually using the material.

So that this indirect occupational exposure in the construction industry, in the shipyards, became of concern to us.

This concern had another dimension rapidly introduced and that is that we find when we examine the lungs of people living in large cities, that everybody now has some asbestos in his lung.

Q Fine, Doctor, and that is the point I was going to get to. Now --

A We have examined, if I might interrupt you --

Q All right.

A -- we have examined, at our hospital, 50 consecutive autopsies.

We have taken a piece of lung from each, about that much which would fit on the head of a pin, a tiny amount and in technical parlance, "a tenet

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to the minus sixth of the lung."

We examined these tiny pieces by electron microscopy and in every instance we have found fibers of asbestos to be present in the lungs of these people.

None of these were asbestos workers. These were generally citizens in New York City and in a variety of occupations, none of which had any direct exposure occupationally to asbestos.

Here then we had evidence that in all large cities now, people had asbestos in their lungs in small amounts; nothing compared to what an asbestos worker has in his lungs.

With this knowledge, we then began to look at the air of the City of New York and other areas and we are continuing at this in cooperation with the National Air Pollution Control Administration of the government, and we find that the air in New York City and in other cities which we have looked at, like, oh, Port Allegany, Pennsylvania, Philadelphia, the town I live in, Ridgewood, New Jersey, that the air of all of these places now has asbestos to be found if you look for it carefully enough with the

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electron microscope, but again in very much smaller concentrations than what you would find in any factories.

The question then might properly be asked: If there is dissemination of asbestos from industrial or commercial sites, is there the risk associated with that use of asbestos in the industrial sites disseminated with it?

And this is the important problem with which we are all grappling at this time. We are doing many studies and if you would like a summary of my views at this point --

Q Not at this point, Doctor.

A Fine.

Q Because I would like to straighten out one other problem.

A All right.

Q I understand there has been a study done by the United States Department of Health, Education and Welfare and I think this was just done a few years back, and one of their findings is that from one-fourth to one-half of the general population of large cities, they would have asbestos bodies in their lungs which

N32
were inhaled through the ambient air or through direct exposure.

Would you agree with those figures, one-fourth to one-half of the population?

A In our own studies and indeed the Public Health Service studies to which you refer may be our studies because we are doing this research for Public Health Service; we have found asbestos bodies -- now, mind you, these are not asbestos fibers, but asbestos bodies and they are very peculiar structures that have an unusual appearance.

They are golden brown in color. They have a good deal of iron in their coating and they occur when asbestos has been inhaled by an asbestos worker. We have known this since 1927.

They are so unusual that when you see them on a slide, you immediately point and say: "There is an asbestos body."

These unusual structures, in 1963, were found in 25 percent of all people coming to post-mortem in Capetown, South Africa and here these 500 people were not asbestos workers, they were simply residents of that city.

This has been confirmed in every other city in which it has been looked for, whether it has been London or Pittsburgh or Miami or Milan or Montreal, et cetera. These unusual bodies have been found.

Now, we don't actually know that the central fiber within these bodies is always asbestos. It is a very difficult thing to determine. They look just like what asbestos workers have, but since the other people are not asbestos workers, they could conceivably be something else.

In New York City we have found these structures to be present in 50 percent of 3000 consecutive autopsies that we have studied, again looking at a very small amount of lungs, 175 microns by one centimeter square. We find these easily visible with an ordinary microscope in 50 percent of all people coming to post-mortem in recent years at three hospitals in which this study was done.

They raised the same question as the finding of asbestos fibers in the electronic microscope or with the electronic microscope, rather, that some fibers have asbestos are now present in the lungs of people living in cities in the United States, in all

cities.

Q Now, Doctor, what provoked my interest in this subject is, as I would walk from my office to the City Hall here, I noticed one day where a man was standing on the ground level and he had a large hose and emitting from the nozzle of this hose was a white material and I thought it had been concrete and it was sprayed up at various beams.

I recall it, the one man who had come out with the process of sending plaster up through a hose had gone broke three or four years ago and this process isn't working, so I made some inquiries as to what is that that is going on and someone told me that "They are spraying asbestos."

I noticed that this was during the summer and right here in Chicago, downtown, and there had been small fibers floating through the air and they had been emanating from this construction site, from the man who had been using this hose to apply this asbestos material.

Now, the things that I saw would not be the thing that would cause the harm. The thing that causes the harm is something that is invisible,

is that correct, Doctor?

A Yes. Every time you see fibers, there are many, many more that you don't see, and the very fine ones, the ones that you don't see, are much more likely to be inhaled into the lungs.

Big particles can't enter the deep recesses of the lungs. In general, the very small fibers are the only ones that are fine enough to reach the depths of the lungs.

Q All right.

So that what I had by chance happened to see that particular day, was causing a problem first of all to that man who was working on this job site?

A Did he have a mask on?

Q I didn't notice any mask on him, but he was wearing boots and some type of a uniform.

A But he didn't have a mask on?

Q No, not at all, and the co-workers who were around there obviously were exposed and the rest of the citizens who would be walking through the area would be obviously exposed.

Now, the question is: Would or would

830 not a danger level be reached at some particular point?

A I don't know the answer to that question because we don't know what the danger level is.

We do know that in fact if controls are not instituted and if men are not protected, these men are at a very serious risk.

We also know that people like ourselves have some asbestos in our lungs, are not dying of asbestosis and we are not dying of mesothelioma and we are not dying of those diseases which are generally attributed to asbestos.

So we can presume that there are levels of asbestos which are so low as not to be associated with any diseases.

So, what science has been able to do in these last six or seven years is to define both ends of the spectrum.

Heavy exposure as in factories, construction work, et cetera: Disease if uncontrolled.

Very few fibers in the general public, no evidence, no evident disease of any kind. Now, in between, there are other levels and what these other

levels are that will be associated with some disease less than the factory workers, but still much more than the no disease of the general population at this point, we do not know.

However, it is obviously very prudent that contamination of the environment such as you have described, carries a very serious risk of giving a dosage to a person inhaling it that could be approaching the level of the factory worker, and let me explain that.

These are fibers. They are minerals. Once inhaled, they stay in your lungs for the rest of your life. Moreover, the fibers you inhale today are added to those you inhaled yesterday and the fibers that you are going to inhale tomorrow will be added to both of those so that it is a cumulative effect.

Q All right.

So that isn't something --

A So you may have gotten, walking past this site, enough in five minutes to last you for the rest of your life in a serious way. Therefore, it is clearly important that all such peak exposures

be absolutely prevented.

Q Now, Doctor, this inhaling of asbestos particles would not be eliminated through the body as other foreign bodies that you might inhale or take in in some manner might be?

A Unfortunately, we have no evidence that this happens. Possibly because of the shape of the particle being long and fibrous, we have no evidence of it happening.

It may very well be that some are, and my presumption would be that indeed some of the fibers that we inhale can dissolve and be excreted by the body, but the evidence is that they are not largely removed from the body because we find them there.

If I were to examine the lung of anyone in this room, I will find asbestos present. We find more the older person will have it suggesting that there is a constant admission with each day that we live.

Q Now, one of the other properties of asbestos as I found in some of the reading that I had done, and I see the term referred to as the fact that it has "A

life of one-half of infinity."

Will you describe what that means to us, please?

A That is -- that refers to a phrase that the physicists use, "a half a life of infinity."

A half-life indicates that period of time in which half the amount of radiation, for example, is still present.

If there is a hundred units of radiation and if in three days there is only three units left, we would say it has a half-life of three days.

Now, a half-life of infinity means essentially that it never disappears and it lasts forever, which is one of the reasons that the industry finds it so useful.

Q It is also one of the reasons that the medical authorities find it dangerous where it is exposed to the health as a hazard?

A Yes. I would think it fair to say that the health authorities believe that asbestos is marvelous in the industrial community, but it has no business being in the human lung.

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Q Doctor, there has been some respectable medical authority which indicates that although there have been findings of asbestosis, there have been findings of mesothelioma and other forms of cancer of the lungs and I think mesothelioma is only one of the cancers --

A It is one cancer.

Q -- one of the cancers connected with asbestos?

A Yes.

Q Now, there are numerous others, but this is the most common?

A Right.

Q But these other forms of cancer have never been found except in a case where the victim were -- well, we can call him one that has always been a heavy smoker.

A You are referring to some research done in my department by myself and Dr. Hammond, which is very interesting.

Q I am glad I said "respectable authority," then.

(Laughter.)

A The facts are as follows, and I think you will find them interesting:

In 1963, we examined 370 asbestos insulation workers in New York, all of whom were in the trade for at least 20 years. In other words, these men were now at risk. They were more than 20 years from onset of their work and they had 20 years of dust in their lungs.

When we examined them each was asked about his smoking habits. 87 men never smoked a cigarette. 283 had a history of regular cigarette smoking.

To the present time, of the 87 men who never smoked cigarettes, we have only seen one lung cancer and that is just about what we would have expected out of 87 nonsmokers watched for seven or eight years.

On the other hand, of the 283 men who were cigarette smokers, we would have expected, by this time, about four cancers of the lung because cigarette smokers develop lung cancer.

We have seen not four, we have seen 40, 37 of whom have already died. So that it is not

asbestos by itself because only one developed lung cancer. It is not cigarette smoking by itself because only four would have occurred, but the combination has a very strong multiplying effect and this gives us added concern, because in large cities, three-quarters of all adult males smoke cigarettes and that is another good reason for keeping asbestos out of their lungs.

Q Was there any degree of smoking connected with these patients? In other words, was it light, moderate or heavy?

A The number we looked at, we have followed, is too small to be sure about this.

We haven't published this data, but I can tell you unofficially that the more the person smoked, the greater the risk of dying of lung cancer and if he was an asbestos worker, just as in general, the heavier the smoking, the greater the risk of dying of lung cancer.

Q Now, assuming that the men or the person who is exposed, doesn't get lung cancer. They were nevertheless, even if they don't smoke, they would be subject to asbestosis?

A Yes. We have seen fatal asbestosis in nonsmokers. We have seen mesothelioma, just as much in nonsmokers as in smokers.

The only thing that we have found highly associated with cigarette smoking has been the lung cancer in asbestos workers. This has a special effect.

Q Now, could you tell us how disabling asbestosis is in the nonfatal cases?

A Asbestosis, it may be completely without effect. I may see a fairly bad looking X-ray and yet the person not be short of breath, not be disabled in any way.

On the other hand, we may see X-rays that are not too bad and yet the person be so ill that he cannot walk across the room because of shortness of breath.

We have even seen instances of, and this is when you see it once, you don't want to see it again -- of many who have to go up the stairs backwards. The reason they have to go up the stairs backwards is that every two steps, then they can sit down again and get their breath back.

So that there is a range of disability from practically nothing or nothing, gentlemen, to serious shortness of breath and indeed fatal shortness of breath.

Q Now, Doctor, could you tell us about the controlled study which you had done with the International Association of Heat and Frost Insulators and Asbestos Workers?

A I have already given you much data of this, derived from this study.

We have, in the last four years, been looking at the experience of all members of the International Association of Heat and Frost Insulators and Asbestos Workers in the country; some 18,000 men that we have been following from January 1st of 1967 to the present and this includes some 700 men in Chicago.

We have now studied some 850 deaths among these men and we find exactly the same unhappy experience as New York to be true in the rest of the country.

We find one out of every four or five deaths due to cancer of the lung. One out of every

10,000 due to mesothelioma and one out of every 10,000 due to asbestosis and in fact, one out of every ten being due to cancer of the stomach and colon.

So it is evident then that the regular occupational exposure to this material is very hazardous and indeed is the spur to which industry is responding in developing safe working conditions so that this occupational hazard will be minimized or eliminated.

Q Doctor, has this study that you have performed with these workers been published?

A Yes, it has been published in the Journal of the American Medical Association which is published here in Chicago.

It has been published in the American Journal of Medicine. It has been published in many other various articles pertaining to this study, that is, many other various articles pertaining to this study have been published in many medical journals.

Q Now, Doctor, we talked about working in factories, working in mines, and in the construction industry and then we have touched upon the fact of -- upon the fact that asbestos is practically

N40 indestructible.

Now, there is another area that we see considerably here in Chicago and that is the demolition of large buildings and we know that in the construction of those buildings, asbestos had been used.

Now, since this mineral is non-destructible, could you tell us what, if any, health hazard there may be as a result of the demolition of these buildings?

A This has been inadequately studied to the present. We suspect that during such demolition, fibrous material, asbestos material, will be disseminated into the air of the community, but I would simultaneously point out that we have already begun to look at this problem and I am sure that the ingenuity of our engineers will be capable of encompassing this problem once they look at it, not only asbestos, but there are many other materials, most of which have not been studied at all, which are a part of the air pollution, particulate air pollution, that can come from demolition.

The whole area of demolition, of waste

disposal, is one which I hope your city and our city will look at as rapidly as possible.

There are very few regulations with regard to enclosure, with regard to wetting, with regard to other mechanisms for prevention of dust dissemination.

There are no regulations with regard to waste disposal of these materials. We have written to the 20 largest cities in the United States to ask about their regulations concerning waste disposal of these materials.

We found none had such regulations and it is of interest to note that in New York City, 10 percent of all solid wastes come from the construction industry.

Q Doctor, you have touched upon another very interesting subject and that is in the application of asbestos say in the construction site.

A Yes.

Q There is a certain percentage of waste, a certain amount of that that doesn't stick on the member, it falls to the ground or it goes somewhere.

Now, that obviously has to be disposed of?

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A Yes.

Q Now, that material that is wasted and must be disposed of, that is just as susceptible to cause these conditions of ill being that you have described as the original application, is it not?

A Absolutely.

The waste which contains asbestos or other harmful materials can be disseminated into the air of the community and there be inhaled and have its deleterious effect.

15 Here, too, however, the industrial hygienists, the engineers, the construction experts have been able, in my experience, to develop methods of management of these wastes that can be effective.

Q That is in construction?

A Yes, sir.

Q All right.

So that they would then be sealed somehow?

A The, will put them in a vinyl plastic bag, sealed and then buried.

Q In a land fill area or something of that nature?

A Yes, sir.

Q All right.

Now, suppose that they should inadvertently be incinerated, it would go into the ambient air again?

A That has not been studied because the incineration heat may be sufficient to destroy the asbestos fibers and in that case, they would cause no trouble.

Q Insofar as the demolition sites are concerned, there is no method of sealing this, of disposing and ultimately burying this?

A Yes, there is.

It is inadequately developed so far, but there is. For example, there was in New York last year a building on 43rd Street and Seventh Avenue that was being demolished.

It was completely enclosed in vinyl plastic sheets and all dusts were contained within this complete and total enclosure and contamination did not occur, so it can be done.

Q Now, Doctor, you have pretty well touched upon the background and history and the uses of asbestos

and its medical hazards connected with this particular mineral.

Could you tell us what, if anything, the industry has done since this problem has become recognized?

A The industry is a misnomer.

Any industry is composed of many companies and sometimes they work together, sometimes they don't. There are various segments of the industry itself.

The asbestos industry is perhaps fortunate in having one company that is not only very large, but also very knowledgeable, and in my experience, very mature in its approach to environmental problems.

I refer to the Johns-Manville Corporation, which, having looked at this problem in their own factories, and in the construction industry on the basis of evidence, I presume, that they brought forth, have decided that the best way to approach this problem is not to literally sweep it under the rug or look the other way, but to work out the methods for correction.

If I might summarize their position, as I know it and they may correct me, but as I know it and as I have heard their President state it, and again, this may no longer be the case, or I may be stating it poorly, but it is that wherever the hazard of environmental control, environmental contamination, rather, exists, either in their factories or in the use of their products, that those areas must be controlled.

Where control is impossible, the use must be abandoned and I can well imagine that there will be uses which cannot be well controlled.

For example, somebody clever in the company looked at the slag heaps outside of their mines and said: "Here is a wonderful magnesium silicate dust and there are magnesium poor soils on a number of farms. Let's bag this and sell it in hundred-pound lots to farmers who need magnesium for their soils."

Well, they got a very good production going and it was pointed out that when farmers would take this material and scatter it over the land, they would be creating a great deal of dust and there would be a danger, not only to everybody else on the

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N52
farm, but certainly to the man scattering this soil-enriching agent.

Now, when this was pointed out and how difficult it would be to control said use, the product was taken off of the market.

I presume that wherever a product cannot be used in a controlled fashion, that such a decision will have to be made.

If control is not possible of any environmental hazards, whether it be a radiation or mercury or carbon monoxide or whatever, if control is not possible, then it must not be used where human beings can be in contact with it.

I suspect that most of the things in our environment that create problems can be controlled by proper engineering.

Now, it might not be economical to do it and in that case, it won't be able to be used.

Q The marketplace will be the judge?

A The marketplace will have to be the judge, but economics decide which safe material, which safe process is the one that will be used and I think that the contractors will take the bit up from there.

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Now, insofar as human beings are concerned, insofar as human health is concerned, none of these materials should be used except under a controlled method.

Controls, however, are possible for most everything if we want to put the money in, if we want to put the engineering time and talent in.

I think there is probably hardly a use of asbestos that cannot be controlled if we look to the appropriate engineering.

Q Now, Doctor, lest someone come here later and taint your testimony with some charge of prejudice, would you please let us know what, if any, connection Johns-Manville has with the insulation industry hygiene research program of which you are the Chairman.

A Yes, I am connected with this and this is a very interesting thing. I am a biological scientist as you gather and I am a physician.

Q Also a medical doctor?

A Also a medical doctor, an epidemiologist. I deal with human diseases.

We, as physicians, however, have often

been pointed to by those who criticize us and there are many nowadays who do, --

Q Join the club. We are criticized every day.

A -- that we are much more interested in rat bites than in doing something about the rats. You know, we like to do research and find out the cause and then we are not interested any more, they say.

Now, in part, this criticism is sometimes justified. When it came to the question of what was happening to our insulation workers, their dying, their being seriously ill, it was rapidly evident that we ought to do something about it and in fact, if we didn't think so, the union thought so, and they came tugging at my elbow and said: "Now, what can we do now that you have identified the risk that we have, the way our men are dying? What can we do to remedy this?"

We said: "Well, we will hire engineers and we will hire industrial hygienists and we will get the equipment either to see whether we can work out methods to use these materials safely; working conditions that will then not be a risk to you."

And we set the insulation industry

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hygiene research program up, and this is now jointly financed by the industry, Johns-Manville and now the contractors in the rest of the country and the union.

Each member of this union, out of his own pocket, took out \$10 and made a fund of almost \$200,000; actually \$180,000, for us to hire the engineers, to hire the physicists, to hire the mineralogists, et cetera, necessary to work out the remedies for this, but this is only one trade, only among the insulation workers in the construction industry and in the shipyards.

This is excellent, however, because it means that this trade, which formerly was a risk not only to themselves, but to other people in the construction industry, will now have safe working conditions, which will safeguard the men actually working in the trade as well as other people working around them.

I would hope that similar programs would be instituted for every asbestos use that we now have.

I understand that the companies do this themselves inside of their own factories. I am

not one that goes inside of their factories, but I presume that they are doing this.

Here, I think you people and by "you people," I mean the administration, politicians, government people, have a special role to play.

I think you are the key to the whole problem, if I may be so bold as to say this.

Q I hope we are the key to the answer.

A I think you are. I hope so, anyhow.

Certainly, if you can't do it, it is not going to be done in this regard.

A safe environment is expensive. It will cost money for any company to use proper precautions to safeguard their working people and to safeguard the community at large, not only -- not to contaminate the community at large.

Now, they have got to put proper exhausts in. They have got to put bag houses in. They have got to change working practices and they have got to do all kinds of things, and it is expensive. One company by itself can't do it.

If one company does it, they wear the white hats and the other companies do not; they

wear the black hats and soon the white hats are going to be out of business, that is, in a short time. They won't be able to afford it and they won't go out of business, what they will do is stop taking precautions because it is too expensive, and therefore, you people have to make sure that there are regulations, that there are laws which insure that everybody does it the same way, that everybody has to bear the expense according to the work he does and according to his business to protect the community.

If you don't insure that everybody does it, nobody is going to do it and the community at large will suffer.

So, the ultimate responsibility really is yours. Our job as scientists is to define what the hazard is, perhaps even work, perhaps even to work out some of the answers. The engineers will work out many more answers than we will.

Industry has got to do the engineering research to show that it is practical and feasible, economically feasible, but the government has to work with us to see that what is found out is actually put

into practice by everybody in the industry and not just a few that we are able to convince that they have a responsibility.

Q Well, Doctor, I certainly must say that it is to your credit and obviously also to the credit of Johns-Manville, if you speak in their behalf, as you may be expected to do, when they recognize that there is a harm and that they will voluntarily withdraw their product or that portion of their product from the marketplace.

I do hope that they support you in that position. That is where no controls are applicable.

A Yes.

Q Now, getting back to your research program that you have that is subsidized by the union membership and by industry and by contractors: Now, some of the things that have come out of there, could you talk about, and I believe, like the respirator protective devices which have come out of there.

Could you comment to some extent on that?

A Yes.

Q And state whether they are adequate or

inadequate.

A I am not very proud of our work in respiratory devices even though it has been very effective and let me tell you why.

I wrote a letter to every insulation worker in the country and asked him whether he wore a mask when there was a lot of dust around.

6 I found to my horror that when they recognized that it was very dusty, only 4 percent wore a mask. Only 4 percent would wear a mask when they knew it was dusty and if they didn't think it was dusty, if they couldn't see the fine fibers, they never wore a mask and when we found out why, it was because the masks were uncomfortable; not that they weren't effective or that the employer wouldn't give it to them if they asked for it, but that they were uncomfortable.

They stated that if they were on a scaffold, the mask was so big that they couldn't see and they could fall off and kill themselves or else they couldn't wear their glasses or else they couldn't talk to the other men working with them and so forth.

We then set up a team to design a mask

not that it would be any more effective because it is easy to design an effective mask, but we wanted to design a mask that was effective and also comfortable at the same time.

We have it now. We have a mask and it will be on the market very shortly. Three companies are making it and it will be both effective and comfortable.

Now, I don't consider that a great achievement because the real achievement should be in the working conditions where a man doesn't have to wear a mask. That is what we have got to do and we have done much work also, for example, in Chicago, in New York and all over, an asbestos worker will often take asbestos cement, pour it out, mix it with water and then use it.

Well, when he pours it out, the whole area becomes dusty. Now, this has been going on for 50 years. When the engineers looked at it, they scratched their heads and said, "Well, this is absurd. Why deliver the cement in a paper bag. Let's deliver it in a plastic bag with a little hole on top and you put the pipe with water in through the hole and mix it

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inside of the plastic bag so there will be no more dust."

Well, we have removed therefore, a major source of the dusty conditions of the trade.

Q All right.

Now, this is another new development that has come out of your research program?

A Yes.

Q And this is comparatively new, is it not?

A Yes, within the past year-and-a-half or the past year, for example, we have visited all over the country, including Chicago, the fabricating shops in the insulation industry and when we went in, every one of the shops had a big table saw to cut these products into blocks for pipe covering, et cetera.

We looked at the table saws and they were creating huge clouds of dust in the room and we found out that out of the 22 companies that made table saws in the United States, not one made one with an attachment where you could attach a dust collector.

So we asked the manufacturers, "Would you be interested in designing a table saw with a dust collector?"

Two out of the 22 said they were interested. We then found out who made the dust collectors. We gave the manufacturers of the dust collectors telephone number and we gave the dust collectors the manufacturers telephone number and they got together and in six months we have table saws with built-in dust collectors and when we did before and after measurements, we found that the amount of dust was reduced 10,000 times what it was before.

Now, new information was needed. All we had to do is look at the things and the engineers are good at that and all we have to do is take them to a building or to a building site and there they see what has to be done.

Now, that is why I am so confident that these problems can be controlled.

Q Now, what you were referring to there was asbestos dust?

A Yes.

Q All right.

Now, this is another problem, sawing the sheet materials on job sites.

A Yes.

Q All right.

Now, Doctor, as I understand it, there are two methods of applying asbestos to these steel beams at construction sites.

One is referred to as a dry method and another as a wet method.

Now, will you tell us what the differences are?

A Well, I will tell you what my inexperienced knowledge in the case is. I am sure you have experts here who might amplify and change it perhaps.

The material that is sprayed onto fireproofing members -- that is, to fireproof the steel girders, for example, consists of anywhere from 10 percent to 20 percent asbestos, about 75 percent of various extruded silicates like rock wool and generally some wetting agents, some clays like bentonite, et cetera.

These are mixed dry in a large mixer and come out of one end of one nozzle and then water comes out of the other nozzle and it mixes, oh, perhaps ten inches from the openings of these two tubes and when

it hits the steel girders it is wet.

Unfortunately, in this process --

Q Is this the wet now, or the dry?

A That is the dry method.

Q All right.

So even the dry method has some liquid application so that it will stick?

A Oh, yes, it is always wet when it hits and most of it sticks.

Q Fine.

A The industry tells me only 3 percent becomes waste and on some jobs, it looks more like 20 percent, but they insist it is only 3 percent.

Q Is this dry process also known as the fibrous application?

A They are all fibrous applications. They are all fibrous.

Q All right.

A The other way is to mix it beforehand with water, and when it comes out of the nozzle, it is wet, just coming through the one hole and our studies indicate that the air in such applications has fewer fibers in it than with the dry method.

In Great Britain, they tend to use the wet method much more than here. Some of the industry people have told me, from an engineering point of view, that the wet method is very difficult.

The wet method has its own problems by the way it gets on the floor, and once it gets onto the floor, it is very hard to get off.

Q And it still has asbestos in it?

A It still does.

Q Fine. Now, Doctor, could you tell us what a cementitious mixer is?

A That is one that has the wet method application. It has cement products and fibers in it and the fibers may or may not be of asbestos, plus other extruded silicates like rock wool.

Q So that it is possible then to have a cementitious mix which contains no asbestos whatsoever?

A Yes, sir, there can be also other fibrous mixes that have no asbestos.

Asbestos has wonderful properties that are not always present in some of the other formulations, but I presume that the sprayed mineral fiber industry has great ingenuity and will develop all kinds of

N66
formulations and so that they will have all kinds of properties so that they can get the properties in there that are desirable.

I would suggest that in all cases appropriate control measures be taken because the other fibers are of unknown quantity, they are of unknown biological effect. Some of them have never been studied in human beings, and therefore, I would suggest that no inorganic particle or particulate matter be used in such a way as to allow it to enter the city's air and potentially enter the human lung.

Q Doctor, we won't be operating in a vacuum here because as I understand it, all of these materials, whether they be dry or whether they be wet or whether they be a cementitious mix or whatever other categories are used, they are all classified in some manual that is published by Underwriters' Laboratories as being safe from the standpoint of the fire hazards?

A I understand that there is such a manual, yes.

Q And all of these are classified and are published and are rated?

A For fireproof, for their fireproofing

qualities.

Q Right?

A Right.

Q Now, Doctor, has your program that you have worked on, also developed any other techniques such as apparel that would be worn other than respiratory devices?

A We are looking at this now. We are aware of research in this regard in Great Britain, and we have utilized what they have done there.

The Cape Asbestos Company in Great Britain, which has the same approach to this problem as does Johns-Manville, has done research in such apparel.

Some materials have the dust cling to them much more than others, and we don't think they are very suitable. Indeed, there are now available disposable overhalls which would then be discarded at the end of the day and not subject the workman to the risk of taking them home, taking home this dust to his family on his clothes.

Q All right.

Doctor, other than the application on

the steel beams, is there also a problem with application of asbestos in plenums involving air conditioning and heating?

A Yes. These plenums or the under surface of floors, et cetera, are a part, very often, of the open spaces of the buildings between the floors.

It is now not an uncommon practice to use these open spaces as return air or collecting plenums from which the air in some instances is recirculated back through the entire building.

Q And is that a health hazard?

A I consider this an important potential hazard in that the fibers or loose material from that surface is being contaminated and recirculated with the air through the building and people who have to be in these buildings eight hours a day or more would then be breathing this contaminated air.

Now, I don't know whether you do that in this building, but if you do, then we are sitting in this room and we would be breathing air that has passed through spaces that are lined and coated with asbestos and the potential for the entrainment of fibers into the airstream which would then subject

B6
all of us to the risk of inhaling these fibers.

Q But is there a method of application in these plenums that is safe or is there some recommendation that you can make in that regard?

A Recommendations have been made to seal these surfaces with various sealants.

I have yet to see evidence that this is effective, and I do not consider that until this is very carefully and definitely and clearly demonstrated that air in public buildings should be passed across surfaces from which they can take fibers and so become contaminated.

Q So this is a serious problem which we should address ourselves to, particularly in urban areas?

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A I consider it very potentially serious. We have not demonstrated that you or I would have any health hazard from it, but we will have to watch you and me for 25 years before we can tell.

Q Now, insofar as the application at the construction site is concerned, are there enclosures laid out and has your research program come up with any enclosures?

A Yes.

It is possible to enclose areas in which asbestos is being used, even sprayed, which will prevent the visible emission of fibers from that area.

These have been, in the past year, designed and introduced in the New York construction industry. I understand that they are quite expensive, but nevertheless we have also found them quite effective.

We have done dust counts outside of the construction sites in which they are being used and we have that they are indeed effective in preventing the emission of particulate matter to the outside air.

Q Now, Doctor, isn't that a serious problem where under a lot of our new schools or architecture we have exposed columns and exposed beams.

Now, if you are going to fireproof exposed beams, it would be very difficult, if not impossible, to enclose a spraying process on the outside of a building.

A Oh, I don't think that -- well, speaking as an amateur engineer now, I don't think that you can have fireproofing of the sprayed variety and leave it exposed on a beam.

I don't think that the architects would design it this way.

Q I see. So that they would either have to leave the beam exposed or they would have to provide some other type of covering?

A Sheathing.

Q Either by concrete or --

A Steel, brick, aluminum.

The World Trade Center, for example, has an aluminum sheathing on top of the fireproofing that has gone on to the steel beams.

Q Doctor, I know that you were instrumental

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in having some interim order entered in the City of New York and I believe it became effective on April 13th of 1970.

Could you tell us what, if any, experience has had with that interim order?

A Well, I cannot speak for the Department of Air Resources.

Q Just give us your idea.

A But speaking of what I do know about it.

Q Yes.

A A series of regulations based upon engineering studies and experience were developed in which it was considered that spraying could be done and that these would effectively prevent contamination of the City's air.

I understand, too, and herethis is primarily scuttlebutt, that the sprayed insulation industry didn't find in many instances, it convenient to follow these regulations and as a result, they were closed down.

Q In other words, to put it bluntly, the New York experiment has been a failure?

A It was at that point, but from another

point of view, in those instances in which these regulations were follows, we in our scientific measurements found that they were effective.

Now, the industry in New York, then, to my mind has the following choice.

If it spends the money to utilize this technique safely, they can do it and not cause contamination or air pollution.

If they do not spend the money or if they have no desire to go through these difficulties, in addition to the money, then they will have a situation on their hands in which any self-respecting control and regulating organization will not allow it to continue.

Q Well, as I understand, this interim order is still in effect?

A Yes, sir.

Q And I also understand that an ordinance was introduced in the New York City Council last November which, to some extent, adopted or actually expanded to some degree on the interim order.

Could you tell us the status of that particular legislation at this time?

A Yes. The interim order No. 437 was introduced on the 30th of November, 1970.

Q As an ordinance?

A It is now under discussion.

Q In the City Council?

A In the City Council.

The City Council is now holding hearings on it. It is part of the administrative code for air pollution control and this order bans the use of asbestos as a spray material presumably because of their experience with the lethargy of the spraying contractors to utilize the controls, the rigorous controls that they had advised and regulated in April.

Q Well, this is very significant, Doctor?

A Yes.

On the other hand --

Q They started out with an interim order to control and then in April of 1970 that was, and then in November an ordinance is introduced to ban.

A Yes, sir.

Q This is a drastic change of position.

A Yes, sir. I would think that part of this,

at least, is due to the fact that they did not find that the control measures were being obeyed. At least this is what they tell me. The same control measures, however, and they are very vigorous and very detailed, are required for the spraying of any insulation material whatsoever, whatever its nature, whatever its composition, and my guess is that if it is not obeyed for other materials, they too will be banned.

In other words, the City of New York is saying that:

"You cannot spray inorganic particles into the air of the City of New York. We don't want to contaminate the air of the City of New York.

"If you do not follow these guidelines and regulations, you will not be able to ply your trade in our City."

I would think on the other hand, from my knowledge, that if these regulations are followed, then any material can be safely used because that use will be controlled.

If the controls and regulations are not followed, then these uses should be eliminated.

Q Doctor, could you tell us what, if anything,

7-6
has taken place in Philadelphia in connection with the problem of control or banning of asbestos?

A Very much the same experience in New York has been found in Philadelphia where control measures were originally required.

Now, these were not very well followed, and as a result, the spraying has been eliminated, presumably, and here you would have to have the Philadelphia people speak for themselves -- presumably on the theory that if controls are not obeyed, then you can't do it at all.

Q Well, as I understand it, the status in Philadelphia at this point is that they have promulgated an order which has been published and has not yet been acted upon?

A That is correct, sir.

Q And so that there is no effective legislation anywhere?

A One way or the other, right.

Q Just interim proposals?

A That is right.

All of us are looking at this now and the same is true in San Francisco.

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Q And Philadelphia is considering banning asbestos?

A Yes. And I think that every city will consider banning where the control measures are not obeyed.

I don't think we would allow cars on the street if the drivers didn't obey traffic lights.

Q All right. Now, Doctor, you mentioned San Francisco and you anticipated my next question.

Could you tell us what is the status of this type of program in San Francisco?

A Yes. They have given guidelines about, or concerning how many fibers per cubic meter of air or per CC of air they would allow at the point of spraying, 100 feet away, down the road, etc.

I don't think they are very practical. It would require 50 inspectors constantly with machines in their hands at all four corners of the compass to monitor these areas.

I think a much more preferable approach is that utilized by New York, Philadelphia and some other cities, and Boston is now looking at this, which would lay down the way it should be done so that we

won't have dust counts all day long.

Q Well, Doctor, in your experience in the studies of the medical aspects and in your experience in researching and working with industry and municipalities and attempting to control the health hazards, we would reasonably conclude that there are three areas that we must look into.

No. 1: The worker who is involved and who is exposed to this asbestos material quite a bit because of his occupation.

Secondly: The job site where these things take place; and,

Thirdly: The general public, whether it be people who are within a building like this or where we know about the return air plenums, or whether it be a pedestrian on the street who is exposed.

Now, could you tell us what in your opinion would be the best approach that we, as a City Council, could take in protecting all of the segments of the people of the City of Chicago?

A Yes. I have a suggestion, and that is that you -- fortunately here you have the great skills and knowledge of Dr. Brown with his vast experience

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in the Bureau of Occupational Health, Division of Occupational Health of the Public Health Service, so he knows this material and has all of the intimate details at his fingertips so that you are way ahead of the game in that regard.

The important perspective is control at the source where at the point of utilization of any asbestos-containing material you need the control because if we control the use at the source we will protect the workmen engaged in using this material. We will protect all others who could be indirectly exposed at the construction site or in the factory, etc. And certainly we will protect the community at large.

Therefore, all use of asbestos in Chicago should be identified and each use should then be studied to determine whether control at the source is being accomplished.

In essence, what I am suggesting is that you trace every pound of asbestos that comes into your city, where it is warehoused, how it is transported around the city, what kind of use is made of it, what industries use it, and in each instance,

each in a way and we could have a joint source and be sure that controls are adequate at that point source, and we will then protect all these groups who could be at risk.

Q When you say controls are adequate; in some areas the scientific community had not come up with adequate controls.

A Where controls do not now exist then the use should be abandoned unless the industry comes up with these controls pre-emptively and when you put it in them in that way, their engineering talents will come up with the controls that are necessary.

Where they find on the basis of an engineering investigation that they cannot control that use, then that use should be abandoned.

Q All right.

So then, Doctor, you do not recommend a ban such as is being considered in New York and Philadelphia.

You recommend a more moderate approach. First an attempt at control and in areas where we can control, a ban outright.

A Yes, sir. I think this has been my

experience with all environmental hazards.

We know that radiation can be a serious hazard, yet we don't ban X-ray machines in hospitals. We investigate them to make sure that there are no leaks or undue exposure, etc.

We know that carbon monoxide, for example, in some parking garages can reach serious levels, yet we don't ban parking garages nor the use of cars. We require adequate ventilation in parking garages, many cities do at any rate.

In material after material we find that society approaches environmental hazards by requiring that they be controlled and that banning not be considered unless controls cannot be accomplished.

Q And also would you consider, from an economic point of view, that it is better not to ban any useful material so long as it could be properly utilized and not cause any health hazards?

A I think this is implicit in not only everything I say, but in actually the way we act.

For example, in the New York City Code, to which you have referred, there is, in addition to a phrase "banning especially in spraying," another

section which says that asbestos can be used in brake lining of motor vehicles during normal use.

Q All right, fine.

A Because they are not prepared to ask us to do without automobiles.

Q Very good.

Now, Doctor, you touched on another subject that I had asked a question about of many people.

We all know that brake linings are made of asbestos because they can withstand the heat very well.

Now, could you tell us how serious an effect that is on the environment of a city like Chicago, for example, considering the amount of asbestos that may emanate into the ambient air as a result of the use of the brakes on automobiles?

A This is not yet known. We are studying this.

We know that much of the asbestos in brake lining is destroyed during the application of the brakes for a great deal of heat is produced and destroyed the asbestos. There is still some left, however.

The magnitude of this is not yet know and is under study.

The use of asbestos in brake linings is not in terms of total tonnage, but what are the major uses of the material.

Q And is NAPCA handling this investigation?

A Yes, they are doing research, we are doing research for NAPCA on this subject at this time.

Q The most serious problem that we should address ourselves to should be what, Doctor?

A The most serious problems are the identification of those uses of asbestos which have the greatest potential for becoming airborne, and here I would think that the most important is the spraying of the asbestos in fireproofing, which is actually spraying the material into the air.

Q All right.

So that the most important thing, then, would be the control of application on construction sites?

A Yes, sir. Three-quarters of all asbestos used in this country is used in the construction industry.

Q All right. So if we addressed ourselves to that problem, first we would be making a major contribution?

A Yes.

Q No. 2, would you rank demolition of buildings as the second most important?

A As part of the construction problem, the other phase of it, yes, sir.

Q What would you rank as the third most important thing that we should address ourselves to?

A The industrial uses, including warehouses and discharge of waste materials.

Q Both from construction sites and from these industrial uses?

A Yes.

Q There are no regulations at the present time with regard to the handling of wastes that contain asbestos.

A Right.

Q Would there be any other major significant areas in which we could go?

A I think that if these were controlled, all the others would become trivial.

Q Now, Doctor, you made reference to Dr. Brown a little earlier.

I was going to ask you about this before the meeting had concluded, and I had noted in some of my reading, in some of the research that we have done on this subject, the name of Dr. Brown has appeared quite a number of times, and I had seen where he had participated or was participating in some symposiums and studies which you had conducted.

I know he is too modest a man to ask him these questions, but would you tell us something about his ability and qualifications and the assistance that he can give to us in this area?

A His ability and qualifications I am sure you are able to judge very well, and I think anyone who comes in contact with him rapidly gets to know that these are self-evident, but I could tell you something a little bit behind the scenes that may be of interest.

Q That is what we would like to know.

(Laughter.)

A In 1964-1965, when Dr. Brown directed the Division of Occupational Health for the Public Health

Service in those years, he saw to it that almost for the first time that funds were made available to scientists in this country to study the asbestos problem before we had all the information that I have told you where we only had hints, where we began to suspect something was going on, and he looked at the question and he was perceptive enough to know and to say and to make the decision that they should look into this problem immediately because of its importance, the important potential for possible human disease.

So he has been one of the figures in this country who have provided the background for what science is now able to tell you in your deliberations on this problem.

We have him, basically, to thank for much of what we now know.

CHAIRMAN BILANDIC: Are there any questions by any other members of the Committee?

ALDERMAN GABINSKI: Yes.

CHAIRMAN BILANDIC: Alderman Gabinski.

EXAMINATION

BY

ALDERMAN GABINSKI:

Q You discussed earlier a scale by which you looked at asbestos and you described your technique as taking a small part of a lung, examining that with an electron microscope and determining whether or not there was asbestos there, and then you used the other end of the scale where you had large quantities of asbestos in a person's lungs as a result of maybe being a worker.

There is a tremendous gap between the two, as you have described.

Is there any study or are there any possibilities in the near future that we might find new levels?

Are there any techniques available where we might be able to place a person's condition somewhere in between because actually, when we are talking about two extremes, we are very far from any answers regarding just how dangerous a person's condition is.

The reason I ask this is because living

in Chicago you are going to find everything in everyone's lungs. In my lungs I am sure you will find something of everything there.

When we are talking about controls, when we are talking about the seriousness of a problem and when we have two extremes, I am concerned about the fact that we have no in-between. There is no guide, no judgement.

We are really guessing.

A Well, you have touched -- what you have touched on is the critical area, to be able to determine at what level do human beings show disease, the lowest possible level, and at what level do we begin to fail to show any disease.

We are doing such studies now and I can tell you very briefly what they are, and I hope in about two years we will be able to answer the important question you have asked.

We have made a list of every person who lived with an asbestos worker in New York City in 1942, and we are now tracing these 2,000 people.

We know how old they were in 1942, so we can cultivate -- that if we can calculate what their

experience should be by now.

We are tracing these people to see what level of risk there is in living with an asbestos worker; whether one out of every ten is going to die or one out of every thousand.

We are looking at all the tile setters, the ceramic tile setters working in the New York City construction industry in the same buildings as the insulation workers, but not working near them, but still in the same building.

This is some exposure, but no where near as much as really working with the tools.

We are examining all of these people to see whether they have a serious risk.

We have made a list of everybody who lived within a half a mile of one asbestos plant in Patterson, New Jersey in 1942, and some 2,000 people lived within a half mile, 2,000 adult males lived within a half mile of this plant in 1942, and we are now tracing all of these people.

We know we are able to calculate with biostatistics what their fate should have been if they hadn't lived near an asbestos plant, and we are now

tracing them to see actually what did happen to them.

We are looking at the mortality experience of all of the roofers in the United States. They use asbestos containing materials, the roofing felts, but generally not enough to become airborne very much.

We are looking at what has happened to 20,000 roofers in the United States from 1959 to 1973 to determine whether such exposure carries a risk and from such studies, we will be able to tell you, I think, in around 1973, whether there is a risk between the two ends of the spectrum.

Q One other thing --

A It is a great deal of work.

Q I am glad to see that the studies are so very extensive because the problem is so difficult to solve. I can see the great lengths you will have to go to.

Q In the area of demolition the question comes to my mind, and I can see the possibility of some control over demolition.

I didn't hear the very first few

minutes of your testimony.

Did you mention when asbestos first -- when they started to use asbestos in buildings? What year was that?

A As an insulation material, during the first decade of this century.

Fred, am I about right on that?

DR. PUNDSACK: Yes.

THE WITNESS: About 1905, from what the union people tell me, and there are still some men who worked in 1905 who are alive now, and they told me when they went over from wool felt to asbestos-containing paper, and asbestos-containing block, that was about the time they did.

ALDERMAN GABINSKI: Q My question would be related to a situation where maybe a lot of buildings that are being torn down in my area now don't contain asbestos, so in your opinion is the mere demolition of the buildings, the dust producing situation, as dangerous, or would it need the control that a building would having asbestos?

You see, I am thinking about a lot of poor people, and now if you are going to demolish

3-13
the building and use these controls, of course, the cost of demolition is going to rise, which is a burden on the homeowner.

Now, assuming that there is no asbestos in this building, which many of them are very old in there --

CHAIRMAN BILANDIC: I think I can anticipate the answer to that and our judgement would be --

ALDERMAN GABINSKI: Q No, I want to know whether or not, if the building doesn't have any asbestos, do you think then that control would be necessary just with respect to the dust produced from an ordinary building, assuming you just have plaster and so on?

A Well, it is a very difficult question to answer.

We have electron microscopes but no crystal balls, but what I refer to, as we look at the dust in a human being's lungs now, there are 90 percent of the particles I can't identify. I don't know what they are, and I certainly don't know what effect they might be having.

Now, ordinary houses have very

Now, ordinary houses have very little asbestos in them, very little. Most of the asbestos that is used as insulation is used in commercial, public, industrial applications, in those type of buildings, etc.

I wouldn't think that the dusts that are created in demolition of private homes would necessarily be hazardous, but I don't know, and so I have suggested that at least some precautions be taken such as appropriate wetting. Even wetting itself is a very effective thing. I don't think that the rigorous demolition controls that we would recommend for asbestos-containing buildings is now necessary for the non-asbestos-containing homes.

I hope that I would not have to change that statement ten years from now, as we learn more and more, but on the basis of our current knowledge, I would think that that is a fair statement.

ALDERMAN GABINSKI: Q Okay, one other question, Doctor.

A Yes.

Q I got the impression that in New York and in Philadelphia they have a ban on using asbestos.

Do they?

A No, sir.

Q Not in New York?

A Neither city. They are talking about banning the spraying of asbestos because the spraying people did not adequately use the control measures which are rigorous that would have controlled it, so they are now discussing it, but these have not yet been promulgated.

Q Are there any bans in the United States at all?

A Not to my knowledge.

Q Have you appeared before the City Council in New York?

A Yes.

Q And what do you think they are going to be doing in New York?

A That is that same crystal ball and I don't have it.

Q You don't know?

A No.

Q You probably made the same suggestions to them that you made here?

A Yes, sir.

Q All right.

A That there be rigorous control.

Q Over the sources?

A Over the sources and the first one, the one that I think you should look at first is the spraying of insulation materials because that is actually putting the insulation materials into the air.

Q Was there a lot of talk regarding substitutes for asbestos in New York?

A Yes, and in fact there are suitable substitutes.

The New York City Council has required, if this ordinance goes through, and this is one that they are now discussing, the air pollution code and the one that was introduced on November 30th of 1970, that the use of any substitute be utilized only with the same controls that they had already required for asbestos.

Q Which means that a substitute may be harmful also?

A We just don't know, and we are not going to

wait thirty years to find out.

ALDERMAN GABINSKI: No further questions.

CHAIRMAN BILANDIC: I have one further question,
Doctor.

EXAMINATION

BY

CHAIRMAN BILANDIC:

Q Could we anticipate that we will be receiving support in the programs that you are suggesting to us from various manufacturers, from labor unions and from the scientific and health community?

A Support for your deliberations and recommendations?

Q Ultimate enactment of an ordinance.

A Ultimate enactment of an ordinance?

Q Yes.

A I would strongly suggest that you will receive it from the scientific community.

I assume on the basis of my general knowledge that large segments of the industry will support you, and I am sure that labor will support you.

— R71-16

EXHIBIT 9

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Identification and Control of Asbestos Exposures

LEWIS J. CRUTLEY, Ph.D.

Bureau of Occupational Safety and Health, Public Health Service, Department of Health, Education and Welfare, 1015 Broadway, Cincinnati, Ohio 45202

Asbestos can be used safely in modern industrial technology if adequate precautions are taken to prevent excessive and unsuspected exposures. To distinguish between asbestos and other fibers, new techniques must be applied to electron microscopy and diffraction, emission and atomic absorption spectrophotometry, electron microprobe, and neutron activation analytical procedures. Standards and evaluation techniques should be based on airborne fibers and the use of the membrane filter and phase contrast microscopy for sampling and counting. Controls should include procedures for the safe transport of asbestos, exhaust ventilation and personal protection at work sites; the safe disposal of waste dusts; and the prevention of community contamination.

THE UNIQUE PHYSICAL and chemical characteristics of asbestos along with its abundance as a natural resource has contributed much to technological advances since the beginning of the century. This is especially true of industrial fiber technology which has had a tremendous growth since the 1940's.

From data developed through epidemiologic and laboratory research it is now apparent that special health problems may be associated with exposure to designated fibers and that the biologic response to some fibers may be quite distinct from that of particulates of the same chemical composition but of random spherical shape.

The biological consequences of fibers in the lungs must be determined and understood for establishing rational control standards. It is equally important that knowledge of the control of fibers in our environment be developed and applied. Otherwise, adverse experiences from health problems associated with the use of asbestos may inadvertently deprive us of the benefit of this natural resource and also cast a shadow that could impede future advancements in the application of fiber technology.

Identification and Measurement of Exposure

Numerous analytical techniques are available for identifying and distinguishing the physical and chemical characteristics of fibers. Because of the minute size of individual respirable fibers, often under 1 micron in diameter, and the similar characteristics of many of the fibers, as in the amphibole group, one technique by itself is seldom definitive in identifying individual fibers.

The ubiquitous nature of respirable fibrous materials has been recently reported.^{1,2} Mineral fibers may be present and dominant in a number of ores of commercial value, ie, various forms of asbestos, or may be present in lesser but appreciable amounts in commercial minerals such as talc and clays.

No problem is generally involved in the identification and measurement of airborne levels of fibers associated with an industrial process where they are a dominant part of the mineral involved, such as asbestos. This is not true in instances where the fiber is present to a lesser extent in the mineral being processed, where more than one type of mineralogy may be present in the formation of the ore, or where mixtures of minerals may be processed. Without such knowledge sup-

ported by definitive identification and measurements, unsuspected hazards may be present from air-borne exposure to fibrous minerals. Conversely, the mere presence of fibers is not indicative that a hazard exists.

Analytical techniques useful in identifying individual fibers include electron microscopy and diffraction, emission and atomic absorption spectrophotometry, electron microprobe, and laser microprobe in conjunction with a time of flight mass spectrometer, and neutron activation analytical procedures. The use of such techniques requires specialists in the instrumentation involved and is time consuming and costly. This data, however, provides an indispensable bridge of knowledge on the physical and chemical nature and relationships of fibers in the air at industrial operations. Less complicated methods of measurement can then be applied as an index of exposure upon which rational standards can be based.

This aspect of knowledge is especially important since past research has shown that asbestos fibers differ greatly from one type to another in their chemical composition and the extraneous metals associated with the ores. Also, due to their hardness, the fibers may abrade and become contaminated with the metal alloys in which they are processed. The above analytical techniques are useful in determining the changing milieu of the fiber during processing.

Research over the past several years in the United Kingdom and the United States has shown that air-borne fiber levels are a much better index of exposure to asbestos than is an over-all particulate exposure since the ratio of fibers to over-all particulates in the air may vary greatly depending on the nature of the process and the materials involved. Revised standards for asbestos exposures in these countries¹¹ also stipulate methods for the collection and measurement of air-borne samples. The procedures¹² state that the samples should be collected on membrane filters and, after subsequent treatment, the fibers should be counted using phase contrast microscopy at 400X magnification.

Direct measuring dust counters that are capable of discriminating between different

particle size ranges have been used to measure asbestos dust levels.¹³ When such an instrument is used it is essential that the ratio of the asbestos fibers to the over-all particulates remain relatively constant. These instruments should be frequently correlated and calibrated against data from the membrane filter and phase contrast microscope method of fiber measurement. The main advantages of the automated dust counters over the manual method are in their saving of manpower and their capability of giving immediate data for assessing the proper functioning of dust control procedures.

Control of Exposures

Special precautions should be taken to prevent exposure to asbestos fibers when they are handled in a loose, unbound form. Such operations include mining, milling, shipping and warehousing, processing into products, and mixing and blending asbestos fibers with other materials. No special problem is normally encountered when the asbestos is firmly bound with other materials, i.e., vinyl asbestos tile, plastics, cement products, except when these materials are reshaped or are dismantled and the fibers may become uncoated, free, and released through friction, destruction, deterioration, and the like.

The control of air-borne asbestos fiber is not greatly different from that of other particulates having a similar aerodynamic size, though special problems and technologies may be involved in processing and handling the fibers. The basic principles of preventing exposures to asbestos include exhaust ventilation, enclosure and isolation of exposures, wet methods, good housekeeping and maintenance, worker education, good work and sanitation habits, and supplementary personal respiratory protection where indicated. Asbestos should be handled and processed with adequate controls to prevent community contamination.

Exhaust ventilation is the control of choice since the asbestos fiber is captured before it can disseminate and contaminate the immediate and adjoining work areas. Requirements have been established for the proper ventilation design, fiber capture and carrying

velocities and enclosed baghouse collecting systems.¹ Special attention should be given to the ventilation system because of the alternative nature of the asbestos fibers.

Enclosure and isolation have many applications in the control of asbestos dust. This is especially true in the batch making and handling of loose asbestos in setting of asbestos in bags before emptying, dumping and blending operations. This precaution is extremely important in the handling of solid waste containing asbestos. Enclosure and isolation of the operation also prevent the dissemination of the fiber into other areas where workers not related to the operation may be unexpectedly exposed.

Wet methods, where feasible, are an effective way of controlling dust. Examples include such operations as mixing, shelling, sawing, sanding and clean-up. Special precaution must be taken to assure that the sludge is handled properly and is not allowed to dry and become redisseminated into the air.

Good housekeeping and maintenance are essential where asbestos is handled and processed. Without such a program, a hazard may exist in an otherwise well constructed and operated plant. Elements of good housekeeping and maintenance include: (a) repairing leaks and improperly operating equipment; (b) immediate cleaning up of spills; (c) routine cleaning of all in-plant areas including floors, structural members, and equipment using vacuum cleaning and water; and, (d) cleaning of areas outside the plant and keeping dust suppressed by use of water.

Worker education is often neglected as an effective way of minimizing dust exposure. A worker who is knowledgeable of the hazards associated with his job is more motivated to protect himself from related injurious agents. Workers should be instructed in the proper use of equipment and in good practices in the handling of material with which they are working.

Good work and sanitation habits are needed to complement other methods of preventing exposure to asbestos and other materials associated with its processing. Without good work habits, excessive dust can be created at operations otherwise adequately provided with

dust control measures. These dust control measures are necessary. **W**ork clothing may become contaminated and provide intermittent sources of exposure during the work shift. **I**nspect workers before they go to the plant for prolonged periods before skin irritation and penetration by the fiber. Workers should be instructed in the proper procedures for the performance of their jobs and in the observance of good sanitation practices while on the job.

Personal respiratory protection is an effective way of preventing exposure to dust where heavy intermittent work is encountered and other means of protection are not feasible. Examples are equipment break-down and maintenance and repair operations. When respirators are used they should be approved for the exposures encountered. A routine program should also be formulated for selecting, issuing, fitting, cleaning, inspecting, and maintaining the respirators.

Summary

Asbestos can be used safely in modern industrial technology, if adequate precautions are taken to prevent excessive and unsuspected exposures. The following control program is recommended where loose asbestos is encountered:

(1) Recognized engineering control procedures to prevent the dissemination of dust into the air should be used where loose asbestos fibers are handled, processed, mixed with other materials or disposed of to assure that exposure levels are kept within safe limits and that other adjoining sites do not become unsuspected hazards.

(2) Asbestos should be packed in containers that are leak-proof, resistant to breakage, and which can be maintained in a clean condition during shipping, storage, and subsequent use.

(3) Storing of asbestos should be of such nature and in such location to minimize subsequent breakage of bags. Spillage from broken bags and adjacent areas should be vacuum cleaned immediately to prevent dispersion of the asbestos.

(4) Bags containing asbestos should be clearly marked and should have labels cautioning against breathing dust from the bags.

Permit {

— R71-16

EXHIBIT 10

REPORT OF THE PHILADELPHIA DEPARTMENT OF PUBLIC HEALTH

AD HOC COMMITTEE ON ENVIRONMENTAL AND OCCUPATIONAL HAZARDS OF SPRAY ASBESTOS FIBERS

August 31, 1970

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INTRODUCTION:

In the Fall of 1969, Air Management Services (AMS) learned that extensive pollution of the environment with spray asbestos fibers was occurring at a building under construction at 5 Penn Center, as a result of structural steel fireproofing operations involving the use of spray asbestos material. Efforts by AMS to persuade the contractors to institute effective measures to prevent further pollution of the environment met with little success. Subsequently, with the support of its consultants, AMS instituted a permit requirement for the use of spray asbestos materials in future construction and adopted a "total containment" policy, i.e., that spraying operations must be conducted under conditions that prevent the escape of any visible amounts of asbestos into the environment.

By the Spring 1970, construction work had begun on three other steel structures in the city wherein spray asbestos material was used. Although the release from those sites of spray asbestos material was, in general, less serious than that experienced at 5 Penn Center, there were numerous violations of the "total containment" policy and the result was far from satisfactory.

Meanwhile, the Section of Occupational and Radiological Health of the Environmental Health Services (EHS) was alerted to the potentially heavy exposure of construction workers to asbestos fibers. Accordingly, Section personnel carried out numerous inspections of the building sites and eventually concluded that voluntary efforts to control the exposure of workers to airborne asbestos fibers would not succeed without adoption of specific regulations by

the Department of Public Health. It also became apparent that erosion of spray asbestos fibers from the surface of air plenum chambers might occur and contaminate the building air supply and subject occupants to a potential hazard of unknown degree.

These problems were discussed at meetings (November 5, January 7, and April 1) of the Philadelphia Board of Health. As a result, the Board authorized the Occupational Health Section of EHS to study the potential hazards arising from the construction industry's use of spray asbestos materials for building fireproofing in Philadelphia, and to submit to the Board such proposed regulations and recommendations that the Section felt necessary or advisable.

After consulting with a number of experts on the hazards of asbestos exposures, Jesse Lieberman, Section Chief, appointed an ad hoc committee to review the problem and to recommend specific courses of action and proposed regulations. Mr. Lieberman considered it important to appoint to the Committee, individuals highly competent in their respective areas of expertise and representing different interests and viewpoints. The ad hoc Committee was appointed formally on 3 July 1970 with the following membership:

Davis N. Bressler, Esq.
Shapiro & Bressler

Robert D. Gorson, M.S. (Chairman)
Prof. of Medical Physics
Thomas Jefferson University

Thomas McCormick, Union Business Agent
Plasterer's Union Local #8

James F. Morgan
Consultant in Industrial Hygiene
E.I. DuPont de Nemours & Co.

Eugene P. Pendergrass, M.D.
Emeritus Professor of Radiology
University of Pennsylvania

F. Spencer Roach, Architect
Harbeson, Hough, Livingston,
& Larson

Katherine R. Sturgis, M.D.
Woman's Medical College

Harry H. Verdier, Executive Director
Safety Council
Greater Philadelphia Chamber of
Commerce

Henry Wohlers, Ph.D.
Prof. of Environmental Engineering
Drexel University

Herbert E. Wolf, Assistant Manager,
Engineering Dept.
Reliance Insurance Company

The Committee as a whole conducted four sessions: July 10, 23, August 4, and 31. During its first three sessions, the Committee received presentations from members and consultants of the Health Department and from invited experts in building construction, the asbestos mining and milling industry, the plastering industry, the spray mineral fiber industry, industrial hygiene, epidemiology, industrial and preventive medicine, and pathology. The individuals who made presentations at one or more sessions are listed in Appendix A.

Two working groups were appointed, the first to prepare a draft of precautions for controlling the emission of asbestos fiber from spray operations, and the second to prepare a draft of this report. These drafts were discussed extensively at the Committee's fourth meeting, and after modification and editing, were approved. The remainder of this report sets forth the Committee's findings, conclusions, and recommendations.

FINDINGS AND CONCLUSIONS:

(1) Gross environmental contamination with visible amounts of spray asbestos material has been produced on a number of occasions in and around construction sites wherein spraying activity was being conducted. Not only was the public in the immediate vicinity of the sites grossly exposed to spray asbestos material on a number of occasions, but so were the construction workers on the site, especially those spraying operators who failed to use protective masks and/or protective clothing provided them. The Committee is also concerned about the potential:

(a) exposure of maintenance personnel to asbestos fibers when entering asbestos sprayed enclosures;

(b) hazard, if any, to the occupants of those buildings arising from the ventilation of such buildings after erosion or dislodging of

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asbestos fibers in the air plenum chambers;

(c) environmental contamination arising from future alterations to and eventual demolition of the buildings.

(2) The Committee was assured by each person who appeared before it, that each shared to varying degrees the Health Department's concern in this matter and that many steps had already been or were being taken to deal with the problem. All of the representatives of the various interests fully cooperated with the Committee and demonstrated deep interest in working out effective and practical ways of dealing with the problem.

(3) The lack of containment of the spray asbestos material and the exposure to workmen on the site were caused by a number of factors. Among these were difficulties inherent in architectural design, negligence on the part of some sprayers, lack of responsibility or authority on the part of one prime contractor to control the activities and work scheduling of other prime contractors and their subcontractors, indifference or ignorance concerning the potential hazards of exposure to airborne asbestos fibers on the part of some of the spray operators and other construction workers, adverse weather conditions, and lack of personnel with adequate knowledge, ability and authority to exercise overall supervision of all activities that could cause immediate or subsequent escape of spray asbestos material. Contractors, in general, did not allow for the cost of such controls in their bids, nor did architects usually specify measures for such controls.

(4) There is no on-going program for periodic medical examinations, such as chest x-rays, for the sprayers of asbestos fireproofing material nor for those involved in the cleaning up after spraying.

(5) It is difficult to ensure 100% containment of airborne asbestos material during the spraying operation. In the absence of 100% containment, the cementitious process allows considerably less airborne material to escape

the premises than does the competing "fluff technique" process. However, cleaning up is quite difficult since the material adheres firmly where it falls. The Committee is not in the position to judge the other relative merits of the two competing processes.

(6) The coordination of the inspection and enforcement activities of AMS and EHS at times left something to be desired with regard to controlling spray asbestos operations. There also appeared to be a lack of coordination of the actions taken independently by the District Attorney's office with the activity of these Services. The Committee believes that all branches of local government should closely coordinate their efforts in safeguarding the health and welfare of its citizens.

(7) The AMS and particularly the EHS, lack the essential resources, i.e., qualified manpower, instrumentation and measurement capabilities, and supporting funds, necessary to conduct the constant surveillance necessary to safeguard all citizens subject to possible exposure from the potential hazards of airborne spray asbestos fibers. The Committee believes that the segment of the construction industry that utilizes spray asbestos material should bear a major portion of the cost of an adequate surveillance and monitoring.

(8) The prolonged exposure of asbestos workers to high concentrations of airborne asbestos fibers is clearly implicated as a cause for the high incidence of asbestosis, a pneumoconiosis, and bronchogenic carcinoma in such workers. There also is considerable evidence relating the development of pleural and peritoneal mesotheliomas, a very rare and fatal form of malignancy, in patients with history of long exposure to asbestos fibers. Data suggest that asbestos, itself, may not be carcinogenic but that trace elements or other agents associated with natural asbestos or in the environment that become attached to the asbestos fibers during milling, processing, and other operations may be the causative agents alone or in combination with asbestos. The

probability of developing lung cancer is very much greater among asbestos workers who smoke cigarettes than among those who do not smoke.

(9) Data are virtually non-existent concerning the possible deleterious effects of chronic exposure to low concentrations of asbestos fibers, such as that found in the ambient air of most metropolitan areas or around construction and demolition sites. [Nevertheless, the Committee agrees with many experts in the field that, until persuasive evidence to the contrary is forthcoming, it is prudent for public health officials to assume that there is no level of concentration of airborne asbestos fibers below which adverse effects will not occur in population groups chronically exposed. Accordingly, the Committee endorses the consensus of many experts that every reasonable method should be exercised to limit the discharge of asbestos fibers into the environment from all sources.] *Bundled Prof*

(10) With regard to limiting the occupational exposure to asbestos fibers, the Philadelphia Department of Public Health, as well as the Pennsylvania Department of Health use as a performance standard (threshold limit values) the limits specified in Chapter 4 of Article 432 of the Rules and Regulations of the Department of Health of the Commonwealth. In April 1970, the American Conference of Governmental Industrial Hygienists (ACGIH) adopted a proposal to lower the threshold limit values in places of employment, to become effective in approximately two years. It appears unlikely that State Regulation 432 can be amended to reflect the revised standard in the near future. However, the Committee believes that the Philadelphia Department of Health should adopt as soon as possible, its own performance standard, consistent with the ACGIH proposed values, to apply to airborne asbestos fibers from all sources within the working environment.

(11) The Committee believes that in contrast to the control measures practical in manufacturing industries, the methods of controlling the dispersion of spray asbestos fibers available to the construction industry are not adequate to protect all citizens (construction workers, maintenance and operating personnel, future occupants of the buildings and the general public) from some degree of exposure to asbestos fibers from the spray material during construction, throughout the life of the structure and during alteration and final demolition. [Accordingly, the Committee believes that the Philadelphia Department of Health (in cooperation with other health departments and the United States Public Health Service) should effectively encourage the manufacturers of spray fireproofing material to develop acceptable fireproofing substitutes which contain essentially no asbestos. The Committee notes that the manufacturers are actively working to develop acceptable substitutes, which may be forthcoming in six months or less. The Committee believes that a "total containment" policy should apply to any substitute material until such time as the potential hazards of any airborne particles from such materials have been evaluated. If such substitute material is judged by medical experts to be without hazard, the containment policy can be relaxed.]

SPECIFIC RECOMMENDATIONS:

The Committee recommends the Department of Public Health of the City of Philadelphia adopt forthwith the following specific proposals:

A. Elimination of spray asbestos material:

A regulation be adopted to the effect that no spray asbestos material shall be applied in building construction, reconstruction or alteration within the City of Philadelphia three months subsequent to (1) the adoption of the regulation, or (2) at such earlier date on which approved asbestos free spray fireproofing substitutes are commercially available from at least two manufacturers.

B. Performance standards for the protection of workers on the construction site:

Regulations be adopted that:

(1) All necessary precautions shall be taken to prevent anyone within the construction site from being subjected to airborne asbestos fiber concentrations, caused directly or indirectly by the spray material, exceeding the Maximum Permissible Concentrations adopted by the Philadelphia Department of Health.

(2) No person on the construction site shall be exposed to the inhalation of airborne asbestos fibers exceeding the following Maximum Permissible Concentration: 5 fibers/cc of size greater than 5 microns in length average over any eight hour period, provided the concentration in any fifteen minute period does not average more than 15 fibers/cc, as determined by measurement techniques acceptable to the Philadelphia Department of Health.* Airborne concentrations of asbestos fibers exceeding the limits will be permitted in only those controlled areas wherein all persons wear respirators and/or other protection devices which effectively reduce the asbestos fiber concentrations to the permissible limit at the point of inhalation.

C. Prevention of the release of spray asbestos material into the environment:

A public announcement, that the Philadelphia Department of Public Health has adopted a "total containment" policy, i.e., that:

(1) the prime contractor and/or any subcontractors shall take all necessary precautions to prevent the release into the environment of any visible amounts of spray asbestos material, and of invisible amounts exceeding a performance standard to be adopted by regulation.

(2) A regulatory performance standard that airborne concentrations of asbestos fibers at any point beyond the building perimeter shall not be visible to the unaided eye and at any point 20 feet beyond the perimeter shall not exceed

*Phase contrast microscopy at 430X. See for example: "Equipment and Procedures for Mounting Millipore Filters and Counting Asbestos Fibers by Phase Contrast Microscopy" by Bayer, Zumwalde and Brown, USPHS (July 1969).

1 fiber/cc of size greater than 5 microns averaged over any 15 minute period as determined by measurement techniques acceptable to the Philadelphia Department of Health.*

D. Implementation of performance standards for controlling the exposure to spray asbestos fibers

Regulations be adopted that:

(1) All applications for a permit to use spray asbestos material shall be reviewed by both the AMS and the EHS (Occupational Health Section) before such permit may be issued. Such applications shall be submitted by the prime contractor responsible for spray fireproofing at least two calendar weeks prior to the start of spraying.

(2) One person shall be designated by the responsible prime contractor as having full time supervisory authority for all aspects of the spraying operation. He shall be the person directly responsible for monitoring all operations from which the release of asbestos fibers could result, in such manner as to insure compliance with the pertinent asbestos control regulations and work requirements (see Appendix B). The name and qualifications of the individual so designated shall be submitted to the Philadelphia Department of Public Health by the prime contractor at the time of application for the installation permit and operating license.

(3) The prime contractor responsible for spray fireproofing shall provide written assurance at the time of applying for the permit, that each employee engaged in spraying, mixing, handling and clean-up of asbestos material has or will have completed a satisfactory course of instruction on the potential hazards of exposure to asbestos fibers and instruction on the precautions that must be observed to control the hazard before he is assigned to the job. Each such employee shall sign a statement filed with the prime contractor that he will comply with the working requirements that are submitted for approval at the time of the permit application.

* See footnote on page 8.

(4) All other prime contractors shall also apply to AMS for a similar permit and shall likewise provide written assurance that their employees and those of their subcontractors assigned to the job site will have been instructed on the precautions required to prevent the dislodging, dispersion and inhalation of asbestos spray material, and on approved clean-up procedures, before spraying commences or before they are assigned to the job site.

(5) Each prime contractor shall at the time of applying for the permit, post an appropriate bond in the amount of \$_____. Such bond shall be refunded to the contractor at the end of all operations which could cause the release of spray asbestos fibers if it has been demonstrated to the satisfaction of the AMS and the EHS that all reasonable precautions were taken to comply with performance standards and that there have been no substantial violations.

E. Requirements for periodic medical examinations for spray asbestos workers:

Condition precedent to permit
A regulation be adopted that all contractors using spray asbestos material shall provide pre-employment medical examinations, including chest x-ray examinations, of all new employees. All such employees shall have periodic chest x-rays and sputum cytology examinations at intervals not exceeding six months. The results of such examinations shall be reported to EHS of the Philadelphia Department of Public Health.

APPENDIX A

INDIVIDUALS WHO ATTENDED ONE OR MORE COMMITTEE SESSIONS TO PRESENT INFORMATION

1. Jesse Lieberman, Chief, Occupational Radiological Health Section, Environmental Health Services, Philadelphia Department of Public Health.
2. William Reilly, Director of Compliance and Enforcement, Air Management Services, Philadelphia Department of Health.
3. Dr. P.W. Purdon, Professor and Director of Environmental Engineering and Sciences, Drexel University.
4. Karl Schofield, Deputy City Solicitor, Law Dept. City of Philadelphia.
5. Arthur Wallach, Assistant Commissioner of Health, Environmental Health Services, Philadelphia Department of Public Health.
6. A.W. Lookup, Structural Engineer, A.W. Lookup Co. Phila. Pa.
7. H.R. Whitman, Whitman Brothers, Inc., Plastering Contractor.
8. Dr. W.J. Nicholson, Director, Insulation Industry Hygiene Research Program, Environmental Science Laboratories, Mt. Sinai School of Medicine, New York, N.Y.
9. Harold Romer, Assistant Commissioner, Dept. of Air Resources, City of N.Y.
10. Edwin Venzie, Venzie Corp., Plastering Contractor.
11. Stanley Fonfa, Sprayon Research Corporation.
12. Herbert Levine, President, Sprayed Mineral Fiber Manufacturers Association.
13. D.G. Powell, J. Cintani, and H. Waxman, Zonolight Division, W.R. Grace Co. Inc.
14. Charles Scull, Sanitarian I, Environmental Health Services, Phila. Dept. Health.
15. Louis J. Cralley, Ph.D., Director, Division of Epidemiology and Special Services, Bureau of Occupational Safety and Health, U.S. Public Health Service.
16. Paul Gross, M.D., Professor of Pathology and Occupational Health, Graduate School of Public Health, University of Pittsburgh.
17. Jan Lieben, M.D., Medical Director, FMC Corporation.
18. Norman Williams, M.D., Prof. of Preventive Medicine, Thomas Jefferson University
19. Edward Wilson, Chief, Air Management Services, Philadelphia Dept. of Public Health.
20. Sidney Speil, Ph.D., Director of Research, Piping and Mining Division, John Mansfield Corporation.
21. Leon Gonshor, Regional Industrial Hygienist, Pa. State Dept. of Health.
22. Clement Lazenka, Director of Engineering, Air Management Services, Phila. Dept. of Public Health.
23. James F. Morgan, Consultant in Industrial Hygiene, E.I. DuPont deNemours & Co.
24. Charles McBride and Raymond L. Weinmann, General Building Contractors Association, Inc.

APPENDIX B

REQUIREMENTS FOR CONTROLLING THE EXPOSURE TO SPRAY ASBESTOS FIBERS

I. Responsibility

- A. No person shall cause any individual to be exposed to asbestos dust in excess of the limits adopted by the Philadelphia Department of Public Health.
- B. All work performed in the construction, modification, and repair of a building or site where asbestos containing fireproofing materials are employed shall be under the direction of a person responsible for asbestos safety therein. His name shall be reported to the Department.
- C. The person in charge of the asbestos safety program at a construction site shall have the following responsibilities. He shall:
 1. Inform himself of the hazards attendant upon the presence of asbestos at the site, or if necessary, to this end obtain the services of a qualified expert.
 2. Provide or cause to be provided any necessary instruction concerning safe working practices to all employees whose duties necessitate the handling of asbestos containing material or operation of any equipment that emits asbestos dust in amounts that lead to a hazard, and to all other employees who are not regularly employed at such work but who may be exposed to asbestos dust.
 3. Take all necessary precautions that all workers engaged in the spray application of asbestos containing materials and other workers whose duties cause exposure to excessive amounts of asbestos dust, such as sweepers, clean up men, chippers, repair personnel, etc. are provided with U.W. Bureau of Mines approved respirators, for pneumoconiotic-producing dusts and furnished with suitable coveralls which will be left at the site and thereby preclude the removal of asbestos from the site.
 4. Take all necessary precautions that procedures are instituted and supervised that preclude the removal and dispersal of asbestos containing material from the construction site on the clothing or other appurtenances of persons leaving the area.
 5. Take all necessary precautions to provide for the safe disposal of asbestos containing waste materials from the construction sites by providing suitable bags or containers strong enough to resist tearing or breakage for transport to the disposal site.
 6. Inspect all bags containing asbestos fireproofing and waste materials to make certain that they are not torn or broken during use or disposal. In the event any bag is torn or broken, its contents will be immediately transferred to a new bag.
 7. Provide that durable and legible warning signs be conspicuously posted outside of enclosures warning persons of the hazard of entering the enclosure without an appropriate respirator and other apparel.

8. Take all necessary steps and precautions to provide that the area or areas being sprayed with asbestos containing material is totally enclosed in such a manner as to prevent the escape of material from the enclosures.
9. Take all necessary steps to prevent the continuation of the application of asbestos containing material whenever weather conditions are such as to preclude the effective containment of material, e.g. high winds.
10. Provide for continuous inspection of construction site vicinity for escape of asbestos material, halt all operations causing such escape, and immediately provide for clean-up of emissions.

11. Requirements for the Prime Contractor Responsible for Asbestos Spray Fireproofing

- A. Permit applications and the satisfaction of all city regulations shall be completed by the responsible Prime Contractor two (2) calendar weeks prior to the start of spray fireproofing.
- B. Notice of intent to spray asbestos-containing material on any premises shall be given to the Philadelphia Department of Public Health (Department of Air Management Services). Said notice shall be oral, by telephone, and in writing and shall specify, at least two (2) days prior to said spraying the floors on which the spraying shall take place.
- C. The responsible prime contractor shall designate one person as having full-time supervisory authority for all safety aspects of the spraying operation as required under 1 (B). The name and qualifications of the individual so designated shall be submitted to the Philadelphia Department of Public Health at the time of application for the installation permit and operating license.

D. Recommended Control Practices

1. Before the start of spraying operations all floor enclosed areas shall be shoveled clean. Before the application of asbestos containing material commences, the floor of the enclosed area shall be cleared of all objects, material and equipment other than that employed in the application and the asbestos containing insulation or all objects, materials, etc. shall be covered with plastic or other approved tarpaulins in a manner that precludes the subsequent dispersal of asbestos particulates.
2. The entire floor, or the part of the floor to be insulated shall be enclosed with plastic or other approved tarpaulins in a manner which shall preclude the escape of asbestos containing material from the enclosure.* All interior open areas, such as elevator shafts, stairwells, etc., shall be enclosed in a manner which shall prevent the escape of asbestos containing material from the working area.

*Due to variations in architectural design and construction techniques, additional requirements may be imposed for permit approval where full containment appears to be difficult.

"Stack effect" of the shafts, stairwells, etc. shall be considered in providing proper enclosures. An enclosure will be considered satisfactory only if visible insulating material cannot escape from the enclosure.

Roughing?

3. Wet asbestos containing material which has fallen to the floor shall be swept up to prevent dispersal of dried material. Under no condition shall this material be removed later than at the end of the working day. Swept up material shall be placed in a heavy plastic bag strong enough to resist tearing or breaking under normal handling conditions and clearly marked as containing asbestos waste. The contents of the aforementioned plastic bags shall not be transferred to another container. The plastic bags shall be placed upon a vehicle for disposal at an approved site.

4. All floors will be vacuumed shortly after drying. The vacuum cleaner shall contain a strong, single-service, disposable inner bag of durable material which shall be removed from the vacuum cleaner and tightly sealed. The bag shall then be placed in a container of the type described in paragraph 3, which shall thereafter be placed on a vehicle for removal and disposal at an approved site.

5. The materials used to form the enclosure shall be thoroughly vacuumed upon completion of the application of the insulation in the area. The entire floor area, all ledges and surfaces including tarpaulins upon which waste insulation material may have fallen, shall then be vacuumed or revacuumed before removal of the enclosures.

6. Enclosures shall not be dismantled until the area has been thoroughly vacuumed after completion of spraying and clean-up. During the entire construction period, dislodged spray material shall be vacuumed immediately as in paragraph 4.

7. All areas used for opening bags containing asbestos insulating material and/or charging of hoppers shall be enclosed in such a manner that asbestos containing insulating material shall not be permitted to escape from the immediate area in which such activity takes place.

8. Signs shall be posted outside enclosures warning persons of the hazards of entering the enclosures without appropriate mask and other apparel.

9. All persons involved in the spraying of asbestos at the site must be furnished with Bureau of Mines approved respirators for pneumoconiotic producing dust or equipment as required by Pa. State Department of Labor and must be furnished with suitable coveralls and/or four weather gear which will be left at the site and thereby preclude the removal of asbestos from the site. No person shall be permitted in an area in which asbestos spraying or handling has taken place until final vacuuming referred to in paragraph 5, unless such person is furnished with and wears a Bureau of Mines approved respirator for pneumoconiotic producing dust and coveralls of the type described herein. Facilities shall be provided and procedures instituted and

supervised that preclude the removal and dispersal of asbestos containing material from the construction site on the clothing or other appurtenances of persons leaving the area. Water, soap and paper towels will be supplied for washing of hands prior to any eating or smoking. No smoking will be permitted within the spray area.

10. Any plenum or other structures coated with asbestos containing insulation which is intended for use in the circulation of air in the building must be thoroughly cleaned of all debris and waste insulation. (All applied asbestos containing insulation within a plenum or duct must be coated with a sealant which precludes exposure of the asbestos containing material to the circulating air.)

11. In case of emission of asbestos from the construction site, immediate steps shall be taken to cause the cessation of such emissions by either effective control measures or work stoppage at the source of the emissions. There shall then be immediate and complete cleanup of all material that has escaped the construction site by measures that will insure that no further dispersal of any asbestos material into the atmosphere can occur.

Note: Regardless of adherence to the stated control practices, any visible emission of asbestos-containing materials beyond the building perimeter will be considered as a violation of the Regulations of the Philadelphia Department of Public Health and will be subject to appropriate legal actions. In the experience of AMS and EHS to date, extraordinary diligence to the schedule of control methods is required in order to avoid violations.

— R71-16

EXHIBIT 11 (b)

CHAIRMAN BILANDIC: The Meeting will come to order. We are now resuming the recessed session of our study on asbestos which commenced on March the 3rd, 1971.

The first witness that we will call today is Lewis Cralley, Chief, Division of Epidemiology and Special Services, Bureau of Occupational Safety and Health, E.C.A.

What does E.C.A. stand for, Doctor?

DR. CRALLEY: It is now Environmental Control Institute. There has been a change. That is not the current set of initials. It is now the Bureau of Occupational Safety and Health.

DR. BROWN: Of the public health service?

CHAIRMAN BILANDIC: Is that the United States Department of Public Health?

DR. CRALLEY: The Department of Health, Education and Welfare.

CHAIRMAN BILANDIC: Are you speaking here on behalf of the federal agency or are you speaking on your own behalf as an individual?

DR. CRALLEY: On my own behalf as a person knowledgeable in certain areas of the subject under

discussion.

CHAIRMAN BILANDIC: All right. Doctor, will you tell us something about your education, training and background?

A Well, I received my Bachelor of Science degree in chemistry and biology from McKendree College, Lebanon, Illinois, in 1933. I then went on to get my doctorate degree at the State University of Iowa, Iowa City, Iowa, in 1942; in industrial hygiene, specializing in the study of elements of the environment as they relate to health.

CHAIRMAN BILANDIC: Will you tell us something about your experience and additional training subsequent to the obtaining of your degrees?

DR. CRALLEY: All of my experience of 29-1/2 years has been in the United States Public Health Service. I started out back in 1942 doing research and field studies, epidemiological studies related to the environment, to occupational diseases, with respect to standards and the like.

I have had progressive experience in this area with increased responsibility during

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this time and at the present time I am now Director of the Division of Epidemiology and Special Services of the Bureau of Occupational Safety and Health which conducts industrywide studies of occupational diseases including the dust diseases for particulate toxic materials and a broad spectrum of the occupational diseases.

Q Doctor, in the interest of saving time I note you have a statement which has been given to us regarding some of the honorary societies. Is there anything special or unusual about any of these?

A They are all professional societies.

(The above referred to honorary and professional societies are as follows:)

Honorary Societies:

Sigma Zeta, McKendree College, 1932
Phi Lambda Upsilon, State University of Iowa Chapter, 1939
Sigma Xi, State University of Iowa, 1945

Professional Societies:

American Public Health Association - Fellow
Chairman, Occupational Health Section, 1962

Positions: (Cont'd.)

Asst. Chief, Occupational Health Field
Hdqtrs. (Environmental Activities) 1954-64
Assoc. Chief, Occupational Health Program
(Field Studies and Epidemiology) 1964-69
Chief, Division of Epidemiology and Special
Services, Bureau of Occupational Safety
and Health, 1969-

CHAIRMAN BILANDIC: Are they societies to
which one is elected because of professional
achievements?

A That is correct, and the one I am
especially proud of is I was elected as a
consultant to the American College of Chest
Physicians.

This college is made up entirely
of physicians but they have asked a few persons,
knowledgeable of the environment, to translate
this to them as physicians so that they could
have a better understanding of how the environment
interrelates to the medical and I have been very
proud to have been a consultant to them for a
number of years.

Q I notice that you are also the author
or co-author of various research papers. Are
there any that you would select or that you would

like to particularly call our attention to insofar as asbestos is concerned?

A . Yes, I have a few that relate specifically to research on asbestos.

We have made studies in which I have been the prime contributor, in the characterization of asbestos fibers particularly with respect to the metals and other things that are associated with them that might relate to health hazards.

Another area we have made a special study of is talc which is a fibrous material quite closely related to asbestos which I will cover later in my discussion.

Another area which I think you might find interesting, a paper I recently presented at the International Congress of Occupational Health in Tokyo; "Identification of Asbestos Exposures" which will be quite pertinent.

CHAIRMAN BILANDIC: If there is no objection we would like to have copies of your documents that you referred to and the publications that you have referred them and we will have them

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incorporated into the record and made a part of the record if there is no objection.

ALDERMAN FIFIELSKI: No objection.

DR. CRALLEY: I will be glad to leave them with you.

(Here follows the above
referred to publications:)

Reprinted from American Industrial Hygiene Association Journal, Volume 29, November-December, 1968.

"Characterization and Solubility of Metals Associated with Asbestos Fibers," L. J. Cralley, R. G. Keenan, R. E. Kupel, R. E. Kinser and J. R. Lynch, Bureau of Occupational Safety and Health, Environmental Control Administration, Department of Health, Education and Welfare, 1014 Broadway, Cincinnati, Ohio 45202

A series of uniform samples representing blends from different sources and types of asbestos were analyzed for nickel, chromium, cobalt and manganese. The bulk samples and the less than 10-micron size fractions were analyzed separately. The metals soluble

in bovine serum were determined for the less than 10-micron fractions. Free silica content was also determined. The degree of solubility of the same metal varied considerably by source and type of asbestos. Chrysotile had greater amounts of nickel and chromium whereas Amosite was higher in manganese. Crocidolite had the lowest metal content.

Introduction: Earlier research indicates that asbestos workers have been unknowingly exposed to appreciable airborne levels of nickel, chromium and manganese as a result of the release of these metals from the ore during processing and also from the abrasive action of asbestos on the alloys used in processing equipment. To further characterize the biological activity of these metals, information is needed on their comparative solubility in relation to different sources and types of asbestos.

As an aid in promoting data reproducibility in research on the biological response to asbestos, the Committee on Asbestos and Cancer of the Union Internationale Contre le

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Cancer (UICC) prepared a series of uniform reference samples representing blends from different sources and types of asbestos. These are available to researchers. This report presents data on the amounts of designated metals in these samples and on their solubility in bovine serum.

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Procedures: The UICC Committee on Asbestos and Cancer made available for chemical characterization blended, milled asbestos fibers from different sources of chrysotile, crocidolite, Amosite, and anthophyllite both before grinding and after grinding the fibers to increase the percentage of respirable materials in the products. To separate the bulk material into fine and coarse particulates, each of the asbestos samples was screened through a 10-micron sieve. The bulk and under 10-micron sieve size samples were dissolved in hydrofluoric acid and the solutions analyzed for nickel, chromium, cobalt, manganese, and iron by means of atomic absorption spectrophotometric procedures. Free silica was determined colorimetrically with the method of

talvitie and Hyslop.

Under 10-micron sieve size ground asbestos reference samples were used in determining metal solubility in serum.

Approximately 100 milligrams of each of the under 10-micron samples of chrysotile, crocidolite, Amosite and anthophyllite was added to 15 ml of bovine serum in ampoules, which were then flame sealed. The ampoules, held for 63 days at room temperature, were shaken daily.

At the end of the test period, the samples were centrifuged in an ultracentrifuge operating at 25,000 rpm. The supernatant liquid was withdrawn and ashed, and the residue was analyzed for nickel, chromium, cobalt, and manganese by means of atomic absorption spectrophotometric procedures.

Results: The designated metal and free silica contents of the UICC ground and unground reference samples, both for bulk material and under 10-micron sieve size, are given in Table 1.

The UICC samples of asbestos were

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blends of components with varying contents of metals; therefore, different samples of each of the blended bulk materials may have varying metal contents depending on the uniformity of the blend.

Accordingly, the metal contents of the bulk and under 10-micron size asbestos samples reported for each type of asbestos in Table 1 will have a general relationship only since each comes from a different sample of the specific blend analyzed.

In comparing the metal contents of the bulk unground and ground samples, the anthophyllite showed an appreciable increase in the overall metals in the ground sample, an indication that the grinding may have been a source of metal contamination.

Chrysotile "A" showed an increase in the overall metals content of the bulk ground over the unground sample though less than the anthophyllite.

The minor differences in the metal contents in chrysotile "B," crocidolite, and amosite ground and unground bulk samples are

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believed to be within the normal variation expected.

The chrysotile "A" unground sample showed a nickel content of 1844 ug/gm in its under 10-micron sieve fraction size in contrast to 1290 ug/gm for the corresponding fraction of the ground sample.

The level of nickel in the ground bulk sample, however, was 1482 ug/gm in contrast to 1284 ug/gm in the unground bulk sample.

The ratio of nickel in the under 10-micron fraction to that in the bulk in the unground sample was 1.44:1.0 in contrast to a ratio of 0.87:1.0 for the ground sample.

The chromium, cobalt, manganese and iron content of the samples followed the same trend.

There was a 42 percent increase of the combined nickel, chromium, cobalt, manganese and iron in the ground over the unground bulk material. This indicates that although appreciable metal contamination appeared to have occurred in the grinding of the

asbestos it was distributed throughout the sample and not concentrated in the under 10-micron fraction.

The anthophyllite unground sample showed a nickel content in its under 10-micron sieve fraction of 414 ug/gm in contrast to 397 ug/gm for the corresponding fraction of the ground sample.

The level of nickel in the ground bulk sample was 414 ug/gm in contrast to 217 ug/gm in the unground bulk sample.

The ratio of nickel in the under 10-micron fraction to the bulk in the underground sample was 1.91:1.0 in the contrast to a ratio of 0.97:1.) for the ground sample.

The chromium, cobalt, manganese, and iron content of these samples followed the same trend.

There was a 51 percent increase in the combined nickel, chromium, cobalt, manganese and iron in the ground over the underground bulk material. This suggests that appreciable metal contamination may have

occurred in the grinding of the anthophyllite.

The metal content of the different types and sources of asbestos appeared to follow distinctive patterns.

The nickel, chromium, and cobalt in the under 10-micron sieve size were highest in the chrysotile "A" and chrysotile "B" asbestos, followed by anthophyllite, crocidolite, and amosite in descending order.

The manganese in the under 10-micron sieve size was highest in amosite, followed by crocidolite, anthophyllite, chrysotile "A" and chrysotile "B" in descending order.

The iron in the under 10-micron sieve size was highest in the crocidolite and amosite, followed by anthophyllite, chrysotile "A," and chrysotile "B."

Free silica in the under 10-micron sieve size was highest in the amosite, followed by crocidolite, anthophyllite, chrysotile "A," and chrysotile "B" in descending order.

The solubility in bovine serum of the nickel, chromium, cobalt, and manganese

associated with the UICC samples, which represented different types and sources of asbestos, is given in Table II.

Nickel was the most soluble of these metals. Thirty-three percent of the available nickel in crocidolite was dissolved in bovine serum during the test period, followed by 25 percent for amosite, 17 percent for chrysotile "B," 12 percent for chrysotile "A" and 8 percent for anthophyllite.

Chromium solubility ranged from 8 percent for chrysotile "A" and 7 percent for chrysotile "B" to 5 percent for anthophyllite.

The level of cobalt was too low to be measured in terms of solubility for any of the asbestos samples.

The solubility of available manganese was 28 percent for chrysotile "B," 20 percent for chrysotile "A," 7 percent for crocidolite, 5 percent for anthophyllite, and 3 percent for amosite.

Human serum samples were prepared as a comparison series to the bovine serum series

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and were held for the same period to compare the solubility of the metals in the two types of sera.

No appreciable difference was found in the solubility of these metals contained in the asbestos samples in either human or bovine serum.

A weighted solubility factor was calculated for the combined nickel, chromium and manganese in the UICC asbestos samples. Seventeen percent of these three metals available in chrysotile "B" was dissolved in bovine serum during the test period, 12 percent for chrysotile "A," 8 percent for crocidolite, 3 percent for amosite, and 3 percent for anthophyllite.

For comparative purposes, pure nickel oxide and chromite were each added to bovine serum and treated in the same manner as the asbestos samples. Only 0.2 percent of the nickel from the nickel oxide and 0.3 percent of the chromium from the chromite were dissolved in the serum.

The degree of solubility of the

same metal varied considerably by sources and types of asbestos.

The data suggests that the nickel and chromium from the UICC asbestos samples dissolved in the serum were from a source other than the corresponding metal oxides.

The sources of the metals in the asbestos include their presence in alloy systems and other more soluble minerals associated with the asbestos ore, replacement by these metals of part of the magnesium during the formation of the asbestos fiber, and the abrasion of metals from equipment in the processing of the ore.

Conclusions: Data are presented on the nickel, chromium, cobalt, manganese, iron, and free silica content of UICC reference samples of different types and sources of asbestos.

Data are also presented on the solubility of the nickel, chromium, cobalt, and manganese content of these asbestos samples in bovine serum.

Additional attention should be directed to defining the biologic activity of

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different sources and types of asbestos in relation to the soluble metal components associated with the specific types of fibers.

Acknowledgments: The authors acknowledge, with appreciation, the technical assistance of Elsie Schneider for applying the micro-sieving procedure to the various samples, and Nancy Dunn, David Thomas and Harold Norris for the determinations of free silica.

(The second report follows:)

Reprinted from AMERICAN INDUSTRIAL HYGIENE ASSOCIATION JOURNAL, Volume 29, July-August, 1968.

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"Fibrous and Mineral Content
of Cosmetic Talcum Products.

"L. J. Cralley, Ph.D., M. M. Key, M.D.,
D. H. Groth, M.D., W. S. Lainhart, M.D., and
R. M. Ligo, M.D.

"Occupational Health Program, National
Center for Urban and Industrial Health, 1014 Broadway,
Cincinnati, Ohio 45202.

"In searching for sources of fibers
ubiquitous to our everyday environment and of
respirable size, the authors examined 22 talcum
products commonly available on retail shelves and
found fiber contents ranging from 8 to 30 percent
by count, with an average of 19 percent. Fibrous
particulates were generally under 1.0 u in diameter
with lengths ranging from 1.5 to 6.0 u. From 0.3
to 3.0 percent quartz was found in 21 of the samples
and the remaining sample had 54.4 percent quartz.
The samples were also analyzed for metals; with four
exceptions, the levels of cobalt, nickel, chromium
and manganese were low. Further research will be
needed to assess the significance of these findings.

"Reports of finding pulmonary fibrous

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bodies, previously referred to as "asbestos" and now as "ferruginous" bodies, in the lungs of persons coming to autopsy in hospitals in a number of cities have recently been increasing.

The first report of these morphologically distinctive fibrous bodies in the sputum and lungs of asbestos workers was made in 1906 by Marchand. Current interest dates from 1963 when Thompson et al. found these fibrous bodies in the lungs in 26.4 percent of the autopsies in a series of examinations in Cape Town.

Subsequent investigations provide evidence that the occurrence of these bodies in the lungs of urban residents is not restricted to those in isolated localities and is not a one-time chance observation.

The identity of the fibrous core of these coated bodies, however, was never determined by the investigators. The fibers were routinely considered as asbestos bodies on the basis of their morphological structure.

In searching for the sources of these ubiquitous fibers, Cralley et al. reported that talcum

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powder contained a significant percentage of respirable fibers and was a potential source of the ferruginous bodies observed in the lungs of humans. This observation led to further study to characterize cosmetic talcum products.

The major purpose of the investigation reported here was to develop and present data on some of the constituents found in cosmetic talcum products and to discuss their health aspects in the light of today's knowledge. It is not our intent to make a general appraisal of health factors in the use of talcum products because of the many variables involved and the limited data available on the consumption of various "sources of talcs" in the formulation and use of cosmetic talcum products. The potential health aspects of some of the data, however, are discussed.

Twenty-two cosmetic talcum products (representing body powder, bath powder and all purpose powder) purchased off the shelf, were analyzed for fibrous content, selected metals, and quartz. The data and a discussion of their possible significance follow.

Analysis of Talcum Products:

Talc is a natural mineral, hydrous magnesium silicate, with the general formula $(OH)_2-Mg_3Si_4O_{10}$.

Talc mineral is formed by the hydrothermal alteration of serpentine and tremolite or directly from the unserpentinized ultrabasic rocks. Talc may also be formed by the thermal metamorphism of siliceous dolomites.

The characteristics of the mineral deposits vary widely from the pure talc formula and from each other according to the mineralogy involved.

Some deposits may contain varying amounts of tremolite, chrysotile, pyrophyllite, or serpentine, or other basic material from which the talc may be derived.

The deposits may also contain varying amounts of metals such as iron, nickel, cobalt, chromium, and manganese as associated minerals, as well as silica.

Cosmetic talcum may be basically pure talc or may be a formulation of talc with other

materials such as clay, chalk, stearates, et cetera. Zinc, titanium, manganese, and iron compounds may be added as pigments and opacifiers.

The particle-size distribution and percentage by count of fibers in the talcum particulates were determined by dispersing the talcum in water, filtering the mixture through an "AA" membrane filter, and measuring with a phase contrast microscope at 430 magnification.

The percentage of free silica was determined by x-ray diffraction. Cobalt, chromium, nickel, and manganese were determined by means of atomic absorption spectrophotometry.

Zirconium, titanium, zinc, iron, and magnesium were determined by means of semi-quantitative emission spectrography.

Table I gives analytical data on 22 different cosmetic talcum products. Figures 1 and 2 are representative photomicrographs of two talcum specimens showing the presence of fibers.

Size Distribution of Talcum Particulates:

Seven of the twenty-two talcum products were selected for size-distribution measurements of

the fibrous and non-fibrous particulate components.

The diameter of 80 to 95 percent of all the particulates in these samples was under 5.0 microns (u).

The median of the diameter of the non-fibrous particulates in the seven products ranged from 0.7 to 2.0 u, with a median average around 1.0 u.

A fiber is defined as a particulate having at least a 1:3 ratio of diameter to length. The fibrous particulates in the seven products were generally under 1.0 u in diameter, with lengths ranging from 1.5 to 6.0 u.

The 22 talcum products analyzed showed fiber contents ranging from 8 to 30 percent by count of the total talcum particulates with an average of 19 percent.

Although the specific fibrous materials were not identified, they were predominantly fibrous talc, as shown by x-ray diffraction, with the probable presence in minor amounts of other fibrous minerals such as tremolite, anthophyllite, chrysotile, and pyrophyllite.

The electron microscope, with its higher power of resolution, shows a number of submicron diameter particulates not visible by means of phase contrast microscopy.

Free Silica:

In 8 of the 22 talcum products (Table 1), the presence of quartz ranged from 0.3 to 1.0 percent; in 13 products, 1.2 to 3.0 percent quartz, and in one product, 54.4 percent quartz.

Metals:

With the exception of talcum products Nos. 4, 8 and 22 (Table 1), the cobalt content of the products analyzed was under 25 parts per million by weight (ppm), chromium under 22 ppm, nickel under 29 ppm, and manganese under 78 ppm.

Product No. 4 had a nickel content of 1270 ppm; chromium 340 ppm; and cobalt, 67 ppm.

Product No. 8 contained 479 ppm nickel and 329 ppm chromium.

Product No. 22 contained 1210 ppm nickel and 1170 ppm chromium.

No. 6

Qualitative tests showed some of the chromium in the talcum products to be in the hexavalent state. The nickel, chromium, cobalt, and manganese in the talcum products may have come from the talc miner deposit or from the alloy metals of the pulverizing equipment used in reducing the talc.

The zirconium content of the products was all under 10 milligrams per gram (mg/gm) except for products Nos. 9 and 17, which had 20 and 30 mg/gm respectively.

The titanium, zinc, and iron ranged from a few tenths to 50 mg/gm of talcum and were probably present as pigments or opacifiers.

The magnesium content of the products was over 10 mg/gm, except for product No. 9 which had only 0.5 mg/gm.

The magnesium was probably present as an additive in the formulation or as a part of the talc molecule or other amphiboles in the products.

The aluminum and silicas were in all probability associated either with the talc molecule, or additives such as kaolin, or the base

89 material from which the talc was derived.

Known Health Effects of Talc:

Much of our knowledge of the health effects of talc is derived from studies of occupational exposures in its mining, milling, and industrial use. In extrapolating this knowledge to the cosmetic use of talcum powder, it must be recognized that the pattern of exposures in the use of talcum product varies markedly from person to person, not only in frequency of use but also in amount and in location.

In contrast to industrial exposures where the pattern is likely to be more continuous with accompanying peaks, exposure in the use of cosmetic talcum products is very intermittent with peak exposures dominating.

The exposure pattern may continue a lifetime, especially if the use of talcum is established in the earlier years as a part of personal habits.

The air-borne dosage to workers from industrial exposures to talc that have resulted in injury to health were undoubtedly much higher, however, than would be predicted from the use of cosmetic talcum products.

Mining, Milling and Industrial Use:

The clinical entity of talc pneumoconiosis, "talcosis," has been observed repeatedly in workers with long exposure to talc in its mining, milling, and industrial use.

Elongated, terminally clubbed pulmonary fibrous bodies, both segmented and unsegmented and similar in morphology to ferruginous bodies, have been found in talc workers, but these workers have received a mixed exposure -- to talc, tremolite, anthophyllite, and silica.

In one study the mortality rate from cancer of the lung and pleura was four times greater for a group of talc workers than for the general population. In pulmonary cancer from asbestos, the role of fibers and trace metals is uncertain.

Some investigators have assumed the fibers play a dominant and direct role; more recent investigations indicate that the fibers may have been only an index concealing a spectrum of unidentified agents and relationship.

Surgical and Cosmetic Use:

The only reported cutaneous reactions from the use of talcum powder are talc granulomas, and these have been rare. Talcum powder, however, is no longer used on surgical gloves and should not be applied to broken skin. Occasionally, perfume oils used in talcum powder formulations sensitize the skin and produce dermatitis.

Conclusions:

With the exception of 4 of the 22 cosmetic talcum products analyzed, the levels of free silica, cobalt, nickel, chromium, and manganese were generally of a low magnitude and within a narrow range. It is not known whether the four products represent a significant proportion of sales in the industry or to what extent the sources of the talc in these four formulations are the same as sources of talc specified for use in other talcum products in the competitive market.

The levels of silica, chromium, and nickel in these four products are sufficiently high, however, to be of concern in their potential to cause disease.

All of the 22 talcum products analyzed

have an appreciable fiber content, ranging from 8 to 30 percent by count of the total talcum particulates, and averaging 19 percent.

The fibrous material was predominantly talc but probably contained minor amounts of tremolite, anthophyllite, and chrysotile as these are often present in fibrous talc mineral deposits.

Cosmetic talcum products should be included as a source of the fibers, from which may be derived ferruginous bodies observed in the lungs of humans. The meaning of the presence of these ferruginous bodies, however, is uncertain.

The industry has the know-how to safely handle fibrous material as well as toxic metals such as nickel, chromium, cobalt, and manganese once adequate criteria have been established.

Unknown significant amounts of such materials in products that may be used without precautions may create an unsuspected problem. For this reason continued research and investigations and communication of findings are necessary in this area.

Acknowledgments:

The authors acknowledge, with appreciation, the technical assistance of Harrold B. Norris who made the free silica determinations; Patricia L. Maurer who made the atomic absorption spectrophotometric determinations for nickel, cobalt, chromium, and manganese; John R. Carlberg who made the emission spectrographic determinations for zirconium, titanium, zinc, iron, magnesium, and aluminum; and Stephen Bayer and Ralph Zumwalde for the fiber determinations."

(The third publication referred to is as follows:)

"Identification and Control of Asbestos Exposures, Cralley, Lewis J.

"U.S. Department of Health, Education and Welfare, Public Health Service, Environmental Health Service, Environmental Control Administration, Bureau of Occupational Safety and Health, 1014 Broadway, Cincinnati, Ohio 45202.

"Presented at the XVI International Congress on Occupational Health, Tokyo, September, 1969.

"Identification and Control of Asbestos

Exposures.

"The unique physical and chemical characterization of asbestos along with its abundance as a natural resource has contributed much to technological advances since the beginning of the century. This is especially true of industrial fiber technology which has had a tremendous growth since the 1940's.

From data developed through epidemiologic and laboratory research it is now apparent that special health problems may be associated with exposure to designated fibers and that the biologic response to some fibers may be quite distinct from that of particulates of the same chemical composition but of random spherical shape.

The biological consequences of fibers in the lungs must be determined and understood for establishing rational control standards. It is equally important that knowledge of the control of fibers in our environment be developed and applied.

Otherwise, adverse experiences from health problems associated with the use of asbestos may inadvertently deprive us of the benefit of this

natural resource and also cast a shadow that could impede future advancements in the application of fiber technology.

Identification and Measurement of Exposure to Asbestos:

Numerous analytical techniques are available for identifying and distinguishing the physical and chemical characteristics of fibers. Because of the minute size of individual respirable fibers, often under 1 micron in diameter, and the similar characteristics of many of the fibers, as in the amphibole group, one technique by itself is seldom definitive in identifying individual fibers.

The ubiquitous nature of respirable fibrous materials has been recently reported (1,2). Mineral fibers may be present and dominant in a number of ores of commercial value, i.e., various forms of asbestos, or may be present in lesser but appreciable amounts in commercial minerals such as talcs and clays.

{ No problem is generally involved in the identification and measurement of air-borne levels of fibers associated with an industrial process

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where they are a dominant part of the mineral involved, such as asbestos.

This is not true in instances where the fiber is present to a lesser extent in the mineral being processed, where more than one type of mineralogy may be present in the formation of the ore, or where mixtures of minerals may be processed. 7

Without such knowledge supported by definitive identification and measurements, unsuspected hazards may be present from airborne exposure to fibrous minerals. Conversely, the mere presence of fibers is not indicative that a hazard exists.

Analytical techniques useful in identifying individual fibers include electron microscopy and diffraction, emission and atomic absorption spectrophotometry, electron microprobe and laser microprobe in conjunction with a time of flight mass spectrometer and neutron activation analytical procedures.

The use of such techniques requires specialists in the instrumentation involved and is

time consuming and costly. This data, however, provides an indispensable bridge of knowledge on the physical and chemical nature and relationship of fibers in the air at industrial operations. Less complicated methods of measurement can then be applied as an index of exposure upon which rational standards can be based.

This aspect of knowledge is especially important since past research (3) has shown that asbestos fibers differ greatly from one type to another in their chemical composition and the extraneous metals associated with the ores.

Also, due to their hardness, the fibers may abrade and become contaminated with the metal alloys in which they are processed. The above analytical techniques are useful in determining the changing milieu of the fiber during processing.

Research over the past several years in the United Kingdom and the United States has shown that air-borne fiber levels are a much better index of exposure to asbestos than is an over-all particulate exposure since the ratio of

fibers to over-all particulates in the air may vary greatly depending on the nature of the process and the materials involved.

Revised standards for asbestos exposures in these countries (4,5) also stipulate methods for the collection and measurement of air-borne samples. The procedures (6,7) state that the samples should be collected on membrane filters and, after subsequent treatment, the fibers should be counted using phase contrast microscopy at 400x magnification.

Direct measuring dust counters that are capable of discriminating between different particle size ranges have been used to measure asbestos dust levels (8). When such an instrument is used it is essential that the ratio of the asbestos fibers to the over-all particulates remain relatively constant.

These instruments should be frequently correlated and calibrated against data using the membrane filter and phase contrast microscopy method of fiber measurement.

The main advantages of the automated

81 dust counters over the manual method are in their saving of manpower and their capability of giving immediate data for assessing the proper functioning of dust control procedures.)

Control of Asbestos Exposures:

Special precaution should be taken to prevent exposure to asbestos fibers when they are handled in a loose (unbound) form. Such operations include mining, milling, shipping and warehousing, processing into products, and mixing and blending asbestos fibers with other materials.

No special problem is normally encountered when the asbestos is firmly bound with other materials, i.e., vinyl asbestos tile, plastics, cement products, et cetera, except when these materials are reshaped or are diminished and the fibers may become uncoated, free, and released through friction, destruction, deterioration and the like.

The control of air-borne asbestos fiber is not greatly different from that of other particulates having a similar aerodynamic size, though special problems and technologies may be

involved in processing and handling the fibers. The basic principles of preventing exposures to asbestos include exhaust ventilation, enclosure and isolation of exposures, wet methods, good housekeeping and maintenance, worker education, good work and sanitation habits, and supplementary personal respirator protection where indicated.

Asbestos should be handled and processed with adequate controls to prevent community contamination.

Exhaust ventilation is the control of choice since the asbestos fiber is captured before it can disseminate and contaminate the immediate and adjoining work areas. Requirements have been established for the proper ventilation design, fiber capture and carrying velocities, and enclosed baghouse collecting systems (9,10,11,12). Special attention should be given to the ventilation system because of the abrasive nature of the asbestos fibers.

Enclosure and isolation has many applications in the control of asbestos dust. This is especially true in the batch making and handling

of loose asbestos i.e., wetting of asbestos in bags before emptying, dumping and blending operations, et cetera. This precaution is extremely important in the handling of solid waste containing asbestos. Enclosure and isolation of the operation also prevents the dissemination of the fiber into other areas where workers not related to the operation may be unsuspectingly exposed.

Wet methods, where feasible, are an effective way of controlling dust. Examples include mixing, drilling, sawing, sanding, clean-up, and the like. Special precaution must be taken to assure that the sludge is handled properly and is not allowed to dry and become redisseminated into the air.

Good housekeeping and maintenance are essential where asbestos is handled and processed. Without such a program, a hazard may exist in an otherwise well constructed and operated plant. Elements of good housekeeping and maintenance include: (1) repairing leaks and improperly operating equipment, (2) immediate

cleaning up of spills, (3) routine cleaning of all in-plant areas including floors, structural members, and equipment using vacuum cleaning and water, and, (4) cleaning of areas outside the plant and keeping dust allayed using water.

Worker education is often neglected as an effective way of minimizing dust exposure. A worker who is knowledgeable of the hazards associated with his job is more motivated to protect himself from related injurious agents. Workers should be instructed in the proper use of equipment and in good practices in the handling of material with which they are working.

Good work and sanitation habits are needed to compliment other methods of preventing exposure to asbestos and other materials associated with its processing. Without good work habits, excessive dust can be created at operations otherwise adequately provided with dust control or where further dust control is unnecessary.

Work clothing may become contaminated and provide a continuing source of exposure during the work shift. Excessive asbestos dust on the skin

for prolonged periods favors skin irritation and penetration by the fiber. Workers should be instructed in the proper procedures for the performance of their jobs and in the observance of good sanitation practices while on the job.

Personal respiratory protection

is an effective way of preventing exposure to dust where heavy intermittent levels are encountered and other means of protection are not feasible. Examples are equipment breakdown and maintenance and repair operations. When respirators are used, they should be approved for the exposures encountered. A routine program should also be formulated for selecting, issuing, fitting, cleaning, inspecting, and maintaining the respirators.

Summary:

Asbestos can be used safely in modern industrial technology if adequate precautions are taken to prevent excessive and unsuspected exposures. The following control program is recommended where loose asbestos is encountered:

- (1) Recognized engineering control procedures to prevent the dissemination of dust

into the air should be used where loose asbestos fibers are handled, processed, mixed with other materials, or disposed of to assure that exposure levels are kept within safe limits and that other adjoining sites do not become unsuspected hazards.

(2) Asbestos should be packed in containers that are leak-proof, resistant to breakage, and which can be maintained in a clean condition during shipping, storage, and subsequent use.

(3) Storing of asbestos should be of such nature and in such location to minimize subsequent breakage of bags. Spillage from broken bags and adjacent areas should be vacuum cleaned immediately to prevent dispersion of the asbestos.

(4) Bags containing asbestos should be clearly marked and should have labels cautioning against breathing dust from the bags.

(5) All processing, handling, and dust control equipment should be maintained and kept in good repair.

(6) A rigid housekeeping program should be enforced.

(7) Asbestos waste should be disposed in a manner to prevent contaminating other areas in its handling or to prevent subsequent redispersion into the air.

(8) A worker education program should be established. Workers should be made aware of the danger of breathing asbestos dust, the proper use of equipment and controls, and of good sanitation and work habits.

(9) Approved respiratory protective equipment should be used where high intermittent exposures to asbestos occur and other means of controlling the exposure are not feasible. A special program should be established for selecting, fitting, inspecting, and maintaining the respirators in a sanitary condition.

(10) Where excessive contamination of the work clothing is potential, workers should wear special clothing which should be changed at the end of the work shift and handled in a manner to prevent exposure to those laundering the clothes.

(11) Washroom facilities should be available where indicated, and special protective

clothing should be used to prevent excessive contamination of the skin with asbestos fibers.

(12) The air in areas occupied by workers handling asbestos should be routinely monitored to assure that control procedures are adequate and that air-borne levels of asbestos dust are within safe limits.

(13) Workers handling asbestos should receive periodic medical examinations.

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CHAIRMAN BILANDIC: All right, Doctor, will you proceed then, now that we have established your background and training and qualifications in the field, will you proceed to present your statement to us?

A This is our area of environment and health which is extremely important. It is important to us at the moment in this country as citizens. It is important to the country in maintaining its leadership in technology in the world.

Our country has achieved an enviable record of world leadership and research coming in both the biological and the physical sciences. Even more important, this research has been harnessed into technology that is the systematic application of science and knowledge.

The other countries of the world are looking to us for this leadership and the impact it will have upon the health and welfare of their people.

This newer technology has given us the elements of a better standard of living, a better understanding of the nature, the cause, the prevention and the treatment of diseases and a better understanding of the relationship of our environment to health.

It is right that the fruits of our research should promote technology that will benefit the whole of our society. This must be done, however,



specific subject here.

Q Dr. Selikoff told us at the last session that there is no such thing as asbestos, that that is a purely generic term and he broke it down into the various components.

Do you agree with that statement also?

A Yes, indeed. That is all they are, chemicals, fibrous silicates.

Q Now, the significant thing that Dr. Selikoff mentioned to us is that, as he put it scientifically, the hazard of asbestos is the fact that it has a half-life of infinity and then he explained what that means.

In other words, once it gets into the human body, it isn't assimilated, it isn't eliminated, but it stays there, it stays there and then it continues to have a cumulative effect so that if you get a little bit of it, of asbestos into your lungs one day and you get a small dose another time and then later you get a little bit more, it would have a cumulative effect rather than like a small dose of some other things.

Do you agree with that theory?

A Generally speaking, yes.

I think we must define it a little more closely in terms, if you want to get into some specific areas as to the size of the fibers, how they are handled once they are retained in the body. It would be quite different.

You have very, very small submicron fibers and those that are maybe a hundredth of a micron in diameter and less than a micron in length and by definition, have a three to one ratio of length to diameter.

They are still defined as a fiber. They may be handled differently than the longer fibers, but we must realize that the spectrum of the size of the fiber is a long-range one.

So with these small ones you also have the larger ones, so generally speaking, that would be true.

They do remain in the body for a long period of time.

Q All right.

Now, as a scientist, do you agree with Dr. Selikoff that when asbestos bodies are

135 found in the human lungs, that they are clearly and readily identifiable?

A Yes, by a specialist using very special techniques. Most of the techniques used would not do this, but with electron microscopy, electron probe equipment and the very specialized advances in identification, it can be done. It is time consuming, expensive and must be done by a specialist.

Q Well, is it fair to say then that there is no mistake when they use this proper equipment, that when we identify something as an asbestos body in a human lung, it is clearly an asbestos body and not something else?

A It would depend upon the techniques they have used.

Many people will do this using a microscope and will not determine these characteristics. They determine the morphological characteristics of the fiber, how it appears and looks under the microscope and don't magnify it to show the characteristics of the fibers, say in great detail.

So as I say, it depends on the

N3- techniques again. They are available, but they are not always used.

Q Well --

A This identification is not often made, but it may be sort of a moot question as to how far we need to go.

We do know that there are asbestoform minerals and as to whether it is one type or another, it is still a related asbestoform mineral and we can go too far in that direction in trying to be too careful to identify each fiber.

Q Would you agree with Dr. Selikoff's conclusion, based upon his studies of asbestos workers and the general public in the City of New York and in certain other areas of the country that he identified, and I think he had also done some tests here in Chicago; that he establishes that one out of every five asbestos workers and in some cases as many as one out of every four asbestos workers dies of lung cancer which is considerably greater than the incidents of lung cancer in the general public that is not associated with work in the asbestos field?

Do you agree or do you have any comment on any of those studies?

A These are documented technical reports appearing in scientific literature. We all have access to them.

I have no reason not to accept them. I accept them as factual, factual statements.

Q Once we establish that there is a medical hazard, and clearly the studies that Dr. Selikoff made with a controlled group of asbestos workers themselves clearly show that the workers are exposed to this problem and have this problem.

Now, taking it one step further, both Dr. Selikoff and the studies that were made in Cape Town, South Africa, where there are various asbestos mines; in Great Britain and other parts of the world, they seem to carry their conclusions even one step farther where they found that people who live in the community who had no particular traffic with any of the asbestos industry, they found children who had played in various lots in the area where there might have been sand or piles of asbestos, 20 or 30 years later, when

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autopsies were performed, they found that these particular children, or these particular people who had been exposed, died of lung cancer.

They relate it back and attribute these results and these deaths to the exposure to asbestos which then for the first time caused a great deal of stir in the medical world because it showed that the exposure of the general public now has become a health hazard.

Now, whether that is serious or not is one of the judgments that we will have to make here.

Do you have anything to enlighten us about on that particular area?

A Well, I think we must remember that in those cases, these children and the people in the general area were exposed to a concentration of asbestos fibers which is far greater than we have in our industry today.

They had massive exposure to this fiber because they were in the environment of the plants which had no dust control. They were playing in the refuse of the plant material that

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had a high fibrous content to it.

If it is harmful, and of course, we know it is in certain amounts, then we would expect them to have biological effects from living in an environment for a lifetime.

Q Would you say that the concentrations would be greater, for example, to a housewife who was doing her husband's laundry, washing the clothes he wore when he went to the plant, that that would be an excessive or heavy exposure?

A Again, we must define the terms.

If he had a heavily contaminated set of work clothes and the housewife was to shake or remove the dust vigorously in such a way, in a confined area, she could get exposure.

Q Well, Doctor, based upon the studies and the research that you have either done personally or with the health service or that you have had an opportunity to study or read, do you feel that we have any problem and if so, what the degree of that problem would be in say a major metropolitan area such as the City of Chicago?

A May I make this little statement, just

air-cleansing devices of rain and whatnot.

There is a second area we must bear in mind and that is the personal habits of people. This may be a rather important source of exposure to asbestos-forming mineral fibers at times and an example is the cosmetic talc compounds that are used.

We did an analysis of over 22 of these and I have it here and these are fibers and mineral contents, mineral talcum powder and I will leave this with you, in which all of these showed a range of from 8 percent -- maybe I better be exact on this -- all of the 22 talcum powders analyzed have appreciable fiber contents ranging from 8 percent to 30 percent by count of the total particulate with an average of 19 percent.

In other words, 19 percent of all the particulates were fibrous in nature of the fibrous talcum products, and I do not believe that we can ignore this as a potential source, depending upon the habits of the person with respect to face powder and so forth.)

ALDERMAN WIGODA: Are you talking about ordinary

cosmetic powders?

DR. CRALLEY: Industrial powders that you buy and cosmetics, yes.

[I want to caution you when I get through here because we might make too much out of this.

This is a source. I am not saying that there is anything wrong with this.

There are many materials we use around the home which have fillers of fibrous materials in them and I am talking about yard fertilizers, the plant insecticides, the animal insecticides and the like. Many of these will have fibrous materials in them and they come from the filler, that is the talc, the diatomaceous earth, the vermiculite and so forth, which are fibrous clay-like materials used as fillers, and these come into play when you spray or when you dust, so there is a chance of getting close to some fibrous exposures, exposures to fibrous materials and so in these two areas, first I mentioned the ambient level is naturally present in the background and also these personal habits.

There is no information which would indicate to the effect that anybody has been injured by this.

It would be a very exceptional case. There is no data that indicates that these practices have been harmful.

It does add up that these may be another source of fibers which they are finding in the lungs at autopsy.

Also at low level, there is no information saying that this was related to injury to the health.

Now, the third source, of course, is the important one; the manufacture and use of products containing asbestos-type material.

That is where we are apt to get the heaviest of the concentrations or exposures to these materials even in the manufacturing or their subsequent use if we don't use the precautions which need to be taken.

So with that background we have a natural background already in the air. By our own way of living, we do have another additional

845 background. We must keep this in mind when we are thinking in terms of a few fibers in the lung, whether it has -- whether this might relate to an outside or industrial source or not.)

Q Well, Doctor, you are telling us now that we don't have to be too concerned about talcum powder and then to make the rest of our friends here more comfortable --

A Well, I use it.

Q Well, good.

We will give you an opportunity to ask a question in a little while, Alderman Sperling.

ALDERMAN SPERLING: Well, would you have him follow through, Mr. Chairman, with that?

He has just said that he might have covered it, but what precaution is the Doctor talking about?

CHAIRMAN BILANDIC: We will get to that. We first want to establish his position and he hasn't completed his answer on the question that we asked before insofar as asbestos itself is concerned.

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Doctor, have there been any studies yet that you are aware of that would indicate that what concentration asbestos particulate may or may not be harmful?

A We have suggested guides for in-plant, not community, but in-plant and yes, we do.

Q All right.

Now, where are those published?

A The American Conference on Governmental Industrial Hygienists published these.

Q All right.

So that you have in-plant guides?

A Yes.

Q Now, do you have any guidelines for concentrations of asbestos that may be harmful to the general public, say in the ambient air in the metropolitan area such as the City of Chicago?

A I know of no data of this nature.

Q All right. Is NAPCA undertaking a study or do they have a study in progress about this particular problem?

A (No response.)

Q Dr. Selikoff did testify to that fact?

A Yes, they have a project under way, and I believe Dr. Clark Cooper, who may be appearing with you sometime, is Chairman of the Committee which will address itself to this point.

Q But you are not aware of any special figure at this time?

A No, and I know of no such figure anybody else has suggested at this time.

If it were available, I think it would be quite knowledgeable with respect to everyone.

Q All right.

So that we can sort of draw some conclusions at this time, Doctor:

First of all, as I understand it from testimony of certain witnesses last time, and from some of the literature in the field, the problem of asbestos control in plants where they may be manufacturing either plasterboard or masonite or any products that have asbestos, this is pretty well under control.

We have certain protective devices

now for the workmen which are helpful and within the plant, they have certain protective devices and they do try to protect the emissions that would go out into the community so that anything that is done in an enclosure, in a factory, so to speak, is generally reasonably well controlled.

Would you agree with that?

A Would I agree that the procedures and the techniques are available?

I am not in a position to make judgment to what extent they are being used universally in the control.

They are available, and I know in many industries they are being used, but I would not want that to be a general statement to cover the entire industry.

That would be unfair because I have no knowledge that it does.

Q As a scientist -- we don't want to pin you down, but these procedures are available and all of the good men are doing this sort of thing.

All right, then the next problem

N4
to build these buildings, and to the general public which has no other choice than they have to conduct their business by walking by these sites?

A I believe that regulations of good practices have been established and if enforced, they would be effective.

Q You say regulations could be?

A Yes, of good work practices; maybe regulations is not the correct word, but good work practices could be enforced.

Good work practices are available if enforced and they -- that is, they are available, and if enforced, they would control it.

Q As I understand it, last April, New York City had entered some type of an interim order for just such construction sites and that this interim order has been a total failure in New York City.

Are you familiar with any of that?

A I have read the reports but other than --

ALDERMAN SINGER: Could you describe what that is?

CHAIRMAN BILANDIC: At the last session,

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Dr. Selikoff testified to that, that it would require the enclosure of certain areas where asbestos was being applied and certain other protective procedures, which must be taken.

A I do not know.

I did not see -- I have talked with Dr. Selikoff and I have heard him speak before, not here, but at other meetings, technical meetings on this subject area.

I don't know. I have never heard explained why they were not enforced or why it was impossible to enforce these practices.

I understood they were not universally practiced.

Q They failed to such an extent that they are considering, and there is an ordinance pending to ban asbestos entirely?

A That would take wisdom above and beyond my capability at the moment.

Q All right.

You can't help enlighten us on that area?

A No. I would think in philosophy we

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should prevent excessive exposures to the community where they can be controlled by established procedures.

We don't want to needlessly expose people to something where in excess it could be harmful. We shouldn't do that.

We should use the procedures available.

Q Then you agree with the principle that we should control and not ban, but you don't have the specifics to give us as to how we should do this?

A Well, if you bear in mind, as I am speaking, I am speaking as an individual here.

Q Yes.

A I believe that we can make better use of our advanced technology with our natural resources if we use the philosophy of "use it right. If we need it, use it right.

"Be sure that we use it right, but have the benefits of it; if it had benefits above and beyond other situations or other materials, that we can do it," all right.

The other two major areas that Dr. Selikoff talked about is in the demolition of buildings, because of the asbestos being present in the demolished building and given off into the air.

Also, he indicated that there should be some control in that area and the other is the industrial use.

If we control these three areas we would be controlling the great bulk of possible exposure to asbestos and not have to concern ourselves with the thousand and one other things, of very minor nature.

Would you agree with that?

A I agree with that.

If you control those, we have come a long way in solving the problem in construction.

Q All right.

So that basically then, you would agree with the recommendations of Dr. Selikoff and that is that some type of controls should be applied rather than a ban of a product such as

asbestos and that hopefully these controls would be sufficient to make the environment safe for the general public as well as for the workers.

A With the exception, I think I would remove the "hopefully" because I think we can make them safe.

CHAIRMAN BILANDIC: Well, are there any questions by any other aldermen?

Alderman Sperling.

ALDERMAN SPERLING: Following up what the Chairman was just talking about, Doctor, and I hadn't been at the previous session, but with respect to the question of precaution, the question of using -- I haven't heard you say it, but it seems to me that the use of a mask would be important.

CHAIRMAN BILANDIC: Well, I will save you some time because your not having been at the last session, Dr. Selikoff has gone into this and he is not an expert in that particular field.

ALDERMAN SPERLING: Let me just complete the question.

CHAIRMAN BILANDIC: Dr. Selikoff explained

the masks and the other suggestions.

. You can go ahead and ask him if you want, but it is not his field.

ALDERMAN SPERLING: I may proceed?

CHAIRMAN BILANDIC: Yes.

ALDERMAN SPERLING: One of the precautions which you urged had to do with the outside construction sites where there might be asbestos available.

DR. CRALLEY: Well, I did not have specific recommendations except the application of these procedures known to be effective.

Now, in industry, there are a few situations, and they are few and we say this should be very limited, where in addition to being controlled, there may be specific situations where you have a very momentary operation that we would recommend additional protection for, but not as the entire method of protection, but this is supplemental to the engineering and good practices.

Q Well, I wanted to ask you that, if the Chairman would bear with me, because he has

his thoughts based upon the previous hearing and I am sure this probably has been brought up, but the question of the experience in New York, I am totally ignorant as to that and I got the impression there from your answer that there are practices, engineering practices around demolition sites, if properly used, if enforced, you felt could cure the situation.

What does that envision?

Do you enclose an area that is being torn down?

A Enclose it in one situation but in another area, you may control it at its source through certain types of ventilation.

In another area, you may use methods combined with whatever the others may have been designed for.

You must study the operation itself with the experienced person who knows how to control this certain operation. He can come up with the proper procedure and recommend it to do the job.

ALDERMAN SPERLING: All right, thank you, Doctor.

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ALDERMAN SINGER: Mr. Chairman?

ALDERMAN WIGODA: I have one other question.
Is there a known or are they working on a substitute for asbestos?

A I don't know about the substitute.

They are developing mineral synthetic fibers. As to a substitute or not, it is not the same because perhaps they will stand on their own right and their own characteristics to contribute to our technology.

Now, they may have a substitute at times, but that again depends upon the situation and the fiber is usually developed to accomplish a specific purpose.

Q Right.

A And that may not relate to why we are using asbestos or some other fibrous type of material.

It is too general a statement so that I couldn't answer that.

Q I just wondered if they had one or are they working on it, that is, if this is dangerous.

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A Industry is always working on synthetic materials, yes.

Q But they don't work on them to help the general public. They only work on them if they can help their business.

The automobile industry is always working on things, too.

A At times, now industry is changing. They will do it now to help the public affairs less harmful.

Q Well, the public is forcing them to.

ALDERMAN SINGER: Only when it comes to good business.

ALDERMAN WIGODA: Another question:

Is the federal government working on this? We have competent people in the City of Chicago, but is the federal government working on any kind of standards and precautions? Are they coming out with any guidelines or anything? We know that there is research.

A I cannot address myself too much to this area. There is another group who will address themselves to this. I am interested in in-plant study,

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the study of in-plant controls, but there is an overlap area here where knowledge relates to both in-plant and community and I would not like to address myself too specifically to that.

7 I know that they are working in this area, but I cannot be specific on them, but they are interested and they have projects going, research projects under way in those areas.

ALDERMAN SPERLING: Your experience and background is in the field of health and the dangers to health that are caused by the ingredients, rather than the technology and the engineering that can control them, is that correct?

A Yes, with this exception.

Now, I say my area is epidemiology of occupational diseases where we will study an in-plant problem, on what are their problems relating to the cause of ill effects in the plant.

What are the agents? What are the safe levels of exposure? Then we determine what are good medical and environmental prevailing techniques to achieve these levels.

Those are what we try to accomplish,

so one area would encompass engineering, but I would have specialists work within this area.

ALDERMAN WIGODA: What do you do after you get all of this data?

What do you do with it?

Frankly, I am very confused because after you get all of this data, what do you do with it to protect the public?

It goes into standards.

We relate this to industry, to unions, to anybody who needs this type of information, to assist in the application of it.

Q: Nobody is bound by it, are they?

I mean, you can say "This is it, this is dangerous, this has too much fibrous material in it."

A: People are bound by the standards.

Q: Are there standards in effect?

A: For in-plant, not community standards. I am talking about in-plant standards. That is my special area.

Q: Would you explain that?

CHARMAN BILANDIC: I will explain it to you.

N59
ALDERMAN WIGODA: I just don't get it.

ALDERMAN SPERLING: I think what the Doctor is saying, Paul, too, is this:

Our history of our workmen's compensation, industrial accident prevention, pneumoconiosis, silicosis, all of these diseases which are and were indigenous to the manufacturer, the work of a manufacturer and to the processes and this is a source and then they have the medical research and the results have come through and the precautions that are suggested, they are made recognizing that this is a danger and it is a hazard and the precautions that have been made have been translated by state laws into certain precautions and to that extent that is what he is saying.

Now, as far as what Alderman Wigoda is asking, it is:

Does your particular present research on the subject matter we are discussing, is that already translatable to a federal program that is in the works, in being for standards and guidelines.

A I would be glad to send you a copy of that. I don't have it here.

Q What do you do with your information?

Would you just tell us a little bit more about the Act itself, what it encompasses, without trying to be a lawyer?

A Well, the Act encompasses the establishment of safe work places, safe and healthful work places.

Q That would cover exactly the very subject matter that the Chairman has been discussing with you, too, would it not?

A Yes.

Q Various areas?

A Yes, it relates to the in-plant, to the worker and to the community.

CHAIRMAN BILANDIC: Yes, so that we may save a lot of time, Alderman, what he is discussing and what his area of expertise is is something that occurs under a roof, within an enclosure in a factory.

ALDERMAN SPERLING: I understand that.

CHAIRMAN BILANDIC: Where people work as opposed to the other.

ALDERMAN SPERLING: Well, where I seem to disagree

CHAIRMAN BILANDIC: Let me complete my statement, please -- as opposed to some of the other problems which we are also interested in.

ALDERMAN SPERLING: Let me respond.

CHAIRMAN BILANDIC: That is, exposure to the public and to the people who are out in the street in general.

ALDERMAN SPERLING: What I am trying to respond to, as I understand the Doctor's position and what he is saying his particular expertise is in and where he is trying to draw a line, but I don't follow that line because it seems to me that line transfers quickly over to the community, the people or members of the community.

CHAIRMAN BILANDIC: That is what we are to find out here.

ALDERMAN SPERLING: That is what I think Alderman Wigoda and I both are interested in knowing; is how you have to go about this and you have indicated that in the new Act somehow --

A We interrelate very closely.

ALDERMAN SPERLING: Well, say he goes into the plant and he gets sick and then transfers a

86-
A Yes, that is true, but as I understand it, these will transfer over to the new Occupational Safety and Health Safety Act when it becomes effective.

Q Does that Act mandate that new standards be developed in this area or can it merely accept existing standards?

A It can accept existing standards, but it alerts those in the Department of Health, Education and Welfare to do continuing studies and research to keep them updated, to see if these standards are correct, or not or valid.

Q Are the standards contained in the Walsh-Healy Act, which presumably they will be transferred over under the new Act, are these standards relating to asbestos in your opinion, are they consistent with the present technology or present knowledge about the problem?

In other words, are they sufficient, or should, in your opinion, those standards be improved?

A I am not able to give a complete answer to this because we need more data on studying people

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under conditions of exposure over long periods to get the final information.

I do believe that the enforcement of those standards would indicate at least to a very low level, extremely low level, the potential of diseases being existent.

Q To what extent are the Walsh-Healy standards being enforced?

A That is an area out of my own area of responsibility.

G The Department of Labor would have to answer that.

Q Let me ask you just a conclusionary question. In your opinion, assume the existing standards are sufficient to do the job which you said they are, and assuming if they were enforced, would we have a great problem today in in-plant exposure?

A Well, I do not recall that I gave assurances that today's standards were adequate. We are always doing research to find out whether or not the standards are adequate because of new knowledge, new technical information on the relation of insult to biological

A I don't -- I wouldn't say the biggest danger because you have to qualify it in terms of the nature of the exposure. Excessive exposure to asbestos and over longer periods has provided data showing that they have excessive lung cancers.

It has shown that but we have also subsequent data showing that if we control the exposures the lung cancer drops remarkably to a very low level.

Now, we are searching for what is a safe level.

ALDERMAN BIGGS: I see.

CHAIRMAN BILANDIC: Are you finished, Alderman?

ALDERMAN BIGGS: Yes.

CHAIRMAN BILANDIC: All right. Thank you very much, Doctor.

The next witness will be William Bourke of Zenolite Construction Products Division of W. R. Grace & Co.

Do you have a prepared statement?

MR. BOURKE: No, it is just notes. I just put it together.

CHAIRMAN BILANDIC: All right. You can come

— R71-16

EXHIBIT 11 (c)

March 23, 1971

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CHAIRMAN BILANDIC: The Meeting will come to order, please.

The next witness will be Dr. George W. Wright.

GEORGE W. WRIGHT,
called as a witness herein, testified as follows:

CHAIRMAN BILANDIC: Dr. Wright, will you tell us where you are from?

A I am from Shaker Heights, but it is actually Cleveland, Ohio. Shaker Heights just happens to be the community I live in.

Q Are you affiliated with any particular hospital or institutions of higher learning?

A Yes, I am the head of the Department of Medical Research at St. Luke's Hospital which is one of the teaching hospitals of Western Reserve University School of Medicine.

I have a faculty appointment of Associate Clinical Professor of Medicine.

Q Will you tell us something about your training, education and background as it would apply particularly to the field of asbestos and its control?

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A . I graduated in 1932 from Indiana School of Medicine. I had a two-year internship at Barnes Hospital in St. Louis in medicine, then went to Bellevue in New York City as a resident in medicine.

At New Haven Hospital I was as a resident in radiology and back to New York City at Bellevue as resident in diseases of the chest.

I then went two years at Western Reserve University in the Department of Physiology, that is, I spent two years.

From there I went to Saranac Lake, New York to what was then called the Saranac Laboratory; subsequently the Trudeau Laboratory where I was associated with Dr. Gardner for 14 years, the last four of which were subsequent to his death. And this laboratory was devoted almost solely to the study of what one would categorize as the "pneumoconiosis" or the dust diseases of the lung.

At that place we utilized all of the techniques that one normally would use

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3m70
for studying the pneumoconioses including pathology, clinical animal experimentation, radiology and so on.

While there I had the opportunity to study a large number of men exposed to asbestos in the mining and milling process in the Canadian mines and mills and also to a somewhat lesser extent, individuals in the textile industry.

I left there in 1953, went to my current place and although my major interest in the ensuing years was the disease known as emphysema, I still retained an active interest in pneumoconiosis.

In 1965, at the same time that your own Commissioner of Health was becoming a very strong figure in the study of asbestos through his public health service activities, I was asked whether or not I would become the chairman of a committee that was organized in the following way, and I agreed to do so.

The question -- the Quebec Asbestos Mining Association, who are the major producers of

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the Free World's supply of asbestos, realized that there was a need to do more than had been done in the past in the matter of studying workers exposed to asbestos.

They organized the Institute of Occupational and Environmental Health based in Montreal, and they asked me if I would serve as the Chairman of their Scientific Committee; that task being to review and pass upon applications for research and where our Committee felt was important, to try to encourage research, we were charged to do so.

This Committee has been very active and we have been able to encourage a number of investigators, both in Canada and in Europe and in the United States to further study the general problems of the biological effects of asbestos.

Q Is this committee principally active in Canada?

A This committee is based in Canada, but it is United States of America people; Dr. Paul Kotin is a member of the committee. He just left the National Institute of Environmental Sciences.

210
Dr. Marvin A. Cushman is the new Chairman of the Department of Pathology at the Stonybrook University School of Medicine, I think it is called, of New York State.

Dr. John Batey, from Cambridge, England.

Dr. Brown, who was until just recently with U.S. Steel as one of their medical directors.

Dr. Vernon Gregwar of Canada, whose major reason for being on the committee was that -- I don't know the exact title, but he serves as the Medical Chairman or Chairman of the Medical Committee for Workmen's Compensation in the Province of Quebec.

Q Is Irving Selikoff connected with your committee?

A No, sir, he is not on the committee.

Q Is there any specific reason why he wouldn't be?

A No special reason.

Q He has probably other reasons that he is involved in other things?

A He is involved in a great many other things.

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There is no reason other than that our committee was a group of people who had more or less been associated in the past with the exception of perhaps Paul Kotin and we have carried on our work.

Q Are you a member of any professional societies which have a membership consisting of people who are admitted to these organizations because of professional achievements as distinguished from dues-paying memberships?

A If you are asking am I a member of honorary societies?

Q Yes.

A No.

Q Are you a fellow of any one of these societies?

A All the scientific societies that I belong to are dues-paying societies, but you do have to apply and be passed upon, and I am a member of a number of what I would call peer groups.

Q Have you written any books or had any articles published on the subject of asbestos

21 control as we would refer to it as the problem here?

A The engineering side of control.

Q No, the health or the engineering side of it.

A I have not had -- I have not written anything on the engineering side.

I wrote, in 1969, a review of the biological effects of asbestos and I have been privy to, although perhaps not a direct contributor, to the studies of particularly Dr. Corbett McDonald in Montreal.

Q Is this the article that you published then, "Asbestos and Health" in 1969?

A That is correct.

Q And this was published in the "American Review of Respiratory Diseases"?

A Correct.

Q Would you say that this is a reasonably current analysis of the problem?

A Up to 1969, it is.

There are some additional things at the present time.

Q All right.

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A That weren't published then.

Q There has been some evidence admitted at our last hearing about certain substitute products or products which we consider as substitutes for asbestos and one of them has been fiberglass.

A Yes.

Q Have you written or published any articles relative to fiberglass and its health aspects?

A Yes, I examined all of the workers of the Newark, Ohio fiberglass plant of Owens Corning, utilizing their X-rays only. I did not examine the men.

And this is, I think, the second oldest commercial fiberglass producer so that we had a group of men some of whom began as far back as 1938.

I looked at those films with a suitable set of controls and found nothing in those employees' films that would suggest to me any unusual diseases that might be related to any employment.

Q I have here an article, "Airborne Fibrous Glass Particles," written in the "Archives of

3NO
Environmental Health," Volume 16, February, 1968,
by George W. Wright, M.D., Cleveland.

Is that your publication?

A Yes.

Q And does this summarize what you have
just identified for us, your study of the X-rays?

A It does.

Q And you didn't find any adverse health
effects from fibers?

A Well --

Q Let me finish, please.

You didn't find any adverse health
effects from fiberglass particles in the ambient
air on the general public or on workmen involved
with it?

A Mr. Alderman, I didn't examine the general
public, but the workers I did examine and I found
nothing unusual in them.

Q All right.

In the study that you had made on
this fiberglass, how long have these workers been
exposed to fiberglass?

A The longest would have been from 1938 to

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the time of the article.

Q That would be 1968?

A Yes; approximately 30 years.

Q Thirty years?

A Yes.

Q Now, we had evidence at our prior hearings that exposure to asbestos would require about a 20 to a 30-year exposure before any disastrous ill effects are detected.

I believe that the evidence indicated that in certain control groups, they had seen that a man had either been working with asbestos for a long period of time and the detection of something wrong usually came about 20 years later.

The same sort of situations came about with some studies relative to the members of the families of some of these workers who had been handling either the clothing or the children who had played in some asbestos piles or in the community where there were asbestos piles outside.

Also, it took about a 20-year period before some serious health effect came about.

Would you say the same thing would

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generally be true of products that are substitutes for asbestos to some degree that use a lighter form of asbestos?

A Well, in general, it would be correct to say that you oughtn't to give a clean bill of health to a material until you have let 25 to 30 years lapse in the way in which one is exposed.

I think that the kind of reaction that we call asbestosis, which is the scarring in the lungs, does occur before 20 years have gone by.

If the exposures have been heavy enough, you find evidences of change within ten years.

Now, the cancer relationship seems to be a long lapse of time and mesothelioma takes still longer.

Now, the question, as you put it to me, included a number of elements so that I am not sure I am answering what you are getting at, but in general, it would be correct to say that strictly speaking on an operational point of view, we like to have at least a period of 20 to 25 years go by before we say that it is unlikely that anything

ON 3
will show up in an exposed population.

Q Now, as I understand it, it was approximately 1935 when this matter first was called to the public's attention and that was some study regarding an individual down in South Carolina who had been working with asbestos for approximately a 20-year period of time, and I believe that his condition was diagnosed as mesothelioma and I think particularly in view of the fact that they attached significance to this particular study, because if you go back 20 or 30 years from 1935, we didn't have a great amount of cigarette smoking during that -- during those periods as we have had during the subsequent 20 and 30-year period.

Would you say that this would probably be about the beginning of this problem's getting public attention, around 1935?

A There are several manifestations of the effect of asbestos.

The lung scarring that occurs, which we call asbestosis, came to sufficient attention so that a fair amount was known about it by 1930 to 1935 and in 1935, it is true that Dr. Lynch reported

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two cases of bronchogenic cancer in textiles, in men who worked as textile workers.

This was then --

Q That would be asbestos textile?

A Asbestos textile. These were bronchogenic cancers. I think you misspoke yourself when you said mesothelioma.

Q It was not mesothelioma?

A Not in 1935. It was bronchogenic cancers.

That did not direct any great attention to any population because you have to recall in 1935 was when an interest in lung cancer as a whole was just beginning.

It is rather interesting that in 1932 when I was an intern at Barnes Hospital, I was the intern on the first lung cancer ever removed.

Dr. Gilmore, an obstetrician from Pittsburgh who survived many years after that, was the patient.

So that it was a new subject and not much attention was paid to it until about 1948 when Dr. Meriwether reported quite strong evidence of an association between bronchogenic cancer and long-term

3N11 exposure to asbestos in the British textile workers.

So I would take the time of lung cancer as being approximately 1948 to 1950, as having been the time when our attention was directed to it enough so that an extensive study was made in the mines and mills of Quebec to see whether that relationship held and it was not found in the mines and mills.

This study has been repeated, as you know, published in March of this year in "Cancer" on the same population, a much longer period of time now indicating that in the most heavily exposed, there is a relationship to excess cancer, but in the moderate and mild, no relationship.

The mesothelioma came to our attention in 1959 when Wagner reported at the Johannesburg Conference an excess of mesothelioma in a geographic area in northwest Cape of South Africa which he associated with the mining and milling of "persitolite" in that area.

Those are roughly the dates that I think one would hang their hats on.

Q So the significant point is that we have

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various stages of ill being, the least form of which would be asbestosis?

A The least form would be plural plaques.

Q Good, that is a new one. What is that?

A These are little areas of thickening on the inside covering of the chest wall and they can be seen in the X-rays. Sometimes they become calcified and they are rather easily seen.

Q What ill effects has that upon the person?

A No ill effects unless there are very massive -- if they are extremely sensitive, then they can limit the movement of the thoracic bellows, but this is a very unusual extent.

They are common in certain localities and seriously, they are common in individuals who have no other manifestation of asbestosis and by and large, when that man does have the scarring in the lung, he doesn't have the plural plaques, so the plaques have not called much attention to themselves, but that is a fourth manifestation.

Q All right.

Now, this would cause a shortening of breath?

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for them to sit and rest and resume their journey upward."

Do you agree that asbestosis could get that bad?

A Oh, yes, asbestosis can be a fatal disease.

Q Now, the next stage, then, of harm, going up from asbestosis, would it be lung cancer?

A I am a little unhappy with the words "next stage," because they don't lead one to the other.

Q I am speaking as a layman, and I am trying to go into degrees of harm.

A Yes, and I think if I can paraphrase what you say a little; you can have some degree of asbestosis and live a normal length of life. It may not interfere at all with anything you want to do and the longevity would be exactly that as if you didn't have it, whereas if you develop a bronchogenic cancer, this is virtually universally a fatal -- universally fatal. With respect to bronchogenic cancer, the treatment is kind of an ineffective kind of thing.

The people that develop the bronchogenic

311-5
cancer, the excess having arisen out of a certain exposure or certain exposures to asbestos that would be a more severe event than simply getting asbestosis but asbestosis itself can also be fatal.

14 Q Now, speaking again as a layman and you can make whatever adjustment in your answer you wish, recognizing the fact that the question is coming from one not trained in medicine, it would seem that the worst thing that could happen to one that would have a causal connection with asbestos would be mesothelioma which is a much more rare form of tumor.

A It is a much more rare form of tumor but it is no more fatal than bronchogenic cancer is.

There is a remote possibility of curing a bronchogenic cancer, by removing it. The threat for a mesothelioma is exceptionally nil.

Q So that in that regard, it would be obviously worse?

A Yes.

Q So basically then, we have covered the conditions of ill being that might befall a human as a result of exposure to asbestos?

A Yes.

Q Now, the other interesting fact that had come out during our prior hearing, is that the diagnoses which have been made and the studies that have been made usually found that situations where lung cancer developed or where mesothelioma was the diagnosis, that there had also been a connection with cigarette smoking.

A You are correct with bronchogenic cancer.

Now, so far as I know in the reports, in the literature, there has never been a bronchogenic cancer occurring in a man who works with asbestos, who hasn't also been a cigarette smoker.

Mesothelioma, it appears to have no relationship to smoking.

Q I see.

Insofar as the cigarette smoking is concerned, the statistics of the control groups that had been studied, as far as the patient suffering from a form of cancer is concerned, the statistics indicate that the incidence of cancer was so much greater than it would have been in the general public, so that they did definitely make a causal connection to asbestos?

4N17
A Yes.

You have, first of all, an excess from smoking and then if you associate it with substantial exposure to asbestos, there is an added increment of excess.

Q Yes.

So that those situations could not have been ruled out as improper or incorrect because the man had been smoking, also?

A Oh, no, no. I didn't mean to imply that.

Q I didn't suggest that you did. I just wanted to clarify the point.

A Yes. What this leads us to is what we call "carcinogenesis," and the whole thrust is that with cancer there is very few single agents that will produce it. It is usually two or more.

Q E. Carter Hammond, Vice President of the American Cancer Society, there is a statement attributed to him, and it indicates that asbestos could be a greater problem than smoking insofar as cancer is concerned.

Would you agree or disagree with that statement?

4N10

A I disagree strongly with it, and that statement is a misquoted incident. You are quoting it correctly, but the writer misquoted it.

Q Well, I am quoting.

A The writer misquoted E. Carter Hammond.

Q Let me ask you about it as long as we are talking about this same writer and this same writer than talks about yourself and Dr. Selikoff.

He says in here, "Dr. Selikoff is called an alarmist by George Wright, M.D., a pulmonary specialist, pulmonary disease specialist in St. Luke's Hospital in Cleveland."

Did they quote you correctly?

A They did.

(Laughter.)

Precisely.

Q Would you tell why Dr. Selikoff is an alarmist?

A Well, I think Dr. Selikoff gets carried beyond the reasonable extrapolation of the information that he has.

For example, I have heard him say that one fiber in the lung is all it needs to lead

to the development of a malignancy.

Well, now, if you pushed him and said, "Do you really mean that?" he would, I am sure, agree to that because if so, then we are all going to get a malignancy because there are some asbestos fibers in 100 percent of the adult population.

Q You and Dr. Selikoff agree on that point because he did testify that every person that was in the room that day and every person in the City of Chicago has some asbestos in their lungs.

A Surely.

Q He did not testify here, however, that one fiber would cause a malignancy?

A I didn't know whether he did or not, but I have heard him say that.

Q On that point, you have a disagreement, and for that reason, and on that point, you would say he is an alarmist?

A That is one of the reasons.

Q In what other way is he an alarmist?

A For example, he has said on a number of occasions that we can anticipate in the public an

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epidemic of mesotheliomas.

Q He didn't testify to that here.

A I am glad he didn't, but I have heard him say that in my hearing and I just don't think there is any evidence to support that at all.

In the public in general, there isn't even going to be an epidemic of mesotheliomas in the population, to say nothing of the public exposure.

Q Did you ever hear of Dr. J. G. Thompson?

A From Capetown, is that South Africa?

Q Yes, the University of Cape Town.

A I know him well.

Q Well, it says in that very same article "Dr. Thompson is an alarmist as well as Dr. Selikoff according to Dr. Wright."

A That is right, over the same subject.

Thompson also, I know him very well. I have visited with him. Thompson believes that we are going to have an epidemic of mesotheliomas in the general public because they have been able to find asbestos bodies in large bodies of people, in their lungs.

Q Will you tell us some of the studies that

Dr. Thompson did at Cape Town, South Africa?

A Dr. Thompson is the Chairman, professor of pathology at the medical school there and the study for which he is most widely known has to do with having examined the lungs of the general population that came to autopsy in Cape Town and if I recall the numbers correctly, approximately 28 percent of the adults who came to autopsy were found to have what he called "asbestos bodies," which most of us today call "ferruginous fibers," since they are simply fibers that have a metal composition on their surface, some of which are obviously asbestos.

There was a very important piece of work and led to a great many more studies which are still in process to give us a better understanding of the public's exposure to asbestos fibers, but where Dr. Thompson and I parted ways, and which led this interviewer to say that I thought he was an alarmist, was that Dr. Selikoff, or rather, Dr. Thompson has said repeatedly in writing as well as orally that we are going to have a tremendous epidemic of mesotheliomas and this I think is very alarming to say to people unless you can back it up.

4N22
and there isn't anything to support this contention at all.

Q Well, was it Dr. Thompson that performed 3,000 autopsies in Cape Town and I believe as a result of his study, according to what Dr. Selikoff told us here, that 50 percent of them had asbestos bodies which were visible under an ordinary microscope.

A I don't think it was quite that high.

I may be in error on my number, but I had recalled it was on the order of 28 percent.

I don't think he found even 50 percent when he did it in Miami. He repeated the study in Miami.

DR. BROWN: Miami was 20 percent.

CHAIRMAN BILANDIC: Now, was it Dr. Thompson or the studies in Cape Town that called attention to the fact that asbestos does have a detrimental effect on people other than those who work directly with asbestos?

A No, he did not say it had a detrimental effect. He said that the public had been exposed to asbestos and heretofore it had been our view that

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asbestos exposure occurred only in an occupational way and the importance to me of Dr. Thompson's study, and I think he would say the same thing, was to indicate that the public at large did have an exposure to asbestos.

Now, that has led us and others to go on to try to take a look at the nature of that exposure so it was an important piece of work.

Q All right.

A But he did not say it was a detrimental exposure and if I can just say again, this was sort of what led to our parting of the ways on this subject.

We are very friendly socially because he then extrapolated and said that we are going to have an epidemic in mesotheliomas.

I don't believe that what he knew would at all support that, and all I can say is that as of the years having gone by, and we now have a number of years since he made the statement therein, and there has been no increase in the number of mesotheliomas reported in South Africa.

Q I think that there was some evidence given

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last time that approximately 800,000 tons of asbestos are used in the United States annually. That is about a fourth of the world's production; a substantial portion of that is used here in the City of Chicago?

A Yes.

Q And that asbestos is a comparatively new material which really was first started to be used in a big way in the construction of high-rise buildings in about 1958.

So, we now have a greater exposure of the public around construction sites particularly in major metropolitan areas and what we would be looking forward to and what studies like Dr. Thompson had performed, Dr. Selikoff, yourself and some others, to now project a little thought to public bodies such as ours to look into questions such as this because what we must look forward to is what will be the condition of the environment and the condition of the health of the citizens of this community 20 or 30 years down the line, because as we know, people are not giving up smoking cigarettes as quickly as a lot of medical authorities would have

4N-3
liked to have seen us quit, and at the same time, we have this great exposure to the asbestos particulate in the ambient air in urban centers and the combination of the two are working together to present a health hazard.

Now, it is our obligation here to try to ascertain the nature and extent of that hazard and we are seeking help from you and advice from people like yourself.

A Sure.

Q As to what if anything we might anticipate say 30 years down the line, and what if anything we can do now to protect people who have had these exposures since 1958 and those who will have them from this day forward?

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A I think you are absolutely prudent in asking these questions. I think that they should be asked.

Would you like for me to expound?
If so, I will tell you what my thoughts are.

Q Yes.

A I will tell you what my thoughts are about that matter.

Q Yes, please do.

A Let me say at the outset that although the tonnage is high in total asbestos used in the United States of America, it is only a small fraction of that that at the present moment at least is used in insulation work.

The vast bulk of it is not used in that way at all. Nevertheless, it is being used and there is the potential for it to be used more and I think that is what you are concerning yourself about.

Q Yes.

A Well, then if I may have about five minutes?

Q Sure.

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A I will express my views about that.

Q Certainly.

A I think it is important to understand that roughly you would think of three categories of or ways in which people can be exposed to asbestos.

You have first of all the general public who is exposed to asbestos fibers and we know that they are exposed because even the few autopsies done on young people, we find fibers in their lungs.

Now, some of this comes simply from talcum powder which does contain -- most varieties contain some asbestos, and of course, contains talc fibers.

Q Incidentally, there is more talc used in the United States of America than asbestos and no one suggested that we abolish it.

A Not at all.

Q And you aren't either.

A No, and I am not suggesting that you abolish asbestos either.

ALDERMAN FIFILLSKI: Which is worse, talc or

5N3
the baby rash?

(Laughter.)

A But there is, in addition to the increments of asbestos that are cast into the air by industry, there is a natural background already in the air from the weathering of rock and soil.

Serpentine rock is a parent rock, and it contains asbestos fibers as one of the commonest of all of its constituents, that is of the surface of the United States of America, and it is especially strong along the Eastern Seaboard.

Now, in the process of weathering, in the process of tilling the soil and in the process of digging foundations, we have this natural background so that no matter what you might want to do, you will never abolish all asbestos fibers or for that matter, talc fibers in the air of the general public.

Now, the deliberate uses of asbestos undoubtedly present the potential for augmenting this amount of asbestos in the air and so the public does have an exposure.

58
The second kind of exposure that occurs which is an order of magnitude greater than the public, would be what might be called para -- meaning surrounding or around the occupations.

These would be people who had lived in the immediate neighborhood, let us say, of an industry where in the past -- I hope not today -- but in the past, the cleaning up of the in-plant air led to the casting out of asbestos fibers into the air from a stack that you have and thus you have an asbestos emission.

Now, under these circumstances, people living in the neighborhood, especially downwind, have an appreciable exposure.

Para-occupational would be, in the setting that we are talking about here today, would be the men who would work on the same building where the spraying of asbestos products might be going on.

These are individuals not clearly identified as being associated with work involving asbestos, but being in the neighborhood.

Hobbyists come in this category.

51 There are people who have certain hobbies that might involve the use of asbestos products. So that is a second group.

On top of that, now, is the third group, which are those that we can readily identify as being occupationally exposed.

The numbers we know a little bit about in terms of the kind of exposure. Mostly we know about occupational exposure because we have been at it many, many years, and just to use some general numbers, exposures as shown by Dr. Brown's department when he was with the Public Health Service and Dr. Cralley, who is in the room today --

Q He testified this morning.

A -- can check on this.

In the occupational exposures that they looked at, they found fiber counts and I presume he talked about that a little bit because that is the way you categorize exposures -- fiber counts that might run in the neighborhood of 15 to 25 and even 30 fibers per cc in the place where the men works.

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We have reason to believe that they were higher than that in the past and we have reason to believe that there is some places where they are lower.

ALDERMAN FIFIELSKI: Is that in the air that you are talking about?

A That is the number in the air, the number of asbestos fibers per cubic centimeter of a certain size.

They must be longer than five microns and I don't think they count anything over 50 microns in length.

We know a lot about that kind of exposure and I will come back to it in a moment, to what it does to people, because as I say, we have spent our lives since -- I don't say I did, but others and myself since 1949 looking at that.

On the street, if you just go get in a car and drive around and sample city air, there have been a few counts made, and I regret to say that there has not been a systematic publication of this information.

However, I am privy to some of it

51 and these fiber counts are a fraction of one fiber per cc.

These are the light-sized fibers.

In some of the para-occupational exposures, it might be somewhat higher and this would be dependent entirely on the nature of the exposure but it might have the order of magnitude of three or four fibers per cc.

So you see that the public, as we have looked at it so far, as we have looked at it, has an order of magnitude that is a minor fraction of what has occurred during the life of the occupational worker.

The para-occupational, close to the public exposure, it is close to the public exposure, but a little higher.

I might just as well stop here to say that recently there has been discussion of fibers that can only be seen with the electron microscope. Those are called E.M. sized fibers.

Now, here in the number, in the para, it is larger on the order of 30 or 40 fibers per cc, but when you go to the occupational exposures, even

150 today, and look at the E.M. sized fibers, they are of the order of anything from 100 to a thousand per cc -- again very much higher.

Now, I present this background in terms of numbers. I don't want to mislead you. These are not published in the literature as yet. I am privy to them. I hope that they will be published in a systematic way.

I assume that your people have some numbers like that that would be available, Dr. Cralley, if anyone wanted to get them.

ALDERMAN FIFIELSKI: Can I interrupt a moment here and ask when does it reach the danger stage?

A Well, this is what I am going to come to now.

ALDERMAN FIFIELSKI: All right.

CHAIRMAN BILANDIC: Proceed.

A Because we have some frame of reference since then.

Now, where have we seen abnormalities occur? Well, the best place to look for them have been in occupational exposures.

All I can tell you about that is that

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there is a sufficient body of evidence, in my opinion, to give you a very good or some very good -- a very good fix on where you stand.

For example, in Turner Brothers Plant in Manchester, and it is almost an isolated experience because no one else has this kind of information.

It has been possible to look at people exposed only since 1933 and the reason that is is their employment dates from the beginning of 1933 and coming up to the present time, and the interesting thing about that is that in Britain in 1933, without knowing what kind of count to use or anything of that sort, the government indicated that the mills would have to be cleaned up and Turner Brothers in Manchester took this seriously and began to clean it up.

So they have some information going back over that period of time and they have been able to demonstrate to their satisfaction, and I think to anyone who wishes to look at their information, that they have a population of people employed since 1933 in which no asbestosis has

5210 developed, no scarring.

In that same population up to the present time, there has been no excess of lung cancer and no excess of mesotheliomas.

Their fiber counts have ranged around 10 to 12 to 14 per cc.

They are now down to, for the most part, below 8, and they, of course, anticipate getting somewhere between 2 and 5.

The point here is that this is one population that has been under observation by a group of very competent people, in which we have some numbers, and in which we have evidence of freedom from development of scars and bronchogenic cancer and mesothelioma.

In that plant, even today, under their best circumstances, you will find fiber counts running around 5 to 8 fibers per cc.

Now, I submit this is far higher than anybody in the public areas has, at least to the present time been exposed to, because in the public air, it is a fraction of one in contrast to between 5 and 8.

I don't know of any place else where we have this kind of number experience to associate with life experience, but we do have some other information bearing on the subject.

We have, for example, a publication by Dr. Newhouse, examining the population from another textile plant in Great Britain, where absolute numbers are not available, but where she was able to categorize the workers in terms of light, moderate and heavy exposure.

She was able to show, over a long period of time, with her study, no existing lung cancer, no excess lung cancer, no excess mesothelioma in the light to moderate exposures, but definitely an excess of lung cancer in the heavily exposed.

So I can't give you the number as to what is light and moderate because she doesn't have the number.

My educated, and I think I have a right to give an estimate, my estimate would be that she is probably talking about something around ten fibers per cc; for the light and moderate exposure, perhaps as low as five for the light, but

61-2 again, an order of magnitude that is many times higher in that group that has lived successively without hazard, many times higher exposure than in the air we know the general public walks around in.

CHAIRMAN BILANDIC: What is the significance here, Doctor, of your referring to the two studies that have been done in England?

A What is the significance of the two studies I have been referring to that have been done in England?

Q Yes, and how recent are these studies?

A They have been published within the last two or three years.

Q What is the time period during which this control group was studied?

A 1933 to 1966. That is 33 years. That is a long period of observation.

Q Were both of them during that same period?

A Dr. Newhouse's actually included people over a longer period. I can't give you the exact years, but it is over that.

Q How recent is the most recent?

6N-3
Was 1966 the most recent?

A That is the last time that they have published about it.

Q There has been quite a gap from 1966 to 1971?

A Yes.

Q Does Dr. Newhouse pick up from there?

A I think they will continue their studies, but they haven't published anything recently so that I can't tell you about it.

Q What you have testified about with respect to the second group in England, what period did they cover, approximately?

A Roughly -- I wish I had that paper with me -- roughly 1930 to 1965.

Q All right.

A That is about a 35-year period.

Let me tell you one more study which bears on this which is appearing in "Cancer" as of this month.

Dr. Corbett McDonald has looked at a very large population of mining and milling workers dealing only with chrysotile and over the lifetime

6N-4
of these workers, he goes back so that we have an exposure history of over 40 years and he has been able to demonstrate that in the light and moderately exposed, there is no evidence of excessive lung cancer and only in the very heavily exposed is there.

Now, again, I can't give you that in absolute numbers back to those days, but they had to be over 25 to 30 fibers per cc; many times, what you find in the public air.

Q This would be "Cancer" Magazine of March, '71?

A March of 1971. It is supposed to come out then.

Q Is it out yet or not?

A It should be out. I haven't been home enough to look at it, but I have a copy of the paper.

Q Now, this reports a study in England also?

A No, this is in Canada.

Q Canada?

A Yes.

Q All right.

A Now, furthermore, on the mesothelioma question, the Newhouse studies show there was no development of mesothelioma over that period of time

in the light to moderate and Corbett McDonald's group has also shown in the heavily exposed group there has been no mesothelioma.

Now, let me go to one other piece of evidence which I might just tell you has been published also from the Balingero Mine outside of Milan, Italy and the same information that Corbett McDonald has.

Q Yes?

A There is another rather direct piece of interesting evidence on this which goes at it from a 180-degree different approach, which has been reported by Mourrman, a Finnish investigator.

Q In Finland?

A Yes, sir.

Q Regarding the condition in Finland?

A Yes.

Q All right.

A What he did was to take a group of people with lung cancer and age-match them with a group of people who did not have lung cancer.

Of course, he knew nothing about their occupational exposure or anything, but what

61.3
he did not look at the ferruginous bodies or asbestos bodies and said, "That we will use this as our evidence of exposure, the lung having acted as the catchment for catching the fibers," and he found no statistical difference between the number of ferruginous bodies in the group that had lung cancer than in the general public and the group who did not have lung cancer, so this is a piece of direct evidence to support the idea that although the public is exposed to asbestos, the exposure is not of a nature that should lead anyone to believe that it would be a hazard exposure.

Q Well, you seem to have touched upon a rather sensitive area in your evidence as comes from England, Milan, Italy, Finland and Canada?

A Yes.

Q And at our last meeting Dr. Selikoff testified to a control group that he had here in the United States and he had some in New York City where he said he even studied some people here in Chicago, in Newark, New Jersey, and some of the latest areas, and he studied people who are involved directly in the asbestos field.

6N-7
A Yes.

Q The asbestos, I don't have the specific name of the union, but a labor organization.

A Heat and Frost Workers.

Q Heat and Frost Workers Union, and this study revealed that one out of every four or five asbestos workers dies of lung cancer.

A Yes.

Q Do you agree or disagree with those figures?

A I certainly have no reason to disagree with his information.

Now, I take a strong exception to his use of the term "asbestos workers."

They are insulation workers and there is, as far as I -- I think anyone has a right to go, that is about as far as they have a right to go because when there is no such thing really as pure asbestos exposure. It doesn't exist.

The minute you have taken asbestos out of the ground and begun to mine and mill it, you have put trace metals on it.

Now, some of these trace metals are

6N18
known to be carcinogenic in their own right.

Q In 1968, Dr. Selikoff testified before a House Subcommittee on Labor -- of course, he testified to the facts that I just mentioned to you and at the same time he also testified that we may look forward to perhaps 6,000 deaths from lung cancer and to perhaps 15,000 deaths from cancer in general among men in this single asbestos trade in the United States.

Would you agree with that?

A I have no way of disagreeing and I have no reason to think I should.

I don't, in any way, question his data.

Q All right.

A What I am saying is that he talks about asbestos workers and really they are insulation workers.

Q I see.

A Let me just finish one thing which is awfully important.

Not only do you have the problem of waste attached to the asbestos, but these people

are exposed to many other things besides asbestos in the nature of their work.

They are, you know, rather migrant. They work here a while and there a while. They work in different factories.

When they are tearing out insulation, they are tearing out a material that can absorb all kinds of hydrocarbons and so on.

Up to the present time, as far as I know, the actual exposure of the insulation worker has never been categorized.

We don't know what they are exposed to. One of the things they are exposed to, at least some of them, is asbestos, but I am sure they are exposed to many things in addition to that.

Now, to me, this is very important for this reason: You brought it out very well -- the health experience in his insulation worker group is so much more severe than any other group that I know of, so that I have to believe there is something besides asbestos being involved in this.

The same experience does not occur in mining or milling, no matter where you go. It

doesn't occur to the degree, qualitatively, yes, there is an excess of lung cancer in all of these, but the severity of it, which he speaks of, and I have no reason to doubt it, is many times more severe in his insulation workers.

The real question is "What are those insulation workers exposed to besides asbestos?"

Q Well --

A And he has never been able to say and I don't think he can. I think he would like to know.

Q If you categorize Dr. Selikoff as an extremist --

A Yes.

Q At the same hearing before the same Congressional Committee, Dr. William H. Stewart, former Surgeon General of the United States --

A Yes.

Q -- told the same Subcommittee that insulation workers are probably sharing their exposure, to a lesser degree, with more than 3.5 million other construction workers.

These are steamfitters, electricians, carpenters, iron workers, plumbers, masons, tile

setters and so on who are on the job when the spraying takes place.

A Sure.

Q Now, we have evidence based upon the Cape Town studies of Dr. Thompson that we do have these, as you categorize them, para-occupational people.

A Yes.

Q They have had exposures?

A Sure.

Q There have been reports of higher incidents of lung cancer among them?

A Yes.

Q Then down in Cape Town at the same time, they had found that the families of asbestos workers, the wives that would wash the clothes, the children who were exposed to the fathers coming home with some of this asbestos fiber perhaps on their clothing or on their bodies and so forth, and they have found again, whether you agree or disagree, this is what we would like your evaluation on, they have found that in studying those people and even further in Cape Town among the general public, and in the 3,000

61-2
autopsies that have been performed, the conclusions that they brought, from people who had nothing to do with working there, but who lived in the vicinity, not necessarily that close to where asbestos was being used and mined or worked with; they find this causal connection between exposure to asbestos and the conditions of ill being, whether it be asbestosis, lung cancer, or mesothelioma.

17

How do you reconcile that?

A Well --

Q Or are they reconcilable?

A Well, they are reconcilable because the situation isn't quite as you presented it, if I understood you correctly.

Now, they have found evidences of fibers in the lungs. There is no doubt about that, but now I speak of the study done by Dr. Thompson.

He did not relate this in any way to any disease of any kind. His study, he simply said that the fibers are there. He did not relate it to lung cancer. He didn't relate it to mesothelioma or to asbestosis.

Now, the para-occupational groups

7N23 where our relation has been made, run about as follows.

Let us first speak of mesothelioma in South Africa. I visited there and I spent a fair amount of time looking into it. These were all around "Kourinan" in the Northwest Cape. The roads are paved with asbestos tailings.

The children played on the waste heap from the asbestos mines.

The women cobbled asbestos and by is meant that they took the pieces of asbestos in the rock and knocked the rock off and they made a few pennies this way and they showed me a picture taken around some of the mines where the vegetation was nonexistent, covered with blue crocidolite.

Now, you may think of these people as being inhumane, but they weren't inhumane.

They just didn't know that anything was going to happen, but you call that a para-occupational. It is essentially the same as occupational because these people lived in very, what I would consider to be, a very substantial asbestos exposure.

This is where the mesotheliomas

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developed in South Africa.

Now, let me tell you a very curious thing, that at this moment is still unexplained.

The same blue asbestos is mined in the Eastern Transvaal; approximately 20 percent of the blue asbestos produced in South Africa is the Eastern Transvaal. All of the world's amosite is mined in the Transvaal.

This is still in South Africa, now, and there hasn't been a single mesothelioma reported from the amosite workings, and only one in contrast to some of the last count, 160, only one from the blue asbestos mines and mills in the Transvaal.

Now, this is an exceedingly important thing because you then must ask: Why are there 160 mesotheliomas in the Northwest Cape and essentially none and yet the same materials are being looked at in the Transvaal.

Now, that is the mesothelioma story, no one knows.

The bronchogenic cancer story in Africa, at this date, that hasn't been recorded.

Now, as to the mesothelioma story

716
to question that whatsoever.

I would say, however, that I don't think he knows what they have been exposed to in addition to asbestos.

He doesn't even know the severity of the asbestos exposure. If so, he has never published anything about it, and in his first workings or writings, he said that upwards of 80 percent had asbestosis.

I can only believe that they had a very heavy exposure and they probably have had exposure to other carcinogens.

Now, Dr. Stewart's extrapolation to the para-occupational workers, let us say the other men working in the building, is a very important thing to say because those workers must be protected. There is no question about it.

I would expect them to have a much lighter exposure even if you did nothing, because of the dilution factor, and the further you go from the source, the more diluted anything becomes.

However, I think that those people deserve protection by all the means that you use

7N-7
for protecting any other occupational population.

If you have, for example, in a cement plant where you make asbestos pipe, at the dry end where you start, you dump asbestos together with cement and with sand, sometimes a few other things, you have to circumscribe that area so that all the way down the line the rest of the men are not exposed. There is no question that you have to do that; if you are going to work with sprayed asbestos, it is my view that you must control where it is going to go.

You must protect not only the man who is spraying, but the man who works in the neighborhood and if you do do that, you will protect the general public.

Q Doctor, we are certainly grateful for one thing that you have done here today and you started to, and hopefully before you are finished, we hope to get more out of you and that is up until this point, no one who has come here to testify had been able to give us any specific evidence as to what level of exposure to asbestos would be "dangerous" if that is a proper medical term.

You are the first one that gave us some hint at this.

I understand that there is a study being done by NAPCA and I believe that there is some research going on here locally in Chicago probably under contract to NAPCA or some other federal agency and perhaps this information might be released soon.

A Yes.

Q Do you have -- and we, of course, are awaiting very eagerly for the release of this type of information so that it may be presented here and evaluated and determined as to whether it is feasible or not.

Do you have an opinion as to what may or may not be a danger level of exposure to asbestos for the general public?

A Well, I can best answer that by saying the following:

I think that an exposure under five fibers is suitable for the occupational man as a safe level; five fibers per cc and you must remember that the man who works in the plant is

also a public citizen so he has the public exposure plus the in-plant exposure.

Now, I think if five fibers in-plant is adequate, surely then something less than that would be safe for the public and we are nowhere near that in the public air.

If you were to say to keep it below one fiber, I think that would be or could be lived with easily. It could be achieved and would be an absolutely safe level.

ALDERMAN FIFIELSKI: Are there any cumulative effects on this?

A Yes, but that is all taken into consideration. There is some accumulation. The bulk of what you inhale comes back out again with the lung cleansing mechanism.

CHAIRMAN BILANDIC: You are the first --

A Very little is kept in.

CHAIRMAN BILANDIC: You are the first one to give us this information also.

A Well, this I can document for you very well in the following way, very dramatically:

The people exposed to silicates;

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silicosis is a very well-known thing.

Now, a number of men now have analyzed the lung at the time that a man dies who has worked, say, 35 or 40 years in a silica exposure and analyzed for how much silicate is in the lung and by making an estimate of what was in the air, how much air he breathes, could estimate how much he took in and people like Policard and others who have looked at this, have shown quite consistently that approximately 99 percent of what you inhale is brought back out.

You only retain somewhere in the neighborhood of 1 percent of the total that you take in.

Q All right.

That which is taken in and not exhaled, that would remain in the lungs.

Now, that will not be assimilated or eliminated by the body?

A Well, the fact if you want to use that word or you want to now just talk about asbestos?

Q Let's stay right with asbestos.

A Yes, because other things dissolve and go

lung and these materials can sit in there and glass, for example, will sit in there. Asbestos will sit inside those cells until the cell dies and the material is spewed out and then another "macrophage" comes along and picks it up, but the beauty of putting it in the "macrophage" is that it isolates it so that it doesn't do anything to the other cells of the body but silicate free crystalline silicates kills the macrophage itself. It is lethal and asbestos is not.

It is lethal in cell culture and all of this is to say that I wish I knew more about the fate and most of it comes back out.

Some is retained free in the tissues, some within these "macrophage" cells that I have talked about.

Now, there is a great oddity about this because you will find that right next to each other, an asbestos fiber that is coated and another fiber that is naked. Now, why one gets coated and one stays naked, I don't know.

How many fibers does it take to stimulate scar, I don't know.

87 It takes in a substantial number, though.

Q Well, we have found other areas of disagreement now, or at least difference of opinion which we will be able to evaluate when we make our judgment.

Doctor, I would like to pursue another area and that is that there seem to be two schools of thought.

Some people are advocating the abolition --

A Yes.

Q -- of the use of asbestos or asbestos containing materials in, for example, construction sites.

Would you advocate such a thing?

A Under proper conditions, I surely would not advocate it.

Q All right, fine, that is sufficient.

Let's proceed further.

The other school of thought seems to be that we should control the application and the use of asbestos at a construction site, for example?

81
A Yes.

Q So as to minimize the exposure for the workers, their coworkers and for the general public.

This is a recommendation that we have received from various witnesses that we should consider some type of control.

Would you agree with that?

A I think absolutely that you must have a control.

Q Now, would you have any specific suggestions to give to us as to what controls we might consider in order to protect the worker, his coworkers and the general public?

A Well --

Q On construction sites, for example?

A Yes, sure.

I think that for the -- for this you do exactly what you do in any other occupational setting.

You would say that the general public should not be certainly exposed to more than five fibers per cc.

I would think, because it is easy to

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do that that you ought to set a limit lower than that. I would pick two or one because you can live with that.

The para-occupational worker should never be exposed to more than five and the man inside or the man actually doing the spraying, I would hope would not be exposed to more than five, either, because this is good occupational practice in the textile mill or where you do anything else.

I will go one step further with that and that is that we do have some situations where this is true.

In coal it is true. In silica it is true. In asbestos it is true. In every other material that has a potential for being toxic it is true.

You have some situations where, while you are developing the methodology for this kind of control, you permit someone to continue work but wearing an absolutely protective mask even if that requires outside air through, blown through a hood and I stress the word "absolutely

protected," because you shouldn't permit people to work with an ineffective mask, giving the impression that it is protecting the worker. This is not right.

Now, I would hope that you would aim in the place where the man is actually spraying and getting him down to what we aim for in any other occupational place setting, and that is under five.

If it is not possible, then he should be there alone and anyone who is there, including himself, should have the kind of protection that you would insist on in any other occupational setting and that is that inside the mask he shouldn't be getting more than five fibers per cc.

Q Now, Doctor, when you talk about the exposure of five fibers per cc or a lower amount as far as the general public is concerned, is that made optically or electromicroscopically?

A This is optically and we all know at the same time that there are E.M. sized fibers there always at the same time that you look at it optically,

817
but you don't have to count the E.M. sized fibers.

Q Doctor, we would like to get to the root of the problems here in Chicago, and we would like to handle them as quickly and expeditiously as possible.

A You are getting closer to the root than anybody I have ever talked to.

I have never had a drilling like this.

(Laughter.)

CHAIRMAN BILANDIC: We don't like to get carried away with some frivolous aspects.

A Yes.

Q Or some minimal aspects.

A Yes.

Q We would like to get to the major sources and put as reasonable controls as we possibly can which would be consistent with carrying on the business and occupation of these various industries that are involved.

A Yes.

Q And to that end, the evidence has revealed so far that the major sources that we should look to

8.8
and consider as far as protecting the public is concerned, is number one: Construction sites, because they cause a substantial amount of exposure because of the very nature of the high-rise buildings that are being built in the large metropolitan areas.

A Yes.

Q Secondly, that we should look to some means of protection for the public in some form of controls on demolition sites.

A Yes.

Q Because once again we have a high-rise building which has the asbestos affixed to the steel skeleton and in the demolition process, obviously we have the same kind of exposure to the asbestos as we probably would have in the construction, only in reverse order.

A Not quite.

Q Not quite, but it is secondarily and it is also a major source according to the evidence that we have so far.

The third one would be industrial uses. Would you agree with that?

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the public air. It would be applicable, for example, to buildings that have a few -- some do have sprayed asbestos coating in the plenum where there is a return air for part of the air conditioning system, and I think that you should be sure --

Q That is a serious problem?

A -- be sure to keep the air of the general public free of anything over five, and I am sure you can do that because there are some data that indicate when it is properly done it is a fraction of a fraction that comes loose and gets into the public air.

Demolition is interesting because we all have that mental picture of a big steel ball swinging against a building and so on.

Well, it is true that there is a lot of demolition of that kind, they do it now with a big clamshell bucket, but in those circumstances, there is very little free asbestos available and there are some studies going on of that kind of demolition.

318
The cloud of dust is there, but when you look for actual asbestos fibers in the original

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material there wasn't much and it is pretty well locked in.

Q Again, depending upon the age of the building?

A Depending on how far back the building went.

When you talk about this new process of spraying steel girders, I have spoken to a number of demolition people and they say "You will never take one of those buildings down that way because in taking down the girders you don't do it with a swinging ball or grabbing a hold of it with a clamshell bucket."

These are taken down individually. Now, you will have a problem once the girder is down before you have it moved off and you will have a problem getting rid of the material, but there you have the opportunity to wet it down and to take it off.

So I don't really think that demolition 25 years or 30 years down the road is a valid question.

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newspapers play it up as something serious.

Is there any creditable scientific evidence that you have found anywhere that this is a problem?

A Yes, there are two very good pieces of evidence. First of all, the studies that have been done by the Public Health Service, I think initiated when Dr. Brown was there on this very question in which they looked at the degradation products of brake linings as it is used and it is perfectly true that asbestos does go into the brake linings originally and in that occupation, you must protect the workers and the man who puts the brake linings on, especially in the old days when they had to bore holes through it, you had to protect him, but once it is out on the street --

Q That is the point where I am speaking about.

A Now, when you apply the brake at a point source of friction, the heat is extremely high and that is the whole point of having asbestos in it, to hold it together and when the heat gets high enough, you convert asbestos to "forsterite," which

9N
is a totally different material.

It isn't asbestos at all.

Forsterite in its own right, I don't mean to be poetic, has been tested by being put into animals and it is biologically inert as far as scarring is concerned.

Roughly I think the figures that the British who did exactly the same study and was it Lynch -- oh, he is gone -- of the Public Health Service, but less than 5 percent of the total amount of asbestos involved in the brake linings ultimately comes out as free asbestos.

Now, there is a very minor amount --

Q So it is no problem?

A Not in my mind, it isn't, and I think the best evidence for that is that when you go around making studies of how much asbestos is in the air at the highway, on the city streets, you find it well below one fiber per cc. It is a fraction of that.

Q Doctor, now, I have one final question which will somewhat obviously have a direct bearing on your testimony today.

91-5
Your present affiliation is with the so-called asbestos industry, if there is such an industry.

A I am affiliated with them in the following way; as I am with -- I am a consultant for Republic Steel, I am a consultant for the National Gypsum Company. I am a consultant to the Federal Government, let me just make that very clear.

Q This isn't intended to be any criticism.

A No, and as a matter of fact, I had planned to say it when we started.

Q I wanted to save it until the end, because I thought we should give you the opportunity to make your statement.

A The Chairman of the Committee -- I am the Chairman of the Committee of the Quebec Asbestos Mining Association, an independently set up group and the whole purpose of this is to develop and support research.

I am Chairman of that scientific committee and I am not on their Executive Committee or anything else, and all we do is recommend to the

9N
Executive Committee whether or not we think they should fund the research.

I serve as a consultant directly to the President of the Johns-Manville Corporation mostly for their in-plant problems.

Q Yes, and they of course, are a major user of asbestos.

A They asked me to do this because they recognize their in-plant problems and they wanted to do something about it and National Gypsum Company which also uses some asbestos, I read all of their X-rays all over the world.

Q My principal purpose in bringing that out is to bring a specific focus to your point of view.

A Sure.

Q For the opinions that you have expressed here today.

A Sure.

Q And would I then be fair in characterizing it as saying that yours is probably the most conservative type of evidence that we could receive on this subject from that point of view?

98
A I think of myself as being an unbiased --
and, that is, as unbiased as I can be.

I do have some biases as we all do,
but I can give you examples of where facts have
made me change my mind in this total problem.

"Conservative," if you want to
use that word, I will accept that.

Q Conservative as applied to asbestos,
not otherwise?

A Yes.

CHAIRMAN BILANDIC: Do you have any questions?

ALDERMAN FIFIELSKI: I have one. Off the
record.

(Discussion off the record.)

CHAIRMAN BILANDIC: My colleague has been a
little bit concerned because he has been a con-
struction worker who had sprayed asbestos on
steel girders and I happened to frighten him
during the course of this hearing.

ALDERMAN FIFIELSKI: Off the record.

(Discussion off the record.)

CHAIRMAN BILANDIC: Thank you very much, Doctor.

A I wrote a letter to Dr. Fundsack.

CHAIRMAN BILANDIC: You want to put it in the record?

A Would you like a copy of it?

CHAIRMAN BILANDIC: If it is free for publication.

A I asked Dr. Pundseck if I could give it to you and he said yes.

CHAIRMAN BILANDIC: All right, fine.

A So far as I am concerned, you can have it.

CHAIRMAN BILANDIC: Then it is in the public domain.

A The grammar isn't too good because I wrote it just before I left and my secretary signed it, and you will find one sentence repeated there with bad typing and a bad secretary.

CHAIRMAN BILANDIC: Would you want to put it in good grammatical form?

A No, the content is all right.

CHAIRMAN BILANDIC: As we do with most of the work of this committee, we put it in our Municipal Reference Library.

A So long as it goes directly to you.

CHAIRMAN BILANDIC: Thank you, and if there is no objection, we will incorporate it and make it a

part of the record.

A I must say, Alderman Bilandic, that it has been a real pleasure.

I have not had anybody to question me this nicely before and I don't mean myself personally, but you have a good understanding of the question, the problem, and you ask the right questions.

CHAIRMAN BILANDIC: Thank you very much, Doctor. We are searching for the right answers and we thank you for your help.

A You're welcome.

(Witness excused.)

CHAIRMAN BILANDIC: We will take a short recess.

(The following proceedings were had after a short recess:)

CHAIRMAN BILANDIC: Mr. John Quinn, Asbestos Workers Union Local No. 17.

JOHN QUINN,

called as a witness herein, testified as follows:

CHAIRMAN BILANDIC: Q Will you state your name, please?

A John Quinn.

— R71-16 —

EXHIBIT 11 (d) —

May 11, 1971

B2 2

J. MUNROE MC NULTY,
called as a witness herein, was examined and
testified as follows:

EXAMINATION

BY

CHAIRMAN BILANDIC:

Q Mr. McNulty, do you have a prepared
statement?

A I do.

Q Do you have any extra copies for the
other Aldermen?

A Yes, I do.

Q Would you pass them on to the Aldermen?

A There was a slight error which is now
being rectified.

Q Do you have one for Alderman Stemberk?

A Yes.

Q You may proceed, Mr. McNulty.

A My name is J. Munroe McNulty.

I am President of the McNulty
Brothers Company, a firm of lathing and plastering
contractors and we do a lot of spray fireproofing.

I am also President of the International

2na Association of Wall and Ceiling Contractors.

As President of the International Association, I have had contact with spray fire-proofing contractors throughout the country.

In the course of these contacts, an exchange of information has taken place on matters of our present concern.

B3 My comments today concern the control of procedures for the containment or the prevention of the release of asbestos and other fibers to the ambient air during the application of spray coating.

As an illustration, for discussion purposes, I will discuss the regulation of sprayed fiber coatings presently occurring in New York City.

New York City has adopted, through their Environmental Control Agency, an order requiring the containment of asbestos particulate matter in spray coating application; hoping to contain the particulate matter to the immediate vicinity of the application; and they are doing this through the use of tarpaulins followed by

Association of Wall and Ceiling Contractors.

As President of the International Association, I have had contact with spray fire-proofing contractors throughout the country.

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3na Based upon my knowledge of the industry's experience both in New York City and Chicago, containment is not a successful procedure for the control of asbestos fibers from spray coatings.

In my judgment, a ban on the use of asbestos containing spray fiber coatings is superior to containment as a control procedure.

I will be happy to answer any questions which you gentlemen might have.

CHAIRMAN BILANDIC: Yes, Mr. McNulty.

The subject of the Port of New York Authority Building was the subject of much discussion during our prior hearings.

As I understand it, that was supposed to be the world's largest fireproofing job.

A I think that is correct.

Q As I understand it, that job started with the use of an asbestos type of spray and there was an attempt to tarp that building and enclose it in accordance with the Interim Order then in existence in New York?

A I believe that is right.

Q And about halfway through that job, I understand that it was stopped because of the failure of the enclosures.

A That is correct, to my knowledge.

Q All right.

We also had some discussion during the last hearing that we had here, in reference to a building under construction in the Chicago area; namely, the IBM Building which is right up here on the north side.

A I am somewhat familiar with it, although I am not the contractor on it.

Q The job was being done with a type of material that had some asbestos in it, and of course, there were some articles in some medical journals and other periodicals that appeared which indicated that it was "snowing asbestos" around that job site.

I guess, regretfully, the man who was doing the job was doing it on a bad day when the editor of the Sun-Times was going into his office and I guess that is where the big attention factor came to us.

312
However, nevertheless, are you familiar with the attempt to tarp that job?

A I am somewhat familiar with it.

I have discussed the matter with the contractor on an occasion or two.

They attempted a very thorough job of tarping the building, but found that the high winds just made it impossible to maintain their tarpaulins as an integrated covering around the building.

The high winds that we get -- and, of course, New York gets the same winds -- why, it was practically impossible to maintain the integrity of the covering.

The wind was strong enough to tear them loose from the floor, and as a procedure for saving the tarpaulins, they found they were better off to release them on one end and let them fly out because if they didn't do that, the wind was strong enough to tear them apart.

Q I understand the contractor sustained a substantial loss on those tarpaulins.

A Well, he didn't discuss that with me.

B31

I would expect that his loss would be in excess of 50 percent of the tarpaulins that he started with, and I would expect that it would be at least that high from my own experience.

Q So then, based upon your experience in this field, you have found that enclosures of this type have not been successful, and therefore, you are recommending to us that we consider a ban on the use of sprayed asbestos for fireproofing?

A I would say so.

I would like to reinforce my opinion by pointing out that I fireproofed the First National Bank Building and also the John Hancock Building, and I have some knowledge of fireproofing high buildings.

Q Well, I didn't ask you for your qualifications, because your reputation is so eminent that I thought everybody would be familiar with it, particularly in your capacity as President of the Association and also as a top contractor in the field.

I am glad that you did add that for the purpose of the record.

Now, of course, we did go through the question of substitute materials and the question of cost.

Mr. Dubin testified earlier here today and he indicated that the cost factor of asbestos and nonasbestos materials is substantially the same, of course, with the added saving of not having the tarpaulins, that using a nonasbestos material would, of course, hold down the cost of the total job.

A The cost of tarping in New York has been so great that the spray fireproofing contractors have been unwilling to undertake the responsibility for providing it and the responsibility for enclosing the buildings for spray fireproofing operations has fallen on the general contractors and Mr. Bracco indicated to me that they were quite extensive and represent a substantial cost.

Q So based upon the inadequacy of the enclosure and based upon the cost factor, you would then recommend that we consider a ban?

A Yes, sir, I would.

CHAIRMAN BILANDIC: Are there any questions

3N1
by any of the Aldermen?

Alderman Singer?

EXAMINATION

BY

ALDERMAN SINGER:

Q Just briefly, the previous witness testified that he felt that substitute materials, nonasbestos materials, were just as good in terms of fireproofing.

Is that your opinion as well, based upon your experience?

A Well, I don't know that I would state that as an immediate fact.

I think that it is possible to develop comparable materials.

We are using a sprayed fiber material ourselves in one or two places that is an asbestos-free material, and it seems to perform reasonably well, although Tishman had an unhappy experience with that same material in Cleveland.

However, I have had Skidmore, Owings & Merrill look at the material as we

3M3
were applying it in the shopping center in Schaumburg and they felt that it was an acceptable material out there.

As architects go, they are pretty tough. They don't accept marginal products.

Q I see.

A You have a gentleman here from the Zonolite Division of W.R. Grace, who I think can give you information on his product.

Q He testified before.

A He has?

Well, then you have had his statement on that. I don't think that asbestos is an essential ingredient. I think it can be worked around.

CHAIRMAN BILANDIC: We also heard from Underwriters' Laboratory on the subject.

Underwriters' Laboratory always has to approve all of these materials.

A Well, they are a testing laboratory, which provides a testing service. There are other laboratories who have the same equipment and produce a comparable test, but there aren't

3M3 many. They are limited and the places that you can go to -- that is, we are limited in the places we can go to to get recognized fire tests. There are only about four in this hemisphere.

ALDERMAN SINGER: Q What did you use on the Hancock and on the First National Bank Buildings?

A On the First National Bank Building, we introduced a product manufactured by U.S. Gypsum Company, which is almost identical with the material that Zonolite manufactures.

On the Hancock Building we used a material manufactured by the National Gypsum Company, which, instead of using vermiculite, we used perlite as an aggregate, and we experienced some very grave difficulties in pumping the materials or the material, rather, because of the abrasiveness of the aggregate.

The material has been withdrawn from the market for that reason, but for no other reason.

The other ingredients in the mix were very similar to Monocote of Zonolite or U.S. Gypsum.

3N3
CHAIRMAN BILANDIC: It was a good material, but it wrecked your equipment?

A Correct.

Q We can't have too much of that either.

A Well, I can't.

ALDERMAN SINGER: Q Is that asbestos-free?

A There may have been a trace of asbestos in it. I believe that the cementitious materials, as they were then being made, ran between 3 percent and 10 percent asbestos.

ALDERMAN SINGER: Thank you.

CHAIRMAN BILANDIC: Alderman Stemberk?

EXAMINATION

BY

ALDERMAN STEMBERK:

Q Mr. McNulty, you have mentioned that the problem in New York in relationship to the ineffectiveness of the ban is a problem between the Building Department and the Environmental Control Department.

Could you expand on why this problem exists?

A The Environmental Control Department

~~# 1011~~
12

Chapter 17 of Municipal Code Amended Defining
Asbestos Matter and Banning Usage Thereof

The following is said ordinance as passed:

Be It Ordained by the City Council of the City of Chicago:

Section 1. Chapter 17 of the Municipal Code of Chicago, Section 17-2.1, is hereby amended by adding thereto a new definition, to be alphabetically interposed in said section, as follows:

Asbestos: A fibrous, rock-forming mineral including, but not limited to, such amphibole varieties as tremolite, actinolite, anthophyllite, grunerite, richterite, edenite, amosite, crocidolite, and such serpentine varieties as amianthus and chrysolite, as well as synthetic asbestos fibers including, but not limited to, fluor-tremolite, fluor-richterite, and fluor-edenite.

Section 2. Chapter 17 of the Municipal Code of Chicago is hereby amended by adding thereto a new section, Section 17-2.4A, as follows:

17-2.4A. On and after January 1, 1972, it shall be unlawful within the City of Chicago, and within one mile of the corporate limits thereof, for any person, firm or corporation cause or to permit the spraying of any substance containing asbestos, as defined in this Article, in or upon any building, structure, column, frame, floor, ceiling or other portion, part or member thereof, during its construction, reconstruction, alteration or repair; provided, however, that such enclosed factories, buildings or structures in which the fabrication or manufacture of products containing asbestos is carried on shall not be subject to this provision.

Section 3. This ordinance shall take effect ten days from the date of its due passage and publication.

— R71-16

EXHIBIT 13

~~##~~
#13

**RECOMMENDED
HEALTH SAFETY
PRACTICES**

*For Handling
And Applying
Thermal
Insulation Products
Containing
Asbestos*

for additional copies, write:

**Executive Secretary
National Insulation Manufacturers Association, Inc.
441 Lexington Avenue, New York, N.Y. 10017**

nima
NATIONAL INSULATION MANUFACTURERS ASSOCIATION

**Fabrication in
Warehouse and
Job Site Shops**

- 1 Open and remove the material from cartons with care.
- 2 Handle and stack pieces of material in a manner that will avoid generating dust and breakage.
- 3 Mechanical dust collecting systems adequately designed to remove dust at the source should be installed on power-operated, fixed and portable equipment in the warehouse shop and/or in the job site shop.
- 4 All machines should be furnished with suitable waste containers for the collection of scrap and waste to prevent the material from falling on the floor and generating dust.
- 5 Shop-fabricated pieces of Thermal Insulation such as fittings, segments, etc., should be placed in containers. Care should be taken when handling these pieces during piling, stacking and packing.
- 6 Containers should be closed after packing and remain closed until the material is used.

Application Procedures

- 1 Unpacking and application of the material should be done in a manner that will minimize airborne dust.
- 2 Avoid dropping, throwing or unnecessary rough handling of insulation materials.
- 3 Keep floors and surfaces clean of scrap, shavings and debris. Put unusable material into waste containers immediately.
- 4 Hand sawing, cutting, jacket stripping, etc. should be done in a manner that will create minimal airborne dust. Use of proper methods and tools will help considerably towards dust abatement.
- 5 When working in confined spaces, portable dust collectors or exhaust blowers should be provided to remove dust from the source.

(Continued on Next Page)

Application Procedures (Cont'd)

- 6 If conditions prevent collecting or exhausting dust from the source, use exhaust blowers to provide general room air changes.
- 7 Avoid exhausting into other working areas.
- 8 In confined spaces, if mechanical exhaust equipment cannot be used, *Respirators should be worn.
- 9 When wiring or banding insulation and applying jackets or facings, care should be taken to avoid generating excessive dust, using the precautionary measures described above.
- 10 Surplus usable materials should be placed in cartons, which should be closed and returned to the warehouse.

*Bureau of Mines-approved respirators. See page 13.

CEMENTS

Warehousing,
Storage and Handling

Handling and storage of closed bags containing cements do not present health safety hazards provided bags are unopened and not broken.

- 1 When unloading railroad cars and trucks, if there is excessive loose material resulting from opened or broken bags, *Respirators should be worn during clean-up and unloading.
- 2 Spillage should be cleaned up and open or broken bags repacked, resealed or discarded immediately to prevent distribution of dust into other areas.
- 3 If bags are damaged and spillage occurs in the warehouse, material should be cleaned up immediately to prevent distribution into other areas. Vacuum equipment is recommended for this cleaning.
- 4 If large quantities of spillage occurs and cleaning involves sweeping and/or shoveling that could create excessive dust, *Respirators should be worn.

*Bureau of Mines-approved respirators. See page 13.

— R71-16

EXHIBIT 14 —

Fibrous and Mineral Content of Cosmetic Talcum Products

#12
#14

L. J. CRALLEY, Ph.D., M. M. KEY, M.D., D. H. GROTH, M.D.,
W. S. LAINHART, M.D., and R. M. LIGO, M.D.

Occupational Health Program, National Center for Urban and Industrial Health,
10 Broadway, Cincinnati, Ohio 45202

In searching for sources of fibers ubiquitous to our everyday environment and of respirable size, the authors examined 22 talcum products commonly available on retail shelves and found fiber contents ranging from 8 to 30% by count, with an average of 19%. Fibrous particulates were generally under 1.0 μ in diameter with lengths ranging from 1.5 to 6.0 μ . From 0.3 to 3.0% quartz was found in 21 of the samples and the remaining sample had 54.4% quartz. The samples were also analyzed for metals; with four exceptions, the levels of cobalt, nickel, chromium and manganese were low. Further research will be needed to assess the significance of these findings.

REPORTS OF FINDING pulmonary fibrous bodies, previously referred to as "asbestos" and now as "ferruginous" bodies, in the lungs of persons coming to autopsy in hospitals in a number of cities have recently been increasing. The first report of these morphologically distinctive fibrous bodies in the sputum and lungs of asbestos workers was made in 1906 by Marchand.¹ Current interest dates from 1963 when Thompson *et al.*² found these fibrous bodies in the lungs in 26.4% of the autopsies in a series of examinations in Cape Town. Subsequent investigations³⁻⁵ provide evidence that the occurrence of these bodies in the lungs of urban residents is not restricted to those in isolated localities and is not a one-time chance observation.

The identity of the fibrous core of these coated bodies, however, was never determined by the investigators. The fibers were routinely considered as asbestos bodies on the basis of their morphological structure.

In searching for the sources of these ubiquitous fibers, Cralley *et al.*⁶ reported that talcum powder contained a significant percent-

age of respirable fibers and was a potential source of the ferruginous bodies observed in the lungs of humans. This observation led to further study to characterize cosmetic talcum products.

The major purpose of the investigation reported here was to develop and present data on some of the constituents found in cosmetic talcum products and to discuss their health aspects in the light of today's knowledge. It is not our intent to make a general appraisal of health factors in the use of talcum products because of the many variables involved and the limited data available on the consumption of various "sources of tales" in the formulation and use of cosmetic talcum products. The potential health aspects of some of the data, however, are discussed.

Twenty-two cosmetic talcum products (representing body powder, bath powder, and all purpose powder) purchased off-the-shelf, were analyzed for fibrous content, selected metals, and quartz. The data and a discussion of their possible significance follow.

Analysis of Talcum Products

Talc is a natural mineral, hydrous magnesium silicate, with the general formula $(OH)_2$

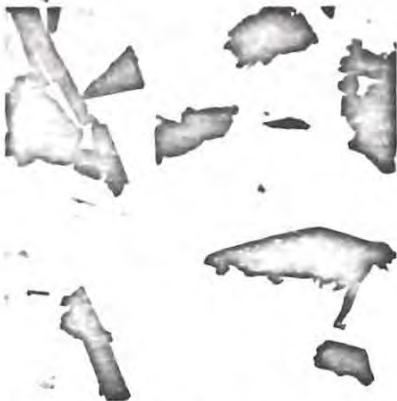


FIGURE 1. Photomicrograph of talcum specimen showing presence of fibers (430 X).

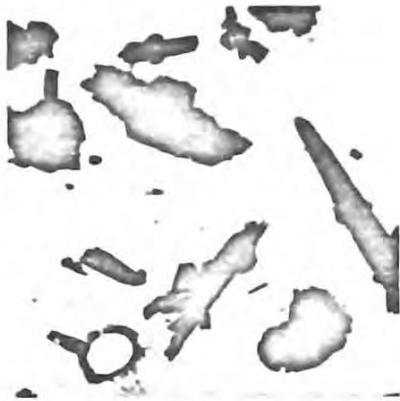


FIGURE 2. Photomicrograph of talcum specimen showing presence of fibers (430 X).

generally under 1.0μ in diameter, with lengths ranging from 1.5 to 6.0μ .

The 22 talcum products analyzed showed fiber contents ranging from 8 to 30% by count of the total talcum particulates with an average of 19%. Although the specific fibrous materials were not identified, they were predominantly fibrous talc, as shown by x-ray diffraction, with the probable presence in minor amounts of other fibrous minerals such as tremolite, anthophyllite, chrysotile, and pyrophyllite.

The electron microscope, with its higher power of resolution, shows a number of sub-micron diameter particulates not visible by means of phase contrast microscopy.

Free Silica

In 8 of the 22 talcum products (Table I), the presence of quartz ranged from 0.3 to 1.0%; in 13 products, 1.2 to 3.0% quartz, and in 1 product, 54.4% quartz.

Metals

With the exception of talcum products Nos. 4, 8, and 22 (Table I), the cobalt content of the products analyzed was under 25 parts per

million by weight (ppm); chromium, under 22 ppm; nickel, under 29 ppm; and manganese, under 78 ppm. Product No. 4 had a nickel content of 1270 ppm; chromium, 340 ppm; and cobalt, 67 ppm. Product No. 8 contained 479 ppm nickel and 329 ppm chromium. Product No. 22 contained 1210 ppm nickel and 1170 ppm chromium. Qualitative tests showed some of the chromium in the talcum products to be in the hexavalent state. The nickel, chromium, cobalt, and manganese in the talcum products may have come from the talc mineral or zirconite or from the alloy metals of the pigging equipment used in reducing the talc.

The zirconium content of the products was all under 10 milligrams per gram (mg/gm) except for products Nos. 9 and 17, which had 20 and 30 mg/gm respectively. The titanium, zinc, and iron ranged from a few tenths to 50 mg/gm of talcum and were probably present as pigments or opacifiers. The magnesium content of the products was over 10 mg/gm, except for product No. 9 which had only 0.5 mg/gm. The magnesium was probably present as an additive in the formulation or as a part of the talc molecule or other amphoteres in the products.

Acknowledgments

The authors acknowledge, with appreciation, the technical assistance of Harold B. Norris who made the free silica determinations, Patricia L. Maurer who made the atomic absorption spectrophotometric determinations for nickel, cobalt, chromium, and manganese, John R. Carlberg who made the emission spectrographic determinations for zirconium, titanium, zinc, iron, magnesium, and aluminum, and Stephen Bayer and Ralph Zumwalle for the fiber determinations.

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Human Factors Association of Canada

On June 1 a group of 29 people gathered in Toronto, Canada, and formed the Human Factors Association of Canada. It is anticipated that the Association will have membership throughout Canada and will represent interests of the many disciplines concerned with man's functioning in his physical and machine environments. The following officers were elected: President, Dr. R. B. Bromiley, Vice-President, Dr. E. Llewellyn Thomas, Secretary-Treasurer, Mr. Ronald E. F. Lewis. The following were named Directors: Dr. J. R. Brown, Dr. A. C. Bryan, Dr. Ruth Hoyt and Mr. J. F. Martin. Correspondence should be addressed to: Secretary-Treasurer, Human Factors Association of Canada, c/o Defence Research Establishment Toronto, Box 2000 Downsview, Ontario, Canada.

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Table III

Mortality experience of 632 members of IAHFIAW, Locals 12 and 32
January 1, 1943 to June 30, 1971

Cause of Death	Number	Per Cent
Lung cancer	85	20.0
Pleural mesothelioma	7	1.6
Peritoneal mesothelioma	20	4.8
Gastro-intestinal cancer	40	9.3
All other cancers	36	8.4
Total cancer = 188		
Asbestosis	32	7.7
All other causes	205	48.2
	425	100.0

Table IV

Asbestos Air Levels in United States Cities

Analysis of 187 Quarterly Composite Samples

Asbestos Air Levels (10 ⁻⁹ grams/m ³)	Number of Samples With Given Value
0.0-0.9	61
1.0-4.9	103
5.0-9.9	12
10.0-19.9	8
20-49	2
50-99	1

— R71-16

EXHIBIT 15

Cigarette Smoking, Asbestos, and Pulmonary Fibrosis¹

WILLIAM WEISS

SUMMARY

A group of 100 asbestos textile workers was examined by chest roentgenogram and a questionnaire concerning age, sex, smoking habits, and duration of occupational exposure to asbestos. The roentgenograms were examined for evidences of pulmonary fibrosis without knowing the results of the questionnaire. Seventy-five workers were cigarette smokers and 25 were nonsmokers. The prevalence of pulmonary fibrosis was 40 per cent in the smokers and 24 per cent in the nonsmokers. The factors of age, sex, and duration of exposure to asbestos did not account for this difference. The prevalence of pulmonary fibrosis increased with increasing amount and duration of cigarette smoking and with increasing duration of exposure to asbestos.

Introduction

The association between pulmonary fibrosis and cigarette smoking has been demonstrated in both pathologic and radiographic studies (1-4). In 1963 Auerbach and co-workers (1), without prior knowledge of smoking habits during life, studied lungs obtained at autopsy and reported an association between smoking habits and pulmonary fibrosis. In the following year, these observations were confirmed by Boucot and associates (2) on a gross radiographic basis during a study of male smokers with lung cancer, matched control subjects who were smokers without cancer, and matched control subjects who neither were smokers nor had lung cancer. Further studies by the present writer (3, 4), who read chest films without knowledge of smoking habits, provided

additional support for the histologic observations of Auerbach in women as well as men. The association between pulmonary fibrosis and cigarette smoking was dose-related, suggesting that the relationship was causal.

Asbestos inhalation also produces pulmonary fibrosis (5), which is probably dose-related. The question arises as to whether smoking and asbestos can produce additive or even synergistic effects with regard to pulmonary fibrosis. Selikoff and associates (6) reported a highly significant observation when they noted that among asbestos insulation workers, a group known to have a high incidence of bronchogenic carcinoma, the only men in whom this disease developed were cigarette smokers. This association of factors might also apply to the development of pulmonary fibrosis in workers exposed to asbestos; therefore, a pilot study was undertaken in an asbestos textile mill.

Materials and Methods

A group of 100 workers in an asbestos (chrysotile) textile mill was examined by chest roentgenogram and a questionnaire concerning age, sex, smoking habits, and duration of exposure

¹From the Department of Preventive Medicine, The Medical College of Pennsylvania, 3300 Henry Avenue, Philadelphia, Pennsylvania 19129; and the Department of Medicine, Hahnemann Medical College, 230 North Broad Street, Philadelphia, Pennsylvania 19102.

TABLE 2
PULMONARY FIBROSIS IN 98* ASBESTOS MILL WORKERS BY
CIGARETTE SMOKING STATUS AND DURATION OF ASBESTOS
EXPOSURE

Smoking Status	Asbestos Exposure (years)	Total No.	Pulmonary Fibrosis				Age Adjusted (%)
			Mild (No.)	Severe (No.)	Total (No.)	(%)	
Nonsmokers		25	5	1	6	24	23
	20	11	0	0	0	0	
	20+	14	5	1	6	43	
Smokers		73	24	5	29	40	40
	20	45	12	1	13	29	
	20+	28	12	4	16	57	

*Two cigarette smokers without information on duration were excluded.

ly significant. Comparing the prevalence of fibrosis, in groups I, IIA, and the two groups in IIB (table 3) by the chi square method, $P < 0.005$.

Four of five instances of severe fibrosis occurred among the persons with more than 20 years of exposure to asbestos, and 3 of these were among the 19 who, in addition, had smoked a pack or more per day for 20 years or more.

It should be noted that all but one of the smokers who had smoked less than 20 years also had less than 20 years of exposure to asbestos. This was explained by the youth of this group. Thirteen (68 per cent) of the 19 workers who smoked less than 20 years were less than 40 years old compared to only

2 (5 per cent) of the 41 who smoked 20 years or more. Duration of smoking is inextricably bound up with age in groups as small as these so it is not possible to age-adjust the prevalences of fibrosis. A similar problem exists to a lesser extent with respect to duration of exposure to asbestos. The data suggested, however, that the duration of exposure was important because among workers less than 50 years of age, the prevalence of fibrosis was 18 per cent of 38 exposed for less than 20 years compared to 37 per cent of 16 exposed for 20 years or more and among workers aged 50 years or more the prevalence was 30 per cent of 20 exposed for less than 20 years compared to 65 per cent of 26 exposed for 20 years or more.

TABLE 3
PULMONARY FIBROSIS IN 73* CIGARETTE-SMOKING ASBESTOS
MILL WORKERS BY DEGREE AND DURATION OF SMOKING
AND BY DURATION OF ASBESTOS EXPOSURE

Smoking Habits	Asbestos Exposure (years)	Total No.	Pulmonary Fibrosis			
			Mild (No.)	Severe (No.)	Total (No.)	(%)
I. <1 pack/day		13	2	1	3	23
	<20	5	1	0	1	20
	20+	8	1	1	2	25
II. 1+ pack/day		60	22	4	26	43
	A. <20 years	19	4	1	5	26
	<20	18	4	1	5	28
	20+	1	0	0	0	0
	B. 20+ years	41	18	3	21	51
	<20	22	7	0	7	32
	20+	19	11	3	14	74

*Two cigarette smokers without information on duration were excluded.

†Per cent not calculated when denominator is less than 10.

Discussion

At the time of this study, the textile plant had 142 workers but 42 refused examination. Unfortunately, the plant was closed soon after the completion of the examinations, the workers disbanded and the records were inaccessible; therefore it was impossible to compare the unexamined workers with the examined ones for possible sources of bias.

The reading of pulmonary fibrosis on roentgenograms is subject to error (7). In this study, however, all the readings were made by the same observer without prior knowledge of either smoking habits or duration of exposure to asbestos. Thus, one would expect any error in the film readings to be of similar magnitude and in the same direction in all cases so that bias would not be expected with respect to the factors studied.

Asbestos exposure is undoubtedly associated with pulmonary fibrosis, and the available evidence (1-4) suggests that cigarette smoking is likewise associated with pulmonary fibrosis. One would expect that pulmonary fibrosis would be most common when both factors are operating. The data in the present study indicate that this is so. There is a higher prevalence of fibrosis in asbestos workers who smoke cigarettes than in those who do not.

It is uncertain whether the effect of these two factors is additive or synergistic. In a previous study (3) of 999 people surveyed by 70-mm chest roentgenograms, the prevalence of pulmonary fibrosis was only 1.5 per cent in 393 nonsmokers and increased progressively with increasing amounts and duration of cigarette smoking to 20 per cent of 55 people who had smoked more than one pack per day for more than 20 years. Information on occupation was not obtained in that survey, but from the nature of the population examined, it is likely that few or none were asbestos workers. Therefore, an estimate is available of the prevalence of pulmonary fibrosis in heavy long-term cigarette smokers not exposed to asbestos.

In the present study, there were 14 people exposed to asbestos for 20 years or more who did not smoke cigarettes and the prev-

alence of fibrosis was 45 per cent. If the effects of smoking and asbestos exposure were additive, a prevalence in the neighborhood of 65 per cent would be expected. In the present study, there were 19 people who had smoked one pack or more per day for 20 years or more and who had also been exposed to asbestos for 20 years or more. The prevalence of fibrosis was 74 per cent. It is not possible to decide whether these data indicate an additive or a synergistic effect because the numbers of people in these groups were too small, and the two surveys were not strictly comparable. For example, the survey of 999 people presumed to be unexposed to asbestos was done with 70-mm films whereas the survey of asbestos workers was done with 14- by 17-inch films. Similar studies of larger magnitude are needed to clarify this matter.

RESUMEN

El fumar cigarrillos, asbestos y fibrosis pulmonar

Un grupo de 100 trabajadores de asbestos textiles fué examinado con radiografías del pecho y con un cuestionario concerniente a la edad, sexo, hábitos de fumar y duración de la exposición ocupacional a los asbestos. Las radiografías fueron examinadas para ver las evidencias de fibrosis pulmonar sin saber los resultados del cuestionario. Setenta y cinco trabajadores eran fumadores de cigarrillos y 25 no eran fumadores. El predominio de fibrosis pulmonar fué de 40 por ciento en los fumadores y de 24 por ciento en los no fumadores. Los factores de edad, sexo y duración de la exposición a los asbestos no fué la razón de esta diferencia. El predominio de fibrosis pulmonar aumentó con el aumento de la cantidad y la duración de fumar cigarrillos y con el aumento de la duración de la exposición a los asbestos.

RESUME

Cigarette, asbestos et fibrose pulmonaire

Un groupe de 100 travailleurs textiles de l'industrie de l'asbeste ont été examinés par des examens radiologiques du thorax et soumis à un questionnaire qui considérait l'âge, le sexe, l'habitude de fumer et la durée de l'exposition professionnelle à l'asbeste. Les clichés radiologiques ont été examinés en vue de détecter

— R71-16 —

EXHIBIT 16

The lake then remained deep for several thousand years and may even have overflowed. About 5,500 BP a change in the hydrologic environment is reflected in an increase in carbonate production and the eventual coarsening of bottom sediments, culminating in the deposition of shallow facies at 3,100 BP. At 3,100 BP water level rose slightly and then fell again, maintaining this low position until about 2,000 BP. Trees found in positions of growth near and below present water level and dated at 1,890 BP (N-390) were submerged by rising waters at this time. The trees belong to the last period when water level was below that of today. The lake rose and remained deep until after European settlement. In the late nineteenth century it began to fall and in 1969 stood at its lowest level for the past 1,900 yr.

In nearby Lake Gnotuk, submerged trees have been dated at $1,865 \pm 85$ BP (GX-0149, E. D. Gill, personal communication). Synchronous oscillations in lakes 20 km apart provide strong evidence that the controlling factor was climate rather than local hydrologic variations. Furthermore, the major drying between 18,000 and 15,000 BP is in close agreement with evidence documented from lakes in different hydrologic settings in north Victoria and south-west New South Wales recorded by J. M. B.² The rise at about 10,000 BP, reflecting an increase in precipitation or decrease in temperature, cannot yet be correlated accurately with events elsewhere in Australia, although Kershaw³ suggests a wet period in north Queensland at this time.

Effects of a Post-Glacial thermal maximum are weakly represented by a slow fall in water level and a corresponding rise in carbonate production from 5,500 to 3,100 BP. The magnitude and implied rates of these changes suggest conditions little different from those in the area today. The data therefore do not support the hypothesis of widespread Post-Glacial aridity so widely claimed in Australia³⁻⁷. Although temperatures from 5,500 to 3,100 BP may have been slightly higher than in the preceding interval, the degree of aridity would almost certainly not have been sufficient to cause the vegetation destruction and widespread landscape instability which are sometimes attributed to events of this time.

Conditions moderated at 3,100 BP when a slight rise of lake level occurred, but the major rise marking the beginning of the next relatively wet period did not occur until 1,900 BP.

Oscillations in Lake Keilambete can be matched in other lakes in a variety of settings in south-east Australia supporting the interpretations of the water level changes discussed here. The sequence of palaeoclimatic conditions probably has general application through south-east Australia and it may help provide a reliable basis for climatic correlations within Australia and between different continents in both hemispheres.

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Asbestos Fibres in Beverages and Drinking Water

The hazards of inhaling asbestos fibres^{1,2}, the high incidence of gastrointestinal cancer^{3,4} and abdominal neoplasms^{5,6} among asbestos workers and the fact that asbestos fibres may penetrate the mucosa of the stomach and the intestine of animals^{7,8} are known and so it is important to study the distribution of asbestos fibres in the environment. Asbestos fibres have already been found in beer⁹, and the occurrence of fibres 0.5 nm in length and 0.01 nm in diameter suggests that such material may find its way through filtering systems into city drinking water supplies, and this we have been able to confirm.

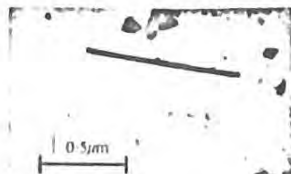


Fig. 1 Electron micrograph of asbestos fibre isolated from beer (enlarged 31,000 times).

The asbestos fibres in 400 ml. samples of beverages and water were concentrated by centrifuging in excess of 16,000g, drying at 100°C and ashing at 500°C¹². The ash was taken up in 1 ml. of distilled water and agitated in an ultrasonic vibrator to disperse the fibres and ash particles¹². Five μ l. of this dispersion was evaporated with the aid of a heat lamp on 200 or 400 mesh carbon coated copper grids. Using a Siemens Elmiskop 1A electron microscope at 80 kV and a magnification of 20,000, the number of fibres in each of five or ten grid holes was counted, and corrected for dilution. Precautions were taken to prevent contamination of samples from extraneous airborne fibres and no fibres were detected in distilled water.

Table 1 Asbestos Fibres in Beverages and Water

Sample	Source*	No. of fibres $\times 10^3$ l ⁻¹
Beer	Canadian 1	4.3
Beer	Canadian 2	6.6
Beer	USA 1	2.0
Beer	USA 2	1.1
Sherry	Canadian	4.1
Sherry	Spanish	2.0
Sherry	South African	2.6
Port	Canadian	2.1
Vermouth	French	1.8
Vermouth	Italian	11.7
Soft drink	Ginger ale	12.2
Soft drink	Tonic water I	1.7
Soft drink	Tonic water II	1.7
Soft drink	Orange	2.5
Tap water	Ottawa, Ottawa river (F)	2.0
Tap water	Toronto, Lake Ontario (F)	4.4
Tap water	Montreal, St Lawrence river (F)	2.4
Tap water	Hull, Quebec, Ottawa river (NF)	9.5
Tap water	Beauport, Quebec, St Lawrence river (6 km below Quebec City) (NF)	8.1
Tap water	Drummondville, Eastern Townships, Quebec, St Francois river (F)	2.9
Tap water	Asbestos, Eastern Townships, Quebec, Nicolet river (F)	5.9
Tap water	Therford Mines, Eastern Townships, Quebec, Lac à la Truite (NF)	172.7
Melted snow	Ottawa, top 30 cm (2-3 weeks precipitation)	33.5
River water	Ottawa river, at Ottawa	9.5

* F, Filtration plant used; NF, no filtration plant used.



Fig. 2 X-ray diffraction pattern of asbestos fibre shown in Fig. 1.

blanks which were treated in the same manner as the liquid samples.

Twelve brands of Canadian beer, wine, sherry and port, six of American beer, six of European wine, vermouth and sherry and two of South African sherry were examined and all were found to contain asbestos fibres. Counts made on some of these brands are reported in Table 1. The fibres in Canadian beer (Fig. 1), sherry and water were identified as being chrysotile asbestos by comparing their electron diffraction pattern (Fig. 2) with that of pure chrysotile asbestos. Some of the European samples contained fibres of amphibole asbestos. Many asbestos fibres were hidden in clumps of debris such as mineral matter in water and diatomaceous earth which is used as a filtering aid in beer. As a result, the number of fibres reported in Table 1 must be considered a minimum estimate of those actually present. Most of the fibres found in Canadian beer and in filtered city water were less than 1 µm in length while most samples of wine, some American beer and unfiltered melted snow or river water also contained a considerable number of asbestos fibres ranging from 1 to 15 µm in length.

Tap water from three major Canadian cities, Ottawa, Toronto and Montreal, combined from 2.3 to 4.4 million fibres per litre. All of these cities have filtration systems and Ottawa tap water, drawn from the Ottawa river, had considerably fewer fibres than unfiltered river water. Many of Canada's major asbestos mines are located in the Eastern Townships of Quebec and filtered tap water from towns in this area did not differ greatly from that in the larger cities but unfiltered tap water in Theford Mines, drawn from a small lake in the heart of an asbestos mining area, was exceptionally high in fibre content. The high level of asbestos fibres in melted snow in the Ottawa area gives some indication of one source of asbestos in surface waters.

The question as to the hazard of orally ingested asbestos has not yet been resolved. The amounts by weight of the asbestos found in these samples were exceedingly small—about 1 µg per 10 million fibres—obtained from counts on 1 mg samples of fibres of approximately the same size as those found in the beverages and water. Further work is required to determine the extent to which the fibres penetrate the walls of the digestive tract, the degree to which they may be transported to other organs of the body and the biological effects of their residence in various tissues. Studies of this nature are now in progress.

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BIOLOGICAL SCIENCES

Molecular Conformation of Deoxyuridine

The structure determination of deoxyuridine was undertaken as part of a series of conformational studies of nucleic acid components. Such studies give information about the preferred conformation of nucleosides and nucleotides^{1,2}, and may clarify the conformational differences and changes that occur in nucleic acids. The conformational parameters of interest in deoxyuridine are the pucker of the sugar ring, the torsion angle of the sugar relative to the base about the glycosidic bond and the orientation of the C(5')-O(5') bond about the C(4')-C(5') bond.

In ribose and deoxyribose nucleosides, the most common type of pucker of the sugar is either C(2') endo or C(3') endo. In the deoxynucleosides the C(2') endo pucker is by far the most common. Since one of the nucleosides with a C(3') endo pucker is thymidine³ it was of particular interest to know whether deoxyuridine would also have this type of pucker. Even the same nucleoside in different environments can have a different type of pucker⁴, but these differences have only been observed in ribose nucleosides. There is therefore a need for further studies of deoxy nucleosides and nucleotides.

Crystals of deoxyuridine were obtained from aqueous solutions. The unit cell is monoclinic with dimensions $a = 7.91$ Å, $b = 6.71$ Å, $c = 18.77$ Å, $\beta = 96.6^\circ$ and the space group is $P2_1$. There are four molecules in the unit cell and therefore two molecules in the asymmetric unit. Intensity data were collected with a Hilger and Watts linear diffractometer using molybdenum K α radiation. The structure was solved by Patterson interpretation methods. The first step was the determination of the orientation of the plane of the pyrimidine bases in the unit cell using the $I(0,0)$ function of Tollin and Cochran⁵. The orientation of the pyrimidine base within the plane was determined by means of a rotation function⁶, and the positions of the molecules relative to the Z_2 axis were found using the $Q(X_0Z_0)$ function of Tollin⁷. Full details of the structure determination will be given elsewhere. The R factor at the present stage of refinement is 0.08.

The conformation of both molecules in the asymmetric unit is very similar. The pucker of both sugars is C(2') endo, or C(2') endo-C(3') exo with respect to the three-atom plane C(1')-O(1')-C(4'). Displacements of the C(2') atoms in molecules I and II relative to this plane are 0.508 and 0.383 Å respectively, and of atoms C(3') they are 0.111 and 0.210 Å respectively. Thus the sugar pucker in deoxyuridine is like that in the majority of deoxynucleosides and differs from that in thymidine.

The glycosidic torsion angle, ϕ_{C4-C5} , defined by Donohue and Trueblood⁸, is -28° in molecule I and -24° in molecule II.

— R71-16

EXHIBIT 17

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tract appears to be a macromolecule with a mass of approximately 30,000 to 50,000 daltons, it is thermostable and destroyed by trypsin. It is apparently not stable on long-term storage, even in the cold.

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Tale-Treated Rice and Japanese Stomach Cancer

Abstract. The Japanese prefer tale-treated rice in their diet. The incidence of stomach cancer in Japan is unusually high. Most tale has some asbestos contamination. Epidemiologic evidence is presented that the asbestos-contaminated tale on rice is the diet carcinogen or cocarcinogen responsible for the high incidence of Japanese stomach cancer.

The high incidence of stomach cancer in Japanese men is a recent, a challenging problem in cancer epidemiology. Study of age-adjusted rates of mortality from stomach cancer in 24 countries showed that from 1962 to 1963 Japanese men had the highest rate for all nations; the rate, 63.96 per 100,000 in the male population, was seven times that for men in the United States (1). Since tale (recently contains asbestos) (2), and rice workers exposed to asbestos show a markedly increased in gastrointestinal cancer, especially stomach cancer (3) the hypothesis that the increased rate of cancer of the stomach in Japan is due to ingestion of rice contaminated with asbestos seems attractive and has been recently suggested in a recent note (4). This report presents evidence for this relationship a somewhat greater length.

Rice grown in California and ancient for the American consumer is milled mechanically. Rice is prepared differently for the Japanese consumer. It is milled and treated with glucose, and then tale is added. The tale is held to the surface of the grain and is said to preserve its flavor better. Japanese consider this rice more tasty, both here and in Japan. Two percent of all tale produced in California from 1959 to 1963 was used as a rice additive, primarily for export to Japan (5). Rice containers in Los Angeles markets intended for the Japanese customer usually bear the legend: "Coated with glucose and tale." Optical microscopy of the ash of such store-bought rice

showed 3.7×10^6 asbestos-form fibers per gram (Fig. 1).

Tale, the mineral, is a hydrous silicate of magnesium. So are some forms of asbestos. The difference between these two minerals is not in their chemical compositions, but rather in their structure, asbestos being fibrous and mineral, tale being flaky or granular. However, commercial tale is composed not only of the pure mineral tale, but also of fibrous silicates, which are in part classifiable as asbestos. There is



Fig. 1. Photomicrograph of the ash of a specimen of rice intended for Japanese consumption and purchased in a Los Angeles market. Arrows indicate asbestos-form fibers. The specimen contained 3.7×10^6 such fibers per gram.

no firm mineralogic definition of commercial tale, and in most areas the mineral tale forms much less than half of commercial tale. The composition of commercial tale varies with the deposit. Vermont mines produce a platy or granular type of mineral relatively free of asbestos minerals (6). New York and California talcs contain more fibrous species, including anthophyllite and tremolite (2, 5).

Repeated asbestos and tale dust cause similar disorders in workmen. Older tale workers show more than four times the expected incidence of lung and pleural cancer (7). Tale or talcose minerals, including contaminating asbestos, are apparently the responsible carcinogens since, where confirmatory evidence could be obtained, all those tale workers with pleural and lung cancer also had tale pneumoconiosis. Autopsies on six tale workers with tale pneumoconiosis showed the presence of the characteristic "asbestos body" in the lungs of every one (8). When tale is examined by phase contrast microscopy, from 8 to 30 percent of the particles are fibrous silicates. Identifiable tremolite, anthophyllite, and chrysotile are also seen (9). The large tale deposits of New York and California regularly contain the amphibole asbestos minerals, tremolite and anthophyllite. These are normal ingredients of commercial tales from these areas. Chrysotile asbestos occurs in veins in talcose mineral deposits and may be then mixed with the tale and create a contaminant (9).

Asbestos is an established carcinogen. Not only does it cause lung cancer, but it is associated with a substantial increase of morbidity from stomach cancer (4). Although chrysotile is the most important commercial fiber of asbestos, there is no reason to regard the amphibole asbestos minerals as safe. To the contrary in one study an amphibole asbestos mineral predominated in asbestos-bodies in the sputum of a group of men exposed to asbestos and having a high incidence of mesothelioma (10). The fact that tale workers exposed to fibrous tale, which contains amphibole asbestos, develop more pulmonary injury than do workers exposed only to tales relatively free of fibrous silicates or amphiboles (11), indicates the pathogenicity of amphibole asbestos.

The Japanese preference for tale-coated rice means that rice eaters of that nation are exposed to the hazard of ingestion of amphibole asbestos, as

RECOMMENDATIONS FOR HANDLING ASBESTOS

(E.E.U.A. Handbook No. 33 : 1969)

This Handbook was prepared by E.E.U.A. Panel M 41 (Handling Asbestos) which was constituted from representatives of the following member organizations:

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Imperial Chemical Industries Ltd.
Lankro Chemicals Limited
Shell International Petroleum Company Limited
United Kingdom Atomic Energy Authority

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FOREWORD

Medical hazards in industry have been known from antiquity and new ones are constantly being discovered. It is the duty of management, sometimes legally and always morally, to take every possible step to protect their workmen.

This booklet relates to asbestos, a substance which makes an interesting study in the evolution of occupational hygiene. The ill-effects of inhalation of the dust were known to the Romans; Pliny in the 1st Century A.D. describes the use of respirators by asbestos miners. Nineteen centuries later, in 1931, the Asbestos Regulations were formulated in this country and now, in view of much recent research, they have been revised in order to give protection to whole groups of workers who were not previously recognized as being at risk.

Naturally, the ideal solution is to use a safe substitute, which is the policy being progressively implemented by members of E.E.U.A. However, for cases where substitution is not practicable, and to deal with existing installations, the following sections describe the nature of the hazards and the ways of overcoming them in accordance with the proposed new legislation. It is not an unattainable counsel of perfection; precautions along these lines are already in use in many factories all over the country, and it is hoped that managements will do all in their power to comply with the spirit as well as the letter of the Law.

NOTE—This handbook contains suggestions as to how users of asbestos may comply with the Asbestos Regulations 1969 (S.I. No. 690), but the Association accepts no responsibility for the duty of users of asbestos to comply with the Regulations, and compliance or failure to comply with the suggestions made in this handbook will in no way relieve from liability any user who fails to comply with the Regulations.

SECTION ONE THE NATURE OF THE PROBLEM

(a) Introduction

The new Asbestos Regulations (see Appendix I) will soon be coming into force. The purpose of this section is to give the background to this, and some general principles which may be adopted to help to comply with the Regulations.

It has long been known that there are two important hazards associated with the inhalation of asbestos dust:

- (i) **Asbestosis** A dust disease of the lung, the main symptom of which is breathlessness and which is usually progressive even after exposure has ceased and may eventually result in death after several years.
- (ii) **Cancer of the lung** This disease is much more prevalent in sufferers from asbestosis than in the rest of the population.

These two hazards normally require a considerable exposure over many years and the Asbestos Regulations of 1931 were designed to protect against them. However, in recent years it has been found that the incidence of asbestosis has been rising (from 43 cases in 1961 to 82 cases in 1965) and that the majority of new cases occur in the user industries not covered by the 1931 Regulations. Thus, out of 315 cases, 58 per cent were in unscheduled occupations such as ladders, sprayers and brake liners (see paper by J. C. Gilson)*. The need has therefore arisen for the 1931 Regulations to be amended to include those user occupations where there is much exposure.

In addition there is a third hazard which the 1931 Regulations do not adequately cover, viz:

- (iii) **Mesothelioma** A very malignant tumour affecting the pleura (lining of the lungs) or peritoneum (lining of the abdominal organs). It is particularly associated with crocidolite (blue asbestos) but it cannot be concluded that only this type of fibre is concerned with these tumours and further investigation of this problem is needed. (See P21 of "Problems arising from the use of asbestos"* and "Asbestos and

* See Appendix IV

health hazards²). Its main feature is that it may develop after relatively slight and intermittent exposure and so the user industries are involved, the risk being not only to their employees actually working with asbestos, but also to those in their vicinity. These workers are not covered by the 1931 Regulations and so the need for the new legislation, to include occupations where the exposure is slight, is evident. In a recently presented series of 31 cases of mesothelioma in the U.K. (Newhouse and Thompson)* only ten were in jobs to which the 1931 Regulations applied.

Mesothelioma has another characteristic: it usually takes a very long time to develop, often as long as 10 years after the initial exposure. In addition to the other two hazards mentioned above, it was made a compensable disease under the Industrial Injuries Act (1966) and a register of all patients who have died is kept by the Medical Inspectorate of the Department of Employment and Productivity. It is of course, important to keep a series of proportion, mesothelioma is still a rare disease, and imports of total asbestos imports. The now less than 1 per cent of total asbestos imports. The following procedures are recommended in order to reduce the hazard to the lowest possible level.

(b) Substitution (See Appendix III)

The problem is solved if a safe, acceptable substitute can be used instead of asbestos. This is sometimes possible, but if not, the type of asbestos should be carefully selected. Crocidolite (blue asbestos) is much more important in the development of mesothelioma than chrysotile (white asbestos) and other forms of asbestos and should never be used unless it is absolutely essential. The requirements of the new Regulations regarding the use of crocidolite are very stringent.

(c) Selection and medical control of workers

All should be medically examined before employment and re-examined annually. Chest X-ray is valuable in the early detection of

asbestos and cancer of the lung, but has limited use in the control of mesothelioma. A register of asbestos workers should be kept. The medical officer should explain to the worker the nature of the hazard and how he is being protected.

Medical officers should exercise particular care when there is a history of chronic bronchitis or other chronic respiratory disease; of rheumatic heart disease or coronary artery disease; of hypertension and of rheumatoid arthritis. The decision to accept or reject the worker will depend on the severity of the illness and the amount and duration of exposure likely.

In spite of every precaution, one cannot be *absolutely* certain that the risk of mesothelioma is reduced to nil. A similar situation exists in other industries which have a cancer hazard. The two following procedures in selection help to reduce an exceedingly small risk still further:

(i) When engaging new staff, men over the age of 40 should be given preference over younger men. This practice has been accepted for many years in the British dyestuffs industry where there is a cancer hazard, and it is recommended by Scott & Williams in their "Code of Conduct for the Manufacture and Use of Products causing Tumours of the Bladder" (See Appendix IV).

(ii) Where possible, the numbers of men exposed should be limited. Smaller numbers of men are more easily protected and controlled and they are more likely to use all the precautions than if they were casually and occasionally exposed. This procedure is also recommended by Scott & Williams.

(d) Monitoring

Environmental dust and fibre concentrations should be measured (see Section Three) and kept within the suggested limits. If this is not possible, the precautions specified in the Regulations must be scrupulously observed. These measurements involve skilled procedures and the necessary advice should be taken.

(e) Working precautions

See Section Four (Page 11).

* See Appendix IV

SECTION TWO LEGAL REQUIREMENTS

a. General

In the past before May 1970 work on an industrial scale with materials containing asbestos has been controlled by the Asbestos Products Regulations, 1931, and in a more general manner by Section 63 of the Factories Act. Removal of dust and fumes. As implied by the title, the old Regulations were directed primarily at the control of dust within factories producing materials or articles containing asbestos.

The Asbestos Regulations 1969 apply to ALL situations where operations entailing the use of dust are carried out on materials comprised wholly, or partly, of asbestos.

The Regulations require *inter alia* that

- i. A. The processes be carried out under an exhaust draught or to secure other equally safe manner designed to ensure that the concentration of airborne asbestos is below a specified limiting value; see below; or
- B. Where A is not practicable, protective clothing and approved forms of respiratory equipment shall be provided.
- ii. All internal surfaces of a building shall be kept free from asbestos waste and dust.

A usual cleaning equipment should be used for this purpose and where this is impracticable personnel undertaking the cleaning and any others in the close vicinity must be provided with protective equipment.

- iii. In any part of a building to be used for the first time for a Scheduled Process, see Regulation 13, for a period exceeding eight hours per week:

- A. All the interior surfaces shall be smooth, impervious, and with minimum dust-trapping surfaces; and
- B. A vacuum cleaning system shall form part of the permanent structure.

iv. Less onerous requirements of the Regulations are:

- A. That all asbestos (both virgin and waste materials) shall be stored and distributed in containers which prevent the escape of dust.

- B. The accommodation and cleaning of protective equipment (see Section Four);
- C. The conditions under which "young persons" may be employed.

b. Implementation

i. The necessity for making use of dust extraction or personal protective equipment will be related to the concentration of asbestos fibres in the atmosphere of the "workplace". For this purpose Threshold Limit Values have been provisionally defined as follows:

Average concentration of asbestos dust		Sampling period		Interpretation of Regulations	
Fibres	mg/m ³	10 min	1 h	Regulations will not be enforced	
Less than 2	Less than 0.1			Each of improvement depends on concentration and duration of exposure	
2 to 12	0.1 to 0.6			Full compliance with Regulations required	
More than 12	More than 0.6	10 min			

The above figures apply to the more usual forms of asbestos but for respirable blue asbestos, respiratory protection will be required if the concentration exceeds 0.01 mg/m³ (0.2 fibres/cc). Because such low concentrations are almost impossible to attain in practice the use of blue asbestos will be impracticable without the provision of protective equipment.

These provisional standards specify concentrations of air-borne fibres but not the magnitude of the fibres. A concentration expressed in terms of mass unit volume (mg/m³) is currently used only when estimating background levels of dust and could include all airborne asbestos particles.

This definition is based upon the assumption that the lungs can deal with fibres less than 5 microns and that the nose will exclude fibres

larger than 100 microns,* but the harmful mode of action of asbestos fibres of differing dimensions is still not fully understood, and the above definition is used primarily for dust sampling and does not necessarily apply to their pathological effects. It may be that fibres which do not come within this range are harmful and the importance of the size factor may vary in different types of asbestos. Methods of collecting and analysing samples are discussed in Section Three of this Handbook, and the Factory Inspectorate interpretation given in Appendix II.

ii) For the purpose of cleaning "workplaces" the Regulations show a preference for vacuum cleaning equipment designed to prevent the escape of dust, or other methods which keep dust below the specified threshold limit value (e.g. damping). Where this is not practicable, cleaning equipment with less than "perfect" filtration will have to be used by an operator who must be provided with respiratory protection.

iii) Regulation 13 requires that where any Scheduled process is to be started in a building not previously used for such purposes, the building or relevant part of it must have smooth, impervious surfaces and a built-in vacuum cleaning system. This requirement will apply only when asbestos dust can be evolved and the specified processing exceeds eight hours in any week.

While the Schedule mainly relates to works manufacturing articles or materials containing asbestos, and although the application or removal of thermal insulating materials is not specified, these operations will be interpreted as "processes incidental thereto" insofar as they may give rise to dust. Hence, if the mixing of asbestos thermal insulation materials exceeds eight hours in any week it must be done either in the open air with adequate precautions, or in a "central room" which complies with Regulation 13. The application of the resultant "wet" mix to the plant would not give rise to dust but the floor must be cleared of waste material. Similarly, with reasonable care the application of pre-formed sections should not give rise to objectionable

*NOTE—The unaided eye has difficulty in resolving individual dots, such as dust particles, less than 100 microns in diameter.

dust concentrations. When old thermal insulation is being "knocked off", dust is inevitable and whether or not the concentration reaches a dangerous level depends on the precautions taken (see Section Four).

(c) Practical consequences

It is readily apparent that to comply with the new Regulations, the spreading of dangerous asbestos dust must be prevented. Their implementation may result in practical difficulties but in relation to the safety of the workers involved the requirements are not unreasonable or excessive. It is expected that as experience is gained some detailed requirements of the Factory Inspectorate may be modified. For example, Regulation 6 requires that the Factory Inspector be given twenty-eight days' written notice (less by agreement) of work involving blue asbestos. In a long-established works much of the thermal insulation will have been in position for many years and it may be almost impossible to ascertain the constituents of the "mix" used. In such cases, provided appropriate precautions have been taken immediately the presence of blue asbestos (crocidolite) is revealed, the Factory Inspectorate would be unlikely to seek to impose any penalty. Similarly Regulation 8 requires that "... there shall be provided for the use of each person employed in any part of the Factory, being a part into which asbestos dust from a process to which these Regulations apply is liable to escape:—

- (i) approved respiratory protection equipment.
- (ii) protective clothing".

Tests have shown that the dust concentration diminishes rapidly as the distance from the source of dust increases. If in a large "open" building there is any doubt about the provision of protection for workers who may be located at considerable distances from any asbestos dust-raising operations, the occupier, on whom falls the onus of proving the safety of the dust concentration, should arrange to monitor the atmosphere in the surrounding area.

It is emphasised that Regulation 5 makes it clear that the "occupier" "contractor" "employer" and "employee" all have responsibilities for

ensuring compliance with the requirements of the Regulations irrespective of whether the workers involved are directly employed or are contractors' men.

The Regulations make no stipulation regarding the emission of asbestos dust to the outside atmosphere; nevertheless the "occupier" has a moral obligation to avoid the dispersion of dust over neighbouring buildings and houses, e.g. from a dump of asbestos waste or exhaust system. Cases of mesothelioma have occurred in people living close to but not working in asbestos factories.

SECTION THREE MEASUREMENT OF CONCENTRATION OF AIRBORNE ASBESTOS DUST

A full account of the methods available for the determination of asbestos dust in air is beyond the scope of these Recommendations and the reader seeking further detail is referred to Appendix IV of the paper by Lane, R. E., *et al.*, "Hygiene Standards for Chrysotile Asbestos Dust" published in "The Annals of Occupational Hygiene", vol. II, No. 1, pp. 47-69, January, 1968, and the Asbestososis Research Council's Technical Note No. 1 (see Appendix IV).

Generally the concentration of asbestos dust can be determined as either the number or weight of asbestos fibres per cubic cm. of air.

The number concentration is determined by collecting all the particles in a given volume of air on to a surface where they can be viewed and counted by a good quality optical microscope, e.g. giving 500 times magnification under phase contrast conditions.

The mass concentration of asbestos dust can be determined typically by drawing a measured volume of air through a tared membrane filter and observing the increase in weight. The use of this simple technique is restricted to situations where asbestos is the major airborne contaminant. Elsewhere, the level of other dust and general atmospheric pollution may be comparable with that of the asbestos present, in which case an analysis of the collected sample will be required.

Whatever the procedure used, experience and expertise are required if reliable information regarding the exposure of workmen to asbestos dust is to be obtained.

The measurement should not be made by untrained or semi-trained staff and it is hoped that courses of instruction in measuring asbestos in air will be started and that companies will co-operate by sending their representatives.

Until such instruction becomes available, assistance can be sought from:—

H.M. Factory Inspectorate,
Industrial Hygiene Division,
Baynards House,
Chepstow Place,
LONDON, W.2.

and

The Secretary,
Environmental Control Committee,
Asbestosis Research Council,
114 Park Street,
LONDON, W.1.

In some circumstances it is likely that the Inspectorate will offer advice directly, but alternatively the enquirer could be referred to one of the recognised consultancy services which are normally closely associated with a University (e.g. London School of Hygiene and Tropical Medicine; the Department of Occupational Health, Manchester University; The North of England Occupational Health Service, University of Newcastle-upon-Tyne; Dundee and District Occupational Health Service, University of Dundee, etc.) and offer a high standard of service.

SECTION FOUR PRECAUTIONS TO BE TAKEN

(a) General

As mentioned in Section One, the ideal solution is substitution by asbestos-free materials which are available for most applications (see Appendix III). Failing this the less carcinogenic forms of asbestos should be used.

Crocidolite (blue asbestos) is the most harmful in this respect and is normally recognized by its grey-blue colour but in certain mixes, e.g. resin-bonded materials or graphited packings, it cannot be so distinguished. This type of asbestos may be found in thermal insulation in the form of cloth, rope and string as well as in sprayed form, sometimes mixed with other fibres. Some reinforced plastics materials contain blue asbestos and it is also used in some gasket and packing materials, particularly where acid-resistance is required.

Block insulation such as 85 per cent magnesia, or calcium silicate, is usually made with white asbestos.

(b) Some potential sources of asbestos dust

For the purpose of recommending working procedures appropriate to the type of asbestos material involved, the following materials groups have been adopted:—

Group A (Applied dry)

- Moulded asbestos
- Moulded calcium silicate/asbestos
- Moulded magnesia (85 per cent)
- Moulded high-temperature insulating block.

Group B (Applied wet)

- Calcium silicate (plastic)
- 85 per cent magnesia (plastic)
- Hard-setting asbestos cement
- Asbestos skimming plasters
- Sprayed asbestos.

Group C Resin-bonded and asbestos-reinforced material, e.g. Fibrous linings and some electrical insulating materials.

Group D General asbestos (hard)

- Roofing sheets
- Building boards
- Rainwater goods
- Drain pipes.

Group E

- Asbestos rope, yarn tape and sealing materials
- Asbestos blankets and clothing
- Asbestos millboard and paper
- Asbestos leathers.

Group F

- Asbestos gland packing (impregnated)
- Compressed fibre jointing.

(c) Protective equipment

(i) Personal

Personal protective equipment should comprise the following:

A. Respirator of a type approved by the Factory Inspectorate. This should be appropriate to the type and duration of exposure to be expected. (See BS. 4273.)

NOTE: In view of the forthcoming revision of the F.I. list of approved respirators, following the issue of BS. 2691 (Canister respirators and dust respirators), and the problems of keeping approved lists up to date, it is considered impracticable to include such a list in this Handbook.

Guidance on the selection and use of respirators should, therefore, be obtained by reference to BS. 4273 (Recommendations for the selection, use and maintenance of respiratory protective equipment), and in cases of doubt advice should be sought from H.M. District Inspector of Factories.

B. Clothing worn outdoors. Weather-protective measure has been found very valuable, and headgear, close-fitting at neck, ankles and wrists.

C. Closely woven, e.g. tyre-tread cotton mixture) scarf, optional.

D. Lightweight plastic gloves (optional).

E. Rubber footwear (optional).

F. A plastic apron may also be worn to prevent gross contamination of the overall.

Clean respirators should be issued to asbestos workers and maintained, serviced and disinfected regularly by a competent department. When not in use they should be stored in dust-proof containers, e.g. a sealed plastic bag. The daily checking and maintenance of the respirators should be the responsibility of a designated person.

ii) Dust extraction

A. All equipment used for dust extraction should be inspected at least once every 7 days and thoroughly examined and tested at least once in every period of 14 months by a competent person. A register of details of such inspections, examinations and tests, state of equipment and repairs (if any) should be maintained by the designated person.

B. Filters used in the equipment should be inspected for damage and blockage at intervals appropriate to the work and replaced at intervals not greater than that recommended by the manufacturers.

(d) Training

Before starting work involving any potential asbestos hazard, operators should receive instruction in the correct precautions to be taken when working with asbestos or asbestos-containing materials. Instruction should also be given in the operational use, care and maintenance of the protective equipment described in Section Four (c).

Other personnel required to enter areas of asbestos hazard inherently, should be required to take adequate precautions.

(c) Hygiene

(i) General

Asbestos fibres are readily swallowed and inhaled and so no worker should be allowed to eat or smoke at his place of work or in the contaminated changing room. All workers should be so instructed.

Asbestos operations should be carried out under high standards of good housekeeping and every precaution should be taken to minimise dust formation and/or transfer of dust and fibres to the person. On leaving an area of asbestos dust hazard, operators should wash their hands and, if necessary, their faces. Persons not directly engaged in work with asbestos should not enter the area: if their presence is essential appropriate precautions should be taken with regard to protective equipment and they should follow the procedure specified in Sub-section (f) (Changing Rooms) even if only a short visit is contemplated.

(ii) Clothing

All persons employed in handling wet or dry asbestos should, before starting work, remove all their personal outer clothing and dress in protective equipment to be supplied, which will include overalls of an approved type. Personal outer clothing and personal effects should be kept in a "clean conditions" room; work clothes and protective equipment should be kept in a "working conditions" room. When personnel leave the work area to go outside the barriers, the protective equipment, before removal, should be thoroughly de-dusted by means of a filtered exhaust system. **Blowing dust off by compressed air must be forbidden.** Temporary changing facilities at the work area may sometimes be useful. When meals are taken or work is finished, all protective equipment should be removed and deposited in the "working conditions" room lockers. Respirators should be the last items of equipment to be removed. The worker should then take a thorough wash or shower. Personal outer clothing should then be put on in the "clean conditions" room.

Care should be taken to ensure that footwear is completely free from dust when leaving the de-dusting chamber.

(f) Changing rooms (See Page 16)

Segregated welfare and toilet facilities should be provided for "Asbestos Workers" to change into the approved protective equipment and for de-dusting on return from duty. They should be monitored for asbestos dust concentration and should comprise:

- (i) Facilities for de-dusting and handling soiled protective equipment.
- (ii) Hot and cold washing facilities (and showers where normally provided), for use before re-dressing into personal clothes.
- (iii) Locker facilities for personal clothes and effects should be completely separate from those required for protective equipment.
- (iv) Vacuum equipment with flexible hose located adjacent to, but separate from, the washing and shower facilities and the locker room.

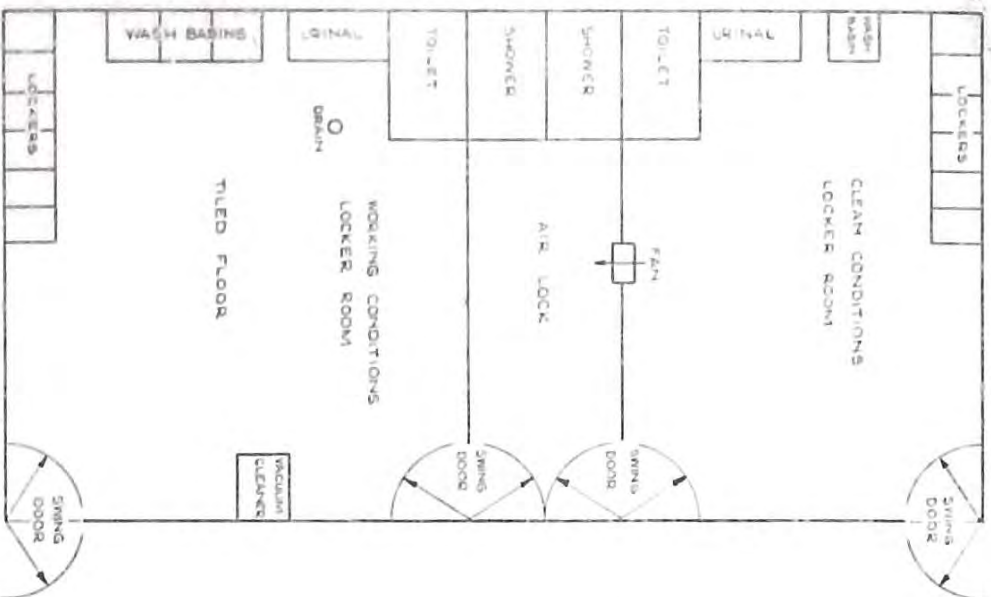
Smoking and the consumption of food and drink should not be allowed in the working conditions changing room.

The changing room attendant, if any, should be provided with protective equipment if proved necessary by the measured asbestos dust concentration.

(g) Laundering

Protective clothing should be vacuum cleaned before collection by the launderer, damped when practicable before dispatch, and packed in plastics bags sealed and clearly marked "Asbestos contaminated clothing—wet before handling".

Workers should not be allowed to take their clothing away for individual treatment by persons who may not be aware of the necessary precautions.



h. Working procedure

1. General

The control of dust is the most important factor in the handling of asbestos products; the dust should be eliminated completely, or kept to a minimum, to ensure the safe handling of these materials. All persons exposed to dust containing asbestos in excess of the limits specified in Appendix II must wear approved respiratory equipment. In confined spaces adequate arrangements for ventilation must be made. As much as possible of the preparatory work should be carried out under controlled conditions in a workshop provided specifically for the purpose.

(ii) Workshop facilities

Dust extraction units shall be provided to exhaust the dust at source from cutting benches, floors, etc. and so prevent its entry into the air of the work place. Hoppers should be used to collect the extracted waste and must be closed when transporting the collected materials. Piped water should be laid into the working area and sprays provided for material dampening, where the nature of the material permits.

(iii) Protective equipment

Protective equipment must be worn by "Asbestos Workers" at all times when dust is raised, such as:

- Carrying, mixing or preparing a mix of any material or composition containing asbestos.
- Cutting by hand, insulating materials containing asbestos.
- Using a portable or other power-driven tool to cut materials containing asbestos.
- Machining resin-bonded asbestos materials.
- Spraying asbestos-containing compositions.
- Applying or stripping insulation containing asbestos.
- Cleaning up locations where asbestos-containing materials have been worked or applied.

H. Emptying or filling hoppers or plastics bags used in this connection (including the emptying of vacuum cleaners).

Unless a particular operation or type of material has been established as non-hazardous, respirators should be worn by all operators and other persons in the area likely to be affected, and must continue to be worn until cleaning-up has been completed and sufficiently low dust concentrations have been obtained.

(iv) **Working of asbestos-containing materials**

A. Group A

The work of applying materials in this group raises dust and adequate precautions should be taken to protect non-asbestos workers in the vicinity as well as the actual workers concerned. In all cases of handling or fitting, the knocking or slapping of sections should be avoided. When cutting or machining "Group A" materials a dust hazard is created. Whenever possible the materials should be wetted before and during shaping or else this operation must be carried out in a segregated area and full precautions observed.

B. Group B

Separate workshop facilities or suitably controlled out-dust facilities should be provided for wet-mixing asbestos compounds. This process should preferably be carried out by adding water to the material in its original container.

C. Group C and F

Where the machining or handling of asbestos-impregnated materials must for practical reasons continue to be carried out in a workshop, a portable dust extraction unit should be used with the point of extraction as close as possible to the work. A clearly identified hopper unit with filter should be provided to collect the extracted waste.

Where small quantities of packing or jointing materials are being cut with sharp hand tools on site, such work is

not normally a hazardous operation and no special precautions are necessary.

D. Group D and E

Materials in this group should, where possible, be cut or shaped in the segregated area provided, using tools equipped with dust exhaust wherever possible. The materials should be wetted with water where practicable immediately before and during cutting or shaping, depending on advice from the material manufacturers.

(v) **Stripping of thermal insulation**

A. General

Before stripping, the ground area should be roped off to a safe distance as mentioned in Section Two (c) and warning notices fixed in prominent positions. Plastics sheeting should be suspended round the job to funnel the stripped insulation on to a suitable plastics ground sheet. Wherever possible, the insulation should be wetted before it is removed from the equipment.

If for operational or other reasons, this is not practicable, care must be taken to keep the scale of the work within the capacity of the safety measures provided. As pieces of insulation are removed they should be immediately placed in plastics bags, and not allowed to fall on the floor, plates or gratings. When full, the bags must be sealed and taken to a designated place for final disposal. A vacuum cleaner, preferably incorporating a disposable bag, or other suitable means e.g. wet methods, should be provided to clear loose particles from the surrounding sheeting and a mobile hopper used to collect the waste for bagging and ultimate disposal.

The cleaner exhaust should be arranged so as not to discharge asbestos dust into the working area. In confined areas a portable dust extraction unit may be required to supplement the vacuum cleaning equipment, particularly where effective damping of the insulating materials cannot

be achieved. No single method of removal can be recommended because of differing applications and situations. The following have been tried and found satisfactory in varying degrees.

B. Wetting Total saturation

This method is useful for large areas of insulation where time can be allowed for the water to soak in. The equipment consists of an injection harness of high rubber hose with right angles, each terminating in an injection head with a down shut-off control. These heads have numerous side holes through which the water is fed, the shut-off only being closed during transfer of the head from one position to the next. The heads are moved progressively at 200 yds intervals as the insulation soaks until the whole area is covered. Water at approximately 2 bar pressure (9.2 MPa) is desirable and can be provided by tapping a higher pressure source through a reducing valve. If total saturation can be achieved no other dust control methods may be necessary. A suitable wetting agent may be added to the water to assist penetration.

When using this method, care should be taken to minimise hazards from any slurry which may form in certain circumstances. Such hazards include slipping on wet floors, cleaning difficulties and possible chloride stress corrosion when working near hot austenitic steel pipes.

C. Wetting By spray

Surface soaking by spray is useful for small areas and where total saturation is not practicable. The equipment may be a spray nozzle fed from a mains supply, or, where no supply is readily available, from a portable pressurised vessel such as a pump-up garden sprayer. It is important that the spray should be copious but fine, so that the water droplets do not generate dust from the surface of the insulation on impact. The spray should be directed at the cutting operation, so it is essentially a two-man job. Dust

is not entirely eliminated but the level is considerably reduced and the remaining dust can be removed with a portable filtered exhaust unit.

Alternatively, a wet-sawing method may be used in which water is directed along the blade of a reciprocating saw.

D. Dry removal

Where it is considered essential to remove asbestos from hot metal, this being one of the worst conditions of removal due to the spread of dust on high convection currents of air, it may be advisable not to use wetting, in order to safeguard the machinery. High-efficiency dust extraction close to the removal operation with efficient filtration must be used. A minimum air velocity of 60 m/min at the extraction point is recommended and the area should be large enough to ensure efficient dust collection.

vi) Spraying

It is recommended that the spraying of asbestos compounds be carried out by specialist contractors using approved equipment. To reduce the risk to "non-Asbestos Workers", work of this nature should when possible be carried out during silent hours.

The area should be defined by warning notices fixed in prominent positions and suitable plastics sheeting hung around the perimeter to contain loose asbestos fibres thrown out by the spraying process. Similarly, plastics ground sheets must be laid on the floor area to collect all waste materials but care should be taken to prevent slipping on the wet floor. Pre-treatment of the fibre may reduce the concentration of dust, and spraying techniques utilising such pre-treatment should be used. Whatever type of machine is being used, the asbestos fibre must not be removed from the dust-proof sacks in which it is supplied until it is fed into the machine in the prescribed manner. Care should be taken in handling the sacks in order not to damage them. Effective means should

be provided to collect and bag waste materials, and scavenge the strutting and ground sheeting of all loose fibres after this work is complete, after which a reasonable delay time must be observed by all Asbestos Workers engaged in the operation, before removing their respirators, which should be done last of all. This "reasonable delay time" is defined as the period after clearing up when the asbestos dust concentration is below the specified threshold limit.

(v) Waste disposal

Asbestos residue and waste, including used filters, and the contents of vacuum cleaners should be packed into robust dust-proof containers, e.g. heavy gauge plastic bags clearly labelled "ASBESTOS: DO NOT OPEN" and completely sealed. The bags should be collected frequently, and care taken to avoid damage to them with consequent spilling of asbestos. Asbestos residues must not be disposed of to incinerators. Where company disposal facilities are not available, arrangements should be made with local authorities for the disposal of all waste asbestos materials.

Asbestos fibres are infestructible and the main object in disposal is to prevent their becoming airborne again; disposal methods such as immediate burial are therefore recommended. Advice is available from the Secretary, Environmental Control Committee, Asbestos Research Council, 114 Park Street, London, W.1.

(j) Labelling, purchasing, storage and transport

(i) Labelling

All materials containing asbestos should be labelled accordingly.

(ii) Purchasing

Yarn, tape, cloth and similar materials should be supplied in shrouded plastic reels. All materials required for wet mixing should be purchased in pack form and supplied in sealed plastic bags, so that the wet mixing can be carried out in the bag. Whenever possible, moulded sections should be purchased and supplied in sealed cartons, in preference to materials mixed on site.

(iii) Storage

A special area should be set aside for the storage of asbestos materials. Products in Groups "A", "B" and "E" should be stored in cartons or sealed bags as delivered. To avoid damage to the containers they should be stacked on sillage above floor level. Where existing stocks of asbestos have not been supplied so packaged, frequent cleaning of the store area with vacuum equipment to prevent the spread of dust should be initiated. Where relatively large quantities of Groups "A", "B" and "E" materials are involved, facilities for dust extraction may be required.

(iv) Distribution and transport

Closed containers should be used for carrying loose asbestos which may give rise to dust.

APPENDIX I ASBESTOS REGULATIONS

STATUTORY INSTRUMENTS

1969 No. 690

FACTORIES

The Asbestos Regulations 1969

Made - - - - - 13th May 1969
Laid before Parliament - - - - - 22nd May 1969
Coming into Operation - - - - - 14th May 1970

ARRANGEMENT OF REGULATIONS

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PART I Application, Interpretation and General	1 - 6
PART II Exhaust Ventilation and Protective Equipment	7 and 8
PART III Grainlines of Premises and Plant	9 - 14
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SCHEDULE Processes specified for the purposes of Regulation 13.	

REGULATIONS

The Secretary of State—

- by virtue of her powers under sections 62, 76 and 180 (6) and (7) of the Factories Act 1961(a) and of all other powers enabling her in that behalf; and
- after publishing, pursuant to Schedule 4 to the said Act of 1961, notice of the proposal to make the Regulations and not having received any objection to the draft in regard to which she is required by the said Schedule to direct an inquiry to be held.

hereby makes the following special Regulations:—

PART I APPLICATION, INTERPRETATION AND GENERAL

Union, commencement and revision

1. (1) These Regulations may be cited as the Asbestos Regulations 1969 and shall come into operation on 14th May 1970.

(2) The Asbestos Industry Regulations 1931(b) are hereby revoked.

3. The Shipbuilding and Ship-repairing Regulations 1960(c) shall have effect as if the following provisions were omitted—

- in Regulation 3(2), the definition of the word "asbestos";
- in Regulation 76(1), subparagraphs (a) to (d); and
- in Regulation 77, paragraphs (a) to (d).

Interpretation

2. (1) The Interpretation Act 1893(d) shall apply to the interpretation of these Regulations as it applies to the interpretation of an Act of Parliament, and as if these Regulations and the Regulations hereby revoked were Acts of Parliament.

(2) In these Regulations, unless the context otherwise requires, the following expressions have the meanings hereby assigned to them respectively, that is to say

"approved" means approved for the time being by certificate of the Chief Inspector;

"asbestos" means any of the following minerals, that is to say, crocidolite, amosite, chrysotile, fibrous anthophyllite and any mixture containing any of the said minerals;

"factory" includes any premises and place to which these Regulations apply;

"protective clothing" means overalls and headgear, which (in either case) will when worn exclude asbestos dust;

"the principal Act" means the Factories Act 1961 as amended by or under any other Act.

(3) References in these Regulations to asbestos dust shall be taken to be references to dust consisting of or containing asbestos to such an extent as is liable to cause danger to the health of employed persons.

(a) 1961 c. 34. (b) S.R. & O. 1931 1140 (Rev. VII, p. 103) (p. 410) (c) S.I. 1960/1922 (1960 II, p. 1427). (d) 1893 c. 69.

Application of Regulations:

3.—(1) These Regulations shall apply to the following premises and places in which any process to which these Regulations apply is carried on, that is to say, factories and all premises and places to which the provisions of Part IV of the principal Act with respect to special regulations for safety and health are applied by the following provisions of that Act, namely, section 124 which relates to electrical stations, section 124 which relates to institutions, so much of section 125 as relates to warehouses other than warehouses within any dock or farming part of any wharf or quay, section 126 which relates to ships, and section 127 which relates to building operations and works of engineering construction.

2.—These Regulations apply to every process involving asbestos or any article composed wholly or partly of asbestos, except a process in connection with which asbestos dust cannot be given off.

3.—The provisions of these Regulations are in substitution for the provisions of so much of section 1-2 of the principal Act as relates to the cleaning of parts of factories, being parts in which any process to which these Regulations apply is carried on or into which any asbestos dust from any such process carried on in the factory is liable to escape, and of so much of section 63 (1) of that Act as relates to the measures to be taken against asbestos dust, but save as aforesaid are in addition to and not in substitution for or in diminution of other requirements imposed by or under the principal Act.

Certification of exemption

4.—The Chief Inspector may (subject to such conditions, if any, as may be specified therein) by certificate in writing (which he may in his discretion revoke at any time) exempt from all or any of the requirements of these Regulations—

- (a) any factory or part of any factory; or
- (b) any class or description of factories or of parts thereof; or
- (c) any machine, plant, apparatus or process or any class or description of machines, plant, apparatus or processes; or
- (d) the employment of any person or class or description of persons.

if he is satisfied that the requirements in respect of which the exemption is granted are not necessary for the protection of persons employed or are impracticable.

Obligation under Regulations

5.—(1) Except in the case of any place where building operations or works of engineering construction are carried on it shall be the duty—

(a) of every employer (whether or not an occupier) who is undertaking in a factory any process to which these Regulations apply to comply with the requirements of these Regulations in relation to any person employed by him, any plant or material under his control, and any part of the factory in which he is carrying on the process or into which asbestos dust in connection with that process is liable to escape;

(b) of every occupier of any factory (whether or not such an employer as aforesaid) where any such process is being carried on to comply with the requirements of Regulations 8, 16 and 19 in relation to any person employed in the factory (other than any person employed by an employer who is undertaking in the factory any process to which these Regulations apply) and with the requirements of Part III of these Regulations; and

(c) of every person who is undertaking in a factory any process to which these Regulations apply and who employs no other person in the carrying on of that process to comply, for the protection of employed persons working there, with the requirements of these Regulations (other than Regulations 8, 14 and 18 to 20) with which he would have to comply if he were the employer of a person doing there the work which he does himself.

(2) In the case of places where building operations or works of engineering construction are carried on it shall be the duty of every contractor and of every employer who is undertaking any process to which these Regulations apply to comply with the requirements of these Regulations in relation to any person employed by him and any person

employed there who is liable to be exposed to asbestos dust in connection with any process carried on by the contractor or employer as the case may be, and in relation to any plant or material under his control and any part of the site where he is carrying on the process or where asbestos dust in connection with that process is liable to escape.

3. It shall be the duty of every person employed to comply with the requirements of such of these Regulations as relate to the performance of or refraining from an act by him.

Vibration

6. (1) Where at the date of the coming into operation of these Regulations a person is undertaking in a factory any process involving considerable jolting the removal from any surface of lagging consisting of or containing woodshole, he shall within 28 days of that date give written notice to the inspector for the district that he is undertaking such a process and of the address of the factory at which he is doing so.

2. Before a person first begins after the coming into operation of these Regulations to undertake in a factory any such process as aforesaid, he shall give not less than 28 days written notice to the inspector for the district, or such shorter notice as the inspector may agree to accept, of the date on which and of the address of the factory at which he will begin to do so.

Part II

EXHAUST VENTILATION AND PROTECTIVE EQUIPMENT

Exhaust ventilation

7. (1) Subject to the provisions of the next following Regulation, no process to which these Regulations apply shall be carried on in any factory unless equipment is provided, maintained and used which produces an exhaust draught which prevents the entry into the air of any workplace of asbestos dust.

Provided that the foregoing requirements of this Regulation shall not apply where any such process is carried on in such a manner as to be as safe as it would be if the said requirements were complied with.

(2) Exhaust ventilation equipment provided in accordance with

this Regulation shall while any work of maintenance or repair to the machinery, apparatus or other plant or equipment in connection with which it is provided is being carried on, be kept in use so as to produce an exhaust draught which prevents the entry into the air of any workplace of asbestos dust.

(3) Exhaust ventilation equipment provided in accordance with this Regulation shall be inspected at least once in every seven days and shall be thoroughly examined and tested by a competent person at least once in every period of fourteen months, and a report of the results of every such examination and test containing approved particulars and signed by the person making or responsible for the carrying out of the examination and test shall be made within fourteen days after the examination and test.

(4) Every such report as aforesaid shall be attached to the general register and be preserved and kept available for inspection by any inspector for a period of two years after it is made.

Protective equipment

8. (1) Where in any factory the requirements of paragraph (1) or (2) of the last foregoing Regulation apply, but it is impracticable to comply with those requirements, there shall be provided for the use of each person employed in any part of the factory, being a part into which asbestos dust from a process to which these Regulations apply is liable to escape—

- (a) approved respiratory protective equipment; and
- (b) protective clothing.

(2) All respiratory protective equipment and protective clothing provided in pursuance of the foregoing paragraph of this Regulation shall be maintained.

(3) All respiratory protective equipment and protective clothing so provided shall be used by the persons for whom they are provided while employed in any such part of the factory as aforesaid.

(4) No respiratory protective equipment so provided which has been worn by a person shall be provided for the use of another person unless it has been thoroughly cleaned and disinfected since last being worn.

3) No person shall be employed to perform any work for which respiratory protective equipment is provided in pursuance of this Regulation unless he has been fully instructed in the proper use of that equipment.

PART III

CLEANING OF PREMISES AND PLANT

Duty to keep clean

9. All machinery, apparatus, work benches and other plant and equipment used in any factory for the purposes of any process to which these Regulations apply, all external surfaces of exhaust ventilation equipment provided in pursuance of these Regulations and all floors, inside walls, ceilings, ledges and other internal surfaces of any part of a building or of any ship, being a part in which any process to which these Regulations apply is carried on or into which asbestos dust from any such process carried on in the factory is liable to escape, shall, so far as is practicable, be kept in a clean state and free from asbestos waste and dust in accordance with the provisions of this Part of these Regulations.

Cleaning by dustless method

10. The cleaning required by the last foregoing Regulation shall, so far as is practicable, be carried out—

- (a) by means of vacuum cleaning equipment so designed and constructed and so used; or
- (b) by some other suitable method such, that asbestos dust neither escapes nor is discharged into the air of any workplace.

Cleaning where cleaning by dustless method is impracticable

11.—(1) In so far as it is impracticable to carry out the cleaning required by Regulation 9 in accordance with the provisions of the last foregoing Regulation, such cleaning shall be carried out in accordance with the provisions of this Regulation.

(2) Protective clothing and approved respiratory protective equipment shall be provided for and used by every person present in any part

of a factory while such cleaning is being carried out in that part otherwise than in accordance with Regulation 10.

(3) No respiratory protective equipment so provided which has been worn by a person shall be provided for the use of another person unless it has been thoroughly cleaned and disinfected since last being worn.

(4) No person shall be employed to perform any work for which respiratory protective equipment is provided in pursuance of this Regulation unless he has been fully instructed in the proper use of that equipment.

(5) Any surface on which asbestos waste or dust is deposited as a result of the carrying out of cleaning in pursuance of this Regulation, not being a surface of the cleaning equipment used, shall be cleaned forthwith.

Vacuum cleaning equipment

12. Vacuum cleaning equipment used for cleaning in accordance with the provisions of this Part of these Regulations shall be maintained and have all external surfaces kept in a clean state and free from asbestos waste and dust.

Requirements as to construction of buildings before first use for certain processes

13. After the date of the coming into operation of these Regulations no process specified in the Schedule to these Regulations shall be carried on in a building by the occupier of the building unless any part of such building where any such process is carried on and has not previously been carried on—

- (a) is so constructed that all its interior surfaces are smooth and impervious and is so designed and constructed as to have in it as few surfaces as is practicable on which asbestos dust can settle; and
- (b) has a vacuum cleaning system which has filters and a source of suction located at a fixed central point with pipes throughout the building fitted with suitably placed inlets to which portable cleaning implements can be attached and is a system which is so designed and constructed that asbestos dust

PART VI
YOUNG PERSONS

Employment of young persons

20. No young person shall be employed—

- a) in any process to which these Regulations apply or in any part of a factory into which asbestos dust from any such process carried on in the factory would otherwise be liable to escape unless the requirements of Regulation 7 are complied with in respect of that process; or
- b) in carrying out any cleaning in accordance with Part III of these Regulations otherwise than by means of vacuum cleaning equipment or other suitable method in accordance with the provisions of Regulation 10.

13th May 1963.

Barbara Castle,

First Secretary of State and Secretary
of State for Employment and Productivity.

SCHEDULE

Regulation 13

Processes specified for the purposes of Regulation 13

1. Breaking, crushing, disintegrating, opening and grinding of asbestos, and the mixing of asbestos (including the mixing of asbestos with any other material) or the sieving of asbestos, and all processes involving manipulation of asbestos incidental thereto.
2. All processes in the ~~preparation~~ manufacture of yarn or cloth composed of asbestos or asbestos mixed with any other material, including preparatory and finishing processes.
3. The making of insulation slabs or sections, composed wholly or partly of asbestos, and processes incidental thereto.
4. The making and repairing of insulating mattresses, composed wholly or partly of asbestos, and processes incidental thereto.
5. Any other process in the manufacture of articles composed wholly or partly of asbestos in connection with which asbestos dust is given off.

EXPLANATORY NOTE

(This Note is not part of the Regulations)

These Regulations impose requirements for the safety and welfare of persons employed in factories and in other premises and places to which the Factories Act 1961 applies, where processes are carried on which involve asbestos or any article composed wholly or partly of asbestos and are capable of giving rise to asbestos dust. The Regulations require that the processes be carried on under an exhaust draught or in some other equally safe way, or that protective clothing and respiratory protective equipment be provided for the use of persons employed. The Regulations also impose requirements as to the cleaning of premises and plant, the construction of buildings newly used for the processes, the storage and distribution of asbestos, the accommodation to be provided for protective equipment and the cleaning of protective clothing. The employment of young persons in connection with processes to which the Regulations apply and in cleaning work is prohibited except in specified circumstances. The Regulations also require that notification of processes involving crocidolite be sent to the inspector for the district.

The Asbestos Industry Regulations 1931 and certain provisions concerning asbestos of the Shipbuilding and Ship-repairing Regulations 1960 are revoked.

APPENDIX II

F.L. INTERPRETATION

In this appendix guidance is given on how H.M. Inspectors of Factories will interpret the expression "dust consisting of or containing asbestos to such an extent as is liable to cause danger to the health of employed persons" and how the measurements may be made. It is emphasised that these notes have been prepared for the guidance of H.M. Inspectors since only the Courts can give binding decisions in these matters. It is important to bear in mind that these standards are provisional and may have to be revised from time to time.

1. Chrysotile, amosite and fibrous anthophyllite

- a. Where the average concentration of asbestos dust over any 10 minute sampling period is less than 2 fibres/c.c. or 0.1 mg m³, H.M. Factory Inspectorate will not seek to enforce the substantive provisions of the Regulations, in particular Regulations 7 and 8. Where the concentration is 2 fibres/c.c. or 0.1 mg m³ or more but not more than 12 fibres/c.c. or 0.6 mg m³ further sampling over a four hour period will be carried out to determine whether the average concentration of asbestos dust still exceeds 2 fibres/c.c. or 0.1 mg m³.
- (b) Where the average concentration of asbestos dust over a four hour sampling period is 2 fibres/c.c. or 0.1 mg m³ or more the extent to which H.M. Factory Inspectorate will require the standard of control to be improved will depend upon the amount by which it exceeds 2 fibres/c.c. or 0.1 mg m³ and the duration of exposure.
- (c) When the average concentration of asbestos dust over any 10 minute period exceeds 12 fibres/c.c. or 0.6 mg m³, Inspectors will normally seek to confirm or otherwise the accuracy of the test by means of a further sample before taking action to enforce regulations 7 or 8 whichever is appropriate.

2. Crocidolite (Blue asbestos)

The Regulations will apply in full wherever workers are engaged in processes involving crocidolite because the concentration of this

mineral that is believed to be liable to be dangerous to health is very small indeed. An approved form of respirator will be required to be worn unless the concentration in the breathing zone of a worker in a crocidolite process can be maintained below 0.2 fibres/c.c. or 0.01 mg m³ when measured as the average concentration over a 10 minute sampling period. Inspectors will normally seek to confirm the accuracy or otherwise of the test by means of a further sample before taking action.

NOTE:

- (1) "fibres" c.c. means particles of length between 5 microns and 100 microns and having a length to breadth ratio at least 3 : 1, observed by transmitted light by means of a microscope at a magnification of approximately 500 ×.
- (2) mg m³ means milligrammes per cubic metre.

The above Appendix is included for interim guidance pending the issue of the booklet "Asbestos: Health precautions in industry".

APPENDIX III ALTERNATIVES TO ASBESTOS

(a) Thermal insulation

E.E.U.A. Handbook No. 12, "Thermal Insulation of Pipes and Vessels" gives details of a wide variety of insulation materials many of which contain no asbestos. These include glass wool, rock wool and slag wool all of which are available as flexible mattresses and rigid or semi-rigid slabs and pipe sections. Mattresses are usually faced with wire netting or expanded mesh and may be tied with asbestos string but wire ties may be specified.

The binder in the rigid and semi-rigid forms is lost at high temperatures so that the inner layer may become mechanically weak in use.

Other materials available as rigid slabs and pipe sections include cellular glass (maximum temperature 427 °C) and polyurethane foam (maximum temperature normally 116 °C, some foams being suitable up to 149 °C).

Loose-fill materials containing no asbestos are exfoliated vermiculite, expanded perlite and the mineral wools referred to above. Some proprietary mixtures based on diatomite contain asbestos.

The table on Pages 40-41 lists materials that commonly contain asbestos, together with suggested alternatives and comments on each. It is emphasised that the alternatives are not equivalent in all respects to the asbestos materials that they may replace and the service conditions in each case should be considered before a substitution is made.

(b) Packing materials

Tape, rope and sheet of various densities, made from refractory fibres are available. They are expensive but have higher limiting temperatures than asbestos. However, glass and rock wool fibres have poor resistance to the combined effects of high-temperature and steam.

(c) Heat-resistant protective clothing

It has not proved possible in all cases to find a non-asbestos substitute for protective clothing hitherto normally made from woven asbestos cloth. This has in some cases been due to poor heat resistance and in others to poor wearing qualities of the alternatives available.

The alternative approach therefore has been to find asbestos clothing which has been suitably treated to minimise the risk to the wearer. The most suitable on all counts appears to be aluminised asbestos cloth which when made up into suits, and suitably lined, creates no dust hazard.

Fire protection suits are designed for absolute protection in fierce heat conditions for short periods and other designs of similar material are available for protection when working close to radiant heat sources such as in steel works, glass works and foundries.

Below are descriptions of fire protection equipment:

(i) Suit

Body fabric	Aluminised asbestos cloth
Body lining	Fabric cotton
Interlining	Wool fleece
Manufactured in accordance with Provisional Specification No. DTD 1255.	

(ii) Gauntlet Gloves

Outer Fabric	Laminated aluminised asbestos cloth
Lining—mitt	Knitted fleece
Lining—Gauntlet	Green canvas
Manufactured in accordance with Provisional Specification No. DTD 1159	

Asbestos-containing materials	Asbestos-free alternatives	Comments
Moulded asbestos (Amosite asbestos bonded with sodium silicate)	None	
85 per cent magnesia (about 15 per cent asbestos)	Asbestos-free magnesia	Ceiling temperature 316°C. One manufacturer only. Coloured pink to distinguish from asbestos-containing materials.
Calcium silicate (about 15 per cent asbestos)	Asbestos-free calcium silicate	Ceiling temperature varies. Generally 600°C.
High-temperature insulating block (about 15 per cent asbestos with diatomite)	Vermiculite-alumina cement block	Some calcium silicates may be suitable alternatives.
Plastics compositions	Asbestos-free versions of magnesia and calcium silicate	Generally satisfactory for gap-filling and small areas.
Sprayed asbestos and asbestos mineral wool	1. Sprayed mineral wool 2. Sprayed ceramic fibres 3. Sprayed vermiculite cement and sprayed concrete	1. At development stage only. 2. Limited experience in this country. Expensive. 3. For fireproofing.
Self-setting and hard-setting finishing cement (up to 40 per cent asbestos)	1. Asbestos-free finishing cements 2. Plastics finishes applied in paste form and reinforced with glass cloth 3. Sheet finishes (metal, plastics, roofing felt, etc.)	1. May be subject to more cracking than asbestos containing finishes. 2. Coloured if required. Less robust and generally more expensive than cement. 3. Cheap for straight pipe lengths but expensive where bends and shapes involved.
Board and sheet	Ceramic fibre board and sheet	Higher ceiling temperature 1,260°C than asbestos material. Expensive.
Rope	1. Glass fibre rope 2. Ceramic fibre rope 3. Silica fibre rope 4. Ceramic fibre felt strip	1. Ceiling temperature 500°C. 2. Ceiling temperature 1,260°C. Expensive. 3. Ceiling temperature 982°C. Very expensive. 4. May be cheaper alternative to ceramic rope in some cases.
Cloth, tape, string, yarn, etc.	1. Glass fibre fabrics and yarn 2. Ceramic fibre and yarn 3. Silica fibre and yarn	1. Ceiling temperatures up to 500°C depending on type of glass. Some cloths may be unpleasant to handle. 2. Ceiling temperature 1,260°C. Expensive. 3. Ceiling temperature 982°C. Very expensive.

NOTE: Manufacturers have incorporated organic fibres in some of the materials that previously contained asbestos. Such materials may no longer be suitable for certain duties, e.g. where contamination with powerful oxidising agents is possible.

(iii) Helmets

Shell
Visor

Glass fibre, resin bonded
Plastic transparent screen,
wire mesh screen

Manufactured in accordance with Provisional Specification
No. DTD 1157 Part 2

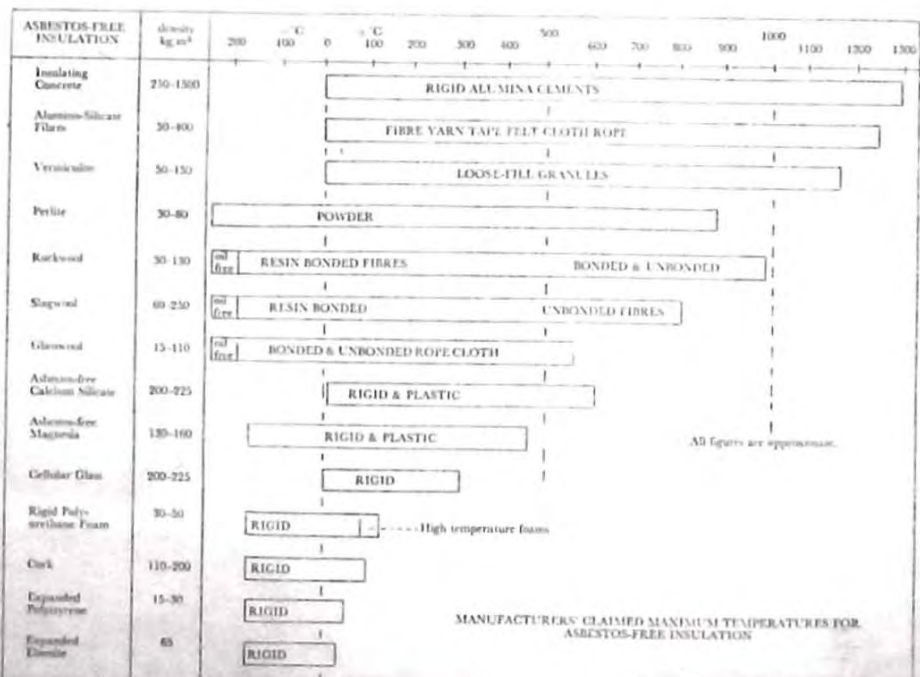
(iv) Boots

Uppers
Soles

Chrome leather
Oil and heat resistant neoprene
Manufactured in accordance with Provisional Specification
No. DTD 1160 Issue 4.

(d) General

Where acceptable substitutes are not forthcoming, consideration should be given to those types of treated asbestos products which by their nature reduce, and in some cases virtually eliminate, the asbestos dust hazard. Examples are grafted, lubricated or foil-wrapped gland packings, rubberised sheet, tapes and joint-rings and plasticised or aluminised cloths.



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EXHIBIT 19

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A I R B O R N E A S B E S T O S

A Report Prepared by the
Committee on Biologic Effects of Atmospheric
Pollutants
of the
Division of Medical Sciences, National Research
Council

National Academy of Sciences

National Academy of Engineering

Washington, D.C.

1971

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PREFACE

The naturally occurring fibrous silicates classified as "asbestos" have become almost indispensable in modern technology.^{67,125,142} The world's annual production has grown from a few thousand tons in 1900 to over 3 million tons in 1968.¹²¹ Annual consumption in the United States averaged nearly 800,000 tons during the period 1965-1969.¹²¹ The potential of asbestos as a hazard to health has been the subject of a number of reviews in recent years.^{22,49,60,134,137,146,148,168}

Although it has been known for a half-century that persons who inhaled large amounts of asbestos dust in the course of their work sometimes developed disabling or fatal fibrosis of the lungs, it has been only within the last three decades that other serious effects, such as cancer, have been associated with occupational exposures. Recently, the likelihood of exposure of the public at large to asbestos has been recognized and has led to a demand for more rigorous control of asbestos emissions into the atmosphere.

"Asbestos" is a generic term for a number of hydrated silicates that, when crushed or processed, separate into flexible fibers made up of fibrils.⁴⁶ Although there are many asbestos minerals, only six are of commercial importance: chrysotile, a tubular serpentine mineral, accounts for 95% of the world's production; the others, all amphiboles, are amosite, crocidolite, anthophyllite, tremolite, and actinolite. The asbestos minerals differ in their metallic elemental content, range of fiber diameters, flexibility or harshness, tensile strength, surface properties, and other

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CHAPTER 1

PATHOGENICITY OF ASBESTOS

The effects of fibers in biologic systems may result not only from the properties of the fibers themselves, but also from contamination with inorganic or organic substances that occur naturally or are added during mining, milling, processing, shipping, or use. Contaminants acquired from the atmosphere or in the respiratory tract may be carried on the surface of fibers. Fibers may act as cofactors; conversely, their action may be modified by other cofactors.

PATHOGENICITY IN MAN

The proven or suspected effects of asbestos minerals on human health include nonmalignant changes, such as pulmonary and pleural fibrosis, and several types of malignancy, notably of the lung, pleura, and peritoneum. Nearly all the positive evidence of an association between asbestos and human disease has come from occupational groups. With few exceptions, these have consisted of workers engaged in the mining and milling of asbestos, the manufacture of asbestos-containing products (such as textiles and construction materials), and the application and removal of asbestos-containing insulating materials.

Asbestosis

Asbestosis, or asbestotic pneumoconiosis, was the first clearly demonstrated adverse effect of asbestos in man. It is characterized by a pattern of roentgenographic changes in the lung consistent with diffuse interstitial fibrosis of variable degree and at times with fibrosis and calcification of the pleura; clinical changes that include fine rales,

finger clubbing, and shortness of breath, each of which may be absent in an individual case; and physiologic changes consistent with a restrictive lung disorder.

The first published mention of a case, in a man who had worked for 10 years in the carding room of an asbestos factory, was by H. Montague Murray in 1907.¹¹¹ Cooke reported a second case in 1924²⁰ and in 1927 provided a more detailed description,²¹ in which the term "asbestosis" was first used. In 1930, Merewether¹⁰⁵ reviewed the salient features of the disease and the environmental exposures of workers, including data derived from an epidemiologic study reported in more detail by Merewether and Price.¹⁰⁶ This led to the promulgation of regulations for environmental and medical control in the United Kingdom, which became effective in 1932.⁷ Cases were first reported in the United States in 1930,^{110,141} and guidelines for acceptable dust concentrations were proposed by Dreessen et al. in 1938.³²

Industrial experience indicates that pulmonary fibrosis sufficient to interfere with respiratory or cardiovascular function can be prevented by reducing asbestos dust concentrations to levels that are still far above any likely to be encountered in community air.

Pleural Calcification

Calcified pleural plaques occur frequently in workers exposed to asbestos.^{5,6,43,103,108,130} When multiple or bilateral, they are regarded by some as almost diagnostic of asbestos-related disease.^{43,71} In asbestos workers, calcified plaques rarely appear until 20 years after first exposure and do not necessarily correlate with parenchymal fibrosis.⁹¹

Calcification appears to differ in frequency in different occupationally exposed groups, but studies are inadequate to verify or explain such differences.

Bronchogenic Carcinoma

No features of bronchogenic carcinoma associated with asbestos are pathognomonic. Primary sites are more often in the lower lobes, in contrast with the usually higher frequency of upper lobe tumors. Peripheral primary sites are common in asbestos-related lung cancer. All cell types are represented in most series.

The first suggestion that asbestos might be causally related to cancer of the lung was made in 1935 by Lynch and Smith,⁹¹ who described squamous cell carcinoma in a South Carolina textile worker with asbestosis. Despite other isolated reports, an association was not firmly supported by epidemiologic evidence until 1947, when Merewether, Chief Inspector of Factories in the United Kingdom, reported 31 instances of cancer of the lung in 235 persons known by his department to have died with asbestosis between 1924 and 1946.¹⁰⁴ That constituted an incidence of 13.2%, compared with 1.32% (91/6884) in persons certified as having had silicosis during the same period. Gloyne in 1951⁵⁰ reported on the pathologic findings in 1205 lungs from workers whose cases were being evaluated for pneumoconiosis. In 132 asbestos workers, he found 121 with asbestosis, of whom 17, or 14.0% had cancer of the lung, compared with 55 (6.9%) of 796 persons who had silicosis. The study was done, however, in a hospital to which suspected tumor patients would have been referred; thus, there may have been an overestimate of risk. Doll in 1955,³¹ after analyzing the causes

of death among 105 men who had worked for at least 20 years in areas of asbestos textile plants defined as dusty, concluded that the 18 cases of lung cancer that occurred indicated a risk about 10 times that in the general male population.

Other studies^{11,13,15,18,23,24,33,34,38-40,59,70,74-76,80-82,87,93,94,98,101,112,131,149,154,155,161,169} have confirmed an association between occupational exposure to asbestos and a higher-than-expected incidence of bronchogenic cancer. Some studies¹³ have demonstrated differences in the degree of risk among different occupationally exposed groups, probably related to dose, as well as to other factors.

Mesothelial Tumors

Primary malignant tumors of the pleura and peritoneum have been regarded as exceedingly rare by most pathologists; until recent years, some even questioned their existence. There are still differences of opinion as to diagnostic criteria.^{18,70,98,149} Therefore, statistics on prevalence or incidence in one geographic area cannot be safely compared with those from another. Adherence to strict diagnostic criteria, including an autopsy complete enough to rule out other primary tumors that could metastasize to or involve serosal surfaces, is a difficult constraint on a retrospective series.

It was after 1960 that serious consideration was first given to asbestos as an etiologic factor in mesothelial malignancies. In that year, Wagner et al.¹⁶¹ reported 33 cases of pleural mesothelioma in a part of South Africa important for crocidolite mining. For all but two of the patients, the authors discovered likely asbestos contacts two decades or

Experimental Asbestosis

Asbestotic pulmonary fibrosis has been produced experimentally in various species of animals, including rats,^{45,58,156,157} guinea pigs,^{29,45,69,157} hamsters,⁵⁵ rabbits,¹⁵⁷ and monkeys.¹⁵⁷ In many of the studies, the disease resembled early asbestotic development in man--e.g., it was multifocal. Diffuse fibrosis has also been produced,¹⁵⁷ but to do so it was necessary to use very high concentrations of asbestos dust and long periods of exposure or observation after exposure. (In contrast, experimental silicosis can be produced with lower cumulative exposure.) In the course of the investigations, it has been asked whether the fibrogenicity of asbestos dust is mostly confined to fibers longer than 5 μm ;^{45,156} the question is still unanswered.

Experimental Neoplasia

Lung cancer from chrysotile dust has been produced experimentally in rats⁵⁸ and in mouse lung implants.¹⁴⁰ Other investigators who used different methods for introducing the dust¹⁴³ did not find lung cancer in the animals they studied. That some asbestos dust has an increased content of trace metals--particularly nickel, chromium, and cobalt--may explain these differing experiences. Rats whose lung clearance had been artificially impaired had twice the lung cancer rate of animals with normal clearance.⁵⁸

Cancer of the pleural surface (mesothelioma) has been reported in rats and hamsters that received intrapleural injections of the three most common types of asbestos.^{139,159,160} The amounts of asbestos dust introduced into the thoracic cavity were very large, and translation of results to human inhalation of asbestos is uncertain.

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polyvinylpyridine N-oxide, a macromolecular chemical that is effective in preventing the cytotoxic damage caused by crystalline silica in experimental conditions.

Although the foregoing types of study have no known relationship to fibrogenic or carcinogenic effects in vivo, they provide systems that may prove useful in determining mechanisms of action and approaches to prophylactic or therapeutic measures.

INFLUENCE OF MAJOR VARIABLES ON PATHOGENICITY

When considering the importance of type of asbestos, fiber size, and cofactors on biologic effects, it is necessary to emphasize that a given attribute may influence in differing ways the respirability, deposition, retention, clearance, translocation, and biologic reactivity. Although some in vitro and laboratory studies yield different responses to different types of asbestos, the results do not justify drawing firm conclusions as to the relative pathogenicity of the different types. Nor do epidemiologic studies conclusively support such differences. All the commercially used forms of asbestos can produce asbestosis. In only relatively few studies has the incidence of malignancies been determined in groups with exposures to a single asbestos type. Where there are data that suggest a lower risk, as in the chrysotile-producing areas of Canada^{13,100} and Italy,¹⁵⁴ there are possible explanations for the difference other than asbestos type. The high incidence of mesothelial tumors in the North Western Cape area of South Africa has led to the suggestion that crocidolite is unusually hazardous, but mesotheliomas have been rare in the Transvaal, where crocidolite is also produced.^{49,138} Although Selikoff et al.¹³² found

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range, compared with fibers in the LM range. It is possible that the relative risks associated with fibers of different sizes are different for nonmalignant and malignant changes. The evidence that bundles of fibrils may be broken down within the body to individual fibrils is important.¹⁴⁷

A number of investigators have postulated that a probable role of asbestos fibers in producing disease is to carry toxic or oncogenic substances to vulnerable sites. Studies bearing on this have included analysis of various types of asbestos to determine the contaminants present, with special emphasis on metals^{26,27} and polycyclic hydrocarbons;^{48,61-64} studies of the elution of contaminants in biologic materials;²⁶ and the concurrent exposure of animals to asbestos and to other materials.¹⁰⁹ The present consensus is that contaminants are present, but a special pathogenetic role is still speculative.

The work of Selikoff et al.¹³⁵ strongly suggests a synergism of cigarette smoking and asbestos exposure in the increased risk of lung cancer in insulation workers. It is not known whether this is because of reduced clearance of asbestos, transportation of cigarette-smoke carcinogens by asbestos fibers, or the promotion by one factor of cancer initiated by another.

CHAPTER 2

EVIDENCE OF HUMAN NONOCCUPATIONAL EXPOSURES

Direct and indirect evidence that persons other than those working directly with asbestos minerals are being exposed to asbestos is of several types. For example, asbestos fibers can be demonstrated in the lungs of persons not occupationally exposed. In a few geographic areas, pathologic changes regarded as representing a reaction to asbestos (e.g., pleural calcification) have been found in populations with no history of occupational exposure. Asbestos fibers have been demonstrated in ambient air.

FIBERS IN LUNG TISSUE

Structures that appear to be fibers coated with a pigmented material were described in lung tissue as early as 1907 by Marchand.⁹⁶ These structures were actually fibers coated with hemosiderin. In 1929, Cooke¹⁹ described such "curious bodies" in association with pulmonary fibrosis. Stewart and Haddow¹⁴⁴ referred to them as "asbestosis bodies." Because those who work with asbestos exhibit them a few months after starting work, it was recognized that they were evidence of exposure, but not of asbestosis. The term "asbestos body" came to be the preferred designation.

As long as the coated fibers were found in persons known to have been occupationally exposed to asbestos, the identity of the central fiber was seldom questioned, although from time to time similar objects were found in persons with no known exposure to asbestos. Meurman¹⁰⁷ in 1966 summarized 19 reports published between 1932 and 1962 in which these objects were associated with exposure to graphite, coal, hornblende, rutile, diatomaceous earth, carborundum, and talc (in which case tremolite asbestos might have been involved). The demonstration by Gross^{54,56} that other fibers may

produce such bodies in experimental animals indicates that they result from a nonspecific reaction to any sparingly soluble fibrous foreign body, as had first been suggested by Cooke in 1929.¹⁹ There is thus ample justification for abandoning "asbestos body" as a generic term; Gough⁵¹ in 1965 suggested the term "mineral fiber-body," and Gross⁵³ in 1966 recommended "ferruginous body."

Thomson et al.,¹⁵¹ in 1963, were the first to show that these coated fibers were present in a high proportion of lungs obtained by routine autopsy. They found that 26.4% of lung smears in 500 consecutive autopsies in Cape Town showed what were called "asbestos bodies."

Reports from many other areas have confirmed a high prevalence in lungs obtained in similar autopsy series.^{3,8,17,30,47,107,118,123,150}

Uttdjian et al.¹⁵³ inferred that, if a sufficient volume of lung tissue were examined in each case, nearly all persons would be found to have such bodies; their study of 100 lungs in Pittsburgh confirmed their suggestion.

Identification of the core fibers has proved to be a formidable technical task.^{57,84,119} Without fiber-by-fiber analysis, all that can be said is that coated fibers resembling those in asbestos workers are present in most persons in our urban centers. Stripping the coating and analyzing the cores by various techniques can sometimes demonstrate that the cores are asbestos, but the process is tedious and often inconclusive.

Attention is now being directed toward study, not of the ferruginous bodies alone, but of the total fiber content of the lungs, whether such fibers are coated or uncoated. In a study of 3,000 consecutive autopsies in New York City, Langer et al.⁸⁴ have found thin, uncoated, optically

visible fibers in two-thirds of the 1,449 lung specimens in which coated fibers were demonstrated and in one-fourth of those in which uncoated fibers were demonstrated. Twenty-eight consecutive samples of lung tissue from the same series examined by electron microscopy were found to contain EM-sized chrysotile fibers.⁸⁵ Pooley et al.¹¹⁹ have reported similar findings.

Evidence is therefore strong that most human lungs harbor thousands or millions of fibers. Some of these are chrysotile asbestos, and other types of asbestos minerals are probably there also. In most persons not occupationally exposed to asbestos, the numbers of fibers are relatively small, compared with the numbers found in the occupationally exposed.¹³³ The systematic application of quantitative techniques, measuring both coated and uncoated fibers, is ~~needed to define~~ a gradient of accumulated fibers for correlation with incidence of disease, on the one hand, and history of environmental exposure, on the other.

Although there appears no doubt that asbestos fibers are present in many human lungs, there are sources of airborne fibers other than asbestos.^{28,57} Some are probably derived from the burning of leaves and plant products, such as paper, wood, and coal. Man-made (mostly vitreous) fibers have also been identified in the sediment isolated from human lungs. Talc, often used generously as a dusting powder, may contain a significant amount of tremolite asbestos fibers.

Information is sparse concerning possible increase of fibers in lungs with increasing use of asbestos and concerning the existence of significant differences between urban and rural populations. Selikoff and Hammond¹³³ compared lung tissues obtained in 1934 and 1967 and found no significant

increase in the proportion containing ferruginous bodies. This suggested that, despite increasing use of asbestos in New York City between 1934 and 1967, fibers of a type producing ferruginous bodies had not been increasing at a corresponding rate. However, Chang-Hyun Um⁷³ reports an increase over each decade in asbestos bodies in samples of lungs from persons who died in London in 1936, 1946, 1956, 1966.

PLEURAL CALCIFICATION IN THE GENERAL POPULATION

Meurman¹⁰⁸ in 1968 reviewed critically the literature related to pleural calcification and asbestos exposure. A number of studies strongly suggested an association between pleural calcification and nonoccupational exposures to asbestos. For example, Kiviluoto⁷⁹ in 1960 reported calcifications in 9% of the adult population detected during mass roentgenographic surveys in a Finnish commune in which there was an asbestos mine and mill; the frequency was low for the remainder of the Finnish population. Naunio¹²⁰ enlarged on these observations in 1966, reporting that, of 633,201 chest films taken in Finland between 1960 and 1965, 1516 showed pleural calcifications; 1232 of the latter were among 43,483 films taken in 10 communes in which there were anthophyllite mines. Rock and soil in such areas also contain much asbestos, so that the demonstration that airborne anthophyllite could be demonstrated over 25 km from the mines is not necessarily relevant. Anspach⁴ reported that, of 244 subjects with pleural calcification found in a chest roentgenographic survey in Dresden, 177 had either worked in or lived near an asbestos factory. Zolov et al.¹⁷⁰ described a 5.1% prevalence of pleural calcification in a rural population in Bulgaria and suggested that the most likely cause was asbestos in the soil. However, Hromek,⁷² Marsova,⁹⁷ and Rous and

Studený,¹²⁶ reporting on a high prevalence of pleural plaques in a rural district of Czechoslovakia, have been unable to demonstrate a source of asbestos exposure. The consensus at present is that calcification alone may not invariably be considered an index of asbestos exposure in the general population, although it may prompt a search for an environmental source of asbestos.

MEASUREMENT OF AIRBORNE ASBESTOS

A more direct method of obtaining evidence on the likelihood of exposure of the general population would be the sampling of air to determine the presence and amount of respirable asbestos fibers. There are, however, many uncertainties as to the best methods of sampling, identifying, and quantitating airborne asbestos and interpreting data so obtained.^{78,90,122,131} Limited information has been derived from measuring fibers on sampling sites, such as that by Laamanen *et al.*,⁸³ who showed asbestos fallout diminishing rapidly beyond 1 km from an anthophyllite quarry, but still detectable at 27 km. Counts of asbestos fibers collected on membrane filters by high-volume air sampling and estimated by light microscopic techniques similar to those used in industrial hygiene have shown small numbers of fibers in a few urban sites.^{10,16} Such results, although showing numbers of fibers detectable by the light microscope that were low by occupational health experience, have been too few and variable to be used with confidence. Alternative methods that are currently under development, including estimations of the number and mass of fibers in the LM and the EM size ranges, have shown measurable concentrations of asbestos in many samples of ambient air.^{1,66,137} Such environmental measurements are in their earliest stages and provide few clues to the extent or significance of the risk from this type of exposure to asbestos or to other mineral fibers.

ESTIMATION OF RISK IN NONOCCUPATIONAL EXPOSURES

Industrial experience has shown that prolonged inhalation of asbestos can increase the risk of neoplastic disease. Examination of lung tissue has made it apparent that a much larger proportion of the general public has inhaled and retained asbestos fibers than had formerly been realized; in fact, most urban dwellers have some such fibers in their lungs. Can these facts be related? Does the general public--as well as persons working near occupational sources, living in the households of asbestos workers, living in the neighborhoods of asbestos plants, or having occasional random exposures--have a detectably increased risk of malignancy or other disease because of airborne asbestos? The limited information we have to answer these questions comes either from direct epidemiologic studies of groups with various levels of nonoccupational exposure or by extrapolation from the experience of industrial populations with direct or indirect asbestos exposures.

EPIDEMIOLOGIC STUDIES RELATED TO NONOCCUPATIONAL EXPOSURES

Two general indices of asbestos exposure are available for use in direct epidemiologic studies of groups not known to be occupationally exposed to asbestos. The first is based on knowledge of each member's place of work and place of residence; because of the long latent periods of asbestos-related disease, this knowledge must cover each person's whole lifetime. The second is a quantitative estimate of each member's lung content of asbestos fibers. There are few such direct epidemiologic studies, and they are inadequate to answer the questions at issue.

The only studies that appear to implicate asbestos in the development of malignancies in persons not occupationally exposed are those involving diffuse mesothelioma, a tumor that is uncommon and that has been the subject of special attention in recent years. Many of the mesotheliomas reported by Wagner *et al.*¹⁶¹ in South Africa were attributed to household and neighborhood exposures in a crocidolite-producing area. Although non-occupational, these exposures have been described as substantial.¹⁶⁸ Newhouse¹¹⁴ studied 76 patients with mesothelioma diagnosed in London Hospital from 1917 to 1964. Of these 31 (40.8%) had occupational exposures to asbestos, 9 (11.8%) had a relative who worked with asbestos, 11 (14.5%) had neither of those backgrounds but had lived within a half-mile of an asbestos factory, and 25 (32.9%) had no known contacts. Corresponding percentages for a group of matched control subjects (patients in the same hospital for other diseases) were 10.5%, 1.3%, 6.6%, and 81.6%. Stumphius and Meyer¹⁴⁵ reported no mesotheliomas in the community near the shipyard in Flushing (Holland), although 17 of 21 mesotheliomas reported in the province of Zeeland in 1964-1967 had been in workers in that yard. Raunio¹²⁰ found no excess of pulmonary, pleural, or peritoneal malignancies in the areas of Finland where pleural calcifications attributed to anthophyllite were present in 6%-9% of routine chest survey films. In a series of 17 mesotheliomas collected by Borow *et al.*,¹² all but two were in persons who had worked in an asbestos mill, although the autopsy series from which the cases were drawn came from an area that included inhabitants of the mill's environs. Lieben and Pistavka⁸⁷ found that of 42 persons with mesotheliomas reported in Pennsylvania, 10 had worked in asbestos plants, 8 lived or worked close to an asbestos industry, and 3 were members of families that

included asbestos workers; in 11, no history of exposure could be obtained, and the remaining 10 had questionable random exposures. McDonald et al.¹⁰⁰ collected information on 165 fatal malignant mesotheliomas known to pathologists in Canada between 1959 and 1968. They confirmed an association with occupational exposure to asbestos but concluded that the excess was in the manufacture and industrial application of asbestos, rather than in mining or milling. It is apparent that no quantitative conclusions were possible from these studies, which present serious methodologic problems to the epidemiologist. They suggest a risk in household contacts and in residence in the immediate neighborhood of asbestos plants. There appear to be different levels of risk in different types of occupational exposures, and some of these may be reflected in corresponding household and neighborhood experience.

In no analysis of causes of death in a large population has there been quantitative estimation of the lung content of ferruginous bodies and bare asbestos fibers, to determine whether a detectable gradient of disease can be correlated with asbestos content. The series so far studied have been too small, and methods have been too variable, to permit any conclusions as to the importance of small numbers of fibers in the lung.

EXTRAPOLATION FROM OCCUPATIONAL EXPERIENCE

Another source of evidence of the relative risks associated with inhaling moderate or small numbers of asbestos fibers is the experience of persons who have had occupational exposures below those known to be definitely hazardous. The maximal airborne fiber concentrations recommended for prevention of asbestosis are much higher than any likely to be encountered in

nonoccupational situations. For example, one recommended standard would limit the average concentration of airborne chrysotile to 2000 fibers per liter as determined by light-field count.¹⁴ Another that has been proposed would limit average concentrations of fibers to 5000 fibers per liter.²

Occupation-related asbestosis can be effectively controlled with airborne fiber concentrations much higher than are likely to be encountered in non-occupational situations. It is important to determine whether workers whose exposures have been reduced to levels that prevent or greatly delay asbestosis, as well as others whose exposures are indirect, have a lower risk of lung cancer than those with higher and more direct exposures.

Workers who began employment in a British textile mill after 1933, when implementation of the Asbestos Industry Regulations of 1931 reduced (but did not abolish) dust exposures, were reported in 1968 to show no excess of neoplasms.⁸² The long latent periods of asbestos-related lung cancer and mesothelioma, which would probably be even longer at lower dose levels, are such that it is too soon to draw final conclusions as to the eventual incidence of these malignancies. Nevertheless, reduced exposure seems to be having an effect. Another indication of reduced incidence or delayed onset of disease with lower exposure is in the observations of Newhouse,¹¹² who found that, although there were more deaths from lung cancer and chronic respiratory disease among those who had heavy exposures many years previously in a London asbestos-products plant, this was not true among those who had low or moderate exposures. McDonald *et al.*¹⁰¹ recently reported the mortality experience of men who worked in chrysotile mines and mills of Quebec. There was a slight excess of lung cancer among the 2457 deaths in workers born 1890-1920, but all could be explained by the excess that

occurred in those who had been maximally exposed. This suggests that, insofar as chrysotile miners and millers are concerned, the risk drops off rapidly with decreasing accumulated dosage.

Most series of case reports of mesothelioma include some persons who have worked in the construction or shipbuilding industries, but in trades not involving direct contact with asbestos.¹⁰² Such persons as plumbers, electricians, and metal workers often have more ferruginous bodies in their lungs than do white-collar workers.¹³⁴ Although Dunn and Weir,³³ in a study of occupational groups in California that revealed an excess of deaths from lung cancer in insulation workers, found no excess lung-cancer deaths in other construction trades, the groups they studied were diluted with many persons who were unlikely to have had exposures. Nevertheless, there may be a definable gradient of effect within the construction trades. More thorough studies of groups with indirect exposures are certainly needed.

We cannot extrapolate from the mortality experience of those who are directly and indirectly exposed to asbestos in their employment to the general public who have had moderate or slight exposures from ambient air. There is evidence to suggest a gradient of effect from direct occupational, to indirect occupational, to family and neighborhood situations, in all of which dust concentrations are probably high by comparison with most community air. This suggests that there are levels of asbestos exposure that will not be associated with any detectable risk. What those levels are is not known, but there is no evidence that persons in the general population--without occupational, household, or neighborhood exposures--have any increased risk of neoplasm, even though there may be ferruginous bodies of fibers in their lungs.

CHAPTER 4

SOURCES OF ASBESTOS FIBERS IN AMBIENT AIR

Precise information is not available on tonnages, numbers of fibers, fiber sizes and varieties, atmospheric dispersion, and ultimate fate of the asbestos emitted into ambient air. Although there are no reliable data to justify extrapolation from the more completely studied occupational exposure experience, information regarding actual and potential sources of emissions of asbestos fibers is of value both for directing future studies and for understanding the steps that might be taken now to safeguard the health of the public.

NATURAL SOURCES OF AIRBORNE ASBESTOS FIBERS

Several varieties of asbestos ore and counterpart rock (containing EM-sized asbestos fibers) occur as outcroppings or are just below the surface of the earth throughout the world. Asbestos fibers can become airborne from these formations during road-building, construction, and tilling of the soil, as well as by landslides, erosion, and weathering. Talc, mined and used extensively in the United States, exists in fibrous, as well as platy, form. Like asbestos ore and rock, talc exists on or close to the earth's surface and is subject to disseminating forces. Such naturally occurring talc, as well as the large quantities used as a diluent and carrier for pesticides, can add to the background fiber concentration in the ambient air.^{166,167} It is thought that studies of fibers in glacial and polar ice now under way will permit comparisons of recent deposition with those in the past and thereby provide definite information on the relative contributions of natural and industrial sources.

MINING AND MILLING OF ASBESTOS

Mining and milling of asbestos provides another source of asbestos emissions. In the United States, such activity is presently confined to a few mines in California, Vermont, Arizona, and North Carolina. Fibers are emitted during removal of overburden and preparation of the ore body for open-pit mining. Further release occurs during drilling and ore-breaking. Waste dumps from mining and milling are exposed to wind and to disturbance by bulldozing. Fibers are emitted during drying, crushing, grinding, and screening of the ore. If dust collectors and air-cleaning devices are used, disposal of the collected dust provides a potential source of fiber emission.

TRANSPORTATION OF MATERIALS CONTAINING ASBESTOS

Transportation of asbestos ore, milled asbestos fiber, and asbestos-containing products and wastes is an emission source of varying importance. Movement of asbestos ore from mine to mill in open trucks contributes to the overall emission. The shipment of milled asbestos fiber, usually in bags, can result in emissions. If bags are reused, either in the asbestos industry or elsewhere, they will become a source of fibers. Occasionally, bags are broken and asbestos is spilled during handling. Similar emissions

occur during the shipment of products. Transporting asbestos-containing solid wastes in open vehicles through urban areas can be a more important emission source.

MANUFACTURE OF PRODUCTS CONTAINING ASBESTOS

Industries that must provide ventilation and other dust-control measures for the protection of workers may emit asbestos fibers into the surrounding environment unless effective air cleaning is applied to effluents. Fibers removed by ventilation and filtering devices and not reintroduced into the production process and asbestos-containing waste products of the manufacturing process ultimately are disposed of outside the plant.

USE OF PRODUCTS CONTAINING ASBESTOS

Many products, at times unknown to the user, contain asbestos of one kind or another. There are great variations among such products with respect to the chances of fiber release during the use of the product. The likelihood depends predominantly on the ease with which the fibers can be dislodged by the application of energy and on the degree to which the application of energy actually destroys the fibers during the use of the product. Almost all the asbestos fibers used in the United States for manufacturing products becomes tightly bound within the products and undergoes little actual abrasion or wear before being discarded. Asbestos cement products (accounting for most of the asbestos used in the United States), shingles, and floor tiles are in this category. Some asbestos-containing products, such as brakelinings, are subjected to high energy, and their rate of wear is considerable and at times almost complete. In the case of brakelinings, the application of energy is so intense and the heat created so great that

most chrysotile fibers are destroyed by being converted to another substance, which is nonfibrous.^{65,68,89} Nevertheless, an appreciable percentage (1%-3%) remains as fibrous asbestos. In some products--for example, asbestos cloth, paper, and sprayed fireproofing materials--asbestos fibers are not tightly bound or mixed with other material that holds them in place. Fiber release from these products occurs primarily during application and removal. The spray fireproofing of buildings with asbestos-containing materials is a case in point. This operation can be a serious source of emission, in that it usually occurs in densely populated areas. The total amount of asbestos fiber used in such procedures, however, is relatively small.

Talc is mined and milled and used in greater quantities than is asbestos in the United States. Because it contains asbestos fibers, its uses will add to the total number of fibers (including nonasbestos fibers) emitted. The use of talc in dispensing pesticides over wide areas of the country and its use in cosmetics are two examples of how this material may act as a source of asbestos and other fibers.

DEMOLITION

For years, asbestos has been incorporated in building materials. In some forms of insulation and wallboard, the amount present is less than 20% of the total; but other materials consist mostly or entirely of asbestos. When a building is demolished, areas of loosened asbestos are open to the ambient air and fibers are emitted. In general, single-family residential structures contain only small amounts of asbestos insulation. Demolition of industrial and commercial buildings that have been fireproofed

with asbestos-containing materials will prove to be an emission source in the future, requiring control measures.

SOLID-WASTE DISPOSAL

Solid wastes produced during manufacture of asbestos-containing products, use of such products, and demolition can be emission sources. These waste materials are usually disposed of without regard to their potential as emission sources. Alternate methods of disposal often result in commingling of asbestos-containing wastes with municipal wastes in open dumps and thus create a long-term emission source.

CHAPTER 5
PRINCIPLES OF CONTROL

The natural background level of asbestos fibers is difficult to quantify or modify. The major sources of local ambient-air contamination with asbestos are industrial processing and use of products containing asbestos. It is feasible to identify the sources of emission, select those to which presently available control procedures can be applied, and point out areas that need further study and development of new methods of control.

NATURAL SOURCES OF AIRBORNE ASBESTOS FIBERS

Natural sources of asbestos fibers have been identified in many areas of the United States. It is assumed that emissions of fibers from these reservoirs through erosion and wind make up a natural background of asbestos in ambient air. Few data are available on the magnitude of the contamination from natural sources, variation with geographic location, and seasonal variation. It is desirable to have information on the natural background, because it would assist in evaluating the effects of control measures and permit some estimates of the lower limits of contamination that might be achieved in different areas of the country.

MINING OF ASBESTOS

The standard techniques for dust control in underground or open-pit mines, if not already in use, can be applied in asbestos mines. Roadways in open pits should be treated with dust-suppressive agents; truckloads of ore should be covered with tarpaulins while being transported; handling of ore should be minimized; ore storage piles should be moistened to reduce wind erosion; and waste dumps should be treated with dust-suppressive agents.

MILLING OF ASBESTOS

An in-plant dust-control program for protecting workers in asbestos mills includes exhaust ventilation with efficient dust-collecting and air-cleaning equipment, isolation, enclosure, wet methods, and good housekeeping and maintenance.

MANUFACTURE OF PRODUCTS CONTAINING ASBESTOS

The elements of dust control recommended for the milling of asbestos ore also apply to the manufacture of asbestos-containing products. It is important that a dust-control system be specific to the operation for which it is used and that it be tested to ensure its effectiveness. Only persons especially trained and experienced in dust control should be used to develop and institute dust-control procedures.

USE OF PRODUCTS CONTAINING ASBESTOS

An important emission source is the use of insulating materials containing asbestos. This constitutes only a small fraction of the asbestos used. But the asbestos in some insulating materials is not bound as it is in cementitious products or tiles; construction activities usually are carried on in urban areas where many people are exposed to contaminated air; and control of dust during construction, although feasible, is difficult.

The most effective approach in reducing exposures of the general public to asbestos from this source involves controlling dust production and release at its origin during construction work. The generation of dust should be reduced by changing material-handling methods, work practices, and cleanup procedures. Local exhaust systems should be used for dust collection at points of generation; for example, dust collectors for band

saws are available, and hand-powered tools supplied with exhaust systems are being made. Much developmental work is needed to produce portable air-moving and air-cleaning systems for use in tunnels, crawl spaces, and other confined spaces. This subject has been neglected by industrial hygienists and ventilation engineers, and no satisfactory equipment is available.

DEMOLITION AND WASTE DISPOSAL

Demolition and waste disposal are likely to be emission sources if appreciable amounts of asbestos are used in construction, unless operational procedures are strictly controlled. Isolation, enclosure, and wetting down are useful. Caution must be observed not to demolish during high winds and to keep sludge from drying out and becoming airborne later through natural forces and from being introduced into sources of drinking water.

MEASUREMENT OF AIRBORNE ASBESTOS

An important consideration in the development of a strategy for control is whether there are methods for measuring airborne concentrations of asbestos that are sufficiently sensitive, specific, and reproducible. Present methods of sampling, identifying, and measuring airborne asbestos are not entirely satisfactory, especially if one is dealing with low concentration and unidentified or mixed sources. Only within recent years have methods for determining concentrations of fibers for industrial hygiene purposes been standardized;^{14,35} they use samples collected on membrane filters in which fibers are counted with phase-contrast illumination. Electron microscopic methods give a much more complete indication of the total fiber content

of the air; but when the need for fiber identification is included, they are tedious and expensive for routine use.^{1,66,137} The relative biologic significance of different sizes of fibers is not known, nor is the relative importance of fiber numbers and fiber mass. There appear to be no published data on the efficiency of air-cleaning equipment as related to fibers of different sizes.

In spite of the difficulties, it is possible to sample air, determine the approximate concentration of airborne fibers, and identify the major types of asbestos. It is not desirable to limit environmental measurements to a single method until there is a clearer definition of the critical variables in terms of health. Because of methodologic and other uncertainties, it is not yet feasible to base control on numerical ambient air quality standards.

CHAPTER 6

RESEARCH NEEDS

Two recent reports^{25,167} have discussed in some detail the many kinds of research needed to answer pressing questions concerning the effects of asbestos on health and the degree and nature of necessary controls. Investigations along the following lines should be given high priority.

Study of the mechanism of action of the asbestos minerals should continue, with particular attention to carcinogenic effects. It is important to learn more about the influence of asbestos type and fiber size on respirability, deposition, retention, translocation, and effects at the tissue, cellular, and molecular levels, with and without cofactors. It is especially important that the role of fibers below the LM range be clarified.

Methods of sampling, identifying, and quantitating airborne asbestos need continued development. Coordination with studies in animals and man is essential to ensure that environmental data will be biologically relevant. Similarly, methods for identifying and quantitating asbestos in biologic tissue need development and application.

Quantitative methods for measuring airborne asbestos should be applied widely to determine the natural background and the concentration and distribution of fibers in the air near various sources. Conventional LM methods and EM methods should be applied simultaneously in selected occupational and community situations.

More epidemiologic studies are needed. Populations in several different exposure ranges should be studied, including occupational, household, and neighborhood exposures. Special studies of mesothelioma are needed to determine whether the incidence has been increasing and to determine the current pattern of distribution. A large series of routine autopsies should be studied to determine whether causes of death can be related to amounts of asbestos in the lungs and other organs. All the above are urgent if a range of safe exposure is to be established with confidence.

CONCLUSIONS AND RECOMMENDATIONS

PATHOGENICITY OF ASBESTOS MINERALS

Any of the commercially used asbestos minerals, when inhaled in sufficient numbers, as in uncontrolled occupational exposures, can cause disabling fibrosis of the lungs. An association between occupational exposures to asbestos and bronchogenic carcinoma has been established, but the dose relationship and the role of cofactors have not been defined. Evidence of a causal association between some but not all exposures to asbestos fibers and diffuse malignant mesotheliomas of the pleura and peritoneum is substantial, but evidence of such a relationship with other tumors is inconclusive. Although the different types of asbestos differ in some of their biologic effects, no type can be regarded as free of hazard. The hypothesis that asbestos fibers act as cofactors or carriers of carcinogens is attractive, but as yet unproved.

EVIDENCE OF HUMAN NONOCCUPATIONAL EXPOSURES TO ASBESTOS

The demonstration of ferruginous bodies, similar to those found in asbestos workers, in a large proportion of randomly selected lung specimens in many parts of the world is presumptive evidence that persons with no occupational contact may have inhaled and retained asbestos. Proof has come in some areas with positive identification of chrysotile asbestos fibers. Analyses of community air for asbestos have been too limited to define the sources, concentrations, and distribution of fibers in the environment. The fiber concentrations that have been demonstrated in ambient air are small, compared with those in industry, but data are inadequate for definitive comparisons.

ESTIMATION OF RISK IN HUMAN NONOCCUPATIONAL EXPOSURES TO ASBESTOS

The most important question in the case of persons with nonoccupational exposures to asbestos is whether there is an increased risk of malignancies. Industrial experience indicates that there is no likelihood of significant asbestosis in nonoccupational exposures. The major potential for risk appears to lie in those with indirect occupational contacts, household contacts, or residence in the immediate neighborhood of asbestos sources; and even there, the actual risk is poorly defined. But the fact that there appears to be a gradient of effect in such groups suggests that there are levels of inhaled asbestos without detectable risk. It is not known what range of respirable airborne asbestos fibers will ultimately be found to have no measurable effects on health. At present, there is no evidence that the small numbers of fibers found in most members of the general population affect health or longevity.

NEED FOR AND FEASIBILITY OF CONTROLS

Asbestos is too important in our technology and economy for its essential use to be stopped. But, because of the known serious effects of uncontrolled inhalation of asbestos minerals in industry and uncertainty as to the shape and character of the dose-response curve in man, it would be highly imprudent to permit unrestricted additional contamination of the public environment with asbestos. Continued use at minimal risk to the public requires that the major sources of man-made asbestos emission into the atmosphere be defined and controlled. In the absence of such controls, local fiber concentrations might at times approach those in occupational sites. Analytic methods and epidemiologic data are inadequate for the development of ambient air standards, but emission controls are needed and appear feasible.

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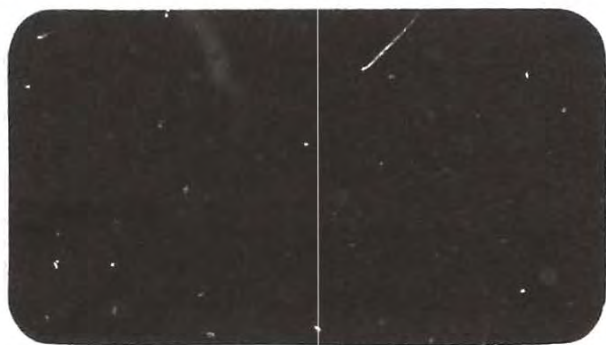
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EXHIBIT 20

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Report No. IITRI-C8216

ASBESTOS AIR POLLUTION CONTROL

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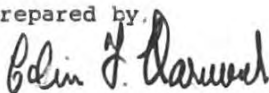
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FOREWORD

This report entitled "Asbestos Air Pollution Control" is issued as a final report on IITRI Project C8216. The report has reviewed the subject of asbestos and the air pollution caused by the production and use of asbestos containing materials. The research program was sponsored by the Institute of Environmental Quality in the State of Illinois.

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ABSTRACT

ASBESTOS AIR POLLUTION CONTROL

Asbestos is a very widely used material for which an estimated 3000 uses have been found. Indiscriminant use of this material may constitute a hazard to the health of the general populace.

This report reviews the many uses of asbestos and the extent of the problem with regard to airborne emissions. The efforts which are made to control these emissions are discussed and detailed. Sections include manufacturing the uses, disposal, alternatives, monitoring, storage and transportation.

The report contains considerable quantities of up-to-date knowledge from which the substantiation of proposed regulations on the use of asbestos may be based.

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ASBESTOS AIR POLLUTION CONTROL

1. INTRODUCTION

The term asbestos applies to a number of fibrous mineral silicates. They can be divided into two groups, serpentine (chrysotile) and amphibole. Amphibole contains anthophyllite, amosite, crocidolite, tremolite, and actinolite.

Asbestos is unique, as a naturally occurring mineral, in the fact that due to its fibrous nature it can be woven. Its industrial usefulness is based on its non-flammability, flexibility, tensile strength, low density, resistance to acids and alkalies, and high electrical resistivity.

In Appendix II, the properties of asbestos have been summarized.

Unfortunately, this unique and useful material is known to have adverse health effects. The subject of this report is to review the sources of emission to the atmosphere and to consider the reasonableness and the rationale of the ways in which the emissions may be curbed.

2. SOURCES OF ASBESTOS

Asbestos deposits of commercial value in the United States are located in Vermont, California, Arizona, and North Carolina. Although not exploited on a commercial scale, small asbestos outcroppings have been reported in many other states. A list of such sources is given in Table 1. Samples of ore from each of these sources are to be seen at the Chicago Museum of Natural History. It is noted that no commercially viable sources are to be found in Illinois.

United States mining of asbestos has increased 180% over the past 10 years with a high percentage of this increase credited to California producers (California now outranks Vermont in total

Table 1
A PARTIAL LIST OF ASBESTOS OUTCROPS

Location	State
Cleburn County	Alabama
Dahl River } Kobak } Shanak River }	Alaska
Yabapia County	Arizona
Garland County	Arkansas
Duval County	Florida
Oscaloose County } Habersham } Pine Mt. Mine, Robin County } Sall Mt. Sautee }	Georgia
Washington County	Idaho
Buchanan County	Iowa
Greenup	Kentucky
New Orleans County	Louisiana
Penobscot	Maine
Blue Mt. Quarry, Baltimore	Maryland
Suffolk County	Massachusetts
Sturgeon Falls	Michigan
Huron County	Michigan
Jackson County	Missouri
Douglass County	Nebraska
Lincoln County } Humboldt County }	Nevada
Cumberland County	New Jersey

Table 1 (cont.)

Location	State
Royal John Mine, Grant County	New Mexico
Dutchess County	New York
Vance County } Bakersville County } Mason County }	North Carolina
Cuyahoga	Ohio
Multnomah } Adam Gordons Place on } Branch Creek, Grant County }	Oregon
Josephus County	Oregon
Lazerne County } Brintons Quarry, Chester County } Easton, E. end of Chestnut Hill }	Pennsylvania
Lawrence County	South Dakota
Davidson County	Tennessee
Bexa County	Texas
Weber County	Utah
Blue Ray County } Bedford County }	Virginia
Berkeley County	W. Virginia
King County	Washington
Albany County } Caster Mt., Natrona County } Encampment }	Wyoming

production). Total US production (1969) was 125,936 tons, valued at an estimated \$10,648,000.

The United States utilizes about one-fourth of the world's production of asbestos, practically all of it is imported from Canada and Africa. Figure 1 graphically illustrates domestic supply and consumption.

3. ASBESTOS EMISSIONS

3.1 Non-Industrial Sources

Before entering into the main source of asbestos emissions, that is, through commercial manufacturers processes, it is worthwhile to consider briefly other less obvious but important mission sources. These may be listed as:

- Rock Outcrops
- Farming
- Construction
- Talcum Powder

3.1.1 Rock Outcrops

It has been pointed out previously in Table 1 that asbestos-containing rock outcrops in very many areas of the United States. It is interesting to note that in 1920, Staten Island was once considered a large enough outcrop that mining on a commercial scale was undertaken there.

Emission from such outcrops will occur due to the natural eroding processes of earthquakes, temperature, wind, and rain. Thus, wind and water-borne erosion products will inevitably contain asbestos particles.

3.1.2 Farming

Farming of the soil in areas containing asbestos rock will cause the emission of fibers. Such actions as tilling of the soil are always dusty operations (unless, of course, it happens to be wet), the extent of the emissions will depend on

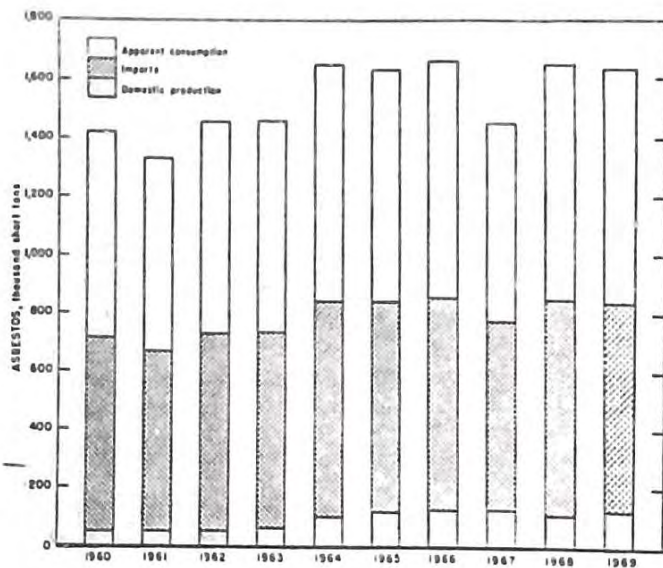


Figure 1
DOMESTIC SUPPLY AND CONSUMPTION OF ASBESTOS

the concentration of asbestos in the soil.

Studies have been conducted on the effect of such emissions on the health of the local population(1). The evidence, though not very conclusive, suggested that asbestosis could be caused by agricultural employment.

3.1.3 Construction

The outcrops of asbestos-containing rock will give rise to emissions whenever construction is undertaken. Hence, such activity as excavating for housing, pipe laying, and road making, must be considered as a source.

3.1.4 Talcum Powders

Talcum powder is a mineral rock dust very closely related mineralogically to asbestos. The main difference lies in the fact that talcum powder has a platy morphology, while asbestos is characterized by its fibrous nature.

The fact that talc is very frequently found with a significant fibrous content now leads to a very important situation since the total quantities of talc mined in the U.S. exceeds that of asbestos.

It was pointed out by Cralley(2) that the majority of cosmetic talcs contain significant quantities of fibrous particles. Virtually everybody, from babies up, is exposed to cosmetic talc and so are exposed to the threat of asbestos bodies.

Apart from the cosmetic use, talc is used very considerably in industry as, for example, a filler and as a lubricant in the production of pharmaceutical tablets and rubber goods. Another large use is as a carrier for pesticides in the spraying of crops.

On a more exotic note, there has been recent publications on the use of talcum for polishing rice(3,4) and its association with stomach cancer because of its asbestos content. Even more

exotic is the quote of Nader that children may be endangered by the talc used as a lubricant in rubber balloons.

The above discussion on talc has been included for completeness in this report concerning asbestos. Certainly it should not be neglected when considering the health effects and hazard of asbestos fibers.

Other studies discussing the presence of asbestiform fibers in the mining of talc are those of Siegal(5) and of Greenberg(6)

3.2 Manufacturing Processes

3.2.1 Introduction

Several tasks preliminary to the manufacture of asbestos products are potential emission sources. These are common to all asbestos processing. They include such functions as unloading and storage of milled asbestos, subsequent transport of the bagged asbestos to the bag opening area, fluffing of the compacted fibers, and a mixing process where asbestos fibers are dry mixed with other materials.

The bagged asbestos is usually unloaded from railway box-cars, moved to storage areas, and finally transported to bag opening areas by fork-lift trucks. Opportunities exist during each of these operations for bags to become damaged and spill fibers to the atmosphere.

The amount of emission from the bag opening and dumping processes depend upon the degree to which the tasks are enclosed. In some instances, the bags are cut open with sharp knives and the contents dumped into a container or onto a conveyor belt.

The compacted fibers, from packing at the mill, are fed through a "willow" or "Moody fluffer" which fluffs up and separates the fibers. This operation may also be employed as a blending stage wherein several grades of fibers are thoroughly mixed.

Emissions will be very bad during this operation if the fluffing is done in an open vessel.

Once separated, the fibers are ready for incorporation into the manufacturing phase. Here the fibers are usually dry mixed with other materials. Fiber emission to the atmosphere will occur where the process is not adequately contained or ventilated.

Each end product requires a unique series of manufacturing steps, any one of which may constitute an emission source. These are discussed individually in the following paragraphs.

3.2.2 Asbestos-Cement Products

The largest user of all grades and varieties of asbestos fibers is the asbestos-cement products industry. The largest segment of this industry is the production of asbestos-cement pipe. This variety of pipe has been utilized to convey a number of fluid and gas products among which are: water supplies (potable, drainage, and irrigation), sewage, industrial products, and air/gaseous products (heating, cooling, gas venting). They have also served as conduit for electric and telephone cables. Pipe sizes range from 3 inches to 48 inches and up in diameter to 13 feet in length.

Other products of the asbestos-cement industry include roofing shingles, insulation board, marine board, laboratory bench tops, etc.

Willowed fibers are dry mixed with fine ground silica and Portland cement. Water is added to the mixture forming a slurry. Once in slurry form, the problem of emission is reduced.

The end products of this industry require finishing steps which produce emission problems.

Completed products require finishing steps which involve one or more of the following operations: sawing, turning, drilling, and sanding. Each operation produces large quantities

of asbestos laden dust. Some of this dust clings to the surface of items being worked on, they, in turn, becoming emission sources.

3.2.3 Asbestos Asphalt and Vinyl Floor Tile Products

The second largest use of asbestos fibers is in the manufacture of asbestos-vinyl floor tile and asbestos-asphalt road paving compounds.

Relatively small additions of asbestos (2 to 3% by weight) to asphalt paving material permit the addition of increased amounts of asphalt (7 to 11% weight of total mix) resulting in a pavement compound of greatly improved quality. Application of this mixture to roadways is by standard paving equipment.

California has been a forerunner in utilizing asbestos-asphalt paving; however, there have been applications in many other areas of the country. The George Washington Bridge, the New Jersey Turnpike, and the Trans-Canadian Highway were constructed with asbestos-asphalt paving. Their use in Illinois is limited at this time.

Asbestos-asphalt and asbestos-vinyl products are processed as wet mixes and as such the major emission problems are limited to the handling procedures prior to processing.

Emission from installed paving compounds occurs due to the normal wear of the road surface. There is no information available at this time as to the nature and extent of these emissions.

Emissions associated with the tile manufacturing include the calendaring process and the destruction and reprocessing of rejected tiles. Cutting and stamping operations also cause minor emissions.

3.2.4 Asbestos Paper Products

Asbestos paper products have perhaps the greatest variety and range of use. A sampling of the variety of applications is

given in the following list excerpted from "Asbestos Fact Book" (Table 2).

Table 2
USES OF ASBESTOS PAPER

Air cell & other pipe covering
Boiler jackets
Asbestos felt roofing
Asbestos built-up roofing
Asbestos-protected metal roofing
Gaskets (plain & metal reinforced)
Wick in oil-burning apparatus
Tubes for electrical insulation
Electrical insulation of wire & cable
Insulation of hot-air pipes
Linings of stoves & heaters
Linings of filing cabinets, cartridges, carpets, auto mufflers, drum controllers, cookers, electrical appliances, armored car roofs, motors, etc.
In enameling ovens to catch drip
Insulation of ovens & dry kilns
Table pads & mats
Heat- and chemical-resistant reinforced pipe & other laminated products
Diaphragms in electrolytic cells
Tank covers
Reinforcing aluminum foil for insulation
Filtering
In window-glass machinery to guide hot sheets & to shield hot glass from flying fragments
In welding & other processes for protection from heat
In annealing (crumpled paper)
In chemical & physics laboratories
Insulating exhausts on automobiles
Clutch facings in automatic transmissions
Baking sheets
Hot-air ducts or lining of paper ducts for hot-air service

The Asbestos Factbook, 1970, p. 27. Published by the Magazine "Asbestos."

The pre-processing of the fibers and the mixing of the asbestos paper ingredients are an emission source. The paper is made utilizing a slurry similar to that used for wood-pulp paper. A mixture is prepared in a pulping mill from approximately

80% asbestos fiber, 18% china clay and 2% starch or sodium silica.

Finished stock, 6-10 feet in width is fed through a battery of knives to slice the sheet into narrower sections. This cutting action releases fibers to the atmosphere.

3.2.5 Asbestos Paint and Coating Fillers

The paint and plastic industries make use of asbestos "shorts" or "floats" recovered from the milling process. The fibers increase the strength and improve the durability of the end product. A further desirable characteristic of the fibers is that they are chemically inert and do not "age". Their small physical size suspended in a paint or plastic matrix increases the density or opaqueness of the coating.

Once the fibers are dispersed in the paint base, the major source of emission is the spraying operation, from which emission may be considerable. The wearing or flaking of the paint due to aging allows the asbestos fibers to be released to the atmosphere and thus constitutes an emission source.

3.2.6 Asbestos Friction Materials and Gaskets

Spinning grade fibers are utilized in fabrics for both friction and gasket materials. Multi-layer crude bands are impregnated by immersion in solutions of plastics, oils, or bitumens; dried; hardened (cured); and sized. Molded fabric-base friction materials are produced by impregnating multi-layer fabrics or strips with high temperature thermosetting resins. They are then molded under pressure and temperature (140-170°C) to give the desired shape.

Apart from the usual emission created during component handling and mixing, further emissions are produced during the finishing of the molded asbestos/matrix product. The molded blocks are cut, drilled, and trimmed to shape. Occasionally extensive trimming is required. Each of these operations produce emissions.

3.2.7 Asbestos Textile Products

Milled fibers are received in a compacted or semi-opened condition. These fibers plus the fibers separated at the textile plant are fed through "Willowers" or "Moody fluffers" which fluff up and separate the fibers.

Once sufficiently separated, the fibers are blended with small percentages (usually less than 5% but up to 25%) of organic fibers such as cotton or rayon which act as carriers or supporting agents to improve spinning properties. The blended fibers are given a final opening and cleaning process by action of the carding machine which combs the fibers into a relatively parallel arrangement forming a tenuous web. This web, or sliver, when separated into ribbons, rubbed mechanically and condensed into untwisted strands, becomes the roving. Roving is the asbestos textile product from which yarns are produced. The variety of products which are then produced are shown schematically in Figure 2.

Blended fibers are carded in machines where the operation is essentially open presenting a serious emission source due to the combing action of the machine. Roving is the output of this operation.

The design of machinery utilized for spinning, twisting, forming, and subsequently, weaving, braiding, plaiting, and knitting is such that multiple strands of asbestos fibers are processed exposed to the work area. The multiple strands present an extremely large surface area from which emissions can take place.

3.3 Product Use

3.3.1 Sprayed Asbestos Insulation

Asbestos insulating materials are applied utilizing specially designed spray guns operating on one of two basic principles. Either a prepared asbestos/cement slurry is pumped to the nozzle of the gun and sprayed directly to the surface or a dry mixture of asbestos/cement is pumped to the gun and is mixed with water during the spraying operation. Figure 3 shows the latter type of spray gun in operation.

Typically, the materials forming the asbestos insulation are asbestos, cement, and rock wool in proportions of 30, 15, and 55%, respectively. These ingredients are mixed (usually away from the construction site) and stored in 3-ply, 50-lb paper bags.

The most commonly used fiber is amosite because of its superior thermal properties. If color is required, crocidolite fibers may be used for blue and chrysotile fibers for white. Occasionally a decorative finish is desired. This may be obtained by tamping the material in the desired pattern before it sets. A coat of PVA or acrylic paint is frequently applied to enhance the appearance and provide surface strength.

Among the unique properties associated with asbestos cement insulation are: low thermal conductivity, inflammability, and high strength. Also the material provides good acoustic insulation. Installation features include low cost, easily controlled thickness of application, and the absence of joints.

Extensive use has been made of this material for fire-proofing of buildings and for thermal and acoustic insulation.

Emissions - The dry mixing of the asbestos, cement, and mineral wool presents emission problems similar to those discussed in previous sections. Care must be exercised during the storage and transportation of the lagged mixture to prevent

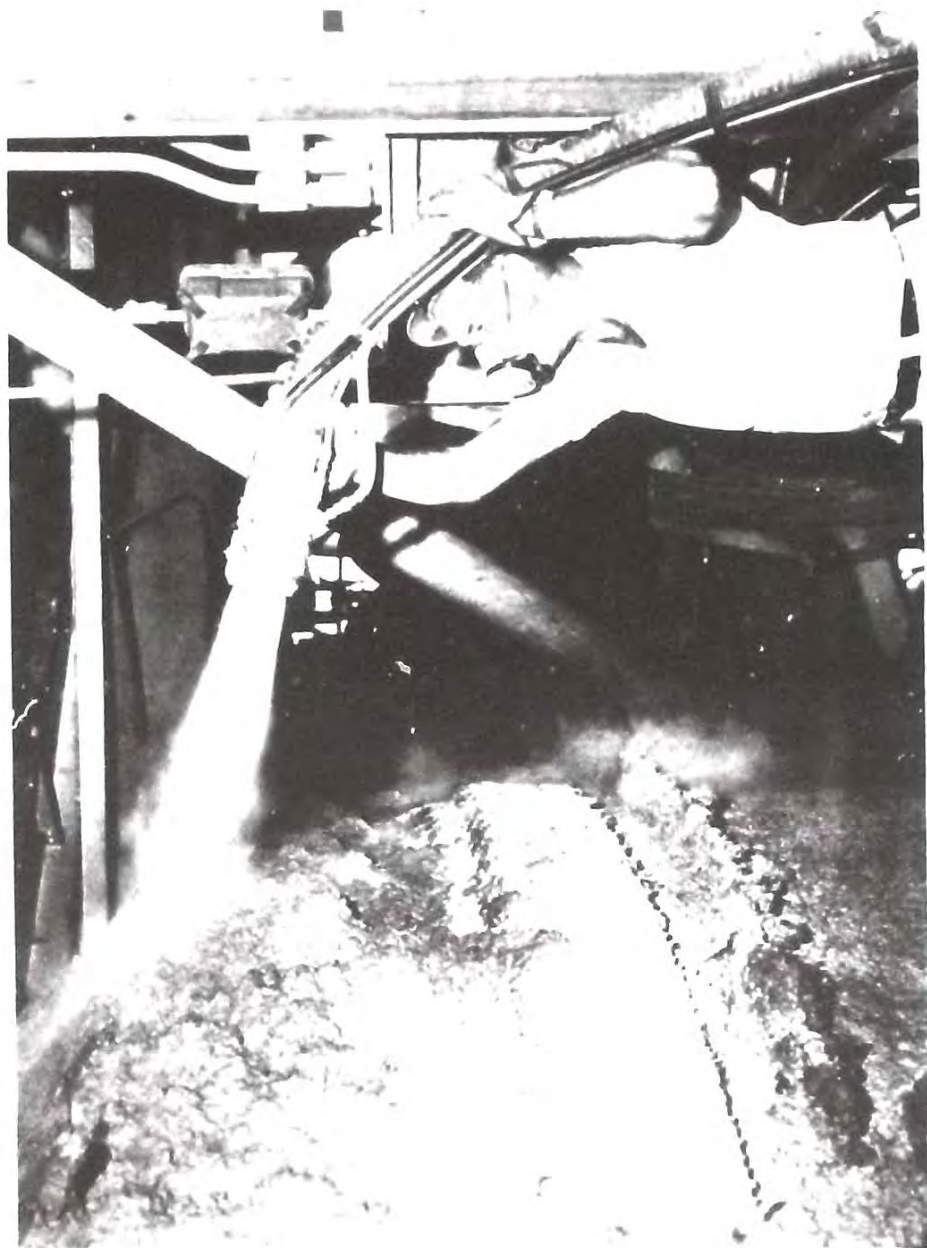


Figure 3. SPRAYING OF ASBESTOS CEMENT INSULATION

damage to the bags. At the construction site the bags are opened and the dry material emptied in hoppers: see Figure 4. Emissions occur at these operations.

The spraying operation causes emission. Studies have been conducted to determine the extent of this emission. Evidence shows high dust concentration can be both seen and measured at construction sites utilizing asbestos spraying methods. Emissions are also created during removal of sprayed asbestos that missed the target area and accumulated on nearby surfaces. Figure 5 illustrates the buildup of asbestos insulation material on surfaces surrounding the target area.

Emissions are also possible from finished surfaces, particularly if the surfaces are abraded or subjected to extreme vibration.

Control Techniques - Air ventilation systems can be employed to minimize emission during the mixing of asbestos-cement fireproofing/insulation materials. Hoods can be provided at bag-opening and emptying areas. The mixing area, if not totally enclosed, can be ventilated. Conveying equipment, to and from the mixer, can be enclosed. Bagging of the mixed material should be done under suction hoods.

On-site controls needed include enclosing the area to be sprayed and employing good housekeeping procedures both before and following the spray operation. The filling of the spray machine hopper must be done carefully to minimize dust. When the machine is not in use, the hopper should be covered to prevent the material from being blown about.

Several states and manufacturing associations have published guidelines for emission control. A summary of their findings and recommendations follows:

1. All areas used for opening bags containing asbestos insulating materials and/or charging of hoppers should be enclosed. Empty bags should be properly disposed of.

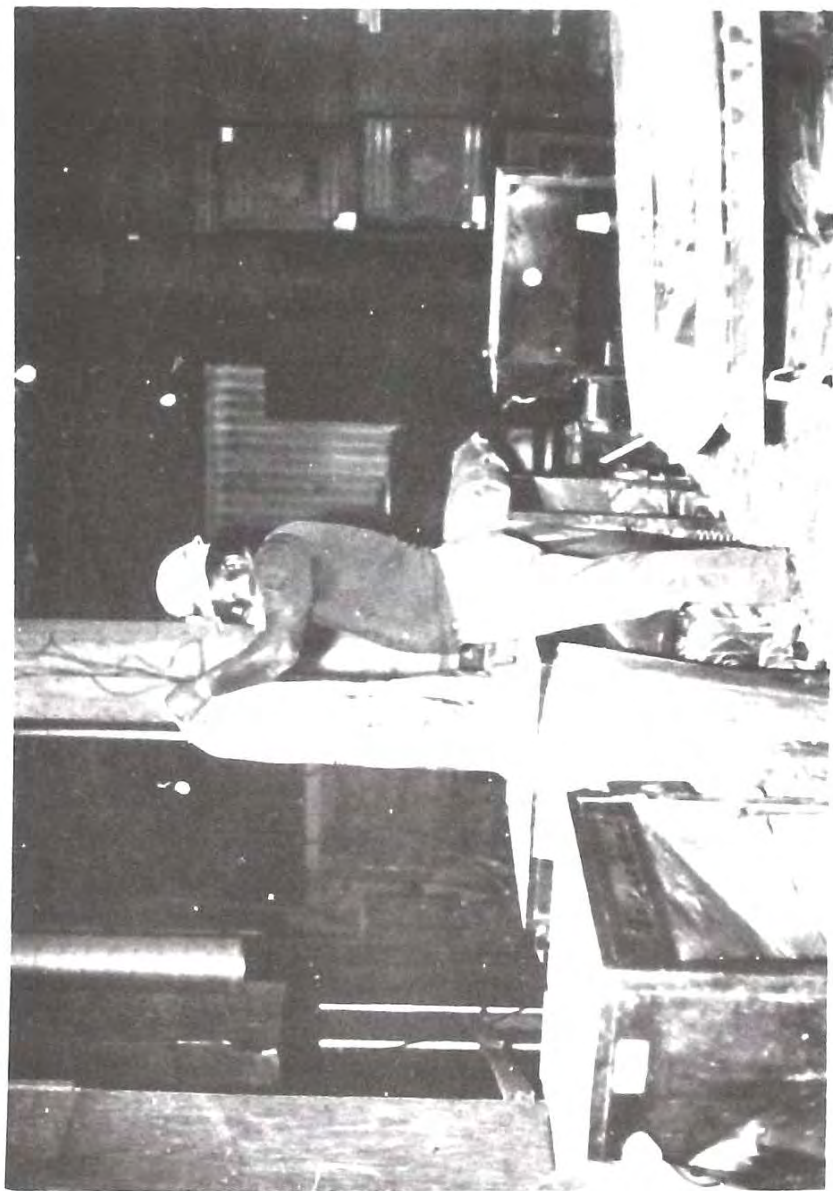


Figure 4. CHARGING OF HOPPER FOR SPRAYING ASBESTOS-CEMENT INSULATION

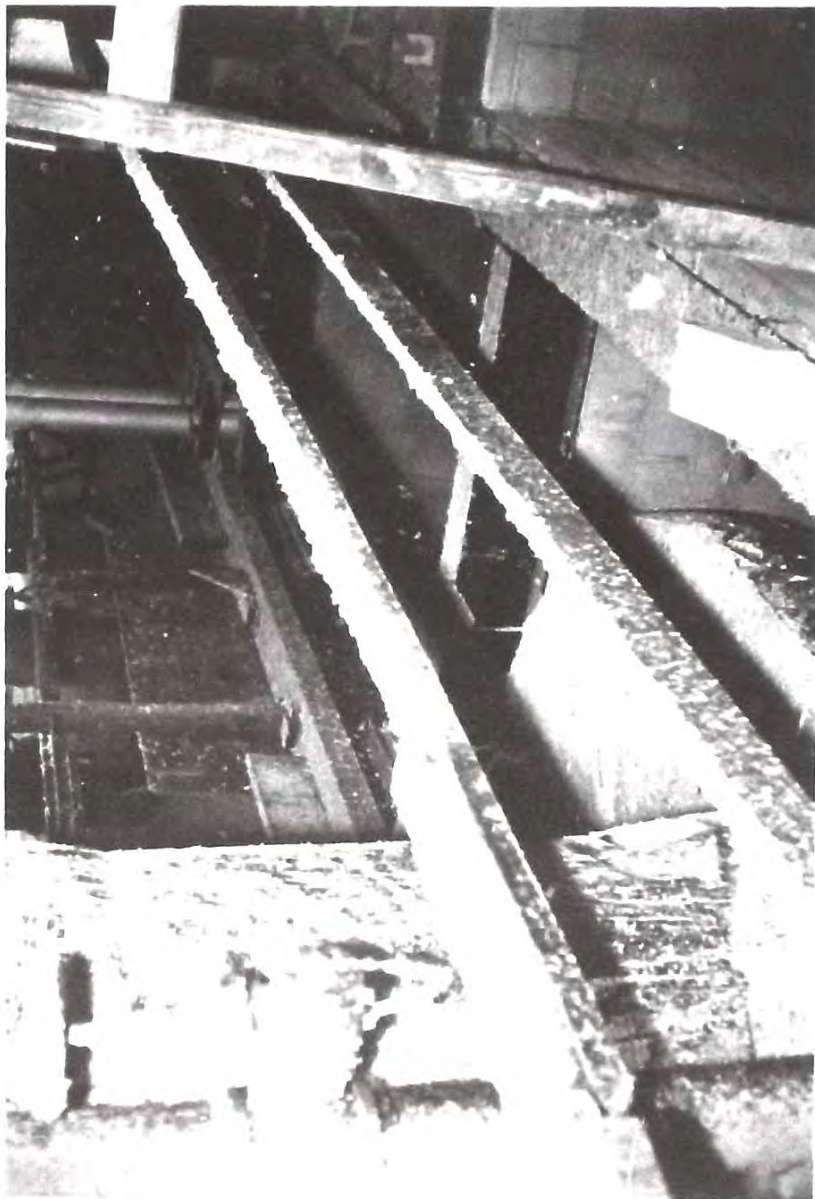


Figure 5. ACCUMULATION OF ASBESTOS INSULATION
ON ARTICLES SURROUNDING THE TARGET AREA

2. Floor areas should be swept broom clean before spraying operations begin. All unnecessary objects in the spray area should be removed or covered with plastic or plastic coated tarpaulins.
3. The entire area to be sprayed should be enclosed with plastic or plastic coated tarpaulins. An enclosure will be considered satisfactory only if visible insulating material cannot escape from the closure.
4. Wet asbestos-containing material that falls to the floor should be swept up immediately and placed in approved disposable containers.
5. When spraying is completed in an area, the entire work area and the materials used to form the enclosure should be thoroughly vacuumed. The vacuum cleaner should contain a strong, single-service, disposal inner bag which shall be removed from the vacuum cleaner and sealed. Disposal is the same as for other bagged waste materials.
6. Any plenum or other structure coated with asbestos-containing insulation and intended for circulation of air in the building must be thoroughly cleaned of debris and waste. Asbestos-containing insulation in a duct or plenum must be coated with a sealer to preclude erosion of the insulation by circulating air.

The recommendations state also that workers are to be informed of the toxicity of asbestos and be provided with coveralls and Bureau-of-Mines approved respirators. Further, the recommendations state that adequate ventilation of the work area shall be provided to hold the concentration of asbestos below the maximum threshold level as recommended by the American Conference of Governmental Industrial Hygienists. This level

has been established as 12 fibers per cubic centimeter.

It can be seen from the above that the spraying of asbestos containing fireproofing requires very elaborate procedures. These procedures are difficult to police since they are scattered very much throughout the state. Further the onus placed upon the average contractor is very often beyond his desires, skills or inclinations.

Alternatives to Asbestos Fireproofing Materials - Intumescent paints are being increasingly used as a fireproofing material. A recent application of these paints was in the construction of the McCormick Place Exposition Center in Chicago.

These paints contain polymeric substances which expand upon being heated to form a heat-shielding and fire-retarding substance for the surface upon which they have been painted.

Other substitute materials for asbestos are mineral wools but the toxic hazard of these and other materials has not been fully evaluated.

3.3.2 Field Fabrication of Asbestos Products

Examples of asbestos product application and the form applicable to the usage are given in the following paragraphs.

Preformed asbestos sections or blocks containing materials which, by nature, are very dusty are used for insulating pipes and/or boilers. These applications may be used for either hot or cold temperature extremes. Examples include:

- Molded asbestos
- Molded calcium silicate/asbestos
- Molded magnesia (85%)
- Molded high temperature insulating block

Another group is made up of materials which are applied in a wet, or slurry state. Frequently these materials are used to seal joints between the blocks or sections discussed in the previous group. When used this way, their composition is similar

to the material being sealed and they are mixed with water just prior to use.

- Calcium silicate asbestos cement
- 85% magnesia
- Hard-setting asbestos cement
- Asbestos skinning plaster

A third group of asbestos materials consist of the asbestos cement products. These products have sealed surfaces which will not dust easily. Considerable energy is required to cut, drill, or break these products. Examples include:

- Roofing shingles
- Building boards
- Drain pipes

Finally a group is listed in which asbestos constitutes a large percentage of the material. Generally the asbestos fibers are loosely bound and the material does not possess a predetermined shape. Examples include:

- Asbestos paper
- Asbestos blanket or clothing
- Asbestos rope, tape, yarn, and sealing compounds

Source of Emissions - Emissions which are created during field fabrication of asbestos containing materials will vary as to: type of material being fabricated; the amount of fabrication required (cutting, sawing, sanding, etc); the location and nature of the fabrication site; and the quantity of material to be fabricated.

The form that the asbestos is in will have an obvious effect on emission as free powdery materials or materials in which the fibers are loosely bound will be more prone to emission than those in tightly bonded composites.

Emissions during fabrication will vary according to the amount of cutting, drilling, or sanding required. Each step will

induce more emission. Cutting may be done utilizing either hand implements or mechanical devices. There is no distinction between the emission when using either system; however, it is easier to fashion emission control hoods to mechanical devices. Allied to the degree of fabrication is the quantity of material to be handled. Large quantities represent greater potential emission sources and thus require more adequate control.

Lastly, the nature of the fabrication site will have a strong bearing on the amount of atmospheric emission. The work area in a closed or partially enclosed structure can be adequately sealed and ventilated with relative ease while installation of asbestos products in open, exposed areas may make emission control impossible.

A side aspect to the installation or fabrication of asbestos products is the transportation of these products to the site and the handling at the site. Emissions can occur unless sufficient care is taken to protect the material against damage.

Control Techniques - The basic control techniques employed by the construction industry consist of isolation of the work area and the arrest of emission at the source.

Isolation of the working area can be accomplished by use of properly installed tarpaulins or plastic sheets. However, even a completely enclosed work area has some shortcoming: the work area requires some ventilation which may propagate fibers unless the air is filtered, dust from cutting, drilling, or sanding operations may collect on materials or clothing of workers which then become secondary emission sources; respirators may be required; and strict housekeeping routines are required which practice has shown to be difficult to enforce.

For these reasons the arrest-at-the-source approach should be employed whenever possible and used in conjunction with work area isolation and strict housekeeping routines. Power tools such as saws, sanders, and drills can be equipped with vacuum

attachments such that the dust created is arrested at the source and collected in filter bags. Commercial devices are available but normally firms with a sincere interest in pollution control, design and fabricate their own devices. Two examples of such devices are shown in Figures 6 and 7. These control hoods work on the high velocity, low volume principle and can be readily adapted to industrial vacuum cleaner systems.

When arrest-at-the-source systems are not possible, techniques similar to those employed by the asbestos spraying industry may be utilized. Rules which relate more specifically to fabrication of asbestos products may be found in such publications as:

1. "Recommended Practices for Fabricating, Handling and Construction Industries," Health and Safety Council-Asbestos Cement Products Association.
2. "Recommended Health Safety Practices for Handling and Applying Thermal Insulation Products Containing Asbestos," National Insulation Manufacturers Association (NIMA).
3. The Asbestos Research Council - Control and Safety Guides (London, England).
4. "Recommendations for Handling Asbestos," Engineering Equipment Users Association (EEUA), E.E.U.A. Handbook No. 33.

3.3.3 Friction Material Applications

Typical friction materials contain 30 to 50% asbestos and may be up to 70%, and the industry that manufactures these materials is ranked third (1969) in the consumption of asbestos fibers. Crysolite is the preferred variety because it has better frictional properties than amphibol asbestos and does not exert so much wear on the opposing surface.

The major uses of asbestos-containing friction materials include brake linings, brake discs and clutch facings. These products have been applied to a wide variety of industrial and commercial products.

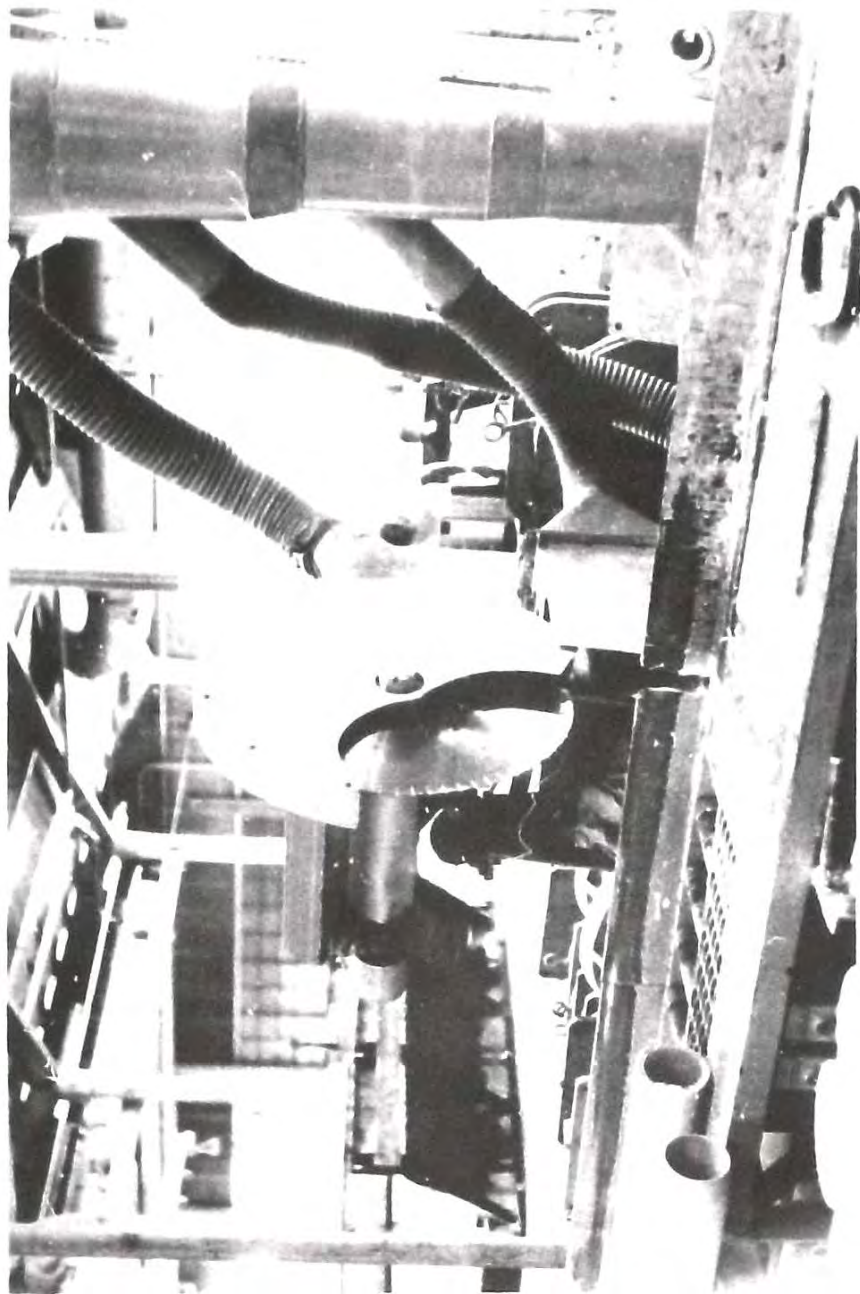


Figure 6. DUST COLLECTION HOODS ADAPTED TO A RADIAL SAW BENCH

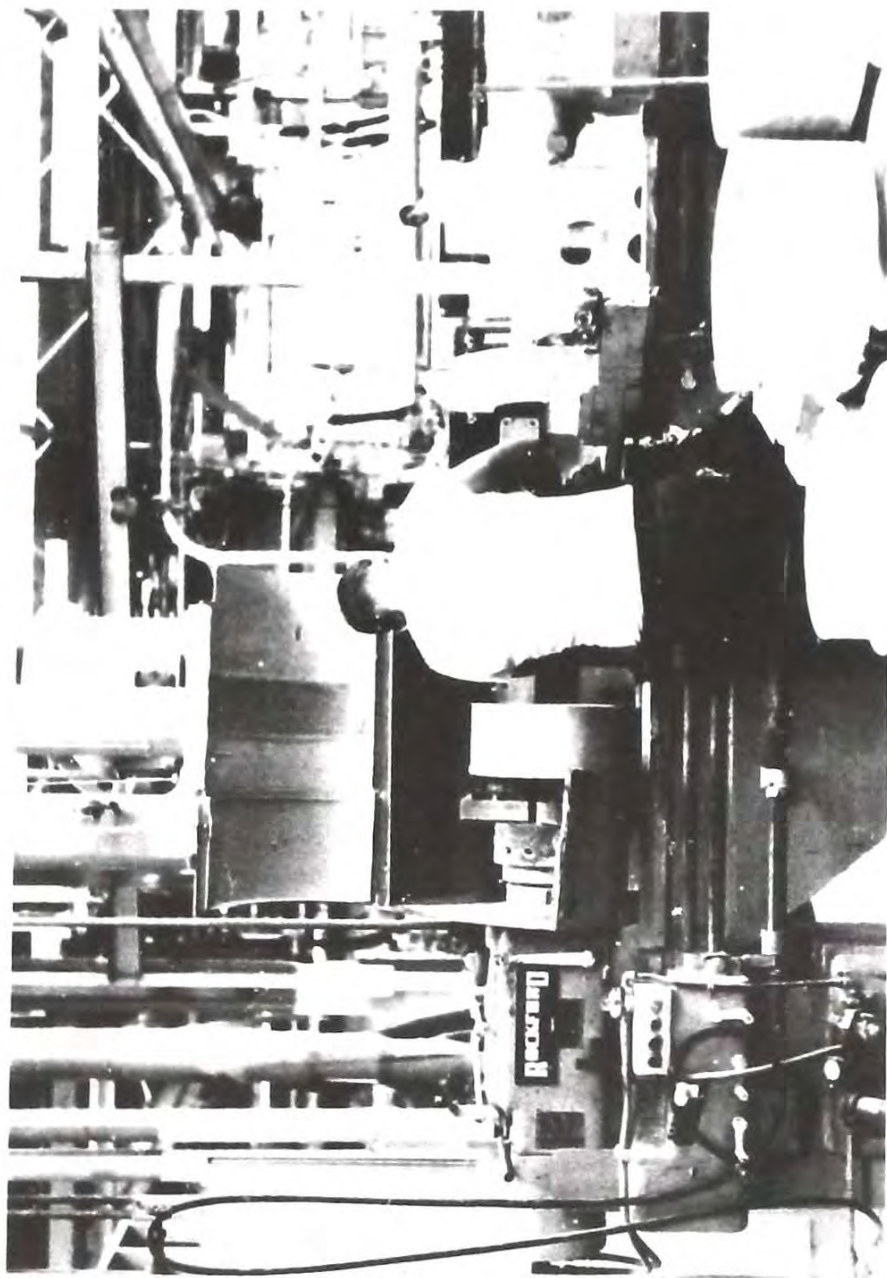


Figure 7. DUST COLLECTION HOODS ADAPTED TO A LATHE OPERATION

Sources of Emission - Emission of asbestos from friction materials results from normal day-to-day usage. An idea of the scope of this source can be gained from the estimation that the average automobile wears out 3 to 4 sets of brake linings, and 1 to 2 sets of clutch facings during its lifetime and that commercial public transport vehicles wear out many more sets. Data concerning motor vehicle brake linings for the whole of the United States are given in the following:

Vehicle miles during 1968	1,010,000,000,000
Mileage life of brake linings	27,500
Brake lining sets on new vehicles	10,718,000
Pounds of asbestos per set of brake linings	3

Tests performed on brake linings have indicated that under conditions of normal usage, considerable alteration of the asbestos occurs. It is reported that most of the dust collected from brake drums appears non-fibrous and is similar in appearance to thermally degraded asbestos. The suggestion is that high temperatures at the brake lining/drum contact points actually reach degradation levels.

Tests on brake linings, brake discs and clutches have demonstrated that the quantity of fiber emitted is some function of the severity of the braking conditions (see Table 3). However, even at the level of 1%, the total emissions must be considerable when the total tonnage in use is considered. Further, the emissions are likely to occur at places of high density population and restricted ventilation. That is, in busy main streets of towns surrounded by large buildings.

The effect of different braking conditions and different types of friction materials on asbestos emissions is the subject of a study being sponsored by the Environmental Protection Agency. Other studies are being conducted in California and some work on roadsides has been done at Mt. Sinai, New York. The EPA study will establish the extent and nature of the asbestos

Table 3

ASBESTOS EMISSIONS FROM BRAKE LININGS
(Test Results by Electron Micrograph)

	Product	Brand	Test Method	No. of Samples	Conditions of Test-F	Presence of Free Fibers	% Free Fiber*
1.	Automobile drum brakes	A	Friction	6	300-800	Few	<1
2.	"	B	Friction	6	250-800	None	0
3.	"	C	Friction	5	300-700	Few	<1
4.	"	C	Friction	1	700-900	Numerous	~10
5.	"	D French	Friction	5	300-800	Few	<1
6.	"	E	Friction	5	300-700	Few	<1
7.	"	F German	Friction	5	300-800	Few	<1
8.	"	G	Friction	2	100-500	Few	<1
9.	"	G	Friction	1	600-700	Numerous	~15
10.	"	H	Friction	2	100-600	Few	<1
11.	Automobile clutch	J	Dynamometer	1	normal driving	None	0
12.	Automobile disk brake	K	Dynamometer	1	normal driving	Few	<5
13.	Bus drum brake	L	Dynamometer	1	city driving	None	0
14.		M	Friction	2	450-550	None	0
15.	Truck drum brake (light)	F	Friction	10	300-800	Few	<1

*Weight estimated from fiber volume.

emitted from brake drums, disc brakes and clutches of vehicles operating under real conditions. Hopefully the situation will be more clearly understood at the end of those studies.

Emission Control Techniques

Emission at Overhaul - To avoid blowing the accumulated dust into the atmosphere at the time of overhaul devices have been suggested for extraction of the dust from brake and/or clutch housings by suction. Of the devices tried, a simple hand-held vacuum cleaner has proven to be the most flexible unit. Changes to the original design have been limited to the addition of a disposable paper bag inserted in the original cloth bag. The paper bag may be easily sealed before removal to prevent emission.

Substitution - Beyond the adaptation of better cleaning practices during brake and clutch maintenance, as mentioned above, additional controls seem to be limited to improve design of brake and clutch assemblies and/or substitution of other materials for asbestos.

The high temperature properties and exceptional tensile strength of asbestos have resulted in very compact and economical design. These same unique properties which make asbestos applicable to friction materials also make the application of substitute materials very difficult. The tensile strength and modulus of rigidity of asbestos as compared to several candidate substitute materials follows:

<u>Tensile Strength lb/in.²</u>		<u>Modulus of Rigidity lb/in.²</u>
Asbestos	550,000	30,000,000
Fiber yarns	180,000	3,000,000
Steel wool	50,000	100,000
Mineral wool	25,000	60,000

There is no doubt that substitution of other materials for asbestos is possible, but the design changes required to accommodate the stresses and temperatures involved would result in

larger, more expensive components.

Brakes - The recent adoption of disc brakes by the automotive industry makes material substitution more plausible. Disc brakes are capable of greater energy dissipation than similar sized drum brakes because the design results in considerably lower heating rates. This is due to the fact that at any one time the friction pad contacts only a section of the disc surface. The lower operating temperatures permit the use of friction materials which do not contain asbestos. Another feature of disc brake design which has reduced the need of asbestos materials is the lower strength requirement of the friction material.

Clutches - The substitution of other material for asbestos in clutch facings of traditional design could only be done by changes in size and design. Two changes in clutch design which have reduced the requirement for asbestos-containing friction materials are the advent of the automatic transmission and the redesign of manual clutch friction surface.

The clutch surface of the automatic transmission is immersed in an oil bath which virtually eliminates airborne pollutants. Further, the cooling affect of the oil bath reduces the requirement for high-temperature capability materials. Automatic transmission clutch facings may be made from either sintered metals or fibrous cellulose materials.

Many manual clutches have redesigned friction surfaces which consist of numerous small circular pads or discs attached to the clutch face as opposed to the standard annular ring friction surface. These small pads act in the same manner as the disc brakes friction pad. Thus, lower stresses and operating temperatures associated with the new design reduce the need for asbestos-containing friction materials.

4. CONTROL TECHNIQUES IN MANUFACTURING PROCESSES

Asbestos emissions are likely in any process in which asbestos fiber is handled or asbestos-containing materials are cut, drilled, or trimmed. The quantity of asbestos dust generated by these operations can be quite large. Since it is impractical to completely seal a factory or a work area, industry has adopted an arrest-at-the-source technique for capturing this dust utilizing a complex ventilation system comprised of capture hoods, ductwork, fans, and filters. Benefits received from this system include:

1. Reduced atmospheric emission when plant is open to ambient conditions (i.e., doors and windows open).
2. Reduced external emission from fibers being carried outside on work clothing and manufactured products.
3. Greater efficiency in plant operation because of reduced internal housekeeping chores (vacuuming, etc.).
4. Better personnel attitudes. The ventilation system may eliminate the need for personal respirator units which limit efficiency and are generally disliked by workers.

Air Ventilation System - The air ventilation system is an integral part of the emission control system, the following several paragraphs are devoted to a discussion of the system components. Examples have been drawn from various asbestos industries.

Hoods - Two types of ventilation systems are employed for dust/emission control; low volume, high velocity; and high volume, low velocity. Each requires a specific type of hood design.

The low volume, high velocity systems induce an extremely high captive air velocity, 10,000 to 12,000 ft/min., at the dust source. The air volume, however, averages only 10-250 cfm.

Report No. IITRI-C8216
(Supplement A)
"Asbestos in Brake Linings"

ASBESTOS AIR POLLUTION CONTROL

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13 October 1971

ASBESTOS AIR POLLUTION CONTROL

1. ASBESTOS IN BRAKE LININGS

In this report on the use of asbestos in brake linings, consideration has been given to two aspects: the extent of the emissions, and, the elimination of emissions. No attempt has been made to estimate the medical significance of the results. Further, neither a detailed cost analysis nor an engineering feasibility study has been attempted when considering systems to eliminate emissions.

1.1 The Extent of the Emissions

The best information available at the present time^{*1,2} indicates that the percentage of asbestos contained in the dust normally emitted from brake linings is of the order of 1%. However, under conditions of severe breaking, this may rise to 15%.

The average brake friction material contains of the order of 50% asbestos, thus an exploration is necessary to explain the discrepancy between the measured and theoretical values. The most likely explanation results from known facts with regard to heat generated at friction surfaces. The work of Bowden and Tabor^{**} has shown that when two surfaces are placed together they only contact at asperity tips and thus the true surface area of contact is dramatically less than the apparent area. This means that very large energy transfer takes place at these contact points and extremely high temperatures are reached.

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- *1. Lynch, J. R., et al., Jnl of Air Polln Control Assn. 18, 12, 824 (1968).
2. Unpublished Study Referred to by Spiel & Leinweber, Env. Res. 2, 166 (1969).

** Bowden, F. P. and Tabor, D., "The Friction and Lubrication of Solids," Pub. Oxford, London, 1950.

In the case of brake linings, some evidence of these temperatures has been generated in unpublished work by the General Motors Company. In this study, a small hole was cut through a brake shoe and the radiation collected from the exposed shoe surface was monitored. The results suggested that under light braking temperatures of the order of 800-900°C were found, while during heavy braking the temperature reached 1700-1800°C.

The significance of this data to the present case is that it is known that asbestos decomposes above 500°C* to give forsterite (Mg_2SiO_4), talc ($Mg_3Si_4O_{10}(OH)_2$) and water vapor.

Acceptance of the value of 1% of asbestos emitted allows an estimate to be made of the effect of this emission on the quality of the atmosphere.

1.2 Estimation of Urban Concentrations of Asbestos from Brakes

Assumptions

- a. There are approximately 10,000,000 motor vehicles.
- b. Motor vehicles have an average approximate weight of 4 lbs of friction material, when new, of which 50% is asbestos.
- c. The average motor vehicle will require replacement of the brakes after three years of use at which time they will have lost 50% of their weight by abrasion.
- d. The dust emitted will contain 1% of asbestos fibers.
- e. Of the wear dust, 80% is emitted and 20% is lodged within the system.

Calculation

The total weight of brake linings in use is

$$\frac{10,000,000 \times 4}{2000} = 20,000 \text{ Tons.}$$

* Bowen and Tuttle, Geol. Soc. America, Bull. 60, 439 (1949).

The quantity worn off these linings per year follows from the assumptions made in c.

$$\begin{aligned} \text{as } 20,000 \times \frac{1}{3} \times \frac{50}{100} \\ = \frac{20,000}{6} \text{ Tons.} \end{aligned}$$

This wear dust contains 1% asbestos, hence emission is

$$\frac{20,000}{6} \times \frac{1}{100} = \frac{200}{6} \text{ Tons.}$$

Allowing for the fact that 20% is not emitted, the total emission is thus

$$\begin{aligned} \frac{200}{6} \times \frac{80}{100} &= \frac{160}{6} \\ &= 27 \text{ Tons/yr.} \end{aligned}$$

Effect on the Atmosphere

There are no facts relating directly to asbestos on which one may judge the effect of these emissions on urban air. However, studies have been made on the diffusion of carbon monoxide and of lead exhausting from automobiles. The figures used here were developed by GM for lead emissions and relate to average city street conditions at street level.

They suggest the following factor:

$$\begin{array}{lcl} \text{Avg. conc. asbestos} & \text{Quantity of} & \\ \text{in city air from} & \text{asbestos} & \times \frac{2 \mu\text{g Pb/M}^3 \text{ city air}}{1.25 \times 10^5 \text{ tons Pb}} \\ \text{automobiles} & \text{emitted} & \text{from cars} \end{array}$$

Applying this figure to the present case and assuming that 20 tons are emitted in city areas, then we get that:

$$\text{The avg. conc. asbestos} \approx 20 \times \frac{2}{1.25 \times 10^5}$$

$$\approx 3.2 \times 10^{-4} \mu\text{g/m}^3$$

$$\text{or } \approx \frac{0.32 \times \text{ng/m}^3}{1}$$

$$\begin{array}{lcl} \text{GM also suggest} & & \\ \text{a high of} & \approx & 0.32 \times 10 = \underline{3.2 \text{ ng/m}^3} \end{array}$$

1.3 The Significance of This Value

The question of whether asbestos containing brake linings constitute a significant source of ambient air background, asbestos levels can now be considered.

Firstly, it is pertinent to review the available evidence on asbestos levels in ambient air. Perhaps the most up-to-date information is reported in a recent paper by Thompson and Morgan of the EPA.* Their data suggests that values 0.5-15 ng/m³ are appropriate to urban sites, while 0.1 ng/m³ is to be found in nonurban sites. They give a figure of 0.01 ng/m³ for remote sites.

The California State Health Department in collaboration with the School of Public Health, Berkeley, California, have tested ambient air for asbestos. Their results suggest that values of 0.06 ng/m³ in remote sites and 3 ng/m³ near a source.

Nicholson and Rohl** of the Mt. Sinai School of Medicine have also been active in the measurement of asbestos in ambient air. New York City air was found to contain 10-60 ng/m³ with most values in the 20-30 range. This value rose significantly to the 45-180 ng/m³ level in the downwind vicinity of asbestos fire-proofing activity.

* Thompson, R. J. and Morgan, G. B., "The Determination of Asbestos in Ambient Air," Int. Sym. on Identification & Measurement of Environmental Pollutants, Ottawa, Canada, June 1971.

**Nicholson, W. J. and Rohl, A., "Asbestos Air Pollution in New York City," presented at Second International Clean Air Congress, Washington, D.C., December 1970.

All of the workers have been at pains to point out that the results are to be considered preliminary. Thus they should be regarded not as final definite figures, but rather as good, considered, preliminary findings. The similarity of the figures reported by the various workers does suggest that at least an order of magnitude has been established.

Consider now the estimate of 0.3 ng/m^3 calculated previously as the contribution from asbestos brake linings. Firstly, it should be stated that this figure is a conservative estimate and allowance has been made for the many factors which could reduce this figure. Secondly, although there is no evidence to doubt the diffusion figure supplied by GM for the diffusion of lead, it should be realized that this figure is an average value for the whole of the city air. Consequently, much higher concentrations are to be expected in certain areas of high brake usage. For example, near intersections, toll-booths, etc.

1.4 Summary

In the light of the evidence here presented, one must conclude that asbestos emitted from brake linings may be a major contributing factor in the overall ambient air concentration.

Further work is necessary to establish more definitely the major contributors to urban asbestos concentration levels.

2. TECHNOLOGY OF ELIMINATING EMISSIONS

The emission of asbestos to the atmosphere from asbestos containing brakes could be prevented in two ways: it could be banned altogether, or, the brakes could be sealed. In the former case, an alternate would have to be found.

2.1 Alternatives to Asbestos

For a replacement to be as suitable as asbestos, it would have to fulfill a list of desirable features as:

- Resist high temperatures without degradation or loss of frictional properties.
- Combine excellent friction characteristics without severe abrasion.
- Provide great strength - asbestos fibers have a greater tensile strength than steel and yet are more flexible.
- Have relative economy.

Of the materials which have been considered, sintered metal with ceramics and fiberglass have been the most likely candidates thus far.

Sintered Metal - This material has been used and indeed has the desirable quality of being fade-free, for this reason it is still used in some racing cars. However, it has several disadvantages. They tend to be noisy and brake screech is quite a problem. They suffer from what is known in the trade as "morning sickness", this means that the first stop is very severe, or, that the cold friction is high. Wear is higher than with asbestos and thus the brakes have to be replaced more frequently. Replacing the brakes is more difficult since all brakes are honed to fit the drum. Honing metal is more difficult than asbestos friction material.

Ceramic Metal (Cermets) - A sintered metal and ceramic composition has been developed and tried. Its use at this time seems to be restricted to the clutches on heavy-duty vehicles (tractors, etc.); in these cases it is usual to use buttons of the material rather than a complete annulus. Although to a lesser degree, it suffers from the same complaints as sintered metal with respect to noise, wear and severe first stop, and in addition, it is relatively expensive. However,

its anti-fade characteristics have led to extensive testing. For example, the Ford Motor Company put inserts of ceramic metal at the position of maximum brake wear on their 1958 Thunderbird. The concept was abandoned, reportedly due to uneven braking leading to a directional pull when braking.

Fiberglass - Attempts to use fiberglass instead of asbestos have been attempted since 1938. So far, no design has met the exacting standards required. As with the other materials, the first stop is harsh. Apparently the design is better for disc brakes than drums. A problem is the sensitivity of the brake to moisture.

Owens-Corning are undertaking development research in this area. They are very guarded with regard to the status of their work, but state that they are actively engaged in developing a product. To date, no vehicles are known to employ fiberglass in their brake friction product.

2.2 Sealed Systems

Sealed systems have been experimented with. Normally, cars rely on air cooling and the movement of air round the brake caused by the motion of the car. However with high performance cars, more cooling is required. This is achieved in a simple manner by fabricating a superstructure which scoops up air and directs it inside the brake drum. If a filter were added on the exiting side, this would cause a substantial reduction in emissions.

A more elaborate design which has been used is to liquid cool the brake system. This enables the braking system to be completely sealed and thus no emission are possible. The one problem area with this design is that the oil seals must be extremely reliable.

2.3 Summary

At the present time, no alternative to asbestos containing friction products is developed to the stage of finesse required by the industry. It is difficult to believe that modern technology cannot overcome the problems presented by the use of alternates. No information is available on the cost or the time factors involved.

The use of sealed brake units is a second possibility. Again there do not seem to be any unsurmountable technological problems, but no information is available as to the cost and time factors.

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1	C. Harwood

With this system it is essential that the feed particles be carried by the air stream. The entrainment force must exceed the gravitational or projectional force.

Low velocity, high volume air ventilation systems, on the other hand, are utilized for operations where localized capture is not possible. For such hoods to be effective, the air velocity to the ducted area must be at least 150 ft/min and should be such that the air flow direction is always toward the collection system.

Ducting - A considerable amount of ductwork is required for a large plant having a central collection point. Circular ducts, having no sharp bends, are recommended. Inspection ports should be provided near bends.

Collectors - Several types of collectors have been utilized by the asbestos industry for dust control. Baghouses represent the most frequently used filtering system. Wet scrubbers are also used. Cyclone precipitators may be used as primary collectors preceding baghouse filters.

Conspicuously absent are electrostatic precipitators. These units have been tried by the asbestos industry; however, they have several deficiencies. Asbestos is an insulator and builds upon the electrodes reducing efficiency. Installation costs are higher than for baghouses. An electrical failure would remove all emission control.

The most commonly used filter system is the fabric filter. This device is found in a wide variety of forms from highly sophisticated systems containing thousands of bags to crude devices containing homemade burlap bags. A typical unit is shown in Figure 8. The baghouse is compartmentalized to allow continuous operation by alternately cutting off sections for cleaning while other sections continue their dust removal function. The bags are "cleaned" by a mechanical shaking action, either by a shaking floor or, more commonly, by a shaking

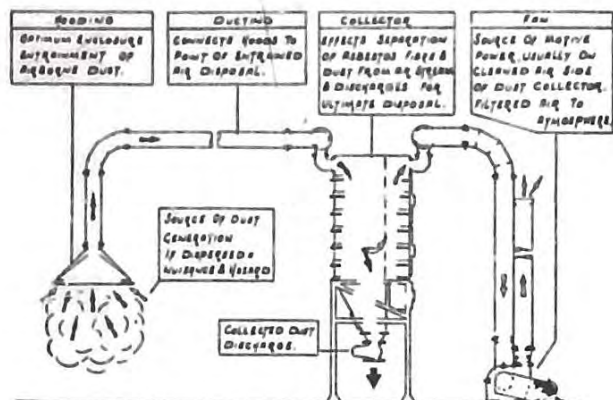


Figure 8

SCHEMATIC REPRESENTATION OF BAGHOUSE SYSTEM

support. More elaborate cleaning systems such as reverse cycle air jets or sonic systems have not been considered worth the extra complication.

The bulky baghouse unit is frequently located outside the factory building, to conserve space. Usually the filtered air is exhausted to the atmosphere. In some cases it is returned to the factory to conserve heat. This air contains some asbestos fibers since no control technique is considered to be 100% efficient. Waste products collected in the baghouse hopper are removed by helical screw conveyor. Design of the hopper emptying mechanism should provide as foolproof an operation as possible. Severe pollution can arise if the mechanism has to be opened for repair.

Other collection devices used either in combination with fabric filters or alone are various types of cyclone separators.

Low efficiency cyclones are used in the asbestos industry as first stage separators preceding fabric filters. They are useful when the waste product is reusable and there is a considerable quantity present. Thus, the load on the fabric filter is relieved. Further the removal of heavy abrasive waste material will prolong the life of the filter bag.

High efficiency cyclones are generally employed in multiple units. They are used where high efficiency is required and where no use is made of backup fabric filters.

Wet cyclones are employed as emission control devices in some special instances where wet processed are used for production of asbestos products. Although generally not as efficient as bag filters, they eliminate the problem of filter blockage caused by condensation.

Fans - The fans which provide the air movement through the dust collection system may be mounted either upstream or downstream of the filter system, the most common arrangement being downstream. This places the ventilation system under a negative

pressure assuring that ambient air will be drawn into the system in case of leaks, rather than dust and fibers being exhausted to the atmosphere.

5. STORAGE AND TRANSPORTATION

All asbestos product manufacturers are faced with the problems of storage and transportation of bagged asbestos fibers. Emission controls for these operations depends to a great extent upon the conscientiousness of the individual user.

Manufacturers should provide clearly defined, clean, dry storage space for the bags where they are protected from accidental damage. A rotation system should be employed to ensure that older bags are used first. Damaged bags should be placed in larger slipover bags and resealed. Small tears in bags should be patched with tape.

Jute bags are permeable and will allow fiber emission. Kraft paper bags are liable to tearing when handled roughly. The best compromise at this time seems to be bags of coated, woven polyolefin. Following is a tabulation of commonly used bagging materials and their relative cost.

<u>Type of Bag</u>	<u>Cost per 1000 Bags, \$</u>	<u>% Used</u>
7 oz Jute Laminated	336.00	9
10 oz Jute	290.00	5
7 oz Jute	210.00	30
5-Ply Kraft Paper	200.00	6
4-Ply Kraft Paper	170.00	20
3-Ply Kraft Paper	130.00	16
Woven Polyolefin (Coated)	245.00	8

Other

Note: Bags are 100 lbs size 6

Bag storage and the bag opening areas should be as close as practical. Fork-lift trucks are usually employed to transport the bags between these areas. The route used by the trucks should be smooth surfaced and free of obstructions which might cause damage to the bags.

The bags should be opened in a hooded area using a sharp knife to slit the bag. Pressure packed fibers will hold together as a unit and can be slipped into an enclosed conveying system where they are transported to a fiber opener (willow or fluffer). All conveyor systems transporting asbestos fibers or mixtures of asbestos and other materials must be completely enclosed.

Bag Disposal - The emptied bags also present an emission source; thus, to prevent emission, steps must be taken to dispose of them. Since there are few manufacturing processes which will accept paper bags as part of the process, asbestos paper being an exception, they are usually bundled into large polyethylene bags and taken to dumps for disposal. Plastic shipping bags can be similarly utilized in some manufacturing processes; the floor tile industry, for example.

6. WORKERS CLOTHING

A possible source of emission from manufacturing processes is the transport of fibers outside by the workers themselves. The control of this source of emission to date is not very prevalent. At best, booths are provided where workers may clean fibers from their clothing. The booths are equipped with extraction fans in the ceiling and a flexible pressure hose. It would be preferable to have flexible suction hoses.

Figure 9 shows a proposed workers change room. Workers would store their work clothes in lockers, pass through a shower room, and then into a "clean" room where their street clothes would be stored in a second series of lockers.

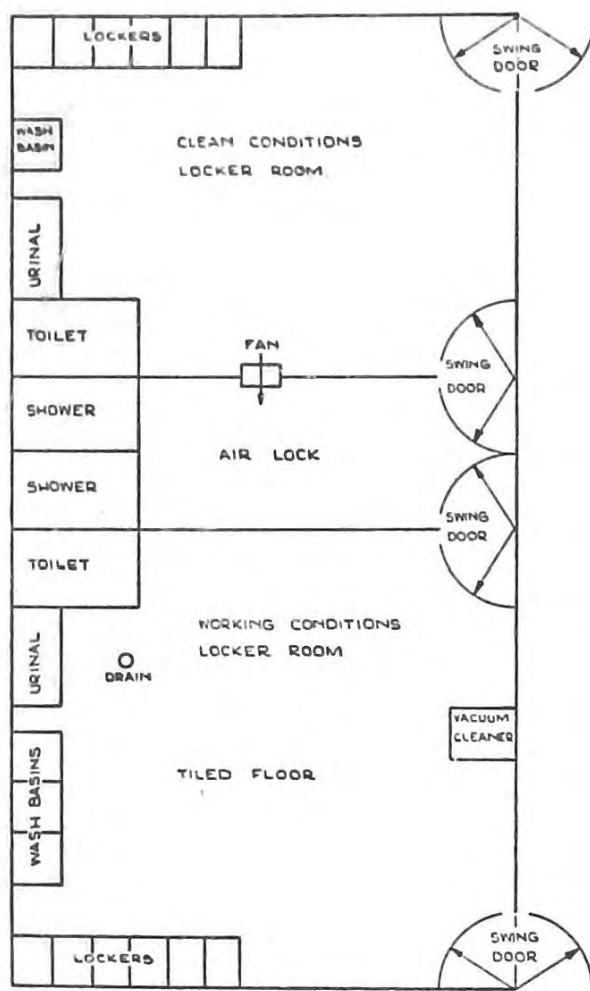


Figure 9
PROPOSED WORKERS CHANGE ROOM

7. ALTERNATIVES TO ASBESTOS

Thermal Insulation - Glass wool, rock wool, and slag wool are possible substitute materials all of which are available as flexible mattresses and rigid or semi-rigid slabs and pipe sections. Other materials available as rigid slabs and pipe sections include cellular glass (maximum temperature 427°C) and polyurethane foam (maximum temperature ranges from 116°C to 149°C).

Loose-fill materials containing no asbestos are exfoliated vermiculite, expanded perlite, and the mineral wools mentioned in the preceding paragraph.

Tables 4 and 5 list materials commonly containing asbestos, together with suggested alternatives and comments on each. It is emphasized that the alternatives are not equivalent in all respects to the asbestos materials they may replace and the service conditions in each case should be considered before a substitution is made.

Packing Materials - Tape, rope, and sheet of various densities, made from refractory fibers are available. They are expensive but have higher limiting temperatures than asbestos.

Heat-Resistant Protective Clothing - It has not been possible to find a non-asbestos substitute for protective clothing normally made from asbestos cloth. The alternative approach therefore has been to treat the asbestos cloth to minimize the risk to the wearer. The most suitable treatment appears to be aluminized asbestos cloth which when made up into suits, and properly lined, creates no dust hazard.

Table 4

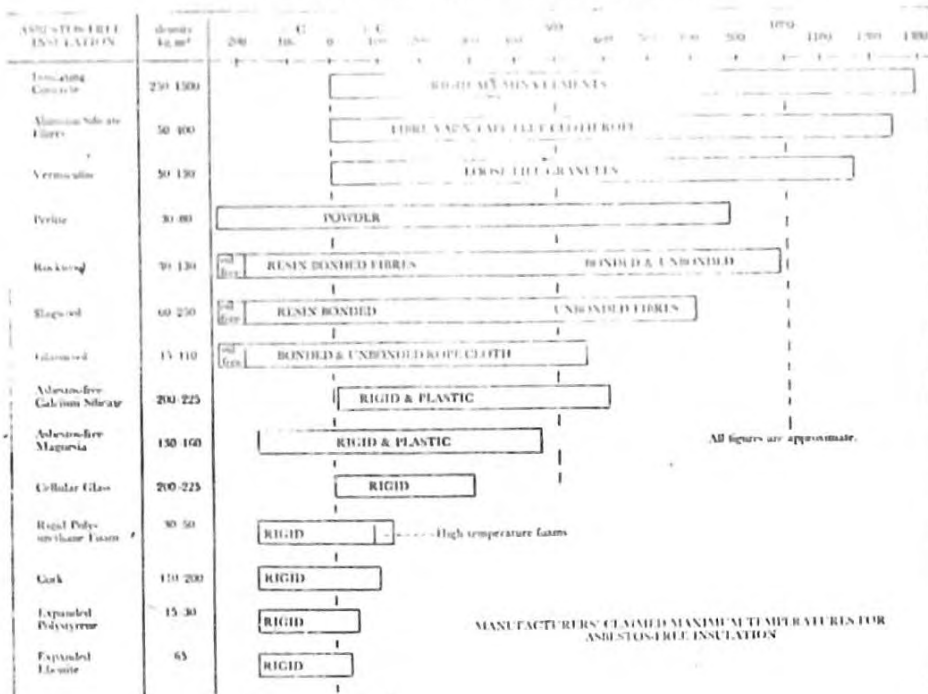
ASBESTOS AND ASBESTOS SUBSTITUTE MATERIALS

Asbestos-containing materials	Asbestos-free alternatives	Comments
Moulded asbestos (Amosite asbestos bonded with sodium silicate)	None	
85 per cent magnesia about 15 per cent asbestos	Asbestos-free magnesia	Coiling temperature 316°C. One manufacturer only. Coloured pink to distinguish from asbestos-containing materials.
Calcium silicate (about 15 per cent asbestos)	Asbestos-free calcium silicate	Coiling temperature varies. Generally 480°C.
High-temperature insulating block (about 15 per cent asbestos with diatomite)	Vermiculite-alumina cement block	Some calcium silicates may be suitable alternatives.
Plastics compositions	Asbestos-free versions of magnesia and calcium silicate	Generally satisfactory for gap-filling and small areas.
Sprayed asbestos and asbestos mineral wool	1. Sprayed mineral wool 2. Sprayed ceramic fibres 3. Sprayed vermiculite-cement and sprayed concrete	1. At development stage only. 2. Limited experience in this country. Expensive. 3. For fireproofing.
Self-setting and hard-setting finishing cement (up to 40 per cent asbestos)	1. Asbestos-free finishing cements 2. Plastics finishes applied in paste form and reinforced with glass cloth 3. Sheet finishes (metal, plastics, roofing felt, etc.)	1. May be subject to more cracking than asbestos-containing finishes. 2. Coloured if required. Less robust and generally more expensive than cement. 3. Cheap for straight pipe lengths but expensive where bends and shapes involved.
Board and sheet	Ceramic fibre board and sheet	Higher coiling temperature (1,260°C) than asbestos material. Expensive.
Rope	1. Glass fibre rope 2. Ceramic fibre rope 3. Silica fibre rope 4. Ceramic fibre felt strip	1. Coiling temperature 500°C. 2. Coiling temperature 1,260°C. Expensive. 3. Coiling temperature 982°C. Very expensive. 4. May be cheap alternative to ceramic rope in some cases.
Cloth, tape, string, yarn, etc.	1. Glass fibre fabrics and yarn 2. Ceramic fibre and yarn 3. Silica fibre and yarn	1. Coiling temperature up to 500°C depending on type of glass. Some cloths may be unsuitable for handling. 2. Coiling temperature 1,260°C. Expensive. 3. Coiling temperature 982°C. Very expensive.

NOTE: Manufacturers have incorporated our own fibres in some of the materials listed previously containing asbestos. Such materials may no longer be suitable for certain duties, e.g. where contamination with potential oxidizing agents is possible.

Table 5

TEMPERATURE RANGES FOR ASBESTOS AND ASBESTOS-FREE INSULATING MATERIALS



8. DISPOSAL OF ASBESTOS WASTE PRODUCTS

8.1 General

Industries that use asbestos fibers or asbestos products have many waste disposal problems. Waste materials may include materials collected in baghouse-type filters where dry-mixing is required. Also the asbestos products may require cutting, drilling, or sanding operations which create dust. Some parts will be damaged during fabrication and these items may constitute emission sources. Spraying asbestos-cement compounds produces large quantities of waste material. And, finally, the emptied shipping bags constitute an emission source.

Fiber emission may occur when filter bag collection systems are emptied into trucks and subsequently unloaded at waste dump sites. Damaged or scrap materials are also discarded on dump sites. Weathering of these waste materials may cause emission. Emission may occur directly from some manufacturing operations which are difficult to ventilate. Asbestos shipping bags may become emission sources due to fibers remaining in the bags following bag opening and emptying operations. Emptying of dust collection devices (baghouses, etc.) can cause atmospheric emission if done exposed to the wind. Shields should be erected around the dust hoppers.

Frequently, waste material from dust collection devices is carted away in trucks, dumped, and buried. Tarpaulins are used to control dust emission during transportation to the dump.

Large waste products (damaged or rejected production products) are sometimes fed through crusher units and broken into smaller pieces. This waste material may then be buried.

Shipping bags present a particular problem since some fibers will remain in them after the asbestos has been emptied. Burning the bags would release the fibers to the atmosphere.

The usual practice is to put the emptied bags into large polyethylene bags and bury them in dumps.

8.2 Water Pollution

Water becomes polluted with asbestos fibers during several manufacturing processes, in particular are the paper and cement product industries. A further stage is the water used in wet cyclones in cleaning exhaust gases from the factories.

The slurry is dealt with in two ways. It is either directed into settling ponds and the water recirculated, or it is dumped directly into convenient sewers, streams, rivers or lakes.

When dumped into settling ponds, it presents a problem in its own right since the settled slurry has to be dried out before being shoveled and transported away for proper disposal. Although it should be stated that a dried out slurry normally contains approximately 30% water and is not too dusty.

If allowed to drain away into streams and lakes, then a considerable amount should eventually settle, but at least the problem of air pollution is solved. It does create a potential water pollution problem though. It is not known whether stomach cancer may be induced in fish by floating asbestos fibers.

With regard to the harmful effects on man, this would depend on the filtration potential of water treating plants. A recent survey has indicated the presence of considerable numbers of fibers in drinking water and beverages even after filtration (see Table 6). Until the full potential of the effects of asbestos in causing stomach cancer are known, this area should be treated with caution.

Table 6

ASBESTOS FIBERS IN BEVERAGES AND WATER

Sample	Source*	No. of Fibers
		$\times 10^6 \text{ l.}^{-1}$
Beer	Canadian 1	4.3
Beer	Canadian 2	6.6
Beer	USA 1	2.0
Beer	USA 2	1.1
Sherry	Canadian	4.1
Sherry	Spanish	2.0
Sherry	South African	2.6
Port	Canadian	2.1
Vermouth	French	1.8
Vermouth	Italian	11.7
Soft drink	Ginger ale	12.2
Soft drink	Tonic water I	1.7
Soft drink	Tonic water II	1.7
Soft drink	Orange	2.5
Tap water	Ottawa, Ottawa river (F)	2.0
Tap water	Toronto, Lake Ontario (F)	4.4
Tap water	Montreal, St. Lawrence river (F)	2.4
Tap water	Hull, Quebec, Ottawa river (NF)	9.5
Tap water	Beauport, Quebec, St. Lawrence river (6 km below Quebec City) (NF)	8.1
Tap water	Drummondville, Eastern Townships, Quebec, St. Francois river (F)	2.9
Tap water	Asbestos, Eastern Townships, Quebec, Nicolet river (F)	5.9
Tap water	Thetford Mines, Eastern Townships, Quebec, Lac a la Truite (NF)	172.7
Melted snow	Ottawa, top 30 cm (2-3 weeks precipitation)	33.5
River water	Ottawa river, at Ottawa	9.5

* Filtration plant used.

NF - No filtration plant used.

Cunningham and Pontefract, Asbestos Fibers in Beverages and Drinking Water, Nature, Vol. 232, July 30, 1971.

9. MONITORING CONSIDERATIONS

9.1 Sample Collection

The aim in sampling is to obtain a representative sample of the total air mass in sufficient quantity to make quantitative assessments statistically significant.

There are two main sources of error when taking particulate samples from an air mass. They are: (1) errors induced at the point of entry into the sampling device, due to changes in air velocity, and due to misalignment of the probe with respect to the air flow, and (2) errors due to loss of sample in the tube conveying the sample to the sampling device.

Sampling should be carried out isokinetically, that is, the velocity in the sampling tube should ideally be the same as the gas velocity. Isokinetic sampling is important for particles larger than 5μ in diameter. Errors due to misalignment of the sampling probe can lead to significant large particle losses. This will bias the collected sample in favor of the smaller particles. Not only will the effective cross-sectional area, seen by the incoming particles, be reduced, but the larger particles which manage to enter the inlet will impact against the far side of the probe. To minimize this error, sampling probes should be aligned within ten degrees of the wind direction.

When sampling airborne particles, eddy currents and wind gusts can lead to significant sampling errors. When designing a sampling probe, attempts should be made to compensate for change in wind direction or velocity.

The second source of error is "line loss," that is, the deposition of particles in the sampling line leading to the collector. The following precautions are recommended to reduce losses in the sampling line.

- (a) As short a sampling line as possible should be used.
- (b) The line should not have sharp bends.
- (c) The tube should be at least 1 in. in diameter.
- (d) Low velocities should be used.
- (e) The tube should have a smooth surface.
- (f) The tube should be electrically grounded to prevent the buildup of static charge.

The accepted apparatus and practical methods of stack sampling have received adequate coverage in previous publications.* For the particular case of sampling from baghouse filters, it is important that there is a sufficient stack length. This is not always the case since in many instances a very short stack is used.

9.2 Membrane Filter Technique

Of the several techniques available for emission sampling, the membrane filter recommended by the U.S. Industrial Hygienists, has become the standard technique for environmental asbestos emission sampling.

Air is drawn through a cellulose membrane filter where the particles are entrained. Although the effective pore size of the filter (0.45μ to 0.8μ) is larger than the diameter of many of the asbestos fibers, the surface charge properties of the filter and the asbestos fibers, plus the circuitous path through the filter, result in collection of virtually all of the particles and fibers to which the filter is exposed.

*
 1. APCO, Source Sampling
 2. ASME, Power Test Code 27, WP-50, 1957.
 3. Air Pollution Manual, Part I, American Industrial Hygiene Association, 1960.
 4. Western Precipitator Corp. Bull. WP-50, Los Angeles, 1968.

9.3 Analysis of Collected Fibers

The collected fibers have to be analyzed. This may be accomplished by using a microscopic or a photographic or electronic technique to count the fibers resulting from a known volume of air. The fibers which are counted are seen at a given magnification. The number will vary. Table 7 shows the number of particles observed within a range of magnifications.

Table 7

EFFECT OF MAGNIFICATION ON OBSERVED FIBER CONCENTRATION

<u>Instrument</u>	<u>Microscope Objective</u>	<u>Fibers* ≥ 5</u>	
		<u>mfpcf</u>	<u>fpcc</u>
Midget Impinger	16 mm	0.11	4
	16 mm	0.06	2
	4 mm	0.37	13
Membrane Filter	2 mm	0.48	17
	16 mm	0.31	11
	4 mm	0.54	19
Thermal Precipitator	2 mm	0.57	20
	Particles Counted 5 μ	0.12	4
Royco Counter			

* Fiber defined as a particle whose length $\geq 3 \times$ width
mfpcf = million fibers per cubic foot
fpcc = fibers per cubic centimeter

Addingly, C.G., "Dust Measurement and Monitoring in the Asbestos Industry," Ann. N.Y. Acad. Sci., 132, 298-305, Dec. 31, 1965.

The standard practice in industrial hygiene is to use a phase contrast microscope at a magnification of X430. The fibers counted are those having a length of greater than 5μ with the ratio of length to breadth at least 3:1.

The use of an electron microscope would allow all the particles to be counted. However, the expense, time and expertise necessary to count all the particles makes this procedure impractical.

Optical Methods of Sample Analysis - Light scattering automatic particle counters are very rapid and have been widely used to determine concentration and size distribution of airborne particles. Their inherent drawback is the inability to distinguish between particles of different shapes and chemical composition. Therefore, the use of automatic particle counters to determine atmospheric concentration of asbestos fibers is limited.

Addingly has carried out comparative tests between a Royco light scattering automatic particle counter and the membrane filter method to determine number-concentration of asbestos fibers. Addingly placed a membrane filter head in the exit line from the Royco counting chamber and counted the dust particles in the same sample of air, by both methods.

The results from the Royco counter, set to read particles greater than 5μ , were 25% lower than the count on the membrane filter of fibers $> 5\mu$ in length. The disparity being due to the varying alignment of the fibers presented to the light beam.

Lowering the threshold of the Royco counter to $> 4\mu$ and again counting fibers $> 5\mu$ on the membrane filter, resulted in a 15% higher count with the Royco counter.

These results show that it is possible to obtain correlation between light scattering particle counts and membrane filter counts.

10. ANSWERS TO SPECIFIC QUESTIONS

10.1 Asbestos Brake Linings

Information on the emissions from brake linings is still insufficient. At the present time there is only one study which has been published. IITRI has been given copies of two unpublished reports detailing experiments made by Johns Manville. The reason the results are unsatisfactory is that they have been done on laboratory test rigs and not on roadways under normal driving conditions. Tests currently being made by the Bendix Company for the EPA and further tests being undertaken in California may well help to solve this question. Tests were performed at Mount Sinai Hospital, New York, by the Selikoff group. These tests measured the asbestos in roadside dusts near heavy braking areas, such as toll booths. Conversations with Nicholson of Mt. Sinai make it apparent that although his results were generally higher than ambient, they were still within the limits of experimental error. The fact that they are not being published tends to make one doubt their scientific credibility.

Other conversations with the Motor Industry, namely, John Kaplan [Exec Director of Laboratories at GM Detroit (313)-575-5286] and John Damian [Head of the Engineering and Materials at Ford (313)322-0450], have been inconclusive. They claim not to have made tests of their own. They cannot see any possible substitute for asbestos that will meet the continually exacting requirements of government regulations. They say they do not try to develop their own products but rely on outside researchers. They gave up using sintered metals for brakes because they were inferior and would sometimes break up under stress.

Ralph Nader and Associates were contacted but they have not entered into this sphere of automobile safety.

10.2 Emission Levels

In understanding control techniques and their efficiencies, it is important not to be confused with efficiencies quoted on a weight basis and those based on a particle count basis.

Firstly, considering efficiency based on a weight basis, it is relatively easy to get a very high efficiency with particles whose size is in excess of 5μ with a variety of control devices (see Figure 10). However, the efficiency does drop off considerably with decrease of particle size, for example, see Figure 11 for 1μ particles. (It should be noted that these graphs refer to spherical particles - information on fibrous particles is not presently available.)

Now consider what these apparent high efficiencies mean in terms of numbers of fibers. It can be assumed that 10^3 average fibers of asbestos weigh approximately 1 ng.

If 1 g of asbestos material approaches a filter rated at even as high as 99.999% efficiency, then the quantity passing through will be 0.00001 g, or 10^4 ng. And since 1 ng $\approx 10^3$ fibers, then a total of 10^7 fibers will pass through the filter for every 1 g of material impinging upon it.

This is a situation not frequently brought out, but is very significant when exposure levels are monitored in terms of fibers per cubic centimeter.

Thus the quoting of efficiency in terms of mass efficiency is a "red-herring" statement that flatters to deceive. It bears no obvious relation to the number of fibers being emitted.

However, Based on tests which actually measure the number of fibers being emitted, it would seem that both fabric filters and high efficiency wet scrubbers are capable of reducing the fiber counts to acceptable levels. British experience is that 0.2 f/cc is routine and Johns Manville finds that 1 f/cc is an acceptable value when the results are averaged over a time period.

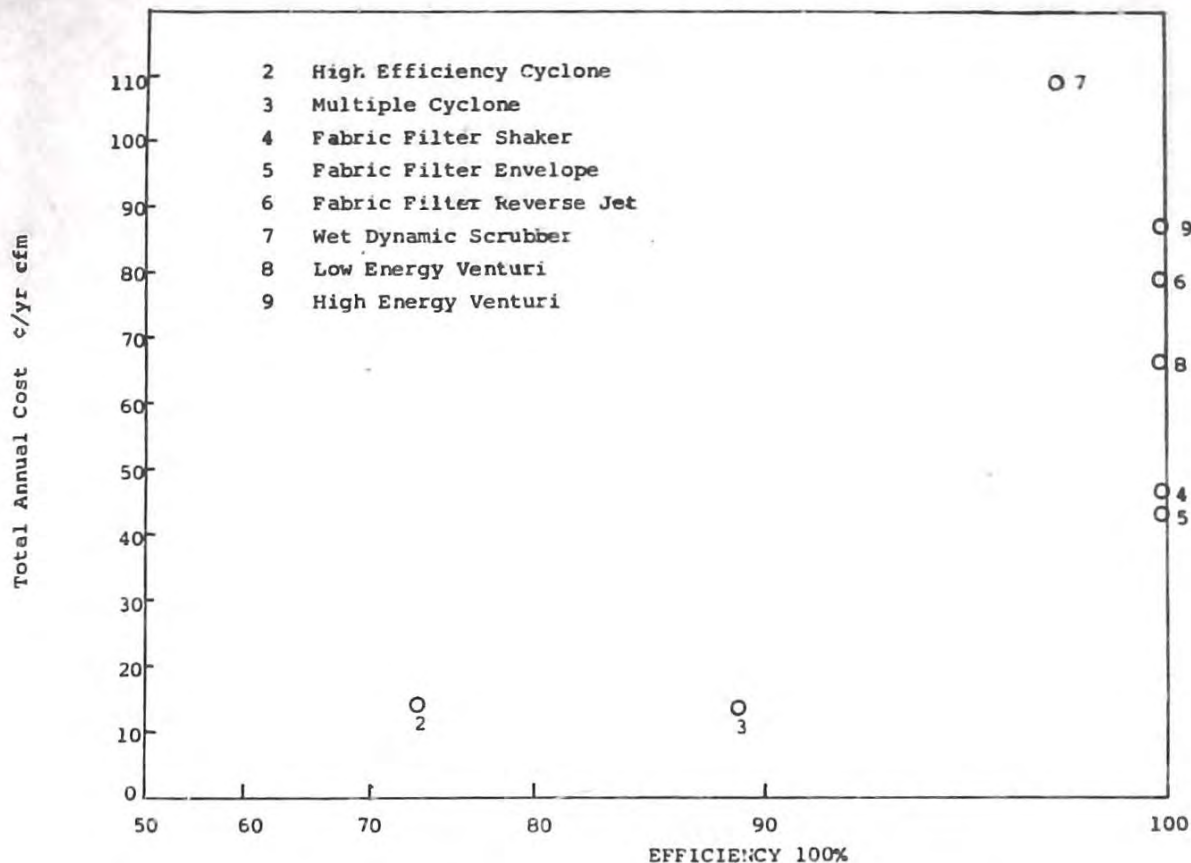
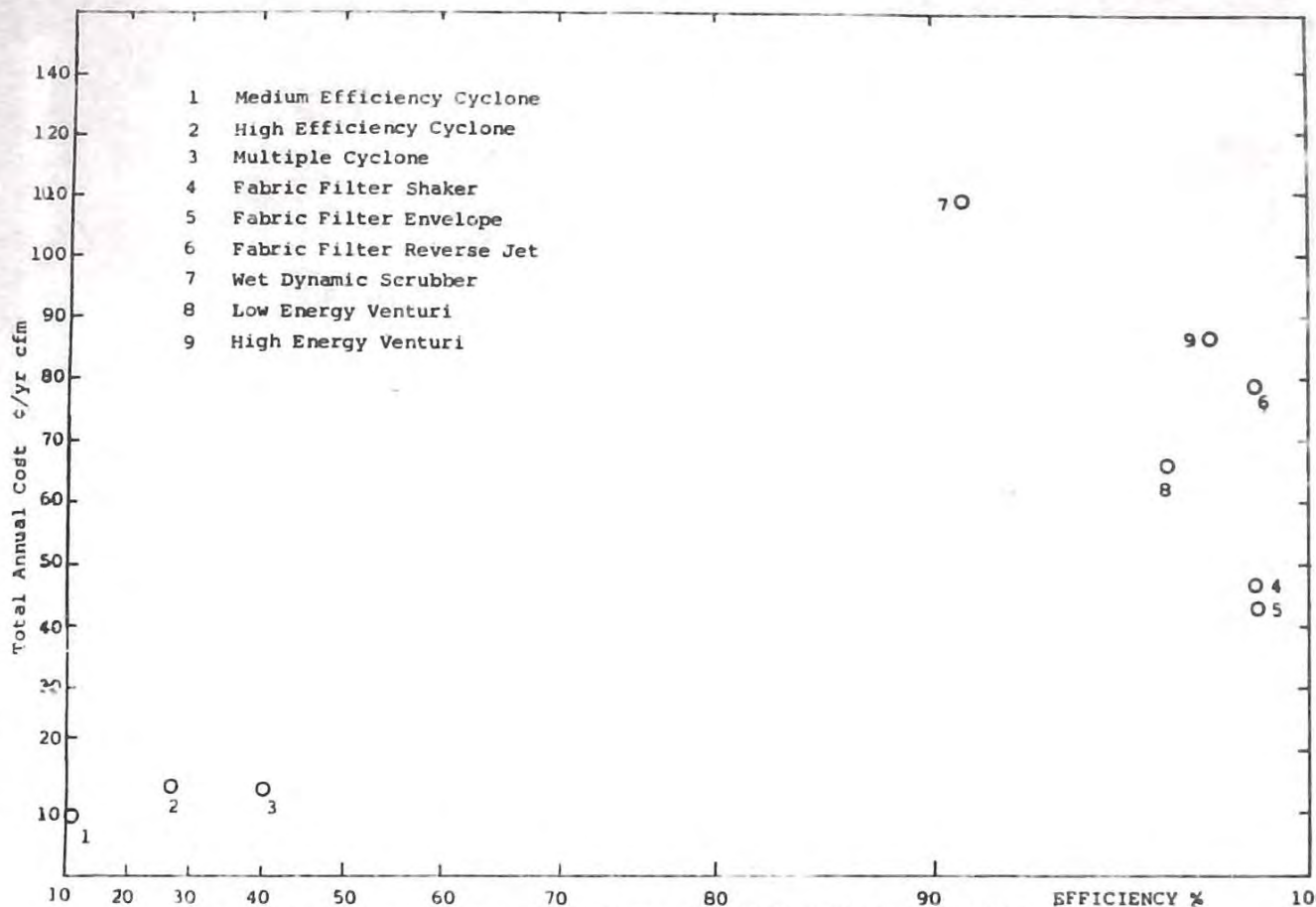


Figure 10. TOTAL ANNUAL COST VERSUS EFFICIENCY FOR 5μ PARTICLES

Figure 11. TOTAL ANNUAL COST VERSUS EFFICIENCY FOR 1μ PARTICLES

The medical evidence as to the size of fiber responsible for adverse health effects is not positive nor is the question of whether fibrils of fibers are most hazardous. Until the medical questions are fully resolved, it would seem premature to impose inflexible or overly rigid regulations.

This has been the view held by the Federal occupational health authorities in assessing their standards. It would seem that their approach in limiting exposure to that which is possible using good modern technology is sensible. The same may be said for their monitoring techniques.

10.3 Water Pollution

Recycling of waste water is possible and is practiced in certain segments of the industry. The question of the damage done to streams, rivers, and lakes by indiscriminant dumping of asbestos containing waste waters needs careful study. One must remember it is a natural material which will appear in water in any case. On the other hand, there is evidence that asbestos particles ingested into the stomach may cause stomach cancer.

Again, until medical evidence is clear, it would seem sensible to recycle water whenever this can be accomplished.

APPENDIX I

ESTIMATES OF THE NATIONWIDE EMISSION QUANTITIES
FROM VARIOUS INDUSTRIES

		PERCENTAGE OF TOTAL CONSUMPTION	NUMBER OF PLANTS	EMISSIONS IF UNCONTROLLED		EMISSIONS WITH PRESENT DEGREE OF CONTROL		EMISSIONS WITH BEST DEGREE OF CONTROL	
	TONS/YR (1969)			EMISSION FACTOR lbs/TON	EMISSION TONS/YR	PRESENT %	EMISSION TONS/YR	BEST %	EMISSION TONS/YR.
MINING (DOMESTIC)	127,000			10	635	50	317	80	121
MILLING (DOMESTIC)	127,000		9	100	6350	80	1210	99	63.5
IMPORTED FIBER	695,000								
TOTAL FIBER	822,000								
<u>PROCESSING</u>									
ASBESTOS CEMENT	412,500	50	361	4	825	75	206	99	8.3
FLOOR TILE	201,200	24.6		4	402	75	101	99	4.0
FRICTION PRODUCTS	104,600	12.8		120	6276	95	314	99	63
TEXTILE	18,100	2.1		40	362	95	18	99	3.6
PAPER	30,200	3.8		4	60	75	15	99	0.6
MISCELLANEOUS	6,800	0.8		10	34	75	9	99	0.3
<u>CONSUMPTIVE USES</u>									
CONSTRUCTION INDUSTRY	5,125	0.6	SEVERAL THOUSANDS	25	64	N/C	64	90	6.4
BRAKE LININGS	38,400	4.7		20	384	N/C	384	90	38.4
STEEL FIREPROOFING	3,050	0.4		40	61	75	15	90	6.1
INSULATING CEMENT	2,025	0.25		40	41	75	10	90	4
TOTAL EMISSIONS TONS/YR.					15,500		2,663		319

APPENDIX II

THE CHEMICAL AND PHYSICAL PROPERTIES OF ASBESTOS

Chemical, Mineralogical, and Physical Properties of the Different Varieties of Asbestos Fibers

Variety	Chrysotile	Crocidolite	Amosite	Anthophyllite	Tremolite	Actinolite
Chemical formula	$3\text{MgO} \cdot 2\text{SiO}_2 \cdot 2\text{H}_2\text{O}$	$\text{Na}_2\text{O} \cdot \text{Fe}_2\text{O}_3 \cdot 3\text{FeO} \cdot 8\text{SiO}_2 \cdot 2\text{H}_2\text{O}$	$1.5\text{MgO} \cdot 0.5\text{FeO} \cdot 8\text{SiO}_2 \cdot 2\text{H}_2\text{O}$	$7\text{MgO} \cdot 8\text{SiO}_2 \cdot 2\text{H}_2\text{O}$	$2\text{CaO} \cdot 5\text{MgO} \cdot 8\text{SiO}_2 \cdot 2\text{H}_2\text{O}$	$2\text{CaO} \cdot 4\text{MgO} \cdot \text{FeO} \cdot 8\text{SiO}_2 \cdot 2\text{H}_2\text{O}$
Essential composition	Hydrous silicate of magnesia	Silicate of Na and Fe with some water	Silicate of Fe and Mg; higher iron than anthophyllite	Mg silicate with iron	Ca and Mg silicate with some water	Ca Mg Fe silicate; water up to 5 %
Percentage chemical composition						
SiO_2 , %	37 - 44	49 - 53	49 - 53	56 - 58	51 - 62	
FeO , %	39 - 44	0 - 3	1 - 7	28 - 34	0 - 30	
Fe_2O_3 , %	0.0 - 6.0	13 - 20	34 - 44	3 - 12	1.5 - 5.0	
Al_2O_3 , %	0.1 - 5.0	17 - 20	---	---	---	
H_2O , %	0.2 - 1.5	---	2 - 9	0.5 - 1.5	1.0 - 4.0	
CaO , %	12.0 - 15.0	2.5 - 4.5	2 - 5	1.0 - 6.0	0 - 5.0	
Na_2O , %	Tr. - 5.0	---	---	---	0 - 18	
MgO , %	---	4.0 - 8.5	---	---	0 - 9	
$\text{CaO} + \text{Na}_2\text{O}$, %	---	---	0.5 - 2.5	---	---	
pH	9.2-9.8	---	---	Neutral	---	---
Resistance to acids and alkalis	Poor	Good	Good	Very good	Good	Fair
Ionizable salts, micromhos (relative electrical conductance)	1.82	0.84	1.34	0.58	---	---
Magnetite content	0 - 5.2	3.0-5.9	0	0	0	---
Mineral impurities present	Iron, chrome, nickel, lime	Iron	Iron	Iron	Lime	Lime, iron
Crystal structure	Fibrous and asbestiform	Fibrous	Prismatic, lamellar to fibrous	Prismatic, lamellar to fibrous	Long and thin columnar to fibrous	Long and thin columnar to fibrous
Crystal system	Monoclinic and orthorhombic	Monoclinic	Monoclinic	Orthorhombic	Monoclinic	Monoclinic
Mineralogical structure	In veins of serpentine, etc.	Fibrous in iron stones	Lamellar, coarse to fine fibrous and asbestiform	Lamellar, fibrous asbestiform	Long, prismatic and fibrous aggregates	Reticulated long prismatic crystals and fibers
Mineral association	In altered peridotite adjacent to serpentine and limestone near contact with basic igneous rocks	Iron rich silicious argillite in quartzose	In crystal-line schists, etc.	In crystal-line schists and gneisses	In Mg lime-stones as alteration product of magnesian rocks, metamorphic and igneous rocks	In limestone and crystalline schists

Chemical, Mineralogical, and Physical Properties of the Different Varieties of Asbestos Fibers (cont'd)

Variety	Chrysotile	Crocidolite	Amosite	Anthophyllite	Tremolite	Actinolite
Property						
Veining	Cross and slip fibers	Cross fiber	Cross fiber	Slip, mass fiber unoriented and interlacing	Slip or mass fiber	Slip or mass fiber
Color	Green, gray, amber to white	Blue	Gray, yellow to dark brown	Yellowish brown, grayish white	Gray-white, greenish, yellowish, bluish	Greenish
Texture	Fine, soft to harsh, also silky	Soft to harsh but flexible	Harsh and coarse but somewhat pliable	Harsh; soft to friable	Harsh or friable	Harsh, brittle
Luster	Silky	Silky to dull	Vitreous, somewhat pearly	Vitreous to pearly	Silky	Silky
Hardness	2.5-4.0	4	5.5-6.0	5.5-6.0	5.5	6+
Flexibility	High	Good	Good	Poor	Poor	Poor
Spinability	Very good	Fair	Fair	Poor	Poor	Poor
Tensile strength, lb/in. ²	80,000-100,000; 824,000 (max.)	100,000-300,000; 876,000 (max.)	16,000-90,000	4,000 and less	1,000-8,000	1,000 and less
Fusion point, °F	2770	2180	2550	2675	2400	2540
Specific heat, BTU/lb/°F	0.266	0.201	0.193	0.210	0.212	0.217
Electric charge	Positive	Negative	Negative	Negative	Negative	Negative
Filtration properties	Slow	Fast	Fast	Medium	Medium	Medium
Specific gravity	2.4 -2.6	3.2 -3.3	3.1 - 3.25	2.85-3.1	2.9 -3.2	3.0 -3.2
Cleavage	010 perfect	110 perfect	110 perfect	110 perfect	110 perfect	110 perfect
Optical properties	Biaxial positive, extinction parallel	Biaxial ± extinction inclined	Biaxial positive, extinction parallel	Biaxial positive, extinction parallel	Biaxial negative, extinction inclined	Biaxial negative, extinction inclined
Refractive index	1.50-1.55	1.7 pleochroic	1.64±	1.61±	1.61±	1.63± weakly pleochroic
Resistance to destruction by heat	Good. Brittle at high temperatures	Poor, fuses	Good. Brittle at high temperatures	Very good	Fair to good	---
Temperature at maximum ignition loss	1800	1200	1600-1800	1800	1800	---

APPENDIX III

INDUSTRIAL COMPANIES USING ASBESTOS
IN THE STATE OF ILLINOIS

Accurate Felt and
Gasket Mfg. Co.
1015 S. California Ave.
Chicago, Illinois
Tel: VA6-3532

Acme Asbestos
Covering and Flooring
141 W. Jackson Blvd.
Chicago, Illinois
Tel: WA2-8220

Asbestos and Magnesia
Materials Co.
2616 Clybourn Ave.
Chicago, Illinois 60614
Tel: 348-4554

Blackhawk Gasket Corp.
216-T Mill
Rockford, Illinois

Chambers Gasket and
Mfg. Co.
4701 W. Rice St.
Chicago, Illinois
Tel: 626-8800

Colonial Kolonite Co.
2232 W. Armitage Ave.
Chicago, Illinois
Tel: 227-3400

Daigger, A. and Co.
161 W. Kinzie St.
Chicago, Illinois
Tel: 644-9438

Excelsior Leather
Washer Mfg. Co., Inc.
726 Chestnut St.
Rockford, Illinois

Farnam, F. D. and Co.
8405 W. 45th St.
Lyons, Illinois
Tel: 277-3515

Fel-Pro, Inc. (A Div. of
Felt Prod.)
7480 N. McCormick Blvd.
Skokie, Illinois
Tel: OR4-7700

Filipaco Industries, Inc.
Filter Media Div.
3837 W. Lake St.
Chicago, Illinois
Tel: 722-2200

Gatke Corp.
228 N. LaSalle St.
Chicago, Illinois
Tel: ST2-0081

Geraghty Gasket & Mfg. Co.
2940 S. Laramie Ave.
Chicago, Illinois
Tel: OL6-3561

Harnisch L. E. & Co.
218 N. Elizabeth St.
Chicago, Illinois
Tel: CH3-3883

Herman, John Mfg. Corp.
1828 S. Canalport
Chicago, Illinois
Tel: TA9-4945

Industrial Hermetic
Materials, Inc.
421 E. Illinois
Chicago, Illinois
Tel: MO4-0220

Kopel Filter Paper Co.
2512-14 S. Damen Ave.
Chicago, Illinois
Tel: VI7-7174

Luse-Stevenson Co.
Melrose Park, Illinois
Tel: MU1-2010

North American Asbestos Corp.
200 S. Michigan Ave.
Chicago, Illinois
Tel: 922-7435

Sall Mountain Co.
176 W. Adams St.
Chicago, Illinois

Singer Safety Prod., Inc.
444 N. Lake Shore Drive
Chicago, Illinois
Tel: 222-1860

Steel Grip Safety Apparel Co.
1013 Garfield
Danville, Illinois

Tapecoat Co.
1521-27 Lyons
Evanston, Illinois 60204
Tel: 328-5220

UNARCO Industries, Inc.
332 S. Michigan Ave.
Chicago, Illinois 60604
Tel: 341-1234

United Gasket Corp.
5107 W. Ogden Ave.
Cicero, Illinois
Tel: BI2-2590

Western Slate Co.
875 Industrial Drive
Elmhurst, Illinois
Tel: 279-6660

Wilson Grant, Inc.
316 S. LaSalle St.
Chicago, Illinois
Tel: 922-8220

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— R71-16

EXHIBIT 21

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— R71-16

EXHIBIT 21

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later studies. In 1947, the Chief Inspector of Factories in Britain found lung cancer in 13 per cent of deaths in which asbestosis was present.

To bring us up to date, 70 years after the first knowledge of asbestos disease, and to complete the picture of occupational risk from exposure to asbestos, let me present some data obtained by Dr. Irving J. Selikoff, Director of the Environmental Sciences Laboratory, Mount Sinai School of Medicine. He has followed since January 1, 1943 the 632 members of the New York and New Jersey Locals of the asbestos workers union, the International Association of Heat and Frost Insulators and Asbestos Workers. These men apply the insulation to high temperature pipes and boilers in apartment, office building and individual plants. Some of the material they use contains a small amount of asbestos -- 7 to 15 per cent, usually. Often it contains none. In comparison to the factory exposures during early decades of this century, their exposures are relatively light.

Knowing their ages in 1943 and using standard tables, it was possible to calculate the expected mortality experience of these 632 workers. This was done by Dr. E. Cuyler Hammond, Chief Epidemiologist of the American Cancer Society, and the results, along with those actually seen, are given in Table I for the years 1943 through 1962.

From all causes, 203 deaths were expected of those who had worked 20 years. In fact, there were 255. More than 50 men who died should not have died. Looking at the table, we can see the causes of these "excess death": 45 died of cancer of the lung, trachea and pleura where only six or seven were expected; 12 died of asbestosis although none

should have died; three times as many died of cancer of the G. I. tract as were expected.

Results of continued observation of the group through June 30, 1971 are shown in Tables II and III. They represent a catastrophe. Over half of the deaths were from some form of cancer or asbestosis. Nearly 40% could be termed "occupational" deaths.

In these latter data, special reference should be made to the high death rate from mesothelioma. Twenty-seven cases of this, as yet, incurable disease were seen. A diffuse cancer of the lining of the chest or abdomen, it is so rare that it has yet to be separately classified in the International Classification of Causes of Death. Its occurrence in the general population may be so infrequent as to cause only one death in 10,000. Here, it was the cause of death in one of every 16 individuals. Moreover, in other studies, its appearance in increased amounts has always been associated with asbestos exposure.

Two other aspects of the research by Doctor Selikoff are important. The first is the evidence of the long lapsed period between onset of asbestos exposure and the incidence of disease. Lung cancer usually appears between 20 and 40 years after first exposure to asbestos, and mesothelioma most often after a 35-year lapsed period.

The second aspect is the strong synergism found between asbestos exposure and cigarette smoking in the incidence of lung cancer. By personal interview in 1962, the smoking habits of 370 of the 632 men were established. By following the mortality experience of these 370 men, a special risk of cigarette smoking was established. From January 1, 1963 through February 1, 1971, in 283 men with a history of cigarette smoking, 40 lung cancers were observed. This is nearly ten times the number ex-

pected in an equivalent cigarette smoking population. In 87 non-smokers, only one lung cancer was seen. Thus, it is not asbestos alone, or cigarette smoking alone, but the combination of the two that produced these grim data. In fact, it is calculated that asbestos workers who smoke run more than 90 times the risk of dying of lung cancer than men who neither smoke cigarettes nor work with asbestos.

These data I have presented to you were from occupational exposures to asbestos. At most, 200,000 men and women in the United States have such exposures. Hopefully, 70 years after the first awareness of asbestos disease, working conditions will be improved to reduce their risk of disease. But were this the only risk of asbestos exposure, it would not be germane to the regulations under consideration here. Unfortunately, in recent years the evidence has accumulated that the problem of asbestos disease has disseminated from the occupational area into the general environment.

Evidence is accumulating that mesothelioma is rapidly increasing among men who worked in the vicinity of asbestos application. Recall that mesothelioma is virtually nonexistent in people who have had no exposure to asbestos. In Holland, 22 cases were found during a 6-year period in workers of the de Shelde shipyard. These cases occurred, however, not in asbestos workers, but in members of other trades, welders, carpenters, or painters, all of whom only worked in an area where asbestos insulation was applied or removed. Similar data have appeared from Great Britain. Here, 80 cases of mesothelioma were found in a survey of the material in the pathology departments of Scottish hospitals and medical schools from the years 1950 through 1967. An investigation of the occupational background of these cases revealed a strong association

with shipbuilding or marine work. Here again, however, the cases were not often found in insulators but in other trades working near the application or removal of asbestos. A further disconcerting feature of the Scottish data is the rapid rise in the rate of occurrence of mesothelioma; 51 of the 80 cases were seen in the years 1964-67, approximately 30 years after the extensive shipbuilding undertaken during World War II.

The spectrum of disease associated with asbestos now extends even to environmental exposures. In 1960, Wagner, Sleggs and Marchand described 47 cases of mesothelioma found during a four-year period in the northern Cape province of South Africa, an area with extensive crocidolite asbestos mines. None were seen in the neighboring Transvaal Province or in 10,000 autopsy cases of workers who had died with known exposures to silica. Of the 47 cases, approximately half were found to have had either industrial or mining exposures to asbestos. Virtually all of the remaining cases were in individuals who simply lived or worked in the vicinity of the asbestos mines or mills.

Confirmation of the possibility of environmental asbestos-associated disease soon appeared. Newhouse and Thompson reviewed all mesothelioma cases at the London Hospital. They corroborated the close association with asbestos, 31 of the 76 cases having had documented occupational exposure. Of the remainder, 11 had lived, decades before, within one-half mile of an asbestos factory. A similar distribution of cases was found by Lieben and Pistawka in Pennsylvania. One insidious form of indirect asbestos exposure is that of families of asbestos workers. In the previously mentioned studies, 9 of the London mesotheliomas were in family members, as were 3 of the 46 Pennsylvania cases.

Unfortunately, these data on the incidence of mesothelioma from environmental or indirect occupational exposure to asbestos are severely limited. The exposures in question took place beginning 20, 30, 40 or more years ago. No knowledge exists of the associated asbestos dust exposure levels. Thus, we have no dose-response information on low-level asbestos exposure. We only have knowledge of a potential risk of cancer, at exposures much below occupational ones. Obviously, additional research and continued surveillance are highly desirable.

Let us now turn to specific items in your regulations. Firstly, the general requirements of Section 2 are excellent, particularly with regard to responsibility and education. We find a great problem to be the ignorance of a potential hazard among marginal users of asbestos. Such ignorance can easily result in lax procedures.

Secondly, the banning of asbestos in spray insulation is entirely appropriate. In New York City we have found this to be a major contributor to ambient air asbestos levels. For the past 10 years, during which spray fireproofing came to be extensively used, it was not uncommon to see extensive snowfalls of asbestos-containing material over widespread areas of New York and other metropolitan centers. In some cases, the fireproofing was even done with no attempt to enclose the spray area by tarpaulins or other means.

To verify that such fireproofing operations were a significant source of respirable asbestos fiber, air sampling was conducted in lower Manhattan about construction sites where extensive spraying of asbestos-containing fireproofing was taking place. This work was done by our laboratory in conjunction with the Department of Air Resources of New York City. The results demonstrated that spray fireproofing can indeed con-

tribute significantly to asbestos air pollution.

Up to $\frac{1}{4}$ mile away, levels exceeding 0.1 micrograms per cubic meter were observed, which exceeds by 100 times background levels found in urban areas distant from construction sites. While 0.1 micrograms may appear to be a small amount, it could represent more than 10 million minute fibers of asbestos. Moreover, the large clumps of asbestos-containing material covering the sidewalk serve as a continuing source to be dispersed by passersby.

As I mentioned earlier, lacking a dose-response relationship, we cannot state that environmental exposure levels from this practice are certain to produce disease. However, it is hard to imagine factory emissions, even 40 years ago, being more excessive. Since the extensive spraying of asbestos-containing fireproofing material has only existed in the construction industry for the last 10 years, disease caused by the exposure resulting from this practice is 10 or 20 years away. However, continuing the uncontrolled spraying practices of the past decade in the face of the mounting evidence of risk to all metropolitan residents would be playing asbestos roulette with the lives of millions of people.

This method of fire protection for high rise buildings presents an additional, continuing risk to the occupants of those buildings. Often, the space between the false room ceiling and the under side of the floor above, which has been sprayed with asbestos, serves as a return plenum for the air supply system. After filtration, inadequate to remove most asbestos fibers that may have eroded into the air stream, this return air is recirculated throughout the building. The magnitude of this problem has yet to be defined. Until recently in New York City, we have not been allowed to analyze the air in buildings that has been in direct contact

with asbestos-containing insulation.

Finally, this spray procedure leaves a legacy of potential risk to future generations. The lifetime of modern office buildings is short and the procedures for the safe future demolition of buildings that could contain more than 100 tons of asbestos as fireproofing have yet to be developed. Perhaps these procedures will be found and perhaps they will be followed but we have no such assurance at this time.

Unfortunately, safe procedures developed for the spray application of asbestos-containing materials have not been followed either voluntarily or under control regulations by contractors. Because of this failure and because alternative procedures or materials are available, action banning this use of asbestos is indeed appropriate.

Moreover, because of uncertain knowledge of the health effects of exposure to mineral fibers other than asbestos, it is entirely appropriate that strict control be exercised over other spraying of such substitute materials. While some forms of "mineral wool" and other mineral fibers have been in use since the 1930s there are still no definitive studies on exposed populations that would, at this time, provide assurance that respirable mineral fibers, other than asbestos, are biologically inert in humans. In the absence of such assurance, caution is warranted and I support, in its entirety, Section 3 of your regulations.

On the other items, in Section 4, you might wish to add a qualifying statement that when there is potential release of asbestos to the air, a special enclosure shall be used. Many asbestos products used in construction can be installed with no release of fiber. Asbestos-vinyl floor tile is one example. In fact, with proper procedures, even asbestos cement can be applied with minimal release of fiber.

I cannot emphasize too strongly the need for stringent controls during demolition. Those listed here may not be adequate and close surveillance should be maintained to verify their adequacy. For example, I am not sure that any asbestos should be transported via chutes to trucks. It is hard to imagine that the debris would be completely wetted.

In reference to Section 6 A, I would like to raise some of the problems associated with air monitoring which may be severe. In ambient air samples, it is difficult, and often impossible, to identify asbestos fibers by optical microscopy alone. There are many birefringent fibers with a similar appearance. Even the sampling of stacks containing only asbestos has its problems. Such sampling does not evaluate any emissions from windows, room exhausts or uncontrolled sources. Moreover, the use of optical microscopy can misestimate actual concentrations, even assuming accurate counting of all fibers longer than 5 microns. Often as many as 100 times more fibers shorter than 5 microns may be present. If those counted were a constant fraction of the total present, this would be no problem. However, such is not the case. In different uses of chrysotile we have found the ratio of the number of fibers longer than 5 microns to the total number to vary from 1/250 to 1/20. Since bag houses have a size dependent efficiency, the results obtained by optical analysis may be misleading and not comparable to other samples analyzed by similar means.

The only way to accurately assess environmental exposures is by electron microscopy. Here, however, an analysis may cost \$200 - \$300 and be very time-consuming. You may wish to consider procedural controls with the requirement of no visible emissions of asbestos from any building or site. If the facility inspected questions the judgement of an inspector they could pay for adequate air analysis by electron microscopy.

I enclose in Table IV data from our work analyzing samples of ambient air from nearby 50 U. S. cities. These data show that a background of some asbestos fiber is found in all cities, but occasional peaks appear. From this occasional occurrence and from the fiber size distribution on high samples, it appears that the contribution of some specific source is important in these high levels.

Finally, one comment on the proposed ban of asbestos in brake linings after 1975. The problem here is there is a dearth of information. We have some very preliminary information that asbestos air levels near sites of extensive braking may be 3 times higher than background. Since wind conditions are highly variable, much more analysis of this question is required. All our data really show is that brakes are not as obvious a problem as asbestos-containing fireproofing spray in New York City. Perhaps you may wish to qualify 7 B by the words "Unless evidence of safety is demonstrated the use of asbestos...."

In summary, your regulations in general are excellent, your intent is proper, and I am pleased to support you.

Table I

Observed and Expected Number of Deaths
Among 632 Asbestos Workers Exposed to
Asbestos Dust 20 Years or Longer, 1943-1962

<u>Cause of Death</u>	<u>Years</u>				<u>Total,</u>
	<u>1943- 1947</u>	<u>1948- 1952</u>	<u>1953- 1957</u>	<u>1958- 1962</u>	<u>1943- 1962</u>
Total, all causes.....					
Observed (asbestos workers)	28	54	85	88	255
Expected (US white males)..	39.7	50.8	56.6	54.4	203.5
Total cancer, all sites.....					
Observed (asbestos workers)	13	17	26	39	95
Expected (US white males)...	5.7	8.1	13.0	9.7	36.5
Cancer of lung and pleura....					
Observed (asbestos workers)	6	8	13	18	45
Expected (US white males)..	0.8	1.4	2.0	2.4	6.6
Cancer of stomach, colon and rectum.....					
Observed (asbestos workers)	4	4	7	14	29
Expected (US white males)..	2.0	2.5	2.6	2.3	9.4
Cancer of all other sites com- bined.....					
Observed (asbestos workers)	3	5	6	7	21
Expected (US white males)..	2.9	4.2	8.4	5.0	20.5
Asbestosis.....					
Observed (asbestos workers)	0	1	4	7	12

Death rates as reported annually by the U.S. National Office of Vital Statistics. A five year average is given here for each period.

Death rates include cancer of lung, bronchus, pleura, mediastinum and trachea, assigned to international list code No. 47b-f prior to 1949 and to No. 160-164 thereafter.

Table II

Expected and observed deaths among 370 asbestos
insulation workers, Jan. 1, 1963 - June 30, 1971.

	<u>Observed deaths</u>	<u>Expected deaths*</u>
Total cancer (all sites)	92	15.1
Cancer of lung, pleura, trachea, bronchus	47	4.5
Lung cancer	43	+
Pleural mesothelioma	4	+
Peritoneal mesothelioma	19	+
Cancer of stomach, colon and rectum	12	3.0
Cancer all other sites	14	7.6
Asbestosis	20	+
All other causes	51	63.5
Total deaths	163	78.6

*Expected deaths are based upon age specific rates for U.S.
white males in 1937. Smoking habits are disregarded.

+United States data not available but figure should be only
slightly less than 4.5.

+United States data not available but these are rare causes
of death in general population.

— R71-16

EXHIBIT 23

23

Asbestos Information Association/North America

22 East 40th Street
New York, N. Y. 10016
(212) 661-8206

ASBESTOS INFORMATION ASSOCIATION/NORTH AMERICA

In the past few years, the subject of occupational and possible public health hazards associated with the manufacture and use of asbestos and asbestos-containing products has become a topic of widespread interest and concern. Many misleading or erroneous impressions of the true nature of this problem have been voiced.

To correct these erroneous impressions and to communicate to various interested groups, both public and private, the true facts about this problem and about industry efforts and accomplishments in identifying and controlling asbestos/health hazards, major companies in the asbestos manufacturing industry have joined together to form the Asbestos Information Association/North America (AIA/NA).

The member companies of the AIA/NA are: Atlas Asbestos Company, Cement Asbestos Products Company, Certain-teed Products Corporation, Flintkote Company, GAF Corporation, Johns-Manville Corporation, National Gypsum Company, Panacem Corporation and Raybestos-Manhattan, Inc.

Basically, the objectives of the Asbestos Information Association/North America are to (1) Provide an authoritative channel of communication to all publics, (2) Publicly rebut irresponsible and uninformed criticism, and (3) Disseminate information on the indispensable uses of asbestos in our modern technological society.

Title: ASBESTOS-CEMENT SIDED FRAME STRUCTURE - Ambient Air
Sampling During Demolition

SUMMARY

On March 11, 1971, a three story building was demolished in the Easton Redevelopment site at S. 4th Street and Washington Court, Easton, Pennsylvania. Air samples were taken the previous day, during demolition, and after demolition. Samples were also taken on March 22, 1971.

The building was of heavy frame construction with three stories and peaked attic. The 25 x 38 ft first story was faced with brick veneer and the remainder of the walls were faced with asbestos-cement shingles. The inner wall of the second floor, rear porch, was also faced with asbestos-cement shingles. Small quantities of other asbestos-bearing materials were also present for a total of:

Asbestos-cement siding Shingles - 2390 sq ft
Asphalt and Vinyl Asbestos Floor Tile - 740 sq ft
Air-cell Pipe Insulation (2 x 1 in.) - 36 linear ft

Heavy visible dust and much shingle breakage were noted during the 1 hr and 25 min. elapsed time of demolition, of which 1 hr was actual working time. The wind varied from calm to light and was variable in direction.

Air sampling for asbestos fiber gave results as follows:

Time	Fibers per cc	
	>5 u	Total
March 10, 1971	0.19	0.42
March 11, 1971 During demolition	0.29 (0.19 to 0.34)	0.56 (0.44 to 0.80)
Ambient measure *	0.08	0.29
After demolition	0.30	0.46
March 22, 1971	0.06 (0.04 to 0.10)	0.10 (0.05 to 0.14)

*160 ft west of house and 15 ft higher than other samplers.
Samples taken during demolition.

The air sampling figures show somewhat high background values on the previous day when no demolition was in progress. In an overall comment, it may be said that no significant increase of airborne fiber was noted during this operation, according to G. L. Swallow of the Environmental Control Department.

Contents: Summary, Investigation, and Appendices I and II.

Reported by



R. H. Neisel

Materials Evaluation Section

jr

INVESTIGATION

On March 11, 1971, a three story building was demolished in the Easton Redevelopment site at S. 4th Street and Washington Court. Air samples were taken the previous day and during and after demolition. Photographs were also taken.

The building was of heavy frame construction with three stories and peaked attic. The 25 x 38 ft first story was brick veneer and the remainder of the walls were faced with asbestos-cement shingles. At least the north face had J-M No. 107 CEDARGRAIN, wavy butt shingles. The inner wall of the second floor porch had painted wet-machine shingles of similar design as did the south wall. Asbestos contents are believed to be 20-25 per cent. The shingles had been painted years ago and the paint was half eroded. The roof was slate.

The 8 x 25 ft second floor rear porch was floored with 1/8 x 9 x 9 in. asphalt tiles with visible asbestos fiber. Two, third floor bedrooms, each 12 x 10 ft, were floored with 1/16 x 9 x 9 in. vinyl tile with visible fiber. In the ground floor was a 25 x 12 ft asphalt tile floor and 12 sections of asbestos Air-Cell pipe insulation. Estimated areas were:

Asbestos-cement siding shingles	- 2390 sq ft
Floor tile	- 740 sq ft
Air-Cell Pipe Insulation	- 36 linear ft

Filters were operated prior to, during, and after demolition at locations shown in Appendix II.

The previous day had been clear with 47 per cent RH at 36F as of 10:30 AM and light (1-3 mph) wind. Light rain fell overnight. At 8:50 AM on the 11th, the weather was cloudy, 82 per cent RH at 36F and calm. At 10:30 AM conditions were 41F, 83 per cent RH with light (4-7 mph) north, northwesterly winds.

Demolition down to ground floor level took 59 min. working time with 1 hr 25 min. elapsed time. A clam shell bucket (3 tons) on a crane was used.

The following is a detailed log of observations:

Time		Observation
Clock	Elapsed	
9:23	0 Min.	North chimney lifted off and deposited on ground.
9:24	1	Dropped bucket 10-15 ft through west roof slope. Repeated three times. Cloud of dark dust.
9:25	2	Gripped north wall peak, lifted and lowered, then dropped 5 ft to ground. Very light SE wind.
9:27	4	Dropped bucket on top of west wall. South dormer still in place.
9:29	6	Tried to lift north wall. Cable frayed and stopped for repair.
9:45	22	Gripped NW corner, tore out, and dropped. Studs noted 2 ft O.C. and 3 x 4 in.
9:49	26	Dropped bucket on floor of third level.
9:50	27	Half shingles of upper west wall off.
9:51	28	Large section shingles fell to 4th Street.
9:52	29	Working on center of N wall. NW corner split open.
9:54	31	Picked off S chimney. SE dormer gone.
9:57	34	All of roof gone.
9:59	36	All of S. peak dropped in building center.
10:00	37	Ten by fifteen foot wall and shingle section dropped inside house.
10:01	38	West wall dropped in 4th Street. Heavy dust toward sampler.
10:03	40	Southwest wall pulled up and into building.
10:08	45	All of attic floor gone. Chimney section 5 ft long dropped in center.
10:10	47	Rest of N wall dropped in parking lot. Heavy dust moving to east.
10:14	48	Crane engine stalled.

Time		Observation
Clock	Elapsed	
10:15	49	Top half of S wall dropped inside.
10:18	52	Entire third floor wall down.
10:22	56	West half of south second floor wall down. One half on street and one half inside.
10:26	60	Dropping bucket through ceiling of ground floor.
10:29	63	Coffee break.
10:35	69	Back to work.
10:38	72	One third of second floor south wall down inside.
10:41	75	East porch wall and rest of north wall into lot.
10:44	78	Inner east porch wall (A-C shingles) into lot.
10:45	79	Work stopped. Rubble down to ground floor brick walls. Plan to cave in with front end loader and truck away.

Walls originally had wood clapboard. Interior plaster on wood lath up to 1-1/4 in. thick. Organic fiber.

APPENDIX I

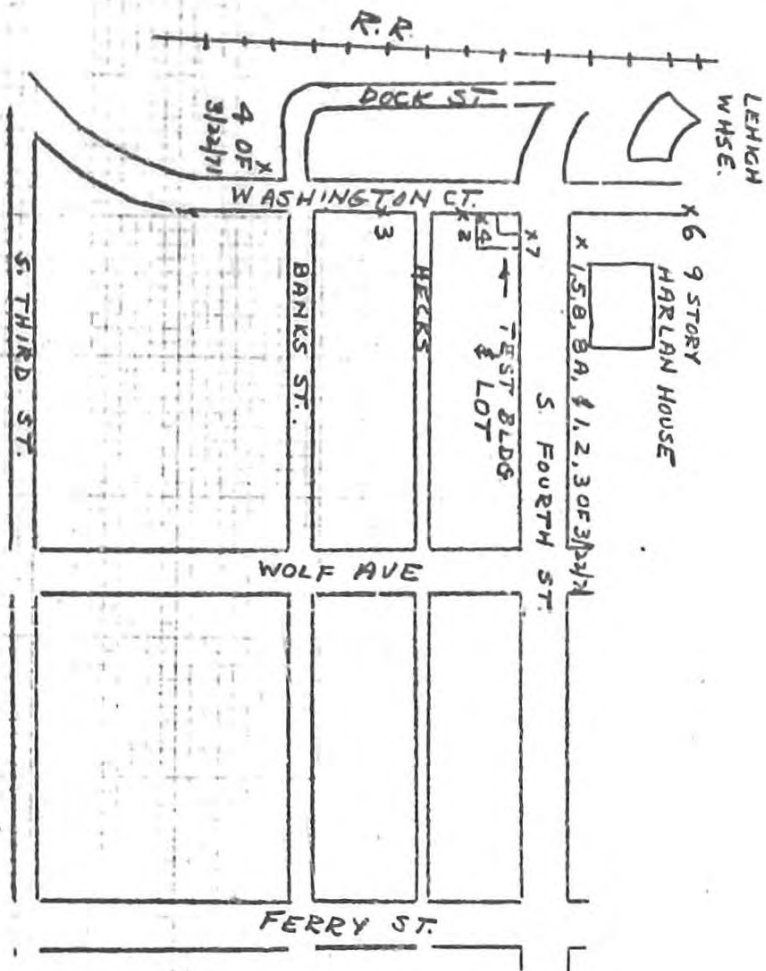
Measurement of airborne fibers collected under the direction of R. D. Hindmarch of the Environmental Control Department, was conducted and reported in his letters of March 17, 1971 and March 25, 1971. The following results are abstracted:

The specimens were collected on 37 mm 0.8 u pore size membrane filters at the stations indicated on the diagram. Count values were as follows:

Sample Number	Date	Results		Remarks
		F/cc > 5 u	Total	
1	3/10/71	0.17	0.41	Before demolition
2	3/10/71	0.21	0.42	Before demolition
3	3/11/71	0.31	0.54	During demolition
4	3/11/71	0.31	0.80	During demolition
5	3/11/71	0.19	0.44	During demolition
6	3/11/71	0.08	0.29	During demolition but 160 ft away
7	3/11/71	0.34	0.53	Personal sample and also mounted on cab of crane
8	3/11/71	0.32	0.55	Taken starting 2-1/2 hr after completion of demolition
8A	3/11/71	0.27	0.37	Same time as No. 8
1	3/22/71	0.07	0.12	Taken later when trucking of debris but no demo- lition was in progress
2*	3/22/71	0.05	0.08	Same
3*	3/22/71	0.04	0.05	Same
4	3/22/71	0.10	0.14	Same

*Samples taken on 8 x 10-in., 1.2 u pore size membrane filters.

APPENDIX II



DEMOLITION AREA IN
EASTON, PA.

NUMBERS REFER
TO SAMPLE SITES
SHOWN IN TABLE

USES OF ASBESTOS

I. Construction Industry - Major Uses

A. Locked-In Products or Uses

1. Asbestos-Cement Building Products
 - Corrugated Sheets
 - Shingles
 - Flat Sheets
 - Decorative Panels
2. Asbestos-Cement Pipe
 - Pressure Pipes
 - Sewer Pipes
 - Irrigation Pipes
 - Conduits
 - Storm Drains
3. Caulking and Sealing Compounds
4. Floor Tiles
 - Asphalt
 - Vinyl
5. Papers
 - Pipeline Protection (Saturated)
 - Roofing (Saturated)
 - Flooring Backing
6. Plastics
 - Filled Plastics
 - Re-inforced Plastics (Fibers, Felts, Papers)
7. Roofing Cements

B. Not Locked-In Products or Uses

1. Acoustical Sprayed Insulations
2. Insulating Cements
3. Insulations
 - Calcium Silicate (Blocks & Pipe Covering)
 - Magnesia
 - Air Cell Paper
 - Calcium Silicate Boards
4. Gypsum Joint Cements
5. Textiles (Not Saturated)
 - Lagging Cloths
 - Welding Curtains
 - Protective Clothing

USES OF ASBESTOS (Con't)

II. Construction Industry - Minor Uses

A. Locked-In Products or Uses

1. Asbestos-Cement Building Products
Electrical Panels
Table Tops
2. Paints
3. Welding Rods

B. Not Locked-In Products or Uses

1. Stucco

III. Non-Construction Industry - Major Uses

A. Locked-In Products or Uses

1. Brake Linings
2. Brake Blocks
3. Clutch Facings
4. Chlorine Cells Usage*
5. Drilling Fluid Usage*
6. Electrical Insulations Saturated Papers
7. Gaskets
8. Packings
9. Plastics
10. Textiles (Saturated)
11. Automobile Undercoatings*

B. Not Locked-In Products or Uses

1. Millboards and Papers (Unsaturated)
2. Textiles (Unsaturated)
3. Electrical Insulations
Rovings
Cable and Cord Insulations

IV. Non-Construction Industry - Minor Uses

A. Locked-In Products or Uses

1. Asbestos-Cement Molded Articles
2. Catalyst Supports

B. Not Locked-In Products or Uses

1. Filtration or Filter Aids*

* May not be locked-in at process mixing stations, but locked-in in actual usage.

RECOMMENDED CHANGES IN ILLINOIS POLLUTION CONTROL BOARD REGULATIONS ON ASBESTOS

PART II: GENERAL REQUIREMENTS

No commercial activity, not otherwise hereinafter prohibited, involving the potential discharge of visible amounts of asbestos fiber into the environment from the construction, alteration, repair or demolition of a structure or from the processing or manufacturing of asbestos-containing products, shall be conducted unless the person or entity in charge of such activity complies with the following regulations:

201. (No recommended changes)

202. Facilities shall be provided and procedures instituted and supervised that prevent the removal of visible amounts of asbestos-containing material from the site on the clothing of employees.

203. (No recommended changes)

PART III: SPRAY FIREPROOFING AND INSULATION

301. (No recommended changes)

302. (No recommended changes, Sections (a) thru (f))

- (g) Any plenum or other structure coated with or containing asbestos-containing insulation and used in the circulation of air in a building shall be thoroughly cleaned of all debris and waste insulation. All applied asbestos fiber-containing insulation within a plenum or duct shall be coated with a sealant which precludes exposure of the asbestos fiber-containing material to the circulating air.

PART IV: CONSTRUCTION, ALTERATION AND REPAIR
OF STRUCTURE

401. (a) Visible emissions from the cutting, trimming, fitting or stripping of asbestos-containing material in an area open to the atmosphere are prohibited.

- (b) Visible emissions of asbestos-containing particulate matter into the atmosphere from the construction, alteration or repair of a structure are prohibited.

402. Loosely-bound asbestos-containing insulation materials used in the construction, alteration or repair of a structure shall be coated with a sealant, provided with a cover or installed in some other manner so as to preclude exposure of the asbestos fiber to the circulating air.

PART V: DEMOLITION

501. Where the risk of public exposure to asbestos fiber from the dialodging of asbestos-containing insulating or fireproofing materials is present, no demolition of a structure shall be initiated unless all safeguards necessary and practical to prevent the emission of visible amounts of dust are taken.

Such procedures shall include, but are not necessarily limited to:

- (a) Boilers, pipes and steel members insulated or fireproofed with asbestos-containing material shall be wetted and stripped before toppling of walls is begun. This procedure shall be followed, where practical, as to all other asbestos-containing insulating products. Asbestos waste from such products shall be immediately bagged and disposed of in accordance with section 203.
- (b) (Eliminate this section as unnecessary)
- (c) Before the demolition or toppling of any section or wall of the structure, adequate wetting to suppress the dust shall be employed.
- (d) (No recommended changes)
- (e) (No recommended changes)

502. (No recommended changes)

PART VI: MANUFACTURING

601. (a) Emissions to the atmosphere from any factory, plant or enterprise which engages in the processing or manufacturing of any asbestos-containing product shall be controlled as follows:

- (1) All direct forced gas streams containing particulate matter resulting from the manufacturing or processing of asbestos-containing products shall be treated in fabric bag filter installations or, where special process conditions exist, in high energy wet collectors.

- (b) Visible emissions of particulate matter from any manufacturing or fabricating operations in an area directly open to the atmosphere are prohibited.

602. (Eliminate this section as unnecessary)

603. Visible emissions from waste sludge containing asbestos collected from settling ponds are prohibited during transport. Such waste sludge shall be disposed by burial at an approved site.

PART VII: ASBESTOS-CONTAINING PRODUCTS

701. No product which may emit asbestos-fiber during its transportation shall be transported unless such product is enclosed in such a manner as to preclude the visible emission of asbestos fiber into the ambient air.

702. (Eliminate this section as unnecessary)

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Brake Lining Decomposition Products

A number of investigators have found asbestos bodies in the lungs of urban residents who were not occupationally exposed to asbestos. A relationship between the carcinogenic properties of asbestos and the urban excess of lung cancer has been suggested. The decomposition products of brake linings have been described as a possible source of these fibers. Brake testing laboratory methods were used to test fiber emission from a variety of friction products. Only a very small fraction of the asbestos escaped as free fiber while the remainder was transformed into some other, nonfibrous, mineral. A significant release of free fiber occurred only under conditions extreme enough to produce brake failure.

Table I. Asbestos bodies in human lungs.

Location	Year	% Positive
Cape Town (2)	1963	26
Miami (2)	1965	27
Pittsburgh (3)	1965	41
Johannesburg (4)	1965	39
Finland (5)	1966	58
Montreal (5)	1966	48
San Francisco (1)	1966	42

The occurrence of coated fibers referred to as "asbestos bodies" in the lungs of urban populations not occupationally exposed to asbestos has been noted by several investigators. The results of several autopsy series, as summarized by Cooper,¹ are shown in Table I.¹⁻⁴ Thompson,⁵ in suggesting possible modes of exposure that could account for these bodies, stated:

The average private motor vehicle wears out three or four sets of brake linings and one or two clutch linings in its lifetime, and commercial and public transport vehicles wear out many more. These linings consist largely of asbestos which is ground to dust as the linings wear, and most wear occurs in built-up areas. This alone in most cities would involve the discharge of many tons of asbestos dust and fibers in the streets each year.

Since asbestos has been implicated as a carcinogen⁶ and lung cancer has a higher incidence⁷ among urban populations, the fate of the asbestos worn from brake linings becomes significant.

Brake Lining Composition

The average composition of typical brake linings of the type tested is shown in Table II. Individual mixes may vary considerably from these averages.

Each of these ingredients performs a particular function. The asbestos provides strength, heat resistance, and selective decomposition under stress. The resins and polymers hold the other ingredients together. Various oxides are added as pigments and to improve the grip of the lining on the drum. Soft metals such as lead and brass improve wear while carbon and graphite serve

as friction modifiers.

Brake lining quality depends to a large extent on the type of binder used. Linseed oil, which begins to decompose at about 450°F, is used for light service brakes. More demanding service requires brakes made from cast-iron type resins or oil modified phenolic resins. These ingredients are mixed, either dry or with a solvent added, formed into shape, and cured in an oven. The hardened lining is then cut, ground, and sanded to the precise dimensions of the finished linings. The dust produced by the abrading operations in asbestos-friction product factories (Figure 1) contains free asbestos fibers that are similar to those in industries where cancer is known to be in excess.¹⁰ The question to be resolved, therefore, is whether the dust produced by the normal wear of brake linings contains this same type of free asbestos fiber.

Test Methods

In an initial exploratory survey, the dust obtained from inside automobile brake drums removed for brake relining was examined by electron micrograph. Figure 2, which is typical of the series, does not reveal any free fibers. However, this finding did not eliminate the possibility that free asbestos fibers were released and that they had escaped into the atmosphere. To examine this hypothesis a series of experiments was devised to permit sampling decomposition products of the lining under simulated operating conditions. The tests (Table III) were performed with the brake testing machines in the laboratory of a major brake lining manufacturer. Most of the tests were performed on a

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Figure 1. Brake lining factory dust.

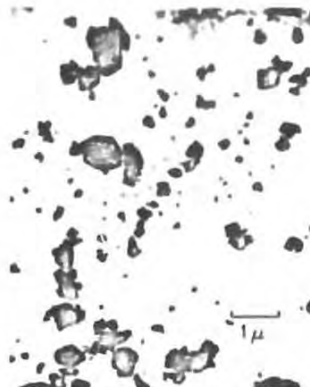


Figure 2. Dust removed from brake drum.



Figure 3. Decomposition product from normal wear of brake lining.

friction-testing machine which used one-inch-square samples of the test lining. Heating and cooling apparatus permitted control of drum temperature from those encountered with intermittent brake use to the maximum temperature which occurred only in extremely rapid or "panic" stops from high speed. Other tests were performed on a brake-testing dynamometer which subjected a complete brake assembly to a series of stops and starts from different speeds and at different deceleration rates to simulate actual driving conditions. In these tests the drum temperatures varied according to the driving condition simulated. During each run at a test condition, a sample was collected on an 0.8 μ pore size membrane filter. Electron micrographs of these filters were examined to determine the presence of free asbestos fibers and to quantify these fibers when more than a few occurred.

Results

The object of the tests was to determine what proportion of the asbestos known to be present in the lining appeared as free fiber in the decomposition product. No attempt was made at a mass balance between the material worn from the lining and the decomposition product since it was not possible to collect all of the decomposition product. However, the percentages obtained in the fraction of decomposition product examined are applicable to the total material worn from the brake and may be compared to the percentage of asbestos originally present.

In all but a few tests the automobile

drum brake linings showed less than 1% free fiber in the decomposition product as compared to about 50% in the lining. An electron micrograph of the decomposition product obtained from a typical test in this group is shown in Figure 3. In those tests where a significant mass of free fiber was released (Figure 4), the temperature was in an extremely high range for the lining in question as evidenced by rapid drops in the coefficient of friction. Had these linings been subjected to like conditions in a vehicle, the brakes would have failed.

Similar results were obtained in the bus and truck drum brake tests and in the clutch test. The experimental model disk brake tested did release some (>5%) free fibers, but no conclusion can be drawn from a single sample.

Discussion

Except in all but the most extreme driving conditions, only a very small fraction of the 30 to 50% asbestos present in a brake lining escapes into the atmosphere as free fiber. The question remains: What happened to the asbestos? A prevalent theory of brake operation holds that wear occurs not by abrasion of the lining by the drum but by the production of minute areas of intense heat at the points of contact between the drum and the lining. Decomposition, not only of the binder but of the asbestos as well, occurs at these locations since the breakdown temperature of asbestos, about 900°F, is exceeded. Decomposition products will include not free asbestos fibers, but a different mineral resulting from thermal metamorphosis of asbestos.



Figure 4. Decomposition product from brake lining at failure.

Table III. Test results by electron micrograph

	Product	Brand	Test Method	No. of Samples	Conditions of Test-F	Presence of Free Fibers	% Free Fiber*
1.	Automobile drum brakes	A	Friction	5	300-800	Few	<1
2.	Automobile drum brakes	B	Friction	6	250-800	None	0
3.	Automobile drum brakes	C	Friction	5	300-700	Few	<1
4.	Automobile drum brakes	C	Friction	1	700-900	Numerous	<10
5.	Automobile drum brakes	D	Friction	5	300-800	Few	<1
6.	Automobile drum brakes	E	Friction	5	300-700	Few	<1
7.	Automobile drum brakes	F	Friction	5	300-800	Few	<1
8.	Automobile drum brakes	G	Friction	2	100-500	Few	<1
9.	Automobile drum brakes	G	Friction	1	600-700	Numerous	<15
10.	Automobile drum brakes	H	Friction	2	100-600	Few	<1
11.	Automobile clutch	J	Dynamometer	1	Normal driving	None	0
12.	Automobile disk brake	K	Dynamometer	1	Normal driving	Few	<5
13.	Bus drum brake	L	Dynamometer	1	City driving	None	0
14.	Bus drum brake	M	Friction	2	450-550	None	0
15.	Truck drum brake (light)	F	Friction	10	300-800	Few	<1

*Weight estimated from fiber volume.

Some independent experiments done at a brake lining research laboratory¹¹ have shown that all of the magnesium present in the chrysotile asbestos originally in the lining can be accounted for in the decomposition products. However, the characteristic X-ray diffraction pattern of chrysotile asbestos had vanished. Thus, it is apparent that under conditions in which asbestos is worn from the lining of a brake, the asbestos is destroyed.

Conclusion

Only a very small proportion of the asbestos worn from brake linings is released as free fiber; the remainder is converted into some other mineral as a result of the extreme temperatures generated at small spots on the lining surface. Thus, although urban air contains a few free fibers as a result of brake lining wear, they represent a very small

proportion of the total asbestos used in manufacture of brakes. Many sources of respirable fibers not associated with asbestos product, have been identified,¹² and free fibers from brake lining wear seem to be an inconsequential health factor in urban air pollution.

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MESOTHELIOMA OF PLEURA AND PERITONEUM FOLLOWING EXPOSURE TO ASBESTOS IN THE LONDON AREA

BY

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A series of 83 patients with histologically confirmed mesothelioma of the pleura or peritoneum, 41 men and 42 women, with biopsy-proven exposure to asbestos dust, was studied. The earliest death recorded was in 1917, the latest in 1976. The mean age at death was 54.0 years (range 40.0 and 70.4).

In 76 of the series full occupational histories were available. Of these 42 (55%) gave a history of exposure to asbestos dust, 34 (45%) to asbestos alone, 12 (16%) to asbestos and other occupational exposures. The remaining 10 (13%) had no history of exposure to asbestos. The mean age at death of the 42 patients with a history of exposure to asbestos compared with the 10 patients with no history of exposure to asbestos was 54.0 years (range 40.0-70.4) and 55.0 years (range 40.0-70.4) respectively. The difference was not significant ($P = 0.0001$). None of the 10 patients with no history of exposure to asbestos, based on pathological grounds, was found to have a history of exposure to asbestos. The remaining 34 (45%) of the series, with a history of exposure to asbestos, were divided into two groups: 30 (88%) of the men and 4 (10%) of the women. The remaining 10 (12%) of the series, with no history of exposure to asbestos, were divided into two groups: 10 (100%) of the men and 0 (0%) of the women. The remaining 10 (12%) of the series, with a history of exposure to asbestos, were divided into two groups: 10 (100%) of the men and 0 (0%) of the women. The remaining 10 (12%) of the series, with no history of exposure to asbestos, were divided into two groups: 10 (100%) of the men and 0 (0%) of the women.

In 47 patients in the mesothelioma series, histological evidence of asbestos exposure was available. In 30 (62.5%), either asbestos or asbestos bodies were present.

In recent years, the association between exposure to asbestos dust and cancer of the lung and other malignant neoplasms has been the subject of much research (leading article, 1964; Wagner, Slone, and Marchand (1966) described the occurrence of mesothelioma of the pleura in those exposed to crystalline asbestos in the mining districts of South Africa, and this has stimulated further studies of the occupational histories of patients suffering from this tumor (Owen, 1964; Fowler, Sloper, and Warner, 1964).

The present investigation concerns patients in whom mesothelioma had been diagnosed at the London Hospital during the past 50 years. After examining the necropsy and biopsy specimens held in the pathology department, Houlding (1964) confirmed a diagnosis of mesothelioma in 83 patients, of whom 41 were men and 42 women. Thirty-one of the men had pleural tumours and 10 peritoneal,

among the women 25 had pleural and 17 peritoneal tumours.

The aim of this study was to investigate the occupational histories of these patients and to trace any other possible exposure to asbestos. It was found that 42 of the patients at the outset of the investigation had no history of exposure to asbestos, and 42 of these subsequently died. The earliest date of death in the series was 1917, 10 died before 1950, 33 between 1950 and 1959, and the remaining 40 in the past four and a half years. The youngest patient died at the age of 43, and nearly all were dead before the age of 65 (Table 1).

Clinical Features

The ward notes, if any, of the patients were available, and gave a picture of a disease with a consistent symptomatology. Aspiration of the pleural

TABLE 1
AGE AT DEATH OF 18 PATIENTS WITH MESOTHELIAL
TUMOURS

Age (yr.)	Male		Female	
	No.	%	No.	%
<34	2	4.9	1	2.4
35-44	7	17.1	6	14.3
45-54	15	36.6	9	21.4
55-64	10	24.4	18	42.9
65+	7	17.1	8	19.0

tumours, the commonest presenting symptom was the rapid onset of extreme shortness of breath due to the formation of a massive pleural effusion. Pain was a prominent feature, either described as a dull ache, sometimes due to invasion of the ribs or vertebrae, or sometimes sharp and radiating, suggesting nerve involvement. Tumours in the chest wall were not uncommon, occurring either in previous operation scars or by direct invasion of the chest wall. The symptomatology of the peritoneal tumours was more varied. In some patients the presenting symptom was pain on defaecation or micturition. Diffuse upper abdominal pain was very common, and swelling of the abdomen due to ascites was always present terminally. The methods of treatment included pneumonectomy, decortication of the lung, deep x-ray therapy, instillation of radioactive gold, and cytotoxic drugs. They were used alone or in combination but had little effect on the course of the disease. Half of the patients suffering from pleural mesothelioma died within one year of the onset of symptoms, a further third within two years, and only one patient survived for more than three years. The course of those with peritoneal tumours appeared to be equally rapid. Eight of the patients died within six months of the onset of symptoms.

Sources of Information on Occupation and Residence

In addition to the ward notes, some of which gave good occupational histories, there were three other sources of information: the patient's general practitioner; the records of an asbestos factory in the area; and personal interviews with patients or their surviving relatives.

As a first step, general practitioners were circulated with an explanatory letter asking them to complete a form giving details of the occupations of patients and their immediate relatives. Within two months 65% of the doctors had replied. In two cases a hitherto unknown exposure to asbestos was revealed; in others the name and address of a surviving relative were given, but in the majority the doctor was unable to give information because, on the death of the

patient, the notes had been returned to the local executive council of the National Health Service, where they were destroyed within a period of three years.

In one of the asbestos factories a file was kept with detailed records of all employees since it started in 1913. The names of all patients were checked with these files. Nine men and nine married women (after their maiden names had been ascertained from relatives) were identified without difficulty. The exact dates of employment of these 18 patients and the jobs they had done were obtained from the records of the factory.

The four patients alive at the beginning of the investigation were interviewed personally. A few relatives were contacted by post, but the relatives of 68 patients were interviewed by one of us (H.T.) at their homes situated mostly in the East End of London. Not only was an occupational history of the patient, the spouse, sons and daughters, and father taken, but past addresses were also recorded. The interviews lasted for at least an hour. To recall events of 30 or 40 years ago it was often necessary to explore the residential and occupational histories of all members of the family.

Exposure to Asbestos in Three Groups of Patients Suffering from Other Diseases

For comparison with the mesothelioma patients, three further groups of patients suffering from other diseases were investigated.

The first group was selected from the patients in the medical and surgical wards of the hospital during the early summer of 1964 ('in-patient' series). Each patient in the mesothelioma series who had been traced was matched with an in-patient of the same sex born in the same five-year period. As there was a dearth of male patients over 75 years of age in the hospital, a sample of six patients of this age and older was taken from a neighbouring geriatric hospital.

The second group were those who had originally been filed in the pathology department of the hospital as cases of mesothelioma but in whom the diagnosis was subsequently rejected on pathological grounds by Hourihane (1964) ('rejected series'). As it had proved extremely difficult to locate the relatives of those who died before 1950, attempts were made only to trace the three survivors in this group and the 14 who died after 1950.

The in-patient series, already described, were all admitted to hospital in 1964. The patients with mesothelioma were admitted to the same hospital over a period of 47 years during which there might have been a substantial change both in the residential

areas and social classes of patients attending the London Hospital. Thus, a third group of patients was taken from the hospital records matched by date of admission as well as date of birth and sex with the patients in the mesothelioma series. Their places of residence and occupations were extracted for comparison with those of the in-patient series.

Results

Details of the 83 patients in the mesothelioma series are given in the Appendix. No information about past domestic and occupational histories was available for seven patients. Among the remaining 76, 40 (52.6%) gave a history of exposure to asbestos compared with only nine (11.8%) of the 'in-patient' series (Table 2). The difference in this proportion is

TABLE 2

TYPES OF EXPOSURE TO ASBESTOS OF 76 PATIENTS WITH MESOTHELIOMA AND 76 IN-PATIENTS WITH OTHER DISEASES

Type of Exposure	Mesothelioma Series		In-patient Series	
	No.	%	No.	%
Employed at one asbestos factory	18	24.0	1	1.3
Delivered goods to factory	1			
Employed at other asbestos factories	4	5.3	1	1.3
Insulators and ladders	8	10.5	4	5.3
Relative worked with asbestos	9	11.8	1	1.3
Dockers handling asbestos cargo	0	—	2	2.6
No history of work or domestic exposure to asbestos	36	47.4	67	88.2

Positive exposures to asbestos in mesothelioma series versus positive histories in in-patient series.

$$\chi^2 = 27.11, P < 0.001$$

*Seven cases of mesothelioma could not be traced and are omitted from this table.

statistically highly significant ($\chi^2 = 27.11$; $P < 0.001$). This result is unlikely to be unduly influenced by the two groups of patients not being matched for year of admission to hospital, since there was no significant difference either in the areas of residence or in the occupations of the patients admitted in 1964 and those admitted between 1917 and 1964. Comparing the confirmed and rejected patients in the mesothelioma group who died after 1950, 36 (50%) of the confirmed and none of the 17 rejected cases gave a history of exposure to asbestos (Table 3). This difference is statistically highly significant ($\chi^2 = 11.83$; $P < 0.001$).

TABLE 3

EXPOSURE TO ASBESTOS OF CONFIRMED AND REJECTED CASES OF MESOTHELIOMA SURVIVING AFTER JANUARY 1950

Exposure to Asbestos	Diagnosis of Mesothelioma	
	Confirmed	Rejected
Exposure positive	36	0
No work or domestic contact	19	16
Untraced	1	1

$$\chi^2 = 11.83, P < 0.001$$

Mesothelioma Series

Occupational Exposures. There are details available for the 18 mesothelioma patients who had worked at one asbestos factory which used crocidolite asbestos with small amounts of chrysotile and first introduced amosite in 1926. Eleven started work before 1933 (the year when the asbestos regulations controlling the manufacture of asbestos goods and the protection of asbestos workers became effective), and seven started work between the beginning of 1933 and the end of 1942. The occupations of these workers and the type of asbestos they used are shown in Table 4. All of the 17 for whom details were available had used crocidolite asbestos. Five of the occupations listed are not scheduled as

TABLE 4

JOBS OF 17* PATIENTS WITH MESOTHELIOMA EMPLOYED AT ONE ASBESTOS FACTORY

Statutory Obligations	Job	Male	Female	Material
Subject to regulations	Spinning	0	4	Crocidolite
	Carding	1	1	Crocidolite
	Clothing and weaving	1	0	Crocidolite, chrysotile, amosite
	Disintegrating and opening	2	1	Crocidolite, chrysotile, amosite
Not subject to regulations	Filter making for A.R.P. masks	2	0	Crocidolite
	Manufacturing of preformed pipe insulation	1	1	Crocidolite
	Manufacturing of brake linings	1	0	Crocidolite
	Rubber compounding	0	1	Crocidolite, chrysotile
	General labourer	1	0	Crocidolite, chrysotile, amosite

*Employment history not available for one female patient.

requiring medical supervision under the Asbestos Regulations (1931).

Four patients were working at other factories making all types of asbestos goods. One had been employed in a wagon works sawing asbestos sheets for partitions. Except for one, a woman employed in insulating electrodes with crocidolite, the types of asbestos used in these other factories are not known.

The eight heating engineers and lagers were all men. Three had been employed in dockyards on ship repairs, two in various types of lagging, one in a power station, one installing hospital sterilizing equipment, and one installing central heating. All were consistently employed on this type of work for more than 20 years, but, in some, exposure to asbestos was intermittent.

Domestic Exposures.—The group of nine, seven women and two men, whose relatives worked with asbestos, are of particular interest. The most usual history was that of the wife who washed her husband's dungarees or work clothes. In one instance a relative said that the husband, a docker, came home 'white with asbestos' every evening for three or four years and his wife brushed him down. The two men in this group, when boys of 8 or 9 years old, had sisters who were working at an asbestos factory. One of these girls worked as a spinner from 1925 to 1936. In 1946 she died of asbestosis. The press report of the inquest states: 'She used to return home from work with dust on her clothes'. Her brother had apparently no other exposure to asbestos; he started work as a shop assistant, then became a sawyer of iron girders until 1948 when he worked as a loader of groceries in the docks for five years (but never on dusty cargoes) and then returned to sawing iron girders. He died in 1956 of a pleural mesothelioma.

In-patient Series

Occupational and Domestic Exposures.—Two of this series had worked in asbestos factories and four had been employed as lagers. The husband of another was employed at an asbestos factory for three years, and two dockers in this group gave histories of handling asbestos cargoes from time to time throughout their working life.

The diagnoses of these patients have been grouped into seven categories (Table 5). The patients with a positive history of exposure are scattered throughout the various diagnostic groups, and there is no indication that exposure to asbestos could be related to the disease which had caused their admission.

Neighbourhood Exposures of Mesothelioma and In-patient Series.—The 36 patients with meso-

TABLE 5
DISEASE GROUPS OF PATIENTS IN THE IN-PATIENT SERIES

Disease Group	No. of Patients	No. with Exposure to Asbestos
Epithelioma	21	7
Mesothelioma	9	0
Respiratory (including cancer)	8	2
Gastro-intestinal (excluding cancer)	6	2
Respiratory (excluding cancer)	3	0
Alveolitis	11	1
Other	11	1
Total	76	9

thelioma and the 67 patients in the 'in-patient' series, who had neither an occupational exposure nor a relative living in the home working with asbestos, might have been exposed to asbestos dust because they lived in the immediate vicinity of an asbestos factory.

One factory, where more than one-fifth of the mesothelioma patients were employed, opened in 1913, having been situated nearer the centre of London for the previous seven years. There were three affected female patients living within half a mile of the factory during the time it was in production at its first site. When it opened they were children between 5 and 7 years old. At the present site, there were eight patients living within a half-mile radius of the factory. One man was born within a quarter of a mile of the factory and remained at the same address for 16 years. The other seven were women, and all except one were children when the factory opened. The seventh was 23 years of age and remained in the same house until she died 48 years later. She is the only patient who had neither occupational nor household exposure but in whom asbestos bodies were found in the lungs at necropsy.

Among the 'in-patient' series, one patient lived near the factory at its previous site and four others lived near its present site. One patient was 22 years old when she moved into the neighbourhood in 1915. She disliked it and, when interviewed, complained impartially about the dust from the asbestos factory and the rats in the house.

TABLE 6
RESIDENCE OF PATIENTS WITH NO OCCUPATIONAL OR DOMESTIC EXPOSURE TO ASBESTOS

Series	Lived Within 1 mile of Asbestos Factory		Lived More Than 1 mile from Asbestos Factory		Total
	No.	%	No.	%	
Mesothelioma	11	30.6	25	69.4	36
In-patient	5	7.5	62	92.5	67

$$\chi^2 = 7.85, P < 0.01$$

Thus, among those with no occupational or domestic exposures to asbestos, there are 11 (30.6%) patients in the mesothelioma series and five (7.6%) in the 'in-patient' series who lived within half a mile of an asbestos factory in the area (Table 6). The difference in the proportion of patients in the two series who lived in the vicinity of the factory and had had no other exposure to asbestos is statistically significant ($\chi^2 = 7.85$; $P < 0.01$).

Date of First Exposure and Interval Before Death in Mesothelioma Series.—Fifty-one men and women had been exposed to asbestos, including those who lived near the main asbestos factory. The duration of exposure varied widely, ranging from two months to over 50 years. The interval between the first exposure and death varied between 16 and 55 years (mean 37.5 years). Among the group of factory workers, where exposure was probably heaviest, the interval was shortest. It was longest among the group living in the neighbourhood of the asbestos factories, where exposure to dust was probably lowest. Table 7 shows the mean age of each group at first exposure and the mean of the interval before death in each group. Although the length of interval

TABLE 7
RELATION BETWEEN TYPE OF EXPOSURE AND LENGTH OF INTERVAL BEFORE DEATH IN 51 PATIENTS IN MESOTHELIOMA SERIES

Type of Exposure	No.	Mean Age at First Exposure (years)	Mean Interval between First Exposure and Death (years)
Factory work	23	22.5	29.4
Domestic	9	17.9	37.9
Laggers and Insulators	8	15.6	38.4
Living in neighbourhood of factory	11	8.5	48.6

varied between 29.4 and 48.6 years, the mean age at death showed less variation; it was 50.6 years in the group of factory workers and between 55 and 57 years in the other groups.

Asbestos Bodies and Asbestosis in the Mesothelioma Series.—Of the 83 patients in the mesothelioma series, lung tissue was available for examination in forty-three. In four other patients, all certified as suffering from asbestosis, with no lung tissue available, examination of sputum during life revealed the presence of asbestos bodies (Table 8). Both asbestos bodies and histological evidence of asbestosis were found in 15 of the 24 factory workers and laggers. A further five showed evidence of either asbestos bodies or asbestosis.

Lung tissue was available in only four patients exposed either through relatives or by living in the neighbourhood of asbestos factories. In two, asbestos bodies were present in lung tissue but there was no evidence of asbestosis.

TABLE 8
EVIDENCE OF ASBESTOS BODIES OR ASBESTOSIS IN 47 CASES OF MESOTHELIOMA WITH LUNG TISSUE OR SPUTUM AVAILABLE FOR EXAMINATION

History of Contact with Asbestos	Asbestos in Asbestos Bodies		Lung Tissue not Available
	Present	Absent	
Positive	22	6	23
Negative	4	8	13
Untraced	4	3	0
Total	30	17	36

Among the 12 patients in whom no definite contact with asbestos could be established, there was one, a merchant seaman from South Africa, whose lungs showed both asbestos bodies and asbestosis; he may have had contact either in South Africa or at sea, but his early history could not be established. There were three others in this group who had asbestos bodies in lung tissue without evidence of asbestosis.

Of the seven patients whose histories could not be traced, three had asbestos bodies and histological evidence of asbestosis and one had asbestosis alone.

Discussion

In the mesothelioma series there are 25 patients in whom no evidence of any exposure to asbestos could be found. A chief source of information was a history taken from a surviving relative. A surprising amount of information was obtained, but in some of those interviewed the memory may have been defective or they may not have known of short periods of exposure during the youth of the deceased. For example, one of the patients was eventually identified as having worked at a large asbestos factory for two months in 1941. This was before he married, and his widow did not know of this episode. It is of interest that asbestos bodies in the lungs were found in only four of this group, and it seems probable that among the remainder there were those who had had no exposure to asbestos.

In the mesothelioma series of patients, both industrial and non-industrial exposures were recognized. Among the men the exposure was predominantly industrial; 22 worked in asbestos factories or as laggers, two were exposed at home, and one lived near the asbestos factory. Among the women only 10 worked in asbestos factories, and a

further 17 had non-industrial exposures, seven in the home and 10 living near asbestos factories.

There is no evidence that the patients with peritoneal tumours differed in their type of exposure from the patients with pleural tumours. The proportion of positive findings of asbestos bodies or asbestosis was similar in both groups. A higher proportion of women, particularly among the factory workers, was affected by peritoneal tumours, but the difference between the sexes was not statistically significant.

The recent increase in the number of patients diagnosed at the hospital may be partly due to an increased interest in mesothelial tumours and partly to the long interval between first exposure and development of the tumour. Those exposed between 1915 and 1925 might be expected to die from about 1950 onwards. Asbestos imports to the United Kingdom have mounted steeply since 1932 (Leathart, 1964) and its uses are more widely diversified in industry. The increasing proportion of the population exposed to asbestos during the past 30 years may be expected to give rise to an increasing occurrence of mesothelial tumours.

The choice of groups for comparison with the mesothelioma series of patients was not ideal. The number of patients it was possible to trace in the 'rejected' series proved to be very small. The 'in-patient' series, although matched for date of birth and sex, differed from the mesothelioma series in that all were admitted to hospital during 1964. Neither of these groups could be interviewed without knowledge of the disease from which they were suffering. This could have led to bias with under-reporting of exposure to asbestos in the in-patient series. However, in the 'in-patient' series the actual patient was interviewed, and more detailed and reliable histories were obtained than was possible from the relatives of those who had died of mesothelial tumours. There was no evidence that, because of their more recent admission to hospital, the in-patient series was less

likely than the mesothelioma series to work in contact with asbestos or to live in closer proximity to asbestos factories.

There seems little doubt that the risk of mesothelioma may arise from both occupational and domestic exposures to asbestos. Wagner and others (1960) described patients with no exposure other than living as a child in the vicinity of the asbestos mines. A high incidence of asbestos plaques of the pleura has been found in the population living near an anthophyllite mine in Finland (Kiviluoto, 1960). More evidence is required of an increased risk to the population living in the neighbourhood of asbestos factories or other areas, such as dockyards, where asbestos is used in quantity.

We should like to thank Dr. D. O'B. Hourihane for his co-operation and for making the results of his investigations available to us.

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Appendix opposite

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APPENDIX
EIGHTY-THREE PATIENTS WITH MESOTHELIOMA TUMOURS

Clin. No.	Site of Primary Tumour	Sex	Year of Birth	Year of First Exposure	Duration of Exposure (years)	Year of Death	How Diagnosed	Asbestos Bodies in Lung Tissue	Pathological Evidence of Asbestosis in Lung Tissue	Interval from Initial Symptoms (months)	Relevant History
<i>Section A: Factory workers</i>											
8	Pleura	M	1901	1921	17	1938	Necropsy	Positive	Positive	5	Brake liner, boiler coverer, asbestos factory, mainly crocidolite
19	Peritoneum	M	1893	1919	22	1941	Necropsy	Positive	Positive	7	Disintegrator and other jobs, asbestos factory, amosite, crocidolite, chrysotile
18	Peritoneum	F	1908	1925	3	1947	Biopsy	Positive (sputum)	N/A	7	Sectional pipe maker, asbestos factory, certified asbestos, mainly crocidolite
24	Pleura	M	1880	1928	12	1950	Necropsy	Positive	Negative	9	Used asbestos boarding in railway carriage construction
14	Peritoneum	F	1893	1917	7	1951	Necropsy	Negative	Negative	7	Asbestos factory, job unknown
16	Peritoneum	F	1906	1922	7	1951	Biopsy	Positive (sputum)	N/A	7	Opener and sectional pipe maker, asbestos factory, certified asbestos, amosite, crocidolite, chrysotile
13	Peritoneum	F	1894	1915	3	1955	Necropsy	Positive	Positive	12	Spinner, asbestos factory, certified asbestos, mainly crocidolite
11	Peritoneum	F	1895	1917	1	1955	Biopsy	Positive (sputum)	N/A	3	Sectional pipe maker, asbestos factory, certified asbestos, mainly crocidolite
15	Peritoneum	F	1901	1918	11	1955	Biopsy	Positive (sputum)	N/A	6	Spinner, asbestos factory, certified asbestos, mainly crocidolite
9	Peritoneum	F	1897	1919	1	1956	Necropsy	Positive	Negative	3	Disintegrator, asbestos factory, amosite, crocidolite, chrysotile
21	Pleura	M	1893	1928	1	1957	Biopsy	N/A	N/A	18	Asbestos mixer, north England asbestos factory
12	Peritoneum	F	1918	1937	2	1958	Biopsy	N/A	N/A	18	Carder, asbestos factory, mainly crocidolite
5	Peritoneum	M	1908	1927	30	1958	Necropsy	Positive	Positive	18	Clothing and weaving, asbestos factory, amosite, crocidolite, chrysotile
7	Peritoneum	M	1923	1946	4	1958	Biopsy	N/A	N/A	3	Filter maker for A.R.P. masks, asbestos factory, mainly crocidolite
2	Pleura	M	1923	1941	1/6	1959	Biopsy	Negative	Negative	11	Filter maker for A.R.P. masks, asbestos factory, mainly crocidolite
20	Peritoneum	F	1907	1921	10	1959	Necropsy	Negative	Negative	7	Electrode coverer, crocidolite
17	Pleura	F	1908	1924	1	1962	Biopsy	Positive	Negative	7	Spinner, asbestos factory, mainly crocidolite
6	Pleura	M	1907	1942	10	1962	Necropsy	Positive	Positive	7	Delivered chemicals to asbestos factory, amosite, crocidolite, chrysotile
10	Peritoneum	F	1899	1923	11	1963	Biopsy	Positive	Negative	5	Spinner, asbestos factory, mainly crocidolite
1	Pleura	M	1899	1916	26	1963	Necropsy	Positive	Positive	7	Labourer, works and buildings, asbestos factory, amosite, crocidolite, chrysotile
23	Pleura	M	1908	1925	1	1963	Necropsy	Positive	Negative	8	Worked in yard of insulating contractors
3	Peritoneum	M	1911	1930	2	1963	Necropsy	Positive	Positive	6	Carder, asbestos factory, mainly crocidolite
4	Peritoneum	M	1917	1937	27	1964	Necropsy	Positive	Positive	24	Disintegrator and other jobs, asbestos factory, amosite, crocidolite, chrysotile
<i>Section B: Luggers and insulators</i>											
25	Pleura	M	1874	1888	53	1935	Necropsy	Negative	Negative	12	Worked in close proximity to boiler repainter in dockyards
28	Pleura	M	1893	1910	22	1935	Biopsy	N/A	N/A	24	Boiler cleaner and stopper, dockyards
22	Pleura	M	1921	1937	22	1959	Necropsy	Positive	Positive	9	Boiler coverer and lugger to various firms
30	Pleura	M	1908	1922	39	1961	Biopsy	N/A	N/A	25	Erecting and fitting central heating
27	Peritoneum	M	1908	1922	36	1961	Necropsy	Positive	Positive	12	Boiler coverer and pipe insulator, contracting firm
26	Peritoneum	M	1911	1927	34	1961	Necropsy	Positive	Positive	13	Lugger and boiler coverer, various firms
31	Pleura	M	1897	1916	41	1962	Biopsy	N/A	N/A	8	Intermittently boiler lugger, ship repairs
29	Pleura	M	1912	1927	34	1962	Biopsy	N/A	N/A	10	Installation of hospital sterilizing equipment

(continued)

APPENDIX (continued)

EIGHTY-THREE PATIENTS WITH MESO-THORIC TUMOURS

Case No.	Site of Primary Tumour	Sex	Year of Birth	Year of First Exposure	Duration of Exposure (years)	Year of Death	How Diagnosed	Asbestos Bodies in Lung Tissue	Pathological Evidence of Asbestos in Lung Tissue	Survival from Initial Symptoms (months)	Relevant History
<i>Section C: Exposure of relatives</i>											
40	Pleura	M	1912	1925	11	1956	Biopsy	N/A	N/A	27	Wife sister worked as spinner at asbestos factory, died 1946, carried asbestos.
34	Pleura	F	1913	1919	2	1937	Biopsy	N/A	N/A	14	Wife sister employed as spinner, looked after patient as child.
38	Pleura	F	1895	1919	41	1960	Biopsy	N/A	N/A	15	For several years husband worked in ship's engine room, close proximity to lagging operations.
37	Pleura	F	1905	1925	14	1960	Biopsy	N/A	N/A	3	Husband worked as boiler coverer; overalls brought home for washing.
35	Peritoneum	F	1890	1921	21	1960	Necropsy	Positive	Negative	24	Husband foreman and executive of asbestos factory.
33	Pleura	F	1916	1930	4	1961	Necropsy	Negative	Negative	*	Husband a docker; frequently handled "white" asbestos.
39	Pleura	M	1919	1928	2	1961	Biopsy	N/A	N/A	16	Wife sister worked as spinner at asbestos factory and suffers from asbestosis.
32	Pleura	F	1900	1941	5	1963	Biopsy	N/A	N/A	18	Daughter worked in asbestos factory for 5 years, patient washed her overalls.
36	Peritoneum	F	1889	1912	18	1963	Biopsy	N/A	N/A	15	Husband railway carriage builder; lined compartments with asbestos sheeting, work clothes washed at home.
<i>Section D: Neighbourhood cases</i>											
44	Pleura	F	1903	1913	7	1959	Biopsy	N/A	N/A	8	Lived within 1 mile of present site of asbestos factory.
48	Pleura	M	1922	1922	16	1960	Biopsy	N/A	N/A	17	Lived within 1 mile of present site of asbestos factory.
42	Pleura	F	1889	1913	48	1961	Biopsy	Positive	Negative	15	Lived within 100 yards of present site of asbestos factory.
50	Pleura	F	1903	1908	6	1961	Biopsy	N/A	N/A	13	Lived within 1 mile of old site of asbestos factory.
43	Pleura	F	1905	1913	13	1961	Biopsy	N/A	N/A	4	Lived within 1 mile of present site of asbestos factory.
47	Pleura	F	1917	1917	10	1961	Biopsy	N/A	N/A	16	Lived within 200 yards of present site of asbestos factory.
64	Pleura	F	1899	1907	6	1962	Biopsy	Negative	Negative	5	Lived within 1 mile of old site of asbestos factory.
71	Pleura	F	1899	1907	6	1962	Biopsy	N/A	N/A	12	Lived within 1 mile of old site of asbestos factory.
41	Pleura	F	1900	1913	32	1962	Biopsy	N/A	N/A	29	Lived within 1 mile of present site of asbestos factory.
45	Pleura	F	1900	1913	7	1962	Biopsy	N/A	N/A	9	Lived within 1 mile of present site of asbestos factory.
46	Pleura	F	1907	1913	7	1963	Biopsy	N/A	N/A	7	Lived within 1 mile of present site of asbestos factory.
<i>Section E: No history of contact with asbestos</i>											
65	Pleura	F	1900			1947	Necropsy	Positive	Negative	7	Worked as nursemaid to manager of gas works in East London, asbestos insulation used in works.
68	Peritoneum	F	1902			1954	Necropsy	Negative	Negative	5	Shortland-typist and housewife, lived Hford area.
49	Pleura	M	1895			1954	Biopsy	N/A	N/A	7	Baker's roundman, army, general labourer, publican for 30 years in East End of London.
75	Pleura	F	1899			1955	Biopsy	N/A	N/A	7	Munitions work before marriage, then housewife, lived Waltham Abbey, Essex.
74	Peritoneum	F	1904			1955	Necropsy	Negative	Negative	27	Tailress and housewife in East End of London.
62	Pleura	M	1899			1955	Necropsy	Negative	Positive	28	Employed as lateman and greaser, Beckton gas works 36 years; persistent discharging sinus R. side of chest following empyema 1925, acute chest infection 1953, subsequent thoracoplasty but deteriorated from this date.

(continued)

MESOTHELIOMA OF PLEURA FOLLOWING EXPOSURE TO ASBESTOS

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APPENDIX (continued)

EIGHTY-THREE PATIENTS WITH MESOTHELIAL TUMOURS

Case No.	Site of Primary Tumour	Sex	Year of Birth	Year of First Exposure	Duration of Exposure (years)	Year of Death	How Diagnosed	Asbestos Bodies in Lung Tissue	Pathology, i.e. Evidence of Asbestosis in Lung Tissue	Survival from Initial Symptoms (months)	Relevant History
61	Peritoneum	M	1913			1955	Biopsy	N/A	N/A	8	Merchant seaman, cargoes from S. Africa 1910-32, no definite evidence of contact with asbestos, subsequently employed on cross-channel boats
54	Pleura	M	1904			1956	Biopsy	N/A	N/A	12	Labourer and builder, finally employed in tunnelling; lived in East End of London
76	Pleura	F	1916			1957	Biopsy	N/A	N/A	19	Employed packing old sacks in Bow, previous current of sacks not ascertained; lived East End of London
58	Pleura	M	1891			1957	Biopsy	N/A	N/A	11	Garage hand, chauffeur, mechanic, lived Wolverhampton and Epping
66	Pleura	F	1896			1958	Biopsy	N/A	N/A	6	Worked as printing machinist before marriage, then as housewife until death; lived East End of London
57	Peritoneum	M	1879			1958	Biopsy	N/A	N/A	3	Stenographer and printer; lived originally in East London, then moved to Ipswich
52	Pleura	M	1890			1959	Biopsy	N/A	N/A	12	Van boy, regular army, cutter and grinder; lived N.E. London and Dagenham
63	Pleura	M	1904			1960	Biopsy	N/A	N/A	18	Bricklayer, cement worker, postman; lived East End of London, moved to Hainault, Essex in 1931
51	Pleura	M	1910			1960	Necropsy	Negative	Negative	18	Filter and turner; lived principally in East End of London
59	Pleura	M	1911			1960	Necropsy	Positive	Positive	+	Coloured S. African merchant seaman, stoker at glass factory, possible contact with asbestos in S. Africa or at sea
73	Pleura	F	1887			1962	Necropsy	Negative	Negative	10	Lived until marriage in Wales, then housekeeper in S.W. London
70	Pleura	F	1888			1962	Biopsy	Negative	Negative	30	Machinist of celluloid collars, housewife and canteen worker; lived East London
72	Peritoneum	F	1898			1962	Biopsy	N/A	N/A	10	Machinist in hosiery trade, Nottingham, housewife, moved to London 1950
55	Pleura	M	1888			1962	Necropsy	Negative	Negative	2	Labourer, merchant seaman, docker, carried only meat
60	Peritoneum	M	1915			1962	Necropsy	Negative	Negative	54	Apprentice printer, hairman, cook, and butcher. For 25 years before death lived East End of London
67	Pleura	F	1897			1963	Biopsy	N/A	N/A	29	Dressmaker and housewife, lived East End of London then Somerset
56	Pleura	M	1912			1963	Biopsy	N/A	N/A	24	Steel erector, army, steel erector, labourer and porter; lived East End of London
69	Peritoneum	F	1901			1964	Necropsy	Negative	Negative	42	Employed as packer in pharmaceutical firm for 12 years, then housewife until death; lived in Dagenham
53	Pleura	M	1892			1964	Necropsy	Positive	Negative	24	Fisherman, fireman, docker, 1915-39 at Tilbury docks, fireman and finally stoker, contact with asbestos not probable at Tilbury docks; lived Tilbury area
Section F: Untraced cases											
78	Pleura	F	1893			1917	Necropsy	?	Positive		Not traced
83	Pleura	M	1860			1922	Necropsy	Negative	Negative		Not traced
81	Pleura	M	1877			1925	Necropsy	Negative	Negative		Not traced
80	Pleura	M	1900			1936	Necropsy	Negative	Negative		Not traced
82	Pleura	M	1893			1938	Necropsy	Positive	Positive		Not traced
77	Peritoneum	F	1910			1945	Necropsy	Positive	Positive		Not traced
79	Pleura	F	1878			1955	Necropsy	Positive	Positive		Not traced

N/A = tissue not available.

Mortality in the chrysotile producing industry of Quebec: A progress report

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Some 30,000 persons have been employed in the Quebec asbestos mines and production mills since their inception at the end of the last century and work records in fair detail have generally been preserved. A cohort was selected for study of mortality comprising all those born between 1891 and 1920 (11,788 in number) who were employed before November 1966 for one calendar month or more. The methods used for the inquiry and mortality findings to that date have been reported (1). A cumulative index of dust exposure was calculated for each man using the observed or estimated dust levels for each job throughout the period and the time worked in each (2). Efforts were made to trace all ex-employees and the certified cause of death was sought for those who had died. The tracing has been continued and we intend to maintain this for some years.

This interim report concerns mortality to 31 December 1969. The size of the original task of tracing 9,304 ex-employees, some employed as much as 50 years previously, had meant that although deaths were fully investigated and certificates obtained in 90%, it was impossible to corroborate thoroughly all reports about surviving persons. In this second period, tracing was easier and more effort could be put into checking and obtaining correct addresses. This has enabled stricter criteria of survival to be

applied. For example, in the earlier tracing period it was assumed that 598 men who were registered within the previous two years in the Central Index of the Unemployment Insurance Commission and whose death had not been reported to the Index, were still alive. No such assumptions have been made in the present follow-up.

As employees whose first names had not been recorded could not be identified with certainty we decided to exclude 50 persons in this category. In the previous analysis every effort was made to bring together the records of men employed in more than one company, but a further 166 matchings have since been made in which a current and in general long-term employee had had an earlier and usually short period of employment recorded elsewhere. These exclusions reduced the total number of persons in the cohort to 11,572.

Despite stricter criteria the proportion traced to the end of 1969 (87.5%) was similar to that obtained to November 1966 (88.4%). A high rate of follow-up in persons who worked for 10 or more years in the industry (99%) and in persons in the highest 3 of the 6 dust exposure groups used (98%) give assurance that risks of exposure in these groups have not been underestimated. In all, 3,270 (32%) of the 10,120 persons traced (Table 1) had died before January 1970. Death certificates were obtained for 2,950 (90%) and reliable information on the cause of death in a further 104 (3%). In 216 (7%) the cause of death has not so far been found; 51 occurred before death registration in Quebec was compulsory in 1976, but efforts are being continued to find death certificates for the other 165. Causes of death were coded according to the 7th Revision of the W.H.O. International Classification of Diseases. Equivalent average death rates were calculated in 5-year cohorts by date of

birth in 6 dust-index groups and 4 groups by duration of employment. This method of age standardization uses equal numbers in each cohort (3).

In the previous report (1), based on the 2,413 male deaths that occurred before 1 November 1966, the age-standardized rate in the highest dust category, comprising 5% of the cohort, was 20% higher than in the other groups. Respiratory, cardiovascular and malignant diseases in equal proportions accounted for the excess. Much of the respiratory excess was ascribed to pneumoconiosis and respiratory cancer accounted for most of the excess in malignant disease. The difference in rates of respiratory cancer for those maximally and minimally exposed was five-fold and, although perhaps an overestimate, was apparently determined by both accumulated dust exposure and duration of employment.

Findings to 1 January 1970

The number of men whose death was certified as cancer of the trachea, bronchus or lung (I.C.D. 162-3) to 1 November 1966 was 94. If these two were stated to be mesotheliomas. One additional malignant mesothelioma had been coded as a benign tumour. Thus in all there were 95 male deaths from respiratory cancer including 3 from mesothelioma. Thirty-seven additional certified cancers of the lung or bronchus were found up to the end of 1969 and 2 cases of mesothelioma giving a further 39 deaths from respiratory cancer. These cases will be verified by seeking pathological information and a study of all deaths in which a post-mortem examination has been performed is presently being made. In this report the cases studied are as certified.

In Table 2, standardized mortality rates to November 1966 are compared with those based on the 804 male deaths which have occurred since then. The trends are very similar in the two periods in all categories except circulatory disease which showed a decline rather than an increase with rising dust index. In particular, the relationship between exposure and respiratory cancer, and all respiratory disease was unchanged.

There were 465 women in the cohort; 428 (92.0%) were traced, of these 54 (12.5%) had died. The certified causes of death were tuberculosis 11, malignant diseases 13, circulatory diseases 13, respiratory diseases 4, trauma 1, other causes 11 and cause unknown 1. Only one death was ascribed to lung cancer and none to pneumoconiosis. However, only 79 women had worked for more than 10 years and only 2 were in the 2 highest exposure groups.

Total male mortality up to the end of 1969 incorporating the new data is shown in Table 3. Rates for all causes show a falling trend in the first 4 dust index groups, which is reversed in the highest dust group. Nearly half of this excess was accounted for by respiratory disease and nearly half of the respiratory disease was pneumoconiosis which was concentrated in the highest dust group. Malignant disease accounted for about a third of the excess and circulatory diseases one sixth. It was previously shown by a parametric analysis ⁽¹⁾ that respiratory cancer depended on both years of exposure and cumulative dust index. This type of analysis has not yet been repeated but the figures in Table 4 using equivalent average rates give smoother trends with both indices.

Conclusions

Data for the further three years, 1967-1969, confirm independently the findings of our first survey which in turn were similar to those of Kogan et al for the chrysotile-producing industry of the USSR (4) and of Vigliani and his colleagues in Italy (5). The consistency of this combined evidence adds weight to the conclusion that even heavy exposure in the mining and milling of this type of asbestos carries only a modest risk of lung cancer and less still of malignant mesothelioma. The extent to which lung cancer is determined also by smoking habits and mesothelioma by other environmental factors requires investigation.

These findings for men and women exposed to a single fibre type under relatively well-defined conditions contrasts sharply with the much higher rates of malignancy, particularly of mesothelioma, experienced by the cohort of American insulation workers studied by Selikoff et al (6) and of London crocidolite factory workers studied by Newhouse and Wagner (7,8). The explanation of this difference, whether due to fibre type as suggested by the work of Trimbell (9), or to some unidentified factor, is urgently needed.

Definition of a safe level for chrysotile production workers cannot be based on mortality alone and involves also questions of what risk is acceptable and the certainty that is required. Tables 3 and 4 suggest to us that any increased risk of death from respiratory cancer or pneumoconiosis at dust-index levels below 200 would not be detectable and still in doubt below 400. To accumulate a dust index of 200 a man could work for 40 years at an average dust concentration of 5 million particles per cubic foot. Assuming the

fibre content of dust to be about 10%, this is equivalent to about 12 fibres per ml. But to prevent less serious degrees of asbestosis much stricter standards would be required.

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Table 1. Subjects of study and results of tracing by age

Year of birth	Persons			Traced			% traced	Dead	% of persons traced
	M	F	Total	M	F	Total			
1891-95	1,535	14	1,549	1,191	13	1,204	<u>77.7</u>	788	<u>65.4</u>
1896-00	1,973	31	2,004	1,583	26	1,609	<u>80.2</u>	867	<u>53.9</u>
1901-05	1,999	51	2,050	1,692	45	1,737	<u>84.7</u>	684	<u>39.4</u>
1906-10	1,851	98	1,949	1,685	86	1,771	<u>90.9</u>	437	<u>24.7</u>
1911-15	1,783	117	1,900	1,674	111	1,785	<u>93.9</u>	296	<u>16.6</u>
1916-20	1,966	154	2,120	1,867	147	2,014	<u>95.0</u>	198	<u>9.8</u>
ALL	11,107	465	11,572	9,692	428	10,120	<u>87.5</u>	3,270	<u>32.3</u>

Table 2. Equivalent average death rates per 1,000 men by cause and dust index in previous and new follow-up

	Before 1 November 1966			From 1 November 1966 and before 1 January 1970		
	<u>Dust Index</u>			<u>Dust Index</u>		
	<u>100</u>	<u>100 -</u>	<u>400 -</u>	<u>100</u>	<u>100 -</u>	<u>400 -</u>
Number of men	6,414	2,150	1,417	6,139	2,131	1,422
Number of deaths	1,595	442	376	549	156	98
Tuberculosis (I.C.D. 001-019)	19.3	27.9	28.1	5.0	3.5	1.3
All malignant neoplasms (I.C.D. 140-209)	38.8	29.7	51.2	19.3	18.3	20.1
Gastro-intestinal (I.C.D. 150-4)	10.8	7.7	18.7	4.4	7.5	7.8
Bronchus trachea & lung* (I.C.D. 162-3)	7.9	10.0	17.5	3.4	4.5	6.5
Other	19.9	11.9	13.2	11.5	6.2	6.2
All circulatory diseases (I.C.D. 400-469)	82.7	75.5	101.8	36.1	42.5	26.8
All respiratory diseases (I.C.D. 470-529)	12.1	18.1	28.5	3.8	6.2	7.1
Trauma (I.C.D. 800-999)	32.2	36.0	25.3	9.0	5.4	2.4
Other causes	49.4	43.2	48.1	14.1	13.9	14.8
Unknown causes	26.3	11.2	5.3	3.7	4.7	2.5
All causes	261.3	242.2	288.2	91.3	94.2	73.1

* including malignant mesothelial tumours

Table 3. Equivalent average death rates to 1 January 1970 per 1,000 men by dust index

	<u>Dust Index</u>						
	<u>10</u>	<u>10 -</u>	<u>100 -</u>	<u>200 -</u>	<u>400 -</u>	<u>800 -</u>	<u>All</u>
Number of men	2,810	3,329	1,124	1,007	837	585	9,692
Tuberculosis (I.C.D. 001-019)	24.7	25.9	37.7	25.1	28.6	28.5	27.0
All malignant neoplasms (I.C.D. 140-209)	57.9	60.8	53.8	40.6	67.1	79.3	58.0
Gastro-intestinal (I.C.D. 150-4)	<u>18.0</u>	<u>13.6</u>	<u>10.7</u>	<u>11.6</u>	<u>26.3</u>	<u>28.7</u>	<u>17.0</u>
Bronchus, trachea & lung* (I.C.D. 162-3)	<u>10.3</u>	<u>13.1</u>	<u>13.4</u>	<u>15.5</u>	<u>21.4</u>	<u>32.1</u>	<u>14.2</u>
All circulatory diseases (I.C.D. 400-469)	122.6	118.8	117.8	116.4	118.3	134.8	121.5
All respiratory diseases (I.C.D. 470-529)	13.6	19.2	24.9	24.0	21.0	48.9	20.5
Pneumoconiosis (I.C.D. 523-4)	<u>1.6</u>	<u>1.5</u>	<u>0.8</u>	<u>4.9</u>	<u>4.9</u>	<u>23.6</u>	<u>3.5</u>
Trauma (I.C.D. 800-999)	48.6	38.1	41.3	41.1	33.7	18.6	39.5
Other causes	62.8	66.6	59.0	55.3	48.8	77.9	63.1
Unknown causes	34.7	25.6	19.4	10.8	5.5	6.6	24.0
All causes	365.0	354.9	353.9	313.2	323.1	394.6	353.7

* Including malignant mesothelial tumours

Table 4. Equivalent average death rates per 1,000 men to 1 January 1970 from respiratory cancer*

<u>Years of employment</u>	<u>Dust Index</u>						
	<u>10</u>	<u>10 -</u>	<u>100 -</u>	<u>200 -</u>	<u>400 -</u>	<u>800 -</u>	<u>All</u>
1	8.2 (20)	5.2 (2)	23.8 (1)	0.0 (0)	0.0 (0)	0.0 (0)	8.0 (23)
1 -	17.3 (12)	13.9 (22)	14.0 (5) <u>1</u>	10.7 (2)	7.6 (1)	0.0 (0)	13.8 (42)
10 -	0.0 (0)	17.7 (13) <u>2</u>	7.8 (4)	11.3 (5)	20.6 (8)	25.3 (7)	17.8 (37)
30 -	0.0 (0)	15.5 (4)	18.0 (3)	24.1 (7) <u>1</u>	19.0 (6) <u>1</u>	38.5 (12)	24.7 (32)
	10.3 (32)	13.1 (41)	13.4 (13)	15.5 (14)	21.4 (15)	32.1 (19)	14.2 (134)

* 129 cancers of the trachea bronchus and lung and 5 malignant mesothelial tumours. Total respiratory cancers are shown in parenthesis and the 5 mesothelial tumours in italics.

Mortality in the Chrysotile Asbestos Mines and Mills of Quebec

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Of 11,788 persons born between 1891 and 1920 employed in the Quebec asbestos mining industry, 88.4% were traced. Of these 2,457 (23.6%) had died. Exposure indexes for each worker were calculated from job dust levels and duration of employment. The overall mortality was lower than expected for the population of Quebec but in the highest dust category, comprising 5% of the cohort, the age-standardized rate was 20% higher than in the other groups. Respiratory, cardiovascular, and malignant disease in equal proportions accounted for the excess. There were 101 deaths from respiratory cancer including three from malignant mesothelioma, an estimated excess of about 15 deaths. The difference in rates for respiratory cancer between those maximally and minimally exposed was fivefold and, though perhaps exaggerated, was apparently determined by accumulated dust exposure and duration of employment.

THE REMARKABLE qualities of the asbestos group of fibrous minerals have been recognized since antiquity, but mining and milling on an industrial scale began only at the end of the 19th century. In the Eastern Townships region of Quebec, deposits of chrysotile asbestos in serpentine rock were

noted in the 1847 Canadian Geological Survey. The first mine was opened at Thetford in 1878, and within 30 years the region was producing most of the world's asbestos. The proportion fell as Russian, South African, and Italian mines came into operation, but Quebec still produces about 46% of the world's supply, now estimated at about 4 million tons a year.¹

There are two main mining areas, one at Thetford Mines and neighboring towns of Black Lake and Broughton, and the other at Asbestos. The Thetford area was developed by many different companies, but with amalgamation the number has now been reduced to six. At Asbestos, the mining has been carried out since 1882 by one large company which also operates a small factory in the town for the manufacture of mixed asbestos products. There is a small mine owned by another company a few miles away.

Concern for the health effects of asbestos has paralleled growth in its production, and the main available evidence was reviewed fully by Wright² in 1969. The first cases of diffuse pulmonary fibrosis after prolonged exposure were noted by Murray in 1907, and by 1930 asbestosis was recognized as an important occupational hazard. Controls introduced since then have reduced considerably the dust concentration to which workers in the mines, mills, and primary manufac-

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Table 1.—Subjects of Study and Results of Tracing by Age and Sex

Year of Birth	Persons			Traced			% Traced	Dead	% of Persons Traced
	M	F	Total	M	F	Total			
1891-1895	1,553	14	1,567	1,179	13	1,192	76.1	611	51.3
1896-1900	1,962	31	2,013	1,588	28	1,616	80.3	655	40.5
1901-1905	2,027	50	2,077	1,724	45	1,769	85.7	498	28.2
1906-1910	1,897	99	1,996	1,741	92	1,833	91.8	130	18.0
1911-1915	1,837	117	1,954	1,773	113	1,886	96.5	221	11.7
1916-1920	2,027	154	2,181	1,976	149	2,125	97.4	142	6.7
All	11,323	465	11,788	9,981	440	10,421	88.4	2,457	23.6

Table 2.—Subjects of Study and Results of Tracing by Dust Index and Years of Employment

Years	Dust Index							All
	< 10	10	100	200	400	800		
< 1	No. 3,043	654	38	3	0	0		3,738
	% traced 76.6	75.1	76.3	66.7				76.3
1	No. 1,025	1,975	450	264	127	64		3,900
	% traced 86.3	88.6	89.3	90.5	91.0	90.6		88.3
10	No. 55	1,117	566	522	437	255		2,947
	% traced 100.0	99.0	98.9	99.0	99.3	99.6		99.1
30	No. 0	204	181	250	303	265		1,203
	% traced	100.0	100.0	100.0	100.0	99.6		99.9
All	No. 4,123	3,950	1,235	1,039	857	584		11,788
	% traced 79.9	89.9	94.9	97.0	98.4	98.6		88.4

There was of course some movement of labor from one mining company to another. This necessitated bringing together and matching the records of all the companies, to obtain complete work histories. In the course of tracing ex-employees, the matching process could often be confirmed or amended. In all, 27,669 men and women were registered, including 6,415 currently employed on Nov. 1, 1966. Of this total, 1,039 persons had been employed by more than one company.

Using the occupational histories, a list was compiled of all named jobs in each company from the beginning of its operation. A description of each of the 13,346 jobs listed in this way was obtained from existing evaluation systems and by interviewing employees with long service. Jobs with several names but involving the same work and dust exposure were combined and the codes finally needed for classification thus reduced to 5,783. For each of these and for each year of operation, the average dust exposure was estimated on a 13-point scale. Throughout the industry in the dryers, crushers, and mills, the concentrations of dust at working places had been measured annually since 1949 by Maurice Lachance, Eng. and since 1946 by various investigators in the small factory. All measurements were made with either a midjet impinger or Greenburg-Smith impinger and recorded in millions of dust parti-

cles per cubic foot (mpcf). Levels of exposure in mining and maintenance jobs and in other operations where no dust measurements had been made were estimated by investigating present-day dust levels and adjusting these according to changes in operating procedure reported by mining and maintenance personnel. A full description of geological and environmental features and of the methods and results of dust measurement are to be published by G. W. Gibbs, MSc, and Mr. Lachance.

Before 1949, few dust measurements were recorded, and estimates of exposure for that period are, therefore, only approximate. Interviews with employees of long service enabled comparisons of dustiness to be made with conditions since 1949. The dates of installation or modification of dust control systems were also taken into account.

Duration of exposure for each person was expressed in years or fractions of years after subtraction of all periods of leave, and was corrected to a working week of 40 hours. A dust index was calculated for each employee by adding together the products of time spent on each job and estimated average dust concentration. For example, suppose the dust index for a man was 600; this might mean that he had worked for five years at 40 mpcf, 15 years at 20 mpcf, and 20 years at 5 mpcf (ie, $200 + 300 + 100$). It would be useful if it could be assumed

had been included incorrectly, 47 were confirmed pathologically, 28 hospital diagnoses were presumed correct, and in 17 no further information was obtained.

The following steps were taken to discover cases of lung cancer that had not been mentioned as such on the death certificate:

1. A letter was sent to the certifying physicians whenever it was stated that an autopsy had been or would be performed. These numbered 306 in all; 33 deaths were certified as due to lung cancer and 273, as due to other causes. In 18 replies, it was stated that there had been no autopsy; in 175, that the certified diagnosis was confirmed at autopsy; and in 18, that it was incorrect. These 18 yielded five additional cases of lung cancer. The diagnoses for these had been certified as renal tumor (ICD 180), infectious hepatitis (ICD 092), pulmonary fibrosis (ICD 519), diabetes (ICD 260), and asbestosis (ICD 523).

2. We asked the physicians in charge of the industrial clinics at Thetford Mines and Asbestos for lists of all deaths known to be due to lung cancer. Their records included only employees who had given up work because of ill health, those who were pensioned, and those who had applied for workmen's compensation. They listed 24 of the 96 lung cancer deaths found by the methods already described plus three additional cases. These three deaths had been certified as due to tuberculosis (ICD 019), asbestosis (ICD 523), and coronary heart disease (ICD 420), and though there had been autopsies for the purpose of compensation, no mention of this was made on the death certificate. In addition, there were three autopsies for compensation purposes in which a small lung cancer was noted as an incidental finding. The pathologist had not considered these cancers to have contributed to death and the causes certified were asbestosis (ICD 523) in two cases and cardiac infarction (ICD 420) in a third.

3. A search was made for any case of primary malignant mesothelial tumor of the pleura that might have been given a code other than 162. One additional case coded as a benign pleural tumor was found.

4. We reviewed cases reported by pathologists in the national survey of primary malignant mesothelial tumors in Canada between 1960 and 1968, and five were in men who had worked in the Quebec asbestos-producing industry. Two were born in 1922 and were, therefore, not in our cohort, and two died in 1967 after the present study had ended. A fifth case, diagnosed by biopsy, was eligible for both studies.

To summarize the results of all these inquiries, three cases were removed from the group of 95 malignant neoplasms of the bronchus, trachea, or lung (ICD 162 and 163) and nine were added, making 101 in all (100 men and one woman). Included in the total were the following: cancer of the trachea, in one case; malignant mesothelial tumor, in three; and cancer of the bronchus or lung, in 97.

Results

Female Mortality.—There were only 465 women in the cohort; almost all had short exposures and all but 33 were in the two lowest dust-index categories. In all, 440 (94.6%) were traced, and of those, 45 had died. Tuberculosis (11), malignant neoplasms (12), circulatory (nine) and respiratory (one) diseases, and trauma (one) were the causes of death in 34. Death was due to other causes in eight cases, and the cause of death was unknown in two; there was one death from lung cancer. Apart, perhaps, from tuberculosis, these figures are not unusual and will not be considered further.

Male Mortality.—To permit comparisons of mortality by dust index and years of exposure, death rates were calculated in five-year cohorts by date of birth. Equivalent average death rates¹³ were then calculated using a standard population with equal numbers in each age group. Since the actual number at risk in each cohort was similar, this method of age standardization was particularly appropriate. The standardized rates are shown by dust index and main disease groupings in Table 3. In the lower five dust categories, equivalent average death rates for "all causes" were approximately the same, but in the top category, containing 5% of the total cohort, mortality was about 20% higher than the rest. Malignant neoplasms, circulatory diseases, and respiratory diseases, in approximately equal proportions, accounted for the excess. Two groups showed the opposite trend, "trauma," probably because rates fell with advancing age, and "unknown causes," probably because death certificates were difficult to find for older men with short periods of employment.

Table 4 shows that the main contribution to the excess in malignant disease was from cancer of the bronchus, trachea, and lung

turing industries are exposed. Though the prevention of asbestosis is far from complete in these industries, it is still generally believed that this can be achieved by more strict environmental control. Another aspect of the problem appeared in 1947 when Merewether³ showed a link between asbestos exposure and lung cancer. The potential extent of this hazard was increased by reports of Wagner and others⁴ during the last ten years that malignant tumors of the pleura and peritoneum are related to certain types of industrial work and perhaps also to neighborhood exposure.

The association between lung cancer and asbestos was first found in the British textile industry. Merewether's inquiry showed an unduly high proportion of lung cancers at autopsy in cases of asbestosis, and other observations among textile workers have confirmed the association.^{5,6} Two studies in particular which take some account of the degree of exposure suggest a dose-response relationship. Knox et al.⁶ found a considerable excess of lung cancer in persons exposed before 1933 when asbestos control regulations were enforced, but none in persons exposed for ten years or more in the most dusty areas since then. These findings must be reviewed when a longer period of observation has elapsed, since Newhouse⁸ (also in a textile factory) found an excess of lung cancer in persons heavily exposed 20 years earlier for periods of less than two years. Workers exposed to light or moderate dust concentrations showed 1.0 excess of bronchogenic cancer even after 20 years of exposure.

Selikoff and his colleagues⁹ found an eightfold increase in lung cancer compared with national figures in a cohort study among the members of an American insulation workers union who had been exposed for 20 years or more. He also found a gross excess of pleural and peritoneal mesotheliomas. On the other hand, in a national survey of all known mesothelial tumors in Canada, 1960 to 1968, McDonald and her colleagues¹⁰ found a history of occupational contact with asbestos in a relatively small proportion of cases. This exposure was mainly in insulation and allied trades, rather than in the asbestos-producing industry.

Of the four main types of asbestos fiber—chrysotile, crocidolite, amosite, and antho-

phyllite, the two textile factories referred to above^{3,5} used mainly chrysotile fiber, together with some crocidolite, and in one of the two, some amosite. As in most industrial applications different types of fibers are mixed, the carcinogenic effects of a single fiber type in practice can only be studied in mining and milling. Chrysotile fiber is of greatest importance because of its qualities and extensive usage. The only studies of lung cancer in chrysotile miners and millers were reported by Braun and Truan in 1958¹¹ and by Kogan et al. in 1966.¹² In the first of these, nine deaths from lung cancer were observed in the Quebec industry compared with six expected from provincial rates. The second was in the Soviet Union and showed that compared with the general population the mortality from lung cancer was increased by a factor of 1.9 for miners, 3.1 for millers, and 2.3 for factory workers. The present investigation forms part of a comprehensive epidemiological survey of the entire Quebec asbestos-producing industry since its inception. Using the considerable volume of data available, our primary aim has been to define as accurately as possible the quantitative relationship between exposure to chrysotile asbestos and the incidence of lung cancer. The results from parallel studies concerned with the relationship of dust exposure to radiographic appearances, pulmonary function, and respiratory symptoms will be reported separately.

Materials and Methods

Registration.—A register was compiled in the personnel department of each asbestos mining company in the Eastern Townships region of Quebec listing all persons currently or previously employed, as of Nov 1, 1968. A card was made out for each employee on which was recorded his name, date of birth, address, and a detailed work history which included the department and mine, dates of starting and finishing, for every job, and all periods of leave.

In one of the 44 mining companies represented in our survey, the records of at least 560 persons had been destroyed. These were of ex-employees and others not transferred when ownership of the company changed in 1964. The work histories of a small number of older men who had been employed partly in this company and partly in others were also incomplete.

that an index of 600 were also equivalent to a working life of 40 years at 15 mpct, but this would entail assumptions which may or may not be justified.

Selection of Cohort.—The study of mortality was based on employees in an age group thought likely to yield the most valuable information. The cohort selected comprised 11,788 persons born between 1891 and 1920 inclusive who had been employed for one calendar month or more. Those born after 1920 would have had periods of exposure well short of a working lifetime and would still not have reached an age of high mortality. Those born before 1891 would have been very difficult to trace, and, as mentioned above, some of their work histories were incomplete or missing. The selected cohort comprised among others 1,203 persons who had worked for 30 years or more, 3,738 who had worked for less than a year, and 1,080 who had worked for more than a year but whose job had not involved any appreciable exposure to dust. Thus, there was a substantial group with minimal exposure available for comparison with others who had been heavily exposed.

Tracing and Ascertainment.—The search for ex-employees was mainly initiated in the mining towns. Telephone or postal inquiries were made first to establish whether the person in question was alive or dead on Nov. 1, 1966. If reported dead, the exact date and place was sought from relatives, friends, and parish registers. For those not traced by these means, a systematic search was made in the provincial death records and certificates, and about 200 persons were found in this way. In addition, a search on our behalf was made in the index of the Canadian Unemployment Insurance Commission for any person not found by means of local enquiries. The names of 598 persons were found who had registered or re-registered between 1964 and Nov. 1, 1966, and 30 more had re-registered after that date. All 628 were presumed to be alive for the purpose of our study.

Information was obtained concerning 10,421 (88.4%) of the 11,788 persons in the cohort: 7,965 were still alive (including 2,484 currently employed) and 2,457 (23.6% of those traced) were reported dead. Copies of death certificates were obtained from Canadian Provincial and US State Registrars for 2,211 (90%) persons. For 59 more (2%), an acceptable cause of death was obtained by other means; 30 were accidental deaths in the mines or during active service with the armed forces and 29 were caused by disease. Twenty-two of the 29 had occurred before the introduction of death registration in Quebec in 1926, and the cause of

death was stated by relatives. In the other seven, the cause of death was obtained from a reliable source. A death certificate could not be obtained for the remaining 187; 47 had occurred before 1926, and 100 were deaths outside Quebec, mostly outside Canada. The cause of death was coded according to the Seventh Revision of the International Classification of Disease (ICD) (World Health Organization, Geneva, 1957). This was done by senior coder of the Department of Demography of Quebec who had recently retired.

The proportion of subjects traced by age, sex, and category of exposure is shown in Tables 1 and 2. Success in tracing depended mainly on duration of employment: 94% of those employed for one year or more and over 99% of all those employed for ten years or more were traced. Information was least satisfactory in persons born before 1900 and in those employed for less than one year. The principal reasons for failure were (1) no relative or friend could be located in the neighborhood and (2) insufficient identifying information, which would have enabled us to make a request to the Unemployment Insurance Commission or to locate a death certificate in Quebec. There were no other obvious reasons for loss towards discovering the living or the dead, but those traced dead or alive are likely to have been long-term rather than transient residents.

Diagnosis of Lung Cancer.—To make our figures for lung cancer as accurate as possible, we investigated and reviewed all certified cases and searched also for cases not described as such on the death certificate. Of 95 deaths coded as due to lung cancer (ICD 162 and 163), one was certified as due to a hydromyoma and another as due to pulmonary fibrosis, both of which had been coded incorrectly. Inquiries about the remaining 93 cases were made from hospitals, physicians, and pathologists, and information was received in 76. In five of these it was reported that there had been an autopsy, but we were unable to obtain pathological details. In another five, though the diagnosis had been made in the hospital, no clinical or pathological record was found. Seventeen more had been certified by family physicians but we did not succeed in finding out where and how the diagnosis had been made.

In the remaining 48 cases, pathological reports were obtained: 25 at autopsy and 23 at biopsy. A diagnosis of carcinoma of the trachea was made in one, of bronchus or lung in 44, and of malignant mesothelioma of pleura in two. In one case the histology was that of a melanocarcinoma, and the pulmonary tumor was thought to be metastatic. Thus, of 95 certified cases, three

Table 3.—Equivalent Average Death Rates per 1,000 Men by Dust Index

	Dust Index						
	<10	10	100	200	400	800	All
No. of men	3,006	3,408	1,148	1,062	842	575	9,981
Tuberculosis (ICD 901-919)	18.8	19.9	31.5	33.8	28.5	25.8	27.5
Malignant neoplasms (ICD 140-209)	38.8	39.3	32.2	27.5	40.1	51.5	38.5
Circulatory diseases (ICD 400-469)	85.0	80.8	82.5	83.5	74.8	107.7	85.9
Respiratory diseases (ICD 470-529)	9.5	15.3	19.5	18.7	15.8	41.6	15.2
Trauma (ICD 800-999)	34.7	30.8	36.3	35.2	23.8	13.7	31.5
Other causes	46.8	52.9	39.0	47.7	38.3	58.9	48.4
Unknown causes	30.6	21.4	16.5	8.2	7.2	3.4	20.8
All causes	264.1	260.4	257.6	240.6	262.9	312.5	263.1

Table 4.—Equivalent Average Death Rates per 1,000 Men for Malignant Neoplasms (ICD 140-209) by Dust Index

Location	Dust Index						
	<10	10	100	200	400	800	All
Esophagus & stomach (ICD 150-151)	10.2 (32)	4.5 (13)	1.7 (2)	7.0 (5)	11.3 (8)	13.7 (8)	7.5 (68)
Intestine & rectum (ICD 152-154)	2.5 (6)	4.9 (15)	3.7 (3)	3.9 (3)	5.6 (4)	6.7 (5)	4.0 (38)
Other abdominal areas (ICD 155-159)	4.0 (13)	3.0 (9)	1.7 (1)	1.3 (1)	2.8 (2)	1.7 (1)	2.9 (27)
Bronchus, trachea & lung (ICD 162-163)	7.6 (25)	8.6 (26)	11.2 (10)	8.9 (8)	15.8 (11)	24.2 (14)	9.0 (94)
Other malignant neoplasms	14.4 (47)	18.6 (54)	14.5 (11)	6.4 (5)	9.5 (6)	13.5 (8)	14.4 (131)
All malignant neoplasms (ICD 140-209)	38.8 (125)	39.3 (117)	32.2 (23)	27.5 (22)	40.1 (31)	51.5 (36)	38.5 (358)

(ICD 162 and 163). In this group there was little difference between rates in the four lower dust categories, but the fifth and sixth groups showed an upward trend. A similar trend with years of exposure was found (Table 5), but the figures within the body of the table suggest a closer relationship with dust than with years. Subtraction of the three incorrectly coded cases and addition of the nine lung cancer cases found at autopsy increased slightly the rate among persons with highest dust and longest exposure, but did not materially change the picture (Table 5). Further analyses to distinguish better the relative importance of years of exposure and dust index are described as follows.

Rates for cancer of the intestine were about one third of those for cancers of the bronchus, trachea, and lung, but showed a very similar trend. Rates for cancer of the esophagus and stomach, on the other hand,

did not appear related to dust in any consistent manner. Other abdominal neoplasms were less frequent still and also unrelated to dust. This is important since it might be expected that unrecognized peritoneal mesotheliomas would be found within this group.

Most of the excess mortality from respiratory disease was ascribed to pneumoconiosis (Table 6). There was little evidence that dust-associated deaths were included in other respiratory categories. Of the 28 deaths coded under the pneumoconioses, one was described as anthracosis, four as silicosis, and the remaining 23 as asbestosis. The greatest excess mortality from pneumoconiosis was among persons in the highest dust group who had been employed between ten and 29 years.

In the circulatory diseases group, there was also excess mortality in the highest two dust categories, mainly in persons employed

between ten and 29 years. It was present equally in the "arteriosclerotic and degenerative heart disease" group (ICD 420 to 422 [which included more than three quarters of all circulatory deaths]) and in the group of "other circulatory diseases" (ICD 400 to 419 and 423 to 469).

Comparison With Quebec Mortality.—The number of deaths from all causes which would have been expected if Quebec death rates had applied was calculated in the following way. Age-specific death rates for the province were applied for each year, 1950 to 1966, to all the men traced who were alive in 1950, with an adjustment for those who started work after 1950. The expected number of deaths among men, thus, was 1,824, whereas the observed number in the cohort was 1,674.

A similar calculation was made for lung cancer deaths. The expected number was 91, whereas 94 male deaths certified as due to this cause were observed. Correction for coding errors and additional autopsy information was not appropriate, since certificates for the general population were uncorrected.

In the six counties of the province which include the mining region (Arthabaska, Beauce, Drummond, Megantic, Richmond, and Wolfe), the lung cancer death rate was about two-thirds the provincial rate and the expected number of deaths was correspondingly lower. However, many ex-employees were no longer living in this area when they died, and most of those who had moved had gone to cities where lung cancer death rates were higher. The best estimate of expected deaths probably lies between the numbers 61, derived from the mining region, and 91, from the province. The excess of observed over expected lung cancer deaths, therefore, lies somewhere between zero and 30 and is most probably between ten and 20.

Factor Evaluation.—Although the numbers of men in each cohort were about the same, their distribution by years of employment and dust index was uneven. In these circumstances, equivalent average death rates could be misleading. Berry¹⁴ recently reviewed some of the methods used for analyzing the importance of factors in multiway tables and described a parametric approach which has the advantage that the adequacy of the model may be checked and statistical

Table 5.—Equivalent Average Death Rates per 1,000 Men for Cancer of Bronchus, Trachea, and Lung*

Dust Index							
Years	<10	10	100	200	400	800	All
As certified							
1	8.8 (16)	1.7 (1)	27.8 (11)	0.0 (0)			5.9 (18)
2	11.2 (9)	8.5 (15)	11.3 (8)	5.8 (3)	7.6 (11)	0.0 (0)	9.6 (30)
10	0.0 (0)	12.4 (20)	6.7 (3)	5.3 (3)	19.9 (16)	28.6 (16)	13.1 (25)
30		1.7 (3)	24.7 (25)	18.9 (4)	16.1 (4)	27.9 (8)	17.2 (21)
All	7.6 (25)	8.6 (28)	11.2 (10)	8.9 (8)	15.8 (11)	24.2 (14)	9.9 (194)
Using all available information							
1	6.7 (17)	1.7 (1)	0.0 (0)	0.0 (0)			5.9 (18)
2	12.0 (8)	10.1 (16)	9.4 (3)	11.5 (2)	7.6 (11)	0.0 (0)	10.1 (31)
10	0.0 (0)	12.8 (18)	7.6 (4)	9.2 (4)	19.9 (16)	28.6 (16)	14.3 (28)
30		12.5 (3)	14.7 (21)	18.9 (4)	18.1 (14)	35.9 (10)	19.1 (23)
All	7.8 (26)	9.1 (28)	10.1 (10)	10.6 (10)	15.8 (11)	27.7 (16)	10.6 (100)

* By dust index and years of employment. Diagnosis was ICD 162 and 163.

significance evaluated. From the number of deaths from any particular cause in groups subdivided by date of birth and year of employment or dust index, expected rates may be calculated assuming no interaction between age and the other two factors. For this analysis, the complementary log log transformation was used.¹⁵ This transformation is appropriate when increasing exposure is associated with proportional increases in the age-specific death rates.

The results of this parametric analysis for respiratory cancer (Table 7) agree remarkably closely with the equivalent average death rates shown in the lower half of Table 5. The rates for the first five dust index levels do not differ significantly but those for the first four are significantly different from the highest exposure group. There are, however, also differences between the rates by years of employment, those for the lower two categories being significantly less than that for men with the longest exposure.

The general fit of the complementary log log model is very good. In no cell of the 24 does the observed number of deaths differ significantly from expectation. Tests for interaction between the 24 cells and the six cohorts gave the following results: likelihood

Table 6.—Equivalent Average Death Rates per 1,000 Men for Respiratory Diseases (ICD 470-529) by Dust Index and Years of Employment

Years	Dust Index						
	<10	10	100	200	400	800	All
Pneumonia and bronchitis (ICD 490-509)							
<1	2.6(7)	8.7(4)	0 (0)	166.7(1)			4.0(12)
1-	7.1(15)	3.7(6)	2.5(1)	4.4(1)	15.5(7)	18.7(1)	5.2(16)
10-	0	6.3(4)	5.9(2)	5.3(1)	5.6(3)	9.5(2)	6.3(14)
30-		0	4.9(1)	5.8(1)	0 (0)	3.4(1)	2.9(3)
All	3.8(17)	4.7(14)	4.2(4)	6.6(4)	5.6(5)	8.0(4)	4.9(45)
Pneumoconiosis (ICD 523 and 524)							
<1	1.4(3)	4.5(2)	0	0			1.9(5)
1-	1.4(1)	1.5(2)	0	5.9(1)		0	1.5(4)
10-	0	2.3(1)	0	1.2(1)	1.9(1)	34.3(8)	6.3(11)
30-		0	0	3.6(1)	7.1(2)	17.9(5)	6.9(8)
All	1.5(4)	2.0(5)	0	3.7(4)	3.0(3)	22.6(13)	3.2(28)
Other respiratory diseases (ICD 470-489, 510-522, 525-529)							
<1	4.5(10)	5.7(3)	0	0	0	0	4.6(13)
1-	3.8(3)	13.2(20)	29.0(9)	9.3(2)	7.9(1)	13.9(1)	12.5(26)
10-	0	2.7(2)	9.2(2)	7.9(2)	11.0(1)	7.8(2)	7.3(11)
30-		0	0	0	0	13.5(4)	3.6(4)
All	4.7(13)	6.6(25)	15.5(11)	6.3(4)	5.9(4)	12.1(2)	7.5(64)
All respiratory diseases (ICD 470-529)							
<1	8.6(20)	18.8(9)	0	166.7(1)			10.5(30)
1-	12.2(9)	18.4(28)	31.5(10)	20.0(4)	23.5(3)	30.6(2)	19.2(56)
10-	0	11.2(7)	15.1(4)	14.4(6)	18.2(2)	51.6(12)	19.9(36)
30-		0	4.9(1)	9.3(2)	7.1(2)	34.7(10)	13.4(18)
All	9.5(29)	15.3(44)	19.5(15)	18.7(13)	15.3(12)	41.6(28)	15.6(137)

ratio test, 88.9 and χ^2 , 90.4, each with 95 degrees of freedom; and $P > 0.5$.

Comment

At face value, the findings suggest that our cohort of workers in the chrysotile mining industry had a lower mortality than the population of Quebec of the same age. This is generally true of employed persons, provided they are not subjected to an occupational hazard sufficient to offset the considerable selective advantage of being and remaining fit for work. This advantage was clearly lost by the men in the highest dust-index category whose standardized mortality was about 20% above that of the rest. Two thirds of the excess mortality in this group was probably due to pulmonary fibrosis, shown on the death certificate as either asbestosis or in the guise of various respiratory or cardiovascular diagnoses, and the remaining third to cancer, mainly of the respiratory tract.

The high rate of lung cancer in men heavily exposed to asbestos might be explained if such men also tended to smoke more heavily than others. We have no direct evidence on this point for ex-employees, since information on smoking was not rou-

Table 7.—Age-Corrected Death Rates per 1,000 Men for Cancer of the Bronchus, Trachea, and Lung by Dust Index and Years of Employment*

Years	Dust Index						
	<10	10	100	200	400	800	All
<1	6.3	2.0	0.0	0.0			5.5
1-	11.4	9.4	8.1	8.3	9.6	0.0	9.5
10-	0.0	10.4	9.8	10.3	16.8	23.4	12.8
30-		13.3	9.9	14.6	13.7	34.4	17.9
All	7.5	8.7	8.9	11.0	14.6	26.3	10.0

* A complementary log-log model was used, and diagnosis was based on all available information.

tinely recorded by the companies or the medical clinics. However, in a survey of a selected random sample of over 1,000 current employees there was little or no relationship, after allowing for age, between smoking habits and either dust exposure or duration of employment.

Our attempts to assess separately the importance of cumulative dust exposure and duration of exposure in relation to lung cancer are capable of more than one interpretation. As shown in Table 5, there is a fivefold difference between the mortality for those with the lowest amount and duration of exposure, 6.7, and that for those with the highest, 35.3. This is confirmed by the parametric analysis (Table 7) which further suggests that accumulated dust exposure and

Table 8.—Comparison of Mesothelial Tumor Findings in Two Previous Studies and the Present Study

	Selikoff et al. ¹⁶	Newhouse ¹⁷	Present Study
No. of men studied	632	4 826	9 981
No. of deaths			
All causes	880	436	2 418
Lung cancer	72	42	97
Mesothelial tumors	22	25	5

duration of employment are about equally important in determining the difference. Unfortunately, there are possible sources of bias and error which may have contributed to both these effects.

In Table 5, the rates for men with less than one year of employment seem remarkably low; 18 deaths were observed, whereas 29 would have been expected on the basis of Quebec rates. It was in this group that tracing was least satisfactory (Table 2). Failure to ascertain even a small number of lung cancer deaths would have made an appreciable difference in the rates. Short-term employees who were traced tended to be those who had stayed in the neighborhoods where, in any case, lung cancer rates were much lower than in the urban areas of the province and elsewhere, to which those untraced may well have gone. Whatever the explanation, it is difficult to accept, without reserve, the low rate of lung cancer in this group.

A second source of error is related to the ascertainment of lung cancer as a cause of death. It is generally believed that this diagnosis is greatly aided by postmortem examination. In our study, 34% of the cases before correction and 40% after correction had had an autopsy, compared with 12% of all deaths. This might not matter if autopsies were evenly distributed in relation to exposure, but this was not so. The autopsy rates in ascending order of dust-index group were 11%, 11%, 9%, 10%, 16%, and 22%, and by years of employment, 12%, 9%, 17%, and 21%. This trend is also likely to have exaggerated the difference in lung cancer rates in relation to exposure, but by how much it is impossible to say.

Taking all these considerations into account we are inclined to conclude that the true difference between those maximally and minimally exposed may well be closer to threefold than fivefold and that this is part-

ly dust-related and partly time-related. We propose in future analyses, when a longer period of observation will have yielded more data, to use an exposure index based on the concept of amount of dust inhaled and the time that it remains in the lung. Our findings so far appear compatible with such a model.

In the Canadian survey mentioned earlier,¹⁸ primary malignant mesothelial tumors were rarely associated with chrysotile asbestos production in Quebec and the present survey bears this out. Three deaths from this cause were found among nearly 2,500 deaths from all causes in the cohort. This is probably more than would be expected in a comparable number of deaths in the general population, but quite out of line with the findings of Selikoff et al.¹⁶ in insulation workers and those of Newhouse¹⁷ in a London asbestos factory. The magnitude of the difference can be inferred from the figures in Table 8. Though these figures are not entirely comparable, because of age, methods of ascertainment, or other factors, they suggest that the results of Newhouse¹⁷ are similar to those of Selikoff et al.¹⁶ for mesothelial tumors and, perhaps, lung cancer. It is clear that the Quebec chrysotile workers have had nothing like the experiences of the American insulation workers or the London factory workers with respect to malignant mesothelioma, and it seems unlikely that they are compatible with respect to lung cancer. These findings strongly suggest either that chrysotile is less likely to cause malignant disease of the lung and pleura than other forms of asbestos, such as crocidolite, or that workers engaged in insulation and processing are exposed to additional factors which explain the difference.

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Paul M. Cantor, MD, medical director of the

Thefted Industrial Clinic, and T. Roger Grainger, MD, medical director of the Canadian Johns-Manville Company assisted and cooperated in this study. Members of unions and management personnel of the Quebec Asbestos Mining Industry gave their support. The Department of Demography, Quebec, and other departments of vital statistics helped us

obtain death certificates. The Department of National Health and Welfare, Ottawa, and the Canadian Unemployment Insurance Commission assisted in the follow-up. Juliette Roy, Fernande Proulx, and Joseph Albert Carrier, and others traced ex-employees, and Geraldine Jacobs and Jean Penabody helped with the data processing.

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Dr. F. L. Pundsack

Presentation to Illinois Pollution Control Board

October 15, 1971

My name is Dr. Fred L. Pundsack, and I am Vice President of Research and Development for the Johns-Manville Corporation. I am an inorganic chemist with a Ph.D. degree from the University of Illinois, and I have been involved in asbestos research since 1952. I am here today representing the Asbestos Information Association, an organization sponsored by nine leading manufacturers of asbestos products to provide information on asbestos and its health implications. The members of this Association are listed in a fact sheet which I have provided.

In the event that my presentation prompts questions relating to environmental control technology or the medical aspects of the asbestos situation, I have asked three experts in these fields to accompany me here today to assist in answering questions of a highly technical or medical nature. They are Dr. Joseph L. Goodman, Associate Professor, Department of Preventive Medicine, Medical University of South Carolina, and Medical Director, Raybestos-Manhattan Corporation; Mr. Isaac H. Weaver, Corporate director for Environmental Control for the Raybestos-Manhattan Corporation; and Mr. George L. Swallow, Manager of Occupational Environmental Control, Johns-Manville Corporation.

Before proceeding, I would like to express the appreciation of the Asbestos Information Association for the opportunity to testify on the proposed asbestos regulations.

Asbestos has numerous important applications in our modern industrial society. Among these, it provides built-in protection against fire and deterioration in scores of common products in daily use. Through the years it has saved countless lives and billions of dollars in property damage by preventing or checking the spread of fires. Asbestos-containing acoustical products make a valuable contribution to noise abatement.

Products containing asbestos are used, among other applications, in the construction of schools, houses, theaters, factories, office and other public buildings, and in the insulation of furnaces, boilers and electrical equipment.

The asbestos industry within the state of Illinois plays an important role in the economic life of the state and its people. The member companies of the Asbestos Information Association own and operate a total of eight plants within the boundaries of the state. Some of the product lines produced at these eight plants include: asbestos-cement pipe, packings, gaskets, friction materials--including both conventional and disc brake linings--asbestos-cement sheet products, asbestos roofing and siding shingles, insulations, and many others. A total of 3,118

Illinois residents are employed at these manufacturing facilities. Last year, these employees received a total of \$ 24.4 million in wages, salaries and fringe benefits. Services and goods purchased within the state, such as water, electricity, telephone service, raw materials and freight amounted to more than \$ 17 million in 1970. In addition, state, county and local taxes, amounting to nearly a million dollars last year, helped to pay for new schools, roads and other benefits to the state and its citizens. The value of goods produced at these manufacturing locations last year totalled \$ 70.1 million, with approximately \$14.5 million worth of these asbestos-containing products being sold within the state.

As you can see, the economic contribution of the asbestos industry in the State of Illinois is by no means small. And the figures I have just given you represent only those companies that are members of the Asbestos Information Association. There are also within the state many other concerns -- both large and small--whose livelihood depends in some measure on the use of asbestos or asbestos-containing products. Such concerns would include many segments of the construction industry, companies engaged in the manufacture of asbestos-containing products, companies which use finished asbestos-containing products in the manufacture of other products, and many others. The economic value to the State derived by the use of asbestos by these other

concerns is difficult to gauge, however it would certainly double or triple the figures I gave you earlier with regard to the contribution of the member companies of the Asbestos Information Association.

The asbestos industry has recognized for many years that there are occupational health hazards associated with the excessive inhalation of asbestos dust over long periods of time. As a result, the industry, beginning in the late 1920s, has sponsored and cooperated in a wide variety of scientific research projects designed to identify these hazards and to eliminate them wherever they may exist, not only in asbestos mines, mills and manufacturing plants, but also among fabricators and applicators of finished asbestos products. Today, the asbestos industry, either as individual companies or through the Quebec Asbestos Mining Association's sponsorship of the Institute of Occupational and Environmental Health in Montreal, is supporting medical research at such highly regarded institutions as McGill University in Montreal, Tulane University in Louisiana, St. Luke's Hospital in Cleveland, the Environmental Sciences Laboratory at Mount Sinai Hospital in New York, the Industrial Health Foundation in Pittsburgh, the University of California at Berkeley, Farleigh-Dickinson University in New Jersey, the University of Pittsburgh, the Medical College of South Carolina, and a number of others, both here and overseas. In addition, the industry has cooperated extensively

in various investigations into asbestos-health conducted by agencies of the Federal Government, including the U.S. Public Health Service, the Federal Environmental Protection Agency, the National Institute of Occupational Safety and Health, and others. In the industry's own research laboratories, scientists and engineers are also engaging in work related to health, developing safer packaging and handling techniques for loosely bound asbestos products and also developing and improving techniques for the measurement and control of asbestos emissions both in the work-place and in the ambient air.

As a result of these activities, over the years the industry has amassed a substantial amount of medical knowledge and technological know-how in the asbestos-health area.

Basically, the known facts about asbestos-related disease can be summed up as follows:

First, asbestos-related health risks are basically confined to the occupational and para-occupational setting.

Second, the effects of excessive inhalation of asbestos are both time and dose related. This means that asbestos-related diseases may develop, generally, only after the inhalation of substantial amounts of asbestos dust over a substantial period of time.

And third, there is presently no evidence of hazard to the general public from exposure to the minute amounts of asbestos that may be present in community air.

Because the general purpose of the regulations under discussion here today is to prevent the emission of hazardous quantities of asbestos fibers into the air that the general public breathes, I will restrict my further comments on health to this area. It is very important to realize in any discussion of a possible public health hazard from asbestos exposure that the only scientific studies we have to go on are those of groups of people in occupational and para-occupational environments. There is no basis for assuming that data on occupational health risks associated with heavy, long term asbestos exposure can be applied to the general public without taking into consideration the vast differences in exposure between the two.

Despite the fact that the only studies in existence are of occupational and para-occupational populations, a careful analysis of the data available can give us some general information about the question of a possible public hazard from asbestos. The substance of this information is that diseases associated with occupational exposure to asbestos are dose related and that the doses below which no measurable increase in disease occurs are far higher than any dose to which the general public is exposed.

This evidence is based on a number of epidemiological studies--- three of the most prominent being one by Dr. J. Corbett McDonald of 10,421 past and present asbestos mining and milling workers in Quebec, a second by Dr. M. L. Newhouse of 4,500 men who were employed in a British asbestos factory between 1933 and 1964 and the Third a study by Drs. Knox, Doll and associates of 878 workers in an asbestos textile factory, also in Great Britain.

Dr. McDonald, in his study, divided his subjects into eight categories, based on length of employment and severity of dust exposure. He noted that only in the highest time-exposure category, comprising five per cent of the total, was there any increase in deaths from respiratory and cardiac diseases, including lung cancer. Taken as a group, the Quebec asbestos workers had a mortality rate from all causes lower than the level expected in the general population.

Dr. Newhouse in her study in Great Britain found that workers who had low and moderate occupational exposure to asbestos dust showed a rate of deaths from diseases of the lung, including cancer of the lung and pleura, that were comparable with that of the general public. On the other hand, another study by Dr. Newhouse of mesothelioma (a relatively rare cancer of the lining around the lungs) in the London area showed a small number of cases among people who lived in close proximity to an asbestos textile plant and also among people living in the household of an asbestos worker, who presumably brought home quantities of the material on his clothes from work. The intensity of exposure experienced by these

so-called para-occupational populations in London is difficult to gauge, but there is every reason to believe that they were in fact higher than the low and moderate occupational levels in factories that have been shown to produce no excess of mesothelioma. These studies do, however, point up the need for proper control of emissions of asbestos from factories so that neighboring areas will not suffer para-occupational exposures to asbestos.

The Knox-Doll study, like the Newhouse and McDonald studies, confirmed the fact that low to moderate levels of asbestos exposure will not produce an excess of disease. It should always be remembered that these low to moderate levels in occupational settings inside factories were certainly higher than any to which the general public in this country is exposed.

In addition to these studies, there are several studies of the effect of various doses of asbestos on laboratory animals which also indicate that there are dose levels below which no measurable disease effect is observed. Dr. William Smith of Fairleigh-Dickinson University studied mesothelioma cancers in hamsters, and his data show that below certain specific doses of asbestos, no cancers were observed to develop over the entire natural life span of the animals. Dr. Merl Stanton of the National Institute of Health, Bethesda, Maryland, conducted a somewhat similar study with rats and observed the same type of dose-related response.

The most well-known of the American epidemiological studies related to asbestos exposure is that made by Dr. I. J. Selikoff of 632 members of the Heat Frost and Asbestos Insulation Workers Union in the New York/New Jersey area. These men, for the most part, are journeymen-craftsmen who work on construction sites and apply insulation to boilers, steam and hot water pipes, heating ducts, etc., during the construction of a building or a power plant. They may also be involved in tearing off old insulation. When one construction job is completed, they move to another construction job.

The occupational environment to which they may be exposed involves not only asbestos but also silica and particulate materials. In addition they are exposed to a variety of fumes from organic solvents and adhesives with which they work. In short, they have a widely varied and, in the past at least, a relatively uncontrolled occupational dust and fume exposure.

Dr. Selikoff found that the death rate from lung cancer was extremely high in this group of 632 workers, but that this very high mortality rate occurred almost exclusively among those workers who were cigarette smokers.

On the other hand, those workers who had no history of cigarette smoking had essentially no greater incidence of lung cancer than the general population of non-smokers.

detectable risk. What those levels are is not known, but there is no evidence that persons in the general population--without occupational, household or neighborhood exposures--have any increased risk of neoplasm, even though there may be ferruginous bodies or fibers in their lungs (end quote).

. In the conclusion and recommendation section of the report for the National Academy of Sciences, the following statements are made:

(quote) At present, there is no evidence that the small numbers of fibers found in most members of the general population affect health or longevity. Asbestos is too important in our technology and economy for its essential use to be stopped.. But, because of the known serious effects of uncontrolled inhalation of asbestos minerals in industry. . . it would be highly imprudent to permit unrestricted additional contamination of the public environment with asbestos (end quote).

I believe that these statements from the report adequately express the position of the asbestos industry with regard to the health hazards of asbestos. We do not believe that at present the general public is in danger from the minute amounts of asbestos that exist in the ambient air. However we also believe that it is prudent and feasible to establish reasonable emission controls that will protect the public and ensure that hazardous levels of asbestos will not develop in the community air in the future.

In general, the mortality rate of insulation workers reported by Selikoff is much higher than the rates found in studies of larger groups of people who work in factories producing asbestos-containing products, and in mines and mills processing asbestos. Why the incidence of certain diseases among insulation workers is so much higher than the other groups studied is a question as yet unanswered.

Unfortunately, as is so often the case with diseases that take from 10 to 40 years to develop, reliable data as to the past occupational exposure of these men to asbestos and other potentially hazardous dust and fumes in their work are not available.

As a result, at the present time we have little information as to what other factors may have influenced their high rate of disease except that those insulation workers who smoked cigarettes developed lung cancer at a rate far higher than the general population who smoked.

In 1970, with a grant from the National Air Pollution Control Administration, The National Academy of Sciences called together a blue-ribbon panel of asbestos-health experts to draft a document on the problem that would serve as the

medical basis for the Federal asbestos emission standards. Among those asked to serve on the panel were Dr. Clark Cooper of the University of California, Dr. Lewis Cralley of the Public Health Service, Dr. George Wright, Dr. Irving Selikoff and others.

The result of their study was a fifty-three page document on asbestos/health. As a conclusion to this part of my presentation, I would like to read a few pertinent excerpts from the report, which constitutes the most up-to-date evaluation of the asbestos-health problem that is available.

On page 20, the report states (quote) We cannot extrapolate from the mortality experience of those who are directly and indirectly exposed in their employment to the general public who have had moderate or slight exposure from ambient air. There is evidence to suggest a gradient of effect from direct occupational, to indirect occupational, to family and neighborhood situations, in all of which dust concentrations are probably high by comparison with most community air. This suggests that there are levels of asbestos exposure that will not be associated with any

With this position in mind, I would now like to discuss the asbestos regulations proposed for the State of Illinois. To begin with, I would like to commend the Illinois Pollution Control Board for its efforts to ensure a safe environment for the State and all its citizens. Your credentials as a progressive yet fair control board are well known and respected in environmental circles throughout the nation.

While the asbestos industry as represented by the Asbestos Information Association is of the position that there is presently no hazard to the general public from the minute amounts of asbestos existing in the community air, we nevertheless are in favor of regulations designed to accomplish one or more of the following three objectives:

One, to ensure the health and safety of those occupationally exposed to asbestos fiber.

Two, to prevent possibly hazardous amounts of asbestos fiber from escaping into the ambient air, and

Three, to eliminate, where possible, the nuisance of even small amounts of asbestos fiber from escaping into the ambient air.

While this third point is not directly related to health because of the small amounts of fiber involved, we wholeheartedly support the position that the environment should be as clean as possible.

For these reasons, we support most of the items in the proposed regulations. However, we do have several specific comments and

suggestions to make regarding the regulations that we hope will be of help to the Board in arriving at a final document that will not only be effective but also practical and reasonable. I will discuss the sections upon which we wish to comment in the order in which they appear in the regulations.

- Our first suggestion is with regard to Part II: General Requirements, Section 201. This section calls for the obtaining of a permit to engage in any activity which could discharge asbestos fiber into the environment. We feel that this section is not specific enough with regard to which activities would be covered. As you are probably aware, in the vast majority of asbestos-containing products used in the country, the asbestos is locked in place with cement, plastics or other binding materials, and does not constitute a source of emissions to the atmosphere. Asbestos-cement pipe and vinyl-asbestos floor tile are examples of such products. Other products and activities do constitute possible emission sources, and under the regulation would require a permit. If it would be of help to the Board, the Association I represent would be most willing to prepare a list of those products in common use where the asbestos is in a non-locked-in or loosely-bound state and thus constitute a possible emission source which would require a permit.

As to Section 201, Sub-section B, requiring a satisfactory course of health instruction for employees in those industries involving the handling of loose or loosely-bound asbestos products, I would like to point out that

the various trade associations in the asbestos industry have over the years produced a number of safety practices manuals and booklets dealing with various sections of the industry. The Asbestos Information Association would therefore recommend for your consideration as instructional material for use under Section 201, Sub-section B, the safety practices booklets which I have submitted to the Board with my presentation. The four booklets are:

1. Recommended Safety Practices for Handling Asbestos Fiber
2. Recommended Health Safety Practices for Handling and Fabricating Asbestos Textile Products.
3. Recommended Practices for Fabricating, Handling and Applying Asbestos-Cement Products in the Building and Construction Industries...and
4. Recommended Health Safety Practices for Handling and Applying Thermal Insulation Products Containing Asbestos.

Under Part V, Section 501, it is our opinion that Subsection B, which requires the enclosure of walls before toppling during demolition, is both impractical and unnecessary. We know of no suitable method for enclosing the walls of a structure during demolition. Even more important is the fact that there is, in reality, very little asbestos used in the construction of a modern medium-sized or high-rise structure. In addition, dust counts taken during the demolition of a building in

Easton, Pennsylvania, which did contain significant quantities of asbestos products, showed very little dispersion of fiber into the surrounding air, both upwind and downwind of the demolition site. This is due to the fact that most asbestos-containing products in which the fiber is locked-in with cement, plastic or other binding materials do not readily release fiber in the atmosphere, even during demolition. Subsection A of Section 501 requires the prior removal before toppling of boiler and pipe insulations....two asbestos-containing products that might release dust from improper demolition. Since this will remove any loosely-bound asbestos products from the structure, we believe enclosure of the walls to be unnecessary, and recommend that simple wetting of the walls to be toppled would be sufficient to control the dispersion of dust from whatever small amount of asbestos-containing products the walls might contain.

With regard to Part VI, Section 601 of the regulations, we would like to recommend for your consideration a completely new approach to the control of asbestos emissions from manufacturing

operations. As presently written, the regulations call for the establishment of a numerical emission standard of .5 fibers per cubic centimeter of air from any manufacturing operation, and .05 fibers per cc of air at the boundary line of the plant or factory.

We would recommend that Section 601 of the regulations be re-written so as to substitute a control practice standard for the present numerical emission standard. The reasons for this recommended approach are varied, but are primarily based on the lack of specific data with regard to safe non-occupational levels and the difficulty of accurate and meaningful emission sampling techniques at very low fiber concentrations.

The Federal Environmental Protection Agency studied this problem very carefully in preparing its national emission standards on asbestos, and arrived at the conclusion that a numerical standard was impractical at this time. In this regard, I would like to read a few excerpts from a document prepared by the EPA as justification for this viewpoint.

The document states that ideally, national emission standards for asbestos would be established on a concentration basis related to health effects and would provide an ample margin of protection to the public health regardless of the number of sources in a given geographical area. There are many practical considerations, the report goes on, which prohibit the establishment of a numerical standard at this time.

Foremost of these problems is the lack of a data base. This base is lacking from both the health-effects standpoint for non-occupational exposure and the emissions standpoint. The majority of data available on asbestos health effects is related to occupational exposure and cannot be readily extrapolated to non-occupational levels.

A little later on, the report states that (quote) a control practice approach to reducing asbestos exposure levels offers a practical alternative to numerical emission limits. A major advantage of a control practice standard would be realized in enforcement. Inspectors could be easily trained to certify compliance with control practice regulations (end quote).

The report goes on to describe some of the many difficulties involved in asbestos sampling and analysis, especially with regard to emissions into the ambient air, and concludes that present techniques are not only time consuming and expensive, but that they

also tend to distort the concentration of asbestos in the sample being analyzed and are hence not meaningful in establishing the actual amount of asbestos being emitted into the atmosphere.

The National Academy of Sciences Committee report on asbestos health, which I quoted from earlier, agrees with the EPA position on numerical standards. It states (quote) because of methodologic and other uncertainties, it is not yet feasible to base control on numerical ambient air quality standards (end quote).

I will not go into detail on the specifics of a control practice standard since they would be primarily of an engineering nature, however, I will submit for your consideration, as soon as they are published, the asbestos emission standards proposed by the Environmental Protection Agency. These standards will contain sufficient information to set up a similar program for the State of Ill.

Section 602 of Part IV of the regulations deals with the discharge of asbestos-containing waste into the sewage systems or waters of the state. We believe that this section is unnecessary. In the first place, the water pollution control systems in use in most asbestos manufacturing plants across the state effectively remove the majority of asbestos wastes from the effluent before discharge into state waters.

Secondly, sewage plants also remove asbestos fibers from waste water during purification. In reality, the high dilution of small quantities of asbestos fibers in water is as effective and safe a method for the disposal of asbestos wastes as could be devised. There is no evidence that the swallowing of small amounts of asbestos constitutes any health hazard whatever.

In addition, studies conducted by Johns-Manville have found small amounts of asbestos fiber in samples of water tested from sources all over the United States, including well and spring water. The reason for this is quite simple. Asbestos-bearing serpentine rock is to be found in a majority of the states of the union, including Illinois, and the simple process of water flowing over and through these rocks erodes a certain amount of asbestos fiber.

Thus, we have all been drinking tiny amounts of asbestos in our water since birth, without any discernable adverse effects.

In Part VII, Section 701 of the regulations, we would recommend the addition of the word "visible" in the next to the last line, so that the amended section would read: "No product which may emit

asbestos-fiber during its transportation shall be transported unless such product is enclosed in such a manner as to preclude the "visible" emission of asbestos fiber into the ambient air." The problem here is once again with analysis techniques and enforcement. The EPA has gone to a (quote) no visible emissions (end quote) standard in circumstances similar to this one, and we would recommend to the Board that this also be applied in Illinois. This would also be useful in other sections of the proposed regulations where measurement and enforcement would tend to be extremely difficult if not impossible.

Our final comment is with regard to Part VII, Section 702, which would prohibit the use of asbestos in the brake lining of vehicles manufactured after January 1, 1975, and sold for use within the State of Illinois. We believe that on the basis of the existing scientific evidence such a prohibition is completely unnecessary. In 1968, this question was investigated thoroughly by the United States Public Health Service, and a report on these investigations by Jeremiah R. Lynch, entitled "Brake Lining Decomposition Products" was published in the Journal of the Air Pollution Control Association. The report stated that (quote) except in all but the most extreme driving conditions, only a very small fraction of the 30 to 50 per cent asbestos present in a brake lining escapes into the atmosphere as free fiber (end quote). Lynch found that the average amount of free fiber released was less than one per cent of the fiber present in the brake lining composition. The percentage was higher only under conditions that would have resulted in brake failure. Lynch further

reported that the majority of the fiber in linings was converted into an inert non-fibrous material by the heat of friction, and concluded that the use of asbestos-containing brake linings was "an inconsequential health factor in urban air pollution." His conclusion is reasonable in light of the fact that hundreds of millions of cars using asbestos-containing brake linings have been stopping and starting in this country for more than half a century, and yet there has been no epidemic of asbestos-related disease among the general public nor have significant quantities of asbestos been demonstrated to exist in the community air. While we consider this evidence to be conclusive, if the Board is still concerned about asbestos emissions from brake linings, you will be interested to know that the Federal Environmental Protection Agency has contracted with the Bendix Corporation to do additional tests in this area. This study is due for completion in June of 1972, and we would recommend that the Board, at the very least, await the outcome of the EPA study before making a final decision on asbestos-containing brake linings. It is our belief that the Bendix study will confirm the conclusion reached by Lynch as to the safety of asbestos-containing brake linings.

This concludes my presentation on the proposed regulations. I have submitted for your consideration copies of all the medical papers, reports, booklets and documents that I referred to in my presentation. I would once again like to express the appreciation of the Asbestos Information Association for the opportunity to appear here this morning. Thank you.

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PHILIP E. RYAN (DEC 8)

November 8, 1971

1123

Mr. Samuel T. Lawton, Jr., Hearing Officer
Illinois Pollution Control Board
189 West Madison Street
Chicago, Illinois 60602

Re: Proposed Regulations on Asbestos
and Spray Installation (#R 71-16)

Dear Mr. Lawton:

On behalf of the Asbestos Information Association/North America, I herewith hand deliver an additional statement of Dr. F. L. Pundsack with additional background material pertaining to his statement as requested at the hearing of the Board October 15, as well as recommended changes in the wording and content of the proposed regulations, to be included in the record.

As suggested in his letter, if you or any member of the Board or staff of the Illinois Pollution Control Board require additional information, or copies of any of the information, please be assured of the complete cooperation of Asbestos Information Association/North America, or any of its member companies.

Sincerely yours,


Harold T. Halfpenny

HTH:am
Enclosures

Asbestos Information Association / North America

22 East 40th Street
New York, N. Y. 10016
(212) 661-8206

November 4, 1971

Mr. Samuel T. Lawton, Jr.
State of Illinois
Pollution Control Board
189 West Madison Street
Suite 900
Chicago, Illinois 60602

Dear Mr. Lawton:

As you requested, enclosed you will find background information and other material pertaining to certain statements and recommendations made during my testimony on behalf of the Asbestos Information Association/North America at the hearing held in Chicago on October 15 concerning the State of Illinois Pollution Control Board proposed regulations on Asbestos and Spray Insulation (#R 71-16).

The enclosed documents include the following:

- (1) A series of recommended changes in the wording and content of the proposed regulations. I have underlined our suggested word changes as they apply to existing sections of the regulations, and have completely rewritten sections of Parts IV and VI in accordance with the testimony presented at Chicago. We firmly believe that the recommended changes are reasonable and acceptable in light of existing technology and today's medical evidence, and do not constitute a weakening of the regulations. In fact, we believe that many of our suggestions will add to the effectiveness of the regulations by making for ease of enforcement. Our suggested deletion of those sections of the regulations regarding waste water discharge and asbestos-containing brake linings is supported by the existing medical evidence with regard to the inhalation and ingestion of extremely low levels of asbestos fiber.

- (2) An updated version of Dr. Corbett McDonald's study of more than 10,000 workers in the chrysotile producing industry of Quebec. Of special interest in this paper is Dr. McDonald's calculations that no detectable increase in deaths from respiratory cancer or pneumoconiosis would develop at asbestos fiber levels below 12 fibers per cubic centimeter of air, a level enormously higher than any to which the general populace of the State of Illinois are exposed. Dr. McDonald's study adds further weight to our contention that the extremely high mortality rates reported by Dr. Selikoff in his study of insulation workers were, at least in part, due to factors other than asbestos exposure, or that the level of asbestos exposure of these men in the past was a relatively high occupational level. In further support of this position, you will also find attached a copy of a letter evaluating this aspect of Dr. Selikoff's study by Dr. George W. Wright, Head of the Department of Medical Research, St. Luke's Hospital, Cleveland, Ohio. Dr. Wright was a member of the asbestos panel that drafted the National Academy of Sciences report on asbestos-health for the Federal Environmental Protection Agency. He also serves as a medical consultant to several companies in the asbestos industry.
- (3) A listing of "locked-in" and "loose, bound" asbestos products. As you recall, we volunteered to provide the Board with such a listing to aid in identifying those activities which constitute a possible source of asbestos emissions into the atmosphere, and would thus require a permit under Section 201 of the regulations.
- (4) A copy of the results of a study undertaken by the Johns-Manville Corporation to measure asbestos emissions during demolition of a house in Easton, Pennsylvania, that contained sizeable quantities of asbestos-containing products. The results indicate that while precautionary measures are certainly in order to prevent the wide dispersion of asbestos dusts during demolition, the problem of control may not be as serious as was once thought.

MORE

Mr. Samuel T. Lawton, Jr.
Page 3
November 4, 1971

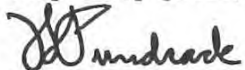
- (5) "Pulmonary Ferruginous Bodies: Studies on their Origin," by Dr. Paul Gross and Dr. Robert T. P. De Treville. As you recall, Dr. Joseph L. Goodman, who appeared with me at Chicago, referred to a section of this study in which rats and hamsters were injected intratracheally with brake drum dust. The results showed no fibrosis in the test animals. The relevant section of the study begins at the top of page 88.

We had hoped to be able to provide you with a copy of the proposed Federal Environmental Protection Agency emission standards on asbestos, which will contain a control equipment standard as opposed to a numerical standard, however, as you probably know, the publication of the Federal regulations has been delayed. Nevertheless, we will send you a copy as soon as they appear in the Federal Register.

I am also enclosing a complete copy of my Chicago presentation and accompanying background material. Ten copies were provided to the Board for inclusion in the record prior to the hearing.

If you require additional copies of any of the material which I submitted with my presentation or with this letter, please do not hesitate to contact us. If you require any additional information, or if I personally, the Asbestos Information Association/North America, or any of its member companies can be of further assistance to the Board, we would be most happy to cooperate in any manner possible.

Very truly yours,



Dr. F. L. Pundsack

Mortality from Lung Cancer and Other Causes among Workers in an Asbestos Textile Factory

J. F. KNOX, S. HOLMES, R. DOLL, and I. D. HILL

From Turner Brothers Asbestos Co. Ltd., Rochdale, and the Medical Research Council's Statistical Research Unit, University College Hospital Medical School, London, W.C.1.

An account is given of work in an asbestos textile factory and of the steps taken to reduce exposure to dust since the introduction of the Asbestos Industry Regulations in 1931.

Measurements of the amount of dust to which men were exposed in the scheduled areas have been made by various methods since 1951, but the data obtained by these methods are not directly comparable. Measurements made since 1960 show that the mean yearly dust level has been fairly constant from year to year and has varied from place to place between 1 and 8 particles (5 to 100 microns long and at least three times as long as they were wide) per c.c. of air.

Chrysotile was used predominantly in the factory, but small amounts of crocidolite were also processed at all relevant periods.

Death rates have been recorded for all the 256 men who were employed for at least 20 years in the scheduled areas since the first man completed 20 years' exposure in 1916, and for the 538 men and 220 women who were employed for at least 10 years in the scheduled areas but were never employed in these areas before 1933.

The results show a substantially increased mortality for men who were exposed for 10 or more years before 1933 (48 deaths from all causes against 17.1 expected).

The increased mortality among these men can be accounted for by an increased mortality from cancer of the lung (12 deaths against 1.2 expected) and from diseases of the respiratory and circulatory systems associated with asbestosis.

Men employed before 1933, but for less than 10 years, showed an increased mortality from lung cancer (5 deaths against 2.6 expected), but no significant increase from other causes. Men and women who were exposed only since January 1, 1933, have had a mortality experience close to the national average.

Analysis of the trends in mortality shows (1) a decrease in mortality from lung cancer and other deaths associated with asbestosis with reduction in length of employment before 1933, and (2) an increase in mortality from lung cancer and other deaths without asbestosis with age.

Twenty-three cases of lung cancer occurred in men who had been employed in the scheduled areas for at least 20 years. One was regarded as a pleural mesothelioma and the rest (approximately 18 in excess of expected) appear to have been ordinary bronchial carcinomas.

Four other mesotheliomas have been diagnosed since 1963, all in men and women with less than 10 years' exposure, one with only seven months' exposure, and one in a man who had been employed in the scheduled areas only since 1937.

The results provide grounds for believing that the occupational hazard of bronchial carcinoma has been largely eliminated, but the data are insufficient to estimate the extent of the risk which may remain.

Many studies of the mortality of asbestos workers have been reported in recent years and it has been

shown that some groups of workers have suffered substantial risks of disability and death from cardio-respiratory disease secondary to asbestosis and from bronchial carcinoma and pleural and peritoneal mesothelioma (New York Academy of Sciences, 1965; Gilson, 1966). In one such study Doll (1955) reported the mortality in a group of 113 men who had worked for at least 20 years in the

Received for publication March 22, 1968

Requests for reprints to Dr. R. Doll, Medical Research Council's Statistical Research Unit, University College Hospital Medical School, 115 Gower Street, London, W.C.1.

'scheduled areas' of a large asbestos textile factory. In that study the mortality experience was reported to the end of 1953. Subsequently the period of observation was extended to the end of 1961 and the scope was broadened to include men and women who had been employed since 1933 and had worked in the 'scheduled areas' for at least 10 years (Knox, Doll, and Hill, 1965; Hill, Doll, and Knox, 1966).

In the present study we have extended the period of observation to the end of June 1966, added further workers who have qualified for inclusion in the defined groups, and examined the trends in mortality attributed to different causes with age and with the duration and period of exposure. We also report, in relation to these findings, the extent to which it has been possible to limit exposure to asbestos dust in the factory.

Places of Work

In an asbestos textile factory the sequence of operations proceeds from the receipt of the raw asbestos fibres to the finished textile product in woven or plaited form. The stages are:

(1) **Fiberizing** The raw asbestos, which has already been prepared at the mines by milling the rock, screening, and separating the fibres by air currents, arrives in bags. These are opened; the fibres are blended, and the fibre bundles are broken down further in enclosed drum-like fiberizing machines with rotary metal beaters. The fibres are then bagged again for transmission to the next process.

(2) **Carding** The formless mass of fibrous material is converted into a gossamer-like web which is divided into tapes and rubbed into sliver consisting of strands of short fibres made parallel to each other (0.7 to 1.0 cm. in length). Much dust is produced, and ventilation at a rate of approximately 2,500 cu. ft. of air per minute (71 m.³ per minute) per carding machine is required to carry it away through ducts, a rate which produced the levels of fibre count at the front of the machine that are shown in the last two columns of Table I. Different grades of sliver are produced, some of which are used in other manufacturing processes without further preparation.

(3) **Spinning** Most sliver is spun into yarn.

(4) **Weaving** Preparations of warp (beaming) and of weft (pirn winding) are preliminary to weaving on looms. A variety of woven materials are

produced from yarns ranging from fine tapes (listing weaving) to wide cloths and including webbing and brake linings.

(5) **Plaiting** Ropes are produced of different specification which are sometimes impregnated with graphite or other lubricant for the manufacture of packings.

'Scheduled areas' are the parts of the factory to which the regulations for the asbestos industry apply (Asbestos Industry Regulations, 1931). They comprise areas for the operation of crushing and opening, textile processes, insulation fabrication, mattress making or repairing, all sawing and grinding, and the cleaning of chambers or appliances for asbestos dust collection.

The report by Merewether and Price (1930) suggested that 'to prevent the full development of the disease (asbestosis) among workers within the space of an average working life-time, it is necessary to reduce the concentration of dust in the air of the workroom to a figure below that pertaining to spinning at the time over which the cases (i.e., those dealt with in the Asbestos Report) were exposed'. To implement the practical application of this principle the asbestos industry in 1931 collaborated with the Factories Department (then in the Home Office, now in the Department of Employment and Productivity) in devising methods of dust control. As a result, it was agreed that the conditions arising from flyer spinning, carried on without exhaust, but under good conditions of room ventilation, should be taken as 'dust datum' for practical purposes (Report on Conferences between Employers and Inspectors, 1931). This level of dust production was accepted as the basis on which to assess the dustiness of other processes, and it is of interest to note that in 1956 Meiklejohn considered that nothing had emerged to suggest the need to depart from this practical standard.

The principal methods laid down to achieve dust control are:

- (a) application of exhaust draught at dust-producing points;
- (b) installation of enclosed mechanical methods for hand conveyance;
- (c) effective enclosure of dust-producing machines and plant; and
- (d) substitution of wet methods for dry. In situations where these methods cannot be applied, respirators must be worn.

The regulations were made in 1931 to come into force by stages during 1932; we have, therefore, regarded January 1, 1933, as the commencing date for workers whose mortality can be related to the

improved conditions. The technical developments necessary for effective dust control were not immediately available, and much research and experiment were needed. Comprehensive accounts of the development of dust control have been given elsewhere by Bamblin (1959) and Hills (1965).

Measurement of Exposure

The type of asbestos that has been used predominantly is chrysotile, which has been imported mainly from Rhodesia and Swaziland. No workers in the factory were exposed exclusively to crocidolite for any considerable part of their working life. It cannot be affirmed that any were not exposed to it at all, but the amount of exposure must have been relatively small.

It is not possible to obtain a single evaluation of the total hazard to which the workers have been exposed by relating time of exposure to dust level in the working area. One man began to be exposed at the end of the nineteenth century and dust records are not available until 1951. The changes made since 1931 have resulted in a steady improvement in working conditions, and the dust concentration has not been constant in any area.

In 1933, ventilation systems were introduced in carding and weaving. In 1939, damping techniques were established in addition in the weaving process; the improvement was considerable and the conditions of this process have remained reasonably constant from that time to the present day. In 1943, the dust settling chambers in the ventilation system were replaced by cotton fabric sleeve filters. This immediately removed the need for the hazardous operation of cleaning out the chambers.

The years immediately following the war saw many other changes. Damping was extended to yarn doubling, and in the period 1953-57 the design of the card extraction system was improved. During this period, oiling of the fibre was introduced at the mixing stage to suppress dust during its subsequent processing.¹ Before 1958, fibre had been blended on an open floor under dust exhaust hoods, but at this time an entirely new and totally enclosed system, covering the whole fiberizing process before carding, came into operation, and it became possible to add the optimum amount of oil necessary for dust suppression, which had not been possible on the old mixing floor.

Various methods of dust sampling were used at irregular intervals until 1951 when routine dust sampling in the principal parts of the factory was introduced. The Casella Thermal Precipitator instrument was used and the samples were incinerated before being counted at a magnification of $\times 1,000$; all visible particles were counted. This system was in use until 1965 and was invaluable in assisting the engineers in the many improvements then being made. Unfortunately, while particles were included down to the limit of visibility (0.2 micron), the instrument was not efficient in capturing the large fibres (above 10 microns long), and these fibres were not generally seen in the dust deposited on the counting slides. The Long Running Thermal Precipitator, which collected the sample by a combination of settlement and thermal precipitation, was therefore adopted. Counting was done without incineration at a magnification of $\times 500$ and only those fibres were counted which were between 5 and 100 microns long and whose length was at least three times the diameter. Correlation between the two sets of results proved difficult because the new fibre count was in a sector of the dust spectrum only partly included by the old sampling method. A subsequent change in 1965 from the Long Running Thermal Precipitator to cellulose membrane sampling did not introduce any further complication, as the results, when the new method was properly established, were directly comparable with those obtained previously.

Table 1 shows average dust levels in parts of the factory where different processes were carried out at the beginning (1952) and the end (1966) of the Casella Thermal Precipitator period, at the beginning of the Long Running Thermal Precipitator period, and with the cellulose membrane fibre counters at the end of the period under observation. There were considerable deviations from the mean figures from time to time, a typical figure for the coefficient of variation in the 1966 data being 40%. The location of the process itself was in some cases changed during the period under review, and sampling points have also changed over the years. It should be noted that sampling, in the past, was geared to the engineering work in progress and not specifically directed towards an assessment of the biological hazard.

The fibre count figures in the Table have been rounded off to the nearest fibre per c.c. These show that considerable improvements were made in the period 1952 to 1960, during which time the factory was modernized. Since then conditions have remained fairly steady, although work on dust control has continued. In particular, engineering

¹The oil used is a white mineral textile oil (Texconox, Shell), solvent refined with low aromatic content, viscous (Redwood) 30-60 sec. at 70°F., specific gravity 0.885 at 60°F.

TABLE I
DUST LEVELS ACCOMPANYING DIFFERENT TEXTILE PROCESSES, 1952-1966

Department	Process ¹	Yearly Mean Dust Levels			
		Casella Thermal Precipitator (particles per c.c.)		Long Running Thermal Precipitator or Cellulose Membrane (fibres per c.c.)	
		1952	1960	1962	1966
Fiberizing	Mixing floor	500	—	—	—
	Opening	440	now totally enclosed		
	Bag slitting	—	110	5	4
	Mechanical bagging	—	120	4	5
Carding	Fine cards	200	200	6	6
	Medium cards	810	400	8	8
	Coarse cards	1,140	420	7	8
	Electrical sliver cards	290	260	5	2
Spinning	Fine spinning	170	110	4	3
	Roving frames	510	150	5	6
	Intermediate frames	530	100	5	6
Weaving	Beaming	190	220	8	4
	Pirn winding	350	130	3	3
	Cloth weaving	180	140	3	2
	Listing weaving	130	110	2	1
Plaiting	Plaiting	140	80	4	4

¹Constituting all the processes in the 'scheduled areas'.

changes at the Electrical Sliver Cards and at Beaming have had an appreciable effect in reducing fibre counts. Consistently low figures have been obtained at Weaving, and the levels at Listing Weaving have been the lowest in the scheduled areas of the factory.

Mortality Experience

The study has been confined to observations on men and women who were employed in the scheduled areas. All men are included who were employed in these areas for more than 20 years at any time since the factory opened, and men and women who were employed for more than 10 years are also included if they were first employed on or after January 1, 1933. Altogether 878 workers have been studied, and the observations extend over a 50-year period from 1916¹ to the end of June 1966. The numbers of men and women studied and the

number of person-years observed are shown in Table II, grouped according to the total duration of exposure, and duration of exposure before January 1, 1933. Men in group 4 (10-19 years' exposure, none before 1933) were transferred to group 3 (20 or more years' exposure, none before 1933) when they

TABLE II
NUMBER OF WORKERS AND NUMBER OF PERSON-YEARS AT RISK IN DIFFERENT EXPOSURE CATEGORIES

Group	Sex	Duration of Exposure in Scheduled Areas (yrs)	Duration of Exposure before 1933 (yrs)	No. of Workers	No. of Person-years at Risk
1	M	20 or more	10 or more	57	918
2	M	20 or more	less than 10	63	984
3	M	20 or more	none	136	621
4	M	10-19	none	538 ¹	4,388
5	F	10 or more	none	220	2,263

¹In Doll's (1955) study the period of observation started from 1922. Further investigation has now shown that one man completed 20 years' employment in what were subsequently scheduled areas in 1916.

¹Including the 136 men in group 3 before they had worked for 20 years in the scheduled areas.

had completed 20 years in the scheduled areas. The person-years that they contributed while in group 4 remain with group 4, but as from the date of transfer they start contributing to group 3 instead. The person-years in Table II are therefore additive, but the number of workers is not additive since all the 136 men of group 3 have passed through group 4, and are included in its total of 528 men.

The personnel records of the company were used to decide who fulfilled the criteria for inclusion, and the Personnel Department carried out the subsequent follow-up. This was relatively simple for the men who had been exposed for 20 years since they either continued working for the company, or were in receipt of a pension until their death. Follow-up of workers who had been employed in the scheduled areas for less than 20 years was more difficult as some had ceased being employed by the company and had not received a pension. All but one man and one woman, however, were successfully traced. Death entries could not be found for the untraced employees at Somerset House, and for the purpose of the present analysis they have been assumed to be alive.

For each of the five groups a three-dimensional table of person-years at risk was constructed, the dimensions being age of the subject, calendar year, and duration of time since the subject entered the survey, using a five-year grouping for each scale. The numbers of person-years were then multiplied by the corresponding national death rates for various causes and the products summed to give the numbers of deaths expected for comparison with the numbers observed.

The computer program for this purpose was written in Hill's (1968) 'Man Years Computer Language' and the calculations were made on the London University Atlas. National death rates for selected causes for quinquennia up to 1956-60 were taken from Case and Pearson's (1957, 1963) tables. Outside the range of these tables, quinquennial death rates were compiled from the annual reports of the Registrar-General; 1961-65 rates were used to estimate the numbers of deaths expected in the first half of 1966.

In previous studies the causes of death were mostly obtained from post-mortem reports and the records of the medical department of the company. Death certificates were obtained only when the cause was in doubt or not known. In the present study death certificates have been obtained relating to all deaths but one (which occurred abroad), and the causes of death have been classified in two ways: first, according to the cause given on the death

certificate using the method of classification in use by the Registrar-General at the relevant period, and, secondly, according to the best information obtainable from all sources.

Table III shows the observed numbers of deaths classified according to the certified cause in comparison with those expected from national rates. As previously reported, the men who were exposed for 10 or more years before 1933 (group 1) are shown to have had a highly significant excess of deaths from lung cancer and also from diseases of the circulatory and respiratory systems. In three instances cancer of the lung was discovered *post mortem* but was not mentioned on the death certificate; these deaths have not been attributed to lung cancer as it is not now possible to discover whether the information was reported to the Registrar-General.¹ The small excess of deaths due to other neoplasms is not statistically significant. It may be noted, however—in view of the finding of an excess mortality from gastro-intestinal cancer in insulation workers who were regularly exposed to asbestos in the United States of America (Selikoff, Churg, and Hammond, 1964)—that the five deaths were attributed respectively to cancer of the stomach (1), colon (2), gall-bladder (1), and prostate (1).

A significant excess of deaths from lung cancer is recorded also among men who worked in the scheduled areas before 1933 but for less than 20 years (group 2). The ratio of observed to expected deaths (3.2:1) is, however, much smaller than for men who were exposed for 10 or more years (10.2:1). In addition to the five men whose deaths were attributed to lung cancer, one other man, whose death was certified as due to myocardial degeneration, was found to have cancer of the lung *post mortem*.² The number of deaths from diseases of the circulatory and respiratory systems (11) is only slightly greater than the expected number (7.94); asbestosis was, however, mentioned on nearly half of the death certificates (5 out of 11).

Among men and women who have worked in the scheduled areas only since 1933 (groups 3, 4, and 5) the observed mortality from all causes is close to the expected (79 against 81.05). The total observed experience is substantial, and the result provides grounds for believing that the occupational hazard has been largely eliminated. Four facts, however,

¹These deaths were attributed to lung cancer in previous reports (Doll, 1955; Knott *et al.*, 1965; Hill *et al.*, 1966).

²Death attributed to lung cancer in previous reports.

TABLE III
 NUMBERS OF DEATHS OBSERVED AND EXPECTED, BY EXPOSURE CATEGORY AND CAUSE

Cause of Death (I.C.D. code numbers)	Group	Observed No. of Deaths			No. of Expected Deaths from National Rates	Probability of finding Observed No. or More
		Asbestos on Death Certificate	Asbestos not mentioned	Total		
Cancer of lung or pleura (162, 163)	1	8	4	12	1.18	<0.001
	2	3	2	5	1.57	0.032
	3	1	1	2	1.35	0.394
	4	0	6	6	6.41	0.018
	5	0	2	2	0.24	0.025
Other neoplasms (140-239, except 162 and 163)	1	0	5	5	2.61	0.124
	2	0	3	3	2.38	0.425
	3	0	1	1	1.55	0.788
	4	0	4	4	9.13	0.981
	5	0	1	1	2.72	0.934
Disease of circulatory system (400-468)	1	7	6	13	5.33	0.003
	2	2	6	8	5.66	0.211
	3	0	9	9	4.03	0.022
	4	1	23	24	22.60	0.412
	5	0	2	2	1.82	0.543
Disease of respiratory system (470-527)	1	11	3	14	2.37	<0.001
	2	3	0	3	2.28	0.399
	3	0	1	1	1.43	0.761
	4	3	5	8	8.59	0.626
	5	0	1	1	0.55	0.473
All other causes	1	2	2	4	5.58	0.807
	2	1	3	4	4.29	0.621
	3	0	2	2	2.26	0.660
	4	1	11	12	15.55	0.849
	5	1	3	4	2.82	0.313
All causes	1	28	20	48	17.07	<0.001
	2	9	14	23	16.18	0.064
	3	1	14	15	10.62	0.120
	4	5	49	54	62.28	0.868
	5	1	9	10	8.15	0.302

suggest that it is necessary to suspend judgement about the extent of the risk that may still remain. First, asbestosis was mentioned on the death certificate in seven instances. Secondly, the observations on men who have worked in the scheduled areas for more than 20 years (group 3) are few, and previous experience has shown that the risk of lung cancer is relatively small until after 20 years' exposure. Thirdly, the ratio of all observed to expected deaths in men in group 3 (1.4:1) is the same as in men who were exposed before 1933, but for less than 10 years (group 2). Fourthly, two deaths from lung cancer occurred in women—an unexpected observation in view of the rarity of the disease in women in the country as a whole ($P = 0.025$).

One explanation of this last finding may be that women who work in factories smoke more than other women of the same ages, so that the national mortality rates provide an unsuitable standard for assessing the existence of a specific occupational hazard. One of the two women who died of lung cancer, however, was a non-smoker.

The only other notable finding is the excess of deaths from diseases of the circulatory system in men who had worked for 20 years in the scheduled areas, but only since 1933 (9 deaths against 4.03 expected). None of the men were known to have had asbestosis and the group showed no corresponding excess of deaths from respiratory disease. It seems probable, therefore, that this is a chance finding of no medical importance ($P = 0.022$).

one-tail test), but the possibility of a continuing hazard must be borne in mind.

One of the difficulties in interpreting these results is that duration of employment before 1933 is closely correlated with the duration of observation and the individual worker's age, both of which may be related to the risk of developing disease. An analysis has, therefore, been made of the trends in mortality in relation to each of these factors after standardizing for the other two. The analysis has been limited to men who were employed for 20 or more years (groups 1, 2, and 3) and the expected numbers of deaths have been calculated for each cell of the three-dimensional table formed by three exposure groups (more than 10 years before 1933, some exposure before 1933 but less than 10 years, and no exposure before 1933); six duration of observation groups (0-4 years to 25-29 years after completing 20 years' exposure); and to five-year age groups (30-34 years to 75-79 years). The analysis has been made separately for three categories of death (lung cancer, other causes with asbestosis, and other causes without asbestosis), and for this purpose—since the comparisons are entirely within the series and not with national mortality data—we have classified death as being due to lung cancer whenever the disease was diagnosed at necropsy, irrespective of the diagnosis on the certificate. The total number of lung cancer deaths available for analysis is, therefore, 23. In these Tables, it will be noted, the 'expected' numbers are derived from the experience of the workers studied as a whole and bear no relation to the 'expected' numbers in Table III which are derived from national death rates. The results show: (1) a statistically significant reduction in mortality

from lung cancer, and other diseases with asbestosis, with reduction in length of exposure before 1933 (Table IV); (2) a highly significant increase in mortality from lung cancer and other diseases without asbestosis with increase in age at exposure (Table V); and (3) no evidence of a change in mortality for any causes of death with the passage of time after completion of 20 years' work in the scheduled areas (Table V¹).

These results are, in large part, those that would be expected if exposure to asbestos dust was responsible for a substantial proportion of the deaths from lung cancer and from other diseases with asbestosis but was unrelated to other diseases without asbestosis. They confirm that there has been a significant decrease in the occupational hazards since 1932.

Two other findings call for comment. First, the trend in the mortality from lung cancer with age at exposure accords with some other human data, but is contrary to the conclusions that have been drawn from animal experiments (Doll, 1964). In part it may be due to the inclusion of four or five cases due to other and non-occupational causes. Secondly, it is surprising to find that, after standardization for age, neither the mortality from lung cancer nor the mortality from other diseases with asbestosis increases with the passage of time after the completion of 20 years' work in the scheduled areas. It must be remembered, however, that exposure to asbestos dust has changed at irregular intervals throughout the whole period of observation and the crude classification of exposures into three groups according to the length of employment before 1933 cannot adequately allow for these changes. How this has biased the results

TABLE IV
DISTRIBUTION OF DEATHS WITH EXPOSURE BEFORE 1933
STANDARDIZED FOR TIME SINCE COMPLETING 20 YEARS' EMPLOYMENT AND AGE

Exposure before 1933 (yrs)	Distribution of Lung Cancer Deaths		Distribution of Other Deaths with Asbestosis		Distribution of Other Deaths without Asbestosis	
	Observed	Expected	Observed	Expected	Observed	Expected
Over 10 (group 1)	15	9.81	17	11.16	16	16.91
Up to 10 (group 2)	6	8.41	5	8.07	12	12.98
None (group 3)	2	4.78	0	2.55	13	11.12
All groups	23	23.00	22	22.00	41	41.01
Test of significance of trend	$\chi^2 = 6.70$ $n = 1$ $P = 0.01$		$\chi^2 = 9.63$ $n = 1$ $P = 0.002$		$\chi^2 = 0.52$ $n = 1$ $P = 0.47$	

TABLE V
DISTRIBUTION OF DEATHS WITH AGE
STANDARDIZED FOR PERIOD OF EXPOSURE AND TIME SINCE COMPLETING 20 YEARS' EMPLOYMENT

Age (yrs)	Distribution of Lung Cancer Deaths		Distribution of Other Deaths with Asbestos		Distribution of Other Deaths without Asbestos	
	Observed	Expected	Observed	Expected	Observed	Expected
30-34	0	0.03	0	0.03	0	0.01
35-39	0	0.36	1	0.37	0	0.26
40-44	0	1.45	2	1.52	0	1.80
45-49	2	4.00	1	3.39	2	4.56
50-54	5	5.79	1	4.53	5	8.45
55-59	4	4.66	7	4.45	10	9.52
60-64	6	3.13	6	3.48	9	8.96
65-69	3	2.45	2	2.70	10	5.57
70-74	3	1.09	2	1.33	3	1.59
75-79	0	0.04	0	0.20	2	0.27
All ages	23	23.00	22	22.00	41	40.99
Test of significance of trend	$\chi^2 = 12.12$ $n = 1$ $P < 0.001$		$\chi^2 = 1.46$ $n = 1$ $P = 0.23$		$\chi^2 = 20.48$ $n = 1$ $P < 0.001$	

TABLE VI
DISTRIBUTION OF DEATHS WITH TIME SINCE COMPLETING 20 YEARS' EMPLOYMENT
STANDARDIZED FOR PERIOD OF EXPOSURE AND AGE

Years after Completing 20 Years' Employment	Distribution of Lung Cancer Deaths		Distribution of Other Deaths with Asbestos		Distribution of Other Deaths without Asbestos	
	Observed	Expected	Observed	Expected	Observed	Expected
0-4	4	5.05	4	4.72	13	12.91
5-9	8	5.06	6	4.20	8	8.37
10-14	7	5.03	4	5.06	5	7.26
15-19	1	3.36	3	4.46	6	6.68
20-24	1	3.28	3	2.81	8	4.46
25-29	2	1.22	2	0.75	1	1.31
All periods	23	23.00	22	22.00	41	40.99
Test of significance of trend	$\chi^2 = 0.97$ $n = 1$ $P = 0.32$		$\chi^2 = 0.20$ $n = 1$ $P = 0.66$		$\chi^2 = 0.77$ $n = 1$ $P = 0.38$	

is not clear, but it emphasizes the need for caution in interpreting the trends with age and duration of observation.

Types of Lung Tumour

Details of the occupational histories of the 23 men who had been employed for 20 or more years in the scheduled areas and who developed lung

tumours are given in Table VII, along with the pathological diagnoses. Twenty-one of the cases were confirmed at necropsy and 22 by histological examination. All but one of the tumours were regarded as bronchial carcinomas, indistinguishable from the type of tumour that has occurred commonly in the general population during the last 30 years. The exception was diagnosed as 'endothelioma of the pleura' in 1936. The expected number of lung

TABLE VII

OCCUPATIONAL AND PATHOLOGICAL DATA RELATING TO MEN DYING WITH LUNG CANCER: MEN EMPLOYED FOR 20 YEARS OR MORE IN SCHEDULED AREAS

Year of Death	Age (yrs)	Occupation	Period of Exposure	Duration of Exposure (yrs) ^a				Necropsy			Death Certificate		Histology of Tumor
				Total	Before Jan. 1 1933	From Beginning to Death	From End to Death	Per- formed	Asbest-osis Present	Asbest-osis Men- tioned	Attributed Cause of Death		
1935	54	Weaver	1909-32	23	23	26	2	+	-	+	Cardiac failure and pericarditis	Epithelioma	
1936	65	Fiberizer	1913-36	22	19	23	<1	+	+	+	Endothelioma of pleura	Endothelioma of pleura	
1939	49	Disintegrator	{ 1910-14 1919-39	23	17	28	<1	+	+	0	Carcinoma of lung	Carcinoma	
1940	52	Disintegrator	{ 1911-15 1919-21 1923-39	21	15	29	<1	+	+	+	Carcinoma of lung	Carcinoma	
1941	52	Weaver	{ 1913-19 1924-38	21	15	28	2	+	+	+	Carcinoma of lung	Oat-cell carcinoma	
1942	59	Bag carrier	1913-41	28	19	29	<1	+	+	+	Pulmonary carcinoma	Oat-cell carcinoma	
1948	59	Weaver	{ 1912-14 1918-48	32	16	35	<1	+	+	+	Asbestosis	Anaplastic carcinoma	
1948	48	Spinner	1922-48	26	10	26	<1	-	+	0	Carcinoma of lung	Carcinoma	
1948	65	Maintenance	1919-48	29	13	29	<1	+	+	+	Tuberculosis	Oat-cell carcinoma	
1951	74	Fiberizer	1917-43	26	15	34	7	-	+	+	Carcinoma of lung	Adenocarcinoma	
1951	60	Weaver	{ 1919-25 1929-50	26	9	31	1	+	+	+	Myocarditis	Carcinoma	
1954	53	Weaver	{ 1923-49	25	9	31	5	+	+	+	Carcinoma of lung	Epithelioma	
1956	67	Spinner	1915-48	32	17	40	7	-	-	0	Carcinoma of lung	Adenocarcinoma	
1956	63	Weaver	{ 1912-14 1919-41	24	16	44	15	+	+	+	Carcinoma of lung	Carcinoma	
1956	55	Braider	{ 1914-19 1932-56	29	6	42	<1	+	0	0	Bronchogenic carcinoma	Oat-cell carcinoma	
1956	55	Fiberizer	1924-48	24	8	32	8	+	+	+	Carcinoma of lung	Carcinoma	
1956	61	Weaver	1923-51	28	9	32	4	+	+	+	Carcinoma of lung	Oat-cell carcinoma	
1957	74	Beamer	{ 1911-19 1919-50 1951-51	39	21	46	6	+	+	-	Carcinoma of lung and pleura	Carcinoma	
1958	71	Weaver	1911-47	35	21	47	11	0	N.A.	0	Carcinoma of lung	Carcinoma	
1960	62	Weaver	{ 1920-33 1940-47	20	12	40	12	+	+	+	Carcinoma of lung	Large-cell adenocarcinoma	
1963	54	Weaver	{ 1929-45 1946-63	33	3	34	<1	0	N.A.	0	Carcinoma of bronchus	Adenocarcinoma (biopsy)	
1963	58	Beamer	{ 1935-37 1939-59	21	0	27	3	+	+	+	Carcinoma of bronchus	Carcinoma	
1966	61	Weaver	1939-66	26	0	26	<1	+	0	0	Carcinoma of bronchus	Carcinoma	

^aIncorporating data from Table 2 in Doll's (1955) paper.^bCompleted years; as a result the sum of the total exposure and the duration from last exposure to death may be less than the duration from first exposure to death, even if the period of exposure was unbroken.

tumours is estimated to have been about four (Table III) or perhaps somewhat more as a high proportion of men who died with respiratory symptoms came to necropsy (see 'mortality experience' page 296 and Doll, 1955). The excess number of deaths from this cause is, therefore, about 19, of which one can be attributed to a pleural mesothelioma and about 18 to bronchial carcinoma.

Awareness of pleural mesothelioma as a separate oncological entity has increased greatly in recent years, particularly since Wagner, Sleggs, and Marchand (1960) drew attention to its characteristic association with exposure to crocidolite asbestos. The possibility must, therefore, be considered that some pleural mesotheliomas have been misclassified as bronchial carcinomas. In our view this is unlikely to have occurred. In nearly every case the tumours classified as bronchial carcinomas were found to have their origin in the substance of the lung at necropsy and were readily identified by their microscopic appearances.¹ Moreover, it may be noted that the distribution of cases of bronchial carcinoma and pleural mesothelioma is not dissimilar from that reported by Selikoff, Hammond, and Churg (1967) among members of the International Association of Heat and Frost Insulators and Asbestos Workers who had first been exposed to asbestos dust more than 20 years previously. In the study by Selikoff and his

colleagues, which covered the period 1962-67, 24 deaths were attributed to bronchial carcinoma against 23 expected and 3 were attributed to pleural mesothelioma out of a total of 94 deaths from all causes.

Since 1935, when the first death from lung cancer in our series was reported, records have also been kept of all coroners' necropsies on men and women known to have been employed at the factory.² Review of these records revealed four other cases which have been accepted as mesothelioma of the pleura by Dr. J. C. Wagner. Occupational histories and other data relating to the five men and women who developed mesothelioma are summarized in Table VIII. Only one man among these had been employed in the scheduled area for more than 10 years. Three died more than 40 years after first exposure to asbestos and one of these had been employed in the factory less than one year. From previous experience (Doll, 1955), it appears that the specific risk of bronchial carcinoma is relatively small with less than 20 years' exposure so that the contrast with pleural mesothelioma in these data is marked. Follow-up information has not been obtained for all men and women who have ever been employed in the scheduled areas, so that it is not now possible to make any accurate estimates of mortality. It is known, however, that over 12,000

¹Including the man shown in Table VII as dying of carcinoma of the lung and pleura in 1957.

²Necropsies on asbestos workers are ordered by the coroner whenever there is, in his opinion, a question of an industrial origin of death.

TABLE VIII
OCCUPATIONAL AND PATHOLOGICAL DATA RELATING TO ASBESTOS WORKERS DYING WITH PLEURAL MESOTHELIOMA:
ALL EMPLOYEES

Year of Death	Sex	Age (yrs)	Occupation	Period of Exposure	Duration of Exposure (yrs)				Necropsy		Death Certificate	
					Total	Before Jan. 1 1933	From Beginning to Death	From End to Death	Per- formed	Asbest-osis Present	Asbest-osis Men- tioned	Attributed Cause of Death
1936 ¹	M	65	Fiberizer	1913-36	23	19	23	1	-	-	-	Endothelioma of pleura
1964	M	57	Carder, spinner	1922-23 ²	1 1/2	1 1/2	42	41	-	+	+	Malignant pleural tumour
1966	F	55	Carder, spinner	1926-35	9	6	40	31	-	0	0	Carcinoma of lung
1966	M	59	Weaver	1937-38 ³ 1960-65	6	0	29	1	-	-	+	Carcinoma of lung
1967	M	71	Weaver	1914 1919-20	0 1/2	0 1/2	53	47	+	0	0	Myeloid leukaemia (carcinoma of lung associated)

¹Also included in Table VII.

²Also employed for 15 years in the rubber department (1929-44) and 20 years in the warehouse (1944-64).

³Also employed in the warehouse, 1929-30.

workers were employed in the scheduled areas at some period between 1930 and 1952.

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A study of the mortality of workers in an asbestos factory

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Newhouse, M. L. (1969). *Brit. J. Industr. Med.*, 26, 294-301. A study of the mortality of workers in an asbestos factory. A cohort study of over 4,500 male workers employed at an asbestos factory making both textiles and insulation materials is described. The main analysis is of the mortality of workers employed between April 1, 1933, the date of the implementation of the Asbestos Regulations, and May 1, 1964. The analysis was made in relation to job, length of exposure, and length of follow-up after first exposure. There was no significant difference between the number of deaths occurring in the factory population and the national figures, until an interval of 16 years or longer had elapsed from first exposure in the factory. There were 1,160 men who fulfilled this criterion. In this group there was no excess mortality among those who worked in jobs where exposure was low or moderate, but among those with jobs which entailed heavy exposure there was a significant excess of deaths from cancer of the lung and pleura, and cancer of other sites, in men with a total period of employment in the factory of less than two years, as well as with those who worked for longer. Excess mortality from respiratory disease was observed only among severely exposed workers with long service.

The commercial use of asbestos dates from the latter part of the 19th century. Power-driven machinery was being rapidly developed and there were increasing demands for this material which is able to preserve heat and resist high temperatures. Italy was exporting asbestos as early as 1870; the Canadian chrysotile mines were in production by 1878. At the end of the century crocidolite asbestos from South Africa was on the market; in 1907 amosite was discovered in the Central Transvaal and commercial production began in 1916. In the United Kingdom, chrysotile was first used in making asbestos products in about 1880, crocidolite in 1895, and amosite soon after its production started in South Africa.

The occurrence of pulmonary fibrosis in asbestos workers was recognized in the 1920s, and the association between the dust and disease was confirmed by a survey of factory workers carried out by Merewether and Price (1930). Later there were reports of pulmonary malignancies associated with asbestosis (Lynch and Smith, 1935; Doll, 1955). Wagner, Sleggs, and Marchand (1960) described

pleural and peritoneal mesotheliomata in those who had been in contact with crocidolite asbestos. By the time of the First International Congress on the Biological Hazards of Exposure to Asbestos (*Biological Effects of Asbestos*, 1965) the carcinogenic and fibrogenic properties of asbestos had been generally accepted.

However, the industry continues to expand and asbestos is a vital ingredient of an increasing number of products. More knowledge is required of the size of the health risk under the varying conditions of the industry. This paper examines the mortality of past male workers at an asbestos factory in relation to their particular jobs in the factory, length of employment, and the duration of follow-up since first exposure.

The factory started production in the East End of London in 1913; at first employing only about 20 workers, it gradually expanded; by the late 1920s there were between 300 and 400 workers, and by the 1950s the pay roll had increased to between 600 and 700. Asbestos textiles and insulation material were

the major products. In the early 1950s the textile departments were moved to another factory. A later development was the employment of ladders, mates, and labourers on insulating contracts at sites anywhere in the country. This activity was developed on an increasing scale from 1950 onwards.

Amosite, crocidolite, and chrysotile asbestos were all used in production, but crocidolite was not used for making sectional pipe segments after 1945. Its use continued in other departments.

Source of data

The data in this study were taken from the factory's personnel records which date back to 1913 and include all who have worked for any time in the factory. The name, address, date of birth, the actual jobs held with the dates of first engagement and final termination of each employee including those currently employed up to May 1, 1964 were abstracted. No attempt was made to trace those employed for less than 30 days. The co-operation of the Ministry of Social Security and the Registrar General was obtained, and each past worker's particulars were sent first to the Records Branch of the Ministry of Social Security. This register was able to identify 75% of the male workers and state whether they were alive or dead at May 1, 1964. The records of those unsuccessfully traced at this Ministry were sent to the National Health Central Register, where 30% of the remainder were identified. Searches of the electoral roll and more local registers were also made, bringing the overall trace rate up to 89% (Newhouse and Williams, 1967). The Registrar General provided the causes of death when the place and date of death had been determined.

The implementation of the Asbestos Regulations on April 1, 1933, which required the provision of exhaust ventilation in many processes, caused very considerable changes in the factory. The workers were therefore divided into three groups (Table 1) according to their period of employment. They consisted of the 4,695 men who worked entirely in the post regulation period, of whom all but 5% were traced; the small group of 140 men who worked before and after April 1, 1933, of whom all but 9% were traced; and 438 who worked entirely before the regulations, of whom only 50% were accounted for.

Studies of the first two groups are described in this

paper; the follow-up of the third was judged to be too incomplete for inclusion.

Classification of jobs

The experience of those with an intimate knowledge of the factory over the past 50 years was relied on to grade the 50 or more different jobs in the factory according to the severity of exposure to asbestos dust. Six grades were adopted ranging from grade 1, which included office workers with very low exposure, to grade 6, which included disintegrators, openers, and others with heavy exposure (Table 2). Jobs in departments making rubber jointings, asbestos building boards, and certain insulating materials were put into grades 1 and 2, as the proportion of asbestos used in these products was low, varying between 5% and 18%. Maintenance workers and those with similar jobs were considered to be moderately exposed (grade 3). The ladders, mates, and their labourers were put in the fourth grade. They worked on a variety of contracts outside the factory and had intermittent and varying exposure, depending on the type of insulating material used and the conditions of the particular job. They are considered separately as not only was the possible period of follow-up shorter than for men in other jobs, but also because the ladders had not infrequently been employed in their trade before entering the service of this particular factory. None of the men in exposure grades 1 to 3 worked regularly in areas where the Asbestos Regulations of 1933 applied. The severely exposed were divided into grade 5 and grade 6. Workers in grade 5 were chiefly makers of sectional insulating material of high asbestos content, sectional pipe covers, and insulation slabs. Men in grade 6 were openers, disintegrators, workers in textile and mattress sections, and those employed in disposal of dust and waste. The environment of all the men in jobs graded as 5 and 6 was controlled by the regulations. The men in grade 6 jobs, but not grade 5, were required under a separate statute to have initial and periodic medical examinations.

Method of estimation of the risk

The main survey was of the post-regulation period, from April 1, 1933 to May 1, 1964. Men qualified to enter the survey by having been on the factory's pay roll for 30 consecutive days at any time during that period. They continued in the survey, irrespective of whether they remained on the pay roll, until May 1, 1964 or the date of death if earlier.

For the analysis it was desired to categorize men by the length of time they were employed in the factory and by the type of job they did.

It was necessary to categorize men only by their past experience at any point, never by the experience yet to come. It was decided, therefore, to split the survey into three separate studies defined as shown in Table 3. It is important to realize that the time scale, of entering and leaving the studies, refers to each man individually, according to his own 'E-day' (when he entered employment at the factory). Thus at any given calendar date there would be a number of men in each study.

If a man died, he was removed at that point from whichever study he was in at the time, and did not enter any later study at all. Similarly, when the survey terminated on May 1, 1964, all men then in the survey were

TABLE 1
NUMBER OF WORKERS TRACED, IN RELATION
TO DATE OF ENFORCEMENT OF ASBESTOS
REGULATIONS

Employment in asbestos factory	Traced alive	Traced dead	Untraced	Total
After April 1, 1933 only	4,183	274	238 (5%)	4,695
Before and after April 1, 1933	71	57	12 (9%)	140
Before April 1, 1933 only	116	105	217 (50%)	438

TABLE 2

JOBS IN THE FACTORY GRADED BY SEVERITY OF EXPOSURE TO ASBESTOS DUST AND SHOWING TYPE OF FIBRE USED IN MANUFACTURE OF THE PRODUCT

Grade 1 (very low)	Grade 2 (Low)	Grade 3 (Moderate)
Staff and office Grounds, gardens, and gateways Canteen Laboratory Stores Mfc. rubber jointing ¹ BC Mfc. asbestos building boards ¹ AC Mfc. brake linings ¹ C	Clothing stores Works and buildings Cleaners Mfc. insulation material ¹ C	Metal shop Engineers Electricians Despatch and packing Laundry
Grade 4 (Moderate-severe)	Grade 5 (Severe)	Grade 6 (Severe)
Laggers ABC	No periodic medical examination Sectional pipe making { B before 1930 AB 1930-45 A 1945 onwards	Periodic medical examination Opening { B before 1930 AB 1930-45 A 1945 onwards
Mates ABC Labourers ABC	Yarn store B A.R.P. tapping B Bulk insulation material ABC	Disintegrating ABC Carding AB Spinning C Doubling B Mattress making { B Before 1936 AB 1936 onwards Crude store AB Dust and waste ABC

¹Asbestos content of product less than 20%.A = Amosite.
B = Crocidolite.
C = Chrysotile.TABLE 3
METHOD OF ALLOCATING WORKERS TO THE
THREE SEPARATE STUDIES

Enter employment at factory	E-day	
Enter survey	S-day	E-day + 30 days
Enter study 1		S-day = 1 year
Leave study 1		S-day + 4 years
Enter study 2		
Leave study 2		S-day + 16 years
Enter study 3		

treated as being removed from their respective studies.

For the analysis of each study the categorization was done by length of time on the payroll and by maximum job grade. For example, if a man started work as a disintegrator, a grade 6 job, he remained in grade 6 in all studies, even if he moved to a job in a lower grade, but if, as rarely happened, a worker started in a job with low exposure grading and later he moved into a higher exposure grade, he would be regraded in subsequent studies. If he remained on the pay roll for a period of months only he remained a short-service man in all three studies, but if he was employed in the factory for a number of years he would be regraded according to his length of time on the pay roll at the beginning of each study.

The workers who survived and who died in each subgroup of each study were listed by computer. The cause of death was taken from the death certificate and coded according to the Seventh Revision of the International Classification of Diseases (W.H.O., Geneva, 1957).

Death rates for England and Wales were taken from the Case Pearson tables (General Register Office, 1957; Case and Harley, 1958). These tables give age specific death rates for cancers of various sites and for respiratory diseases for five-year age groups, for quinquennia from 1911, until 1960-65. The number of years contributed by each man in each subgroup for each five-year period was calculated. Multiplication of the sum of the man years by the relevant death rate in the Case Pearson tables gave the expected number of deaths (Doll, 1955). The computer programme for this purpose was written in Hill's (to be published) *Man Year's Computer Language*.

Results

There was little difference in the mortality experience of men in exposure grades 1, 2, and 3 and they have been grouped together. The severely exposed workers in grades 5 and 6 also had a similar mortality experience, and these have also been grouped together. In study 3, however, there appeared to be a certain difference in the mortality experienced by the two severely exposed groups. The deaths from malignant disease (ICD 140-205) have been subdivided only into cancers of the lung and pleura (ICD 162, 163) and cancers of other sites. More detailed studies of the cancers are reported elsewhere (Newhouse and Wagner, 1969). The respiratory diseases were also grouped together and the figures for the expected number of deaths were taken from

the Case Pearson table which gives death rates for all types of respiratory disease, including the pneumoconioses, but excluding upper respiratory disease, influenza, and cancer of the lung and pleura.

Study 1

The men were categorized by their working experience in the factory during their first year after entry to the survey. It included all who started work between April 1, 1933 and April 30, 1963, and concerned 3,268 men. The mortality experienced between the second and fifth years after entering the study is shown in Table 4. Among those with low to moderate exposure, there were 14 deaths, whereas 19.2 were expected, and among those with severe exposure 10 deaths were found, with 14.6 expected. There was no excess of deaths from either cancers of the lung and pleura or other cancers or from respiratory disease. At this stage the experience of the factory population appeared to be more favourable than that of the general population.

TABLE 4

STUDY 1: MORTALITY BEGINNING OF 2ND TO
END OF 4TH YEAR¹
UNDER 1 YEAR ON PAYROLL
(3,268 men observed)²

Cause of death	Exposure			
	Low-moderate		Severe	
	Obs.	Exp.	Obs.	Exp.
All causes	14	19.2	10	14.6
Cancer of lung and pleura	1	1.1	0	0.7
Other cancers	1	2.6	2	1.7
Respiratory diseases ex- cluding cancer	3	3.5	2	3.0
Other diseases	9	11.9	6	9.2

¹In these tables tests of significance were made by reference to the Poisson distribution and are entered where p is less than 0.05.

²Loggers' mates and their labourers are excluded from the populations whose mortality is described in Tables 4-9.

Study 2

In this study the men were categorized by their experience in the factory during the first five years in the survey, having entered it at any date between April 1, 1933 and April 30, 1959. There were more than 3,300 men in the study. Depending on the date of entry, the number of years of follow-up varied between 5 and 16 years. Tables 5 and 6 show the mortality of those with less than two years and for longer periods of employment in the factory; the deficiency in the observed number of deaths has tended to disappear. There are no significant findings, but the number of deaths from cancers of the lung

TABLE 5

STUDY 2: MORTALITY FROM BEGINNING OF 5TH
TO END OF 16TH YEAR
UNDER 2 YEARS ON PAYROLL
(1,677 men observed)

Cause of death	Exposure			
	Low-moderate		Severe	
	Obs.	Exp.	Obs.	Exp.
All causes	38	38.3	28	32.2
Cancer of lung and pleura	0	4.0	3	5.0
Other cancers	8	4.1	4	1.4
Respiratory diseases ex- cluding cancer	10	6.1	1	5.7
Other diseases	20	24.1	20	20.1

TABLE 6

STUDY 2: MORTALITY FROM BEGINNING OF 5TH
TO END OF 16TH YEAR
OVER 2 YEARS ON PAYROLL
(960 men observed)

Cause of death	Exposure			
	Low-moderate		Severe	
	Obs.	Exp.	Obs.	Exp.
All causes	19	29.9	36	37.4
Cancer of lung and pleura	4	2.4	5	3.1
Other cancers	1	4.3	9	5.4
Respiratory diseases ex- cluding cancer	2	2.8	4	8.8
Other diseases	12	18.4	18	20.1

and pleura among those with longer exposures is higher than the expected figure.

Study 3

This study included only those who had been followed for at least 16 years since entering the factory. Their dates of entry to the survey fell between April 1, 1933 and April 30, 1948. There were 464 men with low to moderate exposure and 696 with severe exposure, comprising 375 in grade 5 exposure jobs and 321 in grade 6.

The group with low to moderate exposure showed no excess mortality either among those with less than two years, or among those with more than two years, employment in the factory (Tables 7 and 8).

Among the severely exposed group employed in the factory for less than two years there was a statistically significant mortality excess both from cancers of the lung and pleura and from cancers of other sites. Among those with more than two years employment in the factory, there was an excess

TABLE 7
STUDY 3: MORTALITY FROM BEGINNING OF 17TH
TO END OF 31ST YEAR
UNDER 2 YEARS ON PAYROLL
(689 men observed)

Cause of death	Exposure			
	Low-moderate		Severe	
	Obs.	Exp.	Obs.	Exp.
All causes	16	16.8	34	19.1 $p < 0.01$
Cancer of lung and pleura	2	1.4	8	1.6 $p < 0.001$
Other cancers	1	2.4	9	2.7 $p < 0.001$
Respiratory diseases excluding cancer	1	2.5	2	2.6
Other diseases	12	10.5	15	12.1

TABLE 8
STUDY 3: MORTALITY FROM BEGINNING OF 17TH
TO END OF 31ST YEAR
OVER 2 YEARS ON PAYROLL
(171 men observed)

Cause of death	Exposure			
	Low-moderate		Severe	
	Obs.	Exp.	Obs.	Exp.
All causes	15	13.5	36	18.3 $p < 0.001$
Cancer of lung and pleura	1	1.1	11	1.5 $p < 0.001$
Other cancers	2	1.9	7	2.6 $p < 0.02$
Respiratory diseases excluding cancer	4	2.0	10	2.8 $p < 0.001$
Other diseases	8	8.5	8	11.4

mortality from cancer of the lung and pleura, cancer of other sites, and from respiratory disease; four of the latter were certified as dying from asbestosis; neither among those with short or those with longer periods of employment was there any excess of deaths from causes other than cancer or respiratory disease.

A more detailed analysis of the mortality of the severely exposed men in study 3. A significant excess of deaths from cancer of the lung and pleura and from cancer of other sites occurred in the short service men in the grade 5 exposure group (Table 9) but not in the grade 6 group. Among the former there were six certified as having died of cancer of the lung and pleura, whereas 1.0 was expected ($p < 0.001$); in the grade 6 group the figures were two deaths observed to 0.6 expected, which is not statistically significant.

In the longer service group (Table 10) both the grade 5 and grade 6 categories showed a statistically significant excess of deaths from cancers of the lung and pleura. The deaths from respiratory disease, however, were concentrated among the men in the grade 5 category (nine observed to 1.8 expected $p < 0.001$). The grade 5 exposure group was larger than the grade 6 group, with 39 more men with short service and 13 more with long service, but the age structure of both groups appeared to be very similar, and there was no evidence that the length of follow-up varied in the two groups.

To determine more precisely the length of follow-up required after first exposure before a significant excess of deaths from cancers and respiratory diseases could be demonstrated, both the observed and the expected number of deaths among the severely exposed were grouped into five-year periods. The first period was from the 17th to the 21st year, the second from the 22nd to the 26th year, and the third from the 26th to the 31st year. In the group who had been followed from 17 to 21 years, among men with less than two years' employment in the factory there was a statistically significant excess of deaths both from lung cancer and from other cancers; among those with longer service there was a significant excess of deaths from cancer of the lung and pleura and from respiratory disease, but not from cancers other than pulmonary cancers. After a follow-up period of more than 21 years there was a statistically significant excess of deaths from cancer of the lung and pleura in men with both long and short periods of employment, there was an excess of deaths from other cancers only among men with shorter periods of employment. There were no deaths observed in this group from respiratory disease.

TABLE 9
STUDY 3: MORTALITY OF WORKERS IN GRADES 5
AND 6 EXPOSURE CATEGORIES
LESS THAN 2 YEARS ON PAYROLL
(429 men observed)

Cause of death	Exposure			
	Severe Grade 5		Severe Grade 6	
	Obs.	Exp.	Obs.	Exp.
All causes	23	11.1 $p < 0.001$	11	8.0
Cancer of lung and pleura	6	1.0 $p < 0.001$	2	0.6
Other cancers	8	1.7 $p < 0.001$	1	1.0
Respiratory diseases excluding cancer	0	1.5	2	1.1
Other diseases	9	6.9	6	5.2

TABLE 10
STUDY 3: MORTALITY OF WORKERS IN GRADES 5 AND 6 EXPOSURE CATEGORIES
MORE THAN 2 YEARS ON PAY ROLL (241 men observed)

Cause of death	Exposure			
	Severe Grade 5		Severe Grade 6	
	Obs	Exp	Obs	Exp
All causes	23	11.6 $p < 0.001$	13	6.7 $p < 0.02$
Cancers of lung and pleura	7	0.9 $p < 0.001$	4	0.6 $p < 0.001$
Other cancers	4	1.6	3	1.0
Respiratory diseases excluding cancer	9	1.8 $p < 0.0001$	1	1.0
Other diseases	3	7.3	5	4.1

TABLE 11
MORTALITY OF MEN WORKING BOTH BEFORE AND AFTER APRIL 1, 1933
OVER 2 YEARS ON PAY ROLL (140 men observed)

Cause of death	Exposure			
	Low-moderate		Severe	
	Obs	Exp	Obs	Exp
All causes	24	15.5 $0.01 < p < 0.05$	28	10.9 $p < 0.001$
Cancers of lung and pleura	4	1.0 $p < 0.02$	3	0.7 $p < 0.05$
Other cancers	4	2.2	7	1.7 $p < 0.001$
Respiratory diseases excluding cancer	7	3.2 $p < 0.05$	15	0.6 $p < 0.001$
Other diseases	9	1.0	3	7.9

The ladders' mates and labourers

This is a group of over 1,200 men in which only 60 were employed between 1933 and 1948: two deaths were observed, both from cardiovascular disease, whereas 2.35 were expected. There were 622 employed on insulating contracts who had been followed for 5 to 16 years. They contributed six deaths, three of which were due to carcinoma of the lung and pleura (three observed - 0.38 expected $p < 0.001$). These three men had only been employed by the firm for short periods, but one was known to have worked as a ladder for the previous 25 years; the past experience of the other two is unknown. Over 1,000 of this group had been followed for from 2 to 5 years, about half of whom were first employed in this firm after 1960. There is no excess mortality (five deaths observed - 7.01 expected).

Workers first employed before April 1, 1933 and continuing after

A group of 140 men was identified who started work before the Asbestos Regulations were implemented and continued into the post-regulation period. Three of these workers joined the factory in 1914, five in 1919, and a few each subsequent year until 1929; between 1931 and 1933 a further 61 (43%) started

work. The workers were grouped by their experience in their first four years after entering the factory, and the same grading of jobs was adopted as in the studies of the post-regulation period.

There were four deaths among the men with less than 10.0 years on the pay roll; 0.39 were expected ($p < 0.001$); one of these deaths was due to asbestosis. The mortality experience of those with more than two years service is shown in Table 11. There was a statistically significant excess of deaths from cancer of the lung and pleura both among those with jobs graded as having low to moderate exposure and among those with severe exposure. In both groups there was also an excess of deaths from respiratory diseases. Eleven of the severely exposed and three of those with lesser degrees of exposure were certified as dying from asbestosis.

In this study the length of follow-up of the group was at most 45 and at least 31 years; this is very much longer than in the studies previously mentioned, but conditions in the factory before the introduction of the asbestos regulations of 1931 cannot now be gauged. It seems probable that all workers, whatever their individual jobs, may have had considerable exposure to asbestos dust.

Discussion

The adoption of the 'three study plan' of analysis enabled the experience of different groups of workers to be examined in terms of job and duration of employment and in relation to their dates of first employment in the factory.

If the worker held more than one job in the factory he was categorized by the job in the highest exposure grade. More than 70% held only one job and, among the remainder, including those with long service, the normal pattern of employment was to start in a job with a high exposure grading and subsequently to change to a job with less exposure. It is possible that some of the workers were categorized by jobs held for only a small proportion of their total service, but this can have occurred only infrequently. Some difficulties were experienced in grading fitters and maintenance men who may have had varying degrees of exposure in their work; men in these jobs were included in the moderately exposed group. In this report there is a marked contrast between the mortality of the group, who were graded as having low to moderate exposure, and the mortality of those who were graded as severely exposed. There is no suggestion of an adverse effect on the mortality of the former group.

Enterline and Kendrick (1967) have shown rising standardized mortality ratios for cancers and respiratory diseases in men employed in asbestos building-product plants, friction material plants, and asbestos textile plants; the highest SMRs occurred in the textile plants which were reported to be the dustiest. In this factory also the statistically significant excess of observed deaths occurred among workers from departments which were believed to be the most dusty.

This excess was only seen in study 3 after a follow-up period of not less than 16 years. In study 1, where

the mortality was observed from the beginning of the second to the fifth year, there were fewer deaths observed than were expected (Table 12). This is not an unusual finding at the beginning of an epidemiological study of an occupational group, as men with severe disabilities are not eligible for recruitment to the industry (Doll and Hill, 1954; Selikoff, Churg, and Hammond, 1964). Pre-employment medical examinations of this group may have helped to exclude unfit men. From the beginning of the post-regulation period in 1933 the workers in grade 6 were subjected to a pre-employment medical examination, and after 1942, when an industrial medical officer was first employed, all workers in the factory were medically examined before starting work. In study 2, where the follow-up period is extended to between five and 16 years, the deficiency in the observed number of deaths has tended to disappear.

In study 3 the excess of observed over expected deaths certified as due to cancer of lung and pleura, and cancer of other sites among men with severe but short exposures was notable (Table 12). Seventeen out of a total of 34 deaths were due to cancer, whereas 3.3 would have been expected (Table 7). The mortality of men with this type of experience has not been examined until recently. Doll (1955), Knox, Doll, and Hill (1965), and Knox, Holmes, Doll, and Hill (1968) observed only workers with 10 or more years' exposure; Mancuso and El Attar (1968), in their study of a 1938-39 cohort of asbestos workers, grouped them according to their length of employment in an asbestos factory. They used the group with less than two years' exposure as an internal control; the incidence of cancer deaths rises with increasing periods of employment, but among those with less than two years' employment the SMR for malignant diseases in certain groups is over 200.

None of the insulation workers studied by Selikoff

TABLE 12
RATIO OF OBSERVED TO EXPECTED DEATHS

Follow-up period (years)	Period on pay roll (years)	All causes of death		Cancer of lung and pleura		Other cancers	
		Low-moderate exposure	Severe exposure	Low-moderate exposure	Severe exposure	Low-moderate exposure	Severe exposure
2-4 (Study 1)	<1	0.7	0.6	0.9	(0.0.7) ^a	0.4	1.8
5-16 (Study 2)	<2	1.0	0.9	(0.4.0) ^a	0.6	2.0	2.9
	>2	0.6	1.0	1.6	1.6	0.2	1.7
17-31 (Study 3)	<2	1.0	1.8	1.4	5.0	0.4	5.3
	>2	1.0	1.9	0.9	7.3	1.1	2.7

^aNo deaths observed in this category. The expected number of deaths is entered.

and Hammond (1968) died from carcinoma of the lung, mesothelial tumours or asbestosis before 20 years had elapsed since first exposure. Among the workers at the asbestos factory reported in the present paper a significant excess of observed deaths from cancer of the lung and pleura, other cancers, and from respiratory diseases was found in certain groups of workers after a follow-up period of between 17 and 21 years, suggesting that the latent period of asbestos related disease may be less than 20 years in these workers. Of the entire group of post-regulation workers studied, 1,160 (25%) have a follow-up period of 16 years or longer. Selikoff, Hammond, and Churg (1968) have revealed that cigarette smoking has an adverse effect on the mortality of asbestos workers; nothing is known of the smoking habits of this group as no direct contact was made with any worker. In future studies it may be possible to learn the smoking habits of selected groups of workers. A study of the mortality after May 1, 1964 is planned. Workers first exposed at later dates will have been followed for a sufficient length of time for asbestos related diseases to contribute to the causes of death. It will be possible to contrast their mortality experience with those employed earlier and to estimate whether comparatively modern methods of asbestos manufacture are reflected in an improved mortality experience.

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September 17, 1971

Mr. Edward Fenner
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Dear Mr. Fenner:

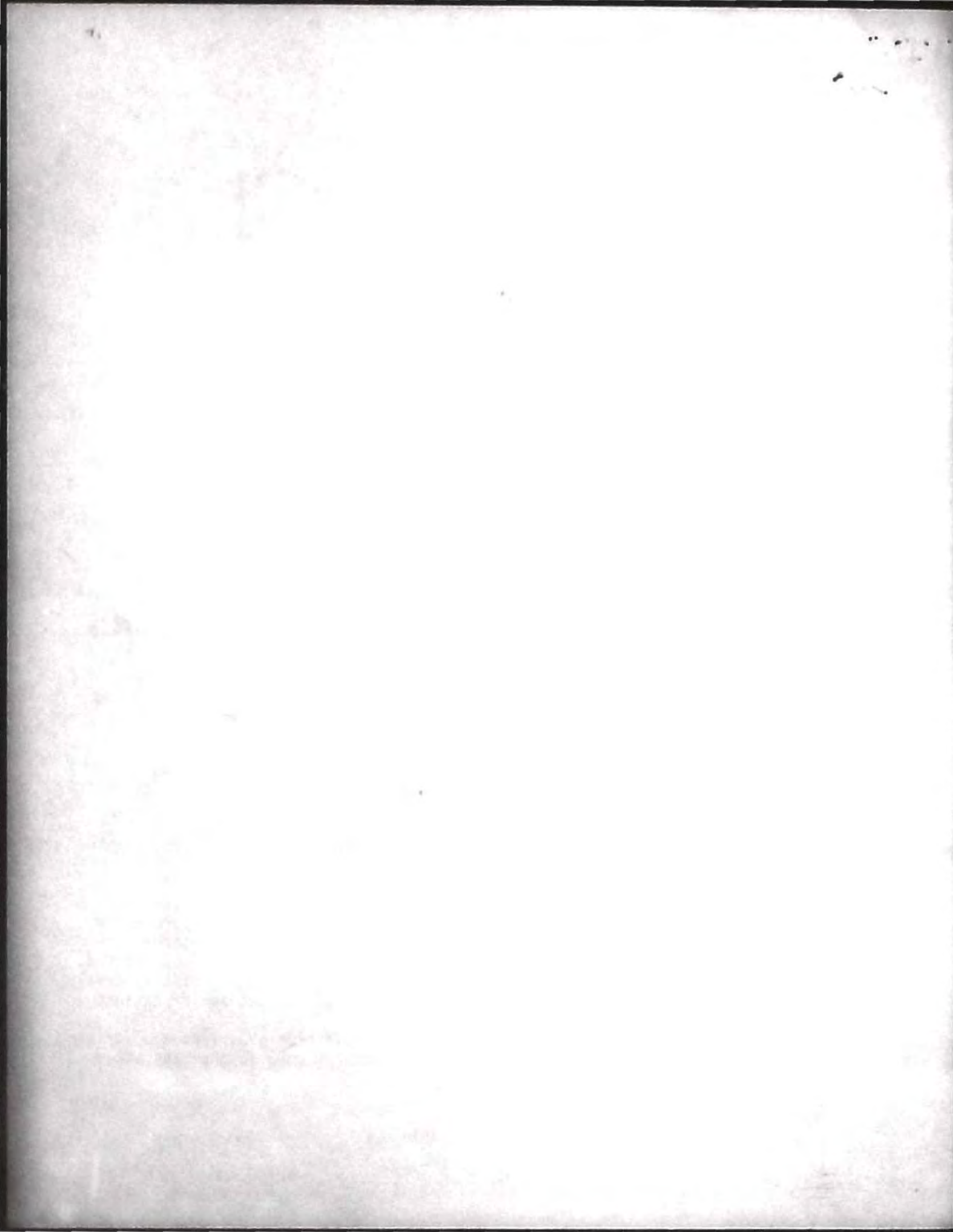
You have indicated to me that during recent convening of the EPA Document Review Committee Meeting, held in Atlanta on August 17, 1971, information was offered to the effect that the severe adverse health experiences of the group of insulation workers under observation by Dr. Selikoff was the result of low intensity of exposure of asbestos fiber - on the order of an average of two to three fibers per cc. Recently Dr. Nicholson, of the Mt. Sinai group, has published remarks to this effect in Insulation Hygiene Progress Reports, Volume 3, No. 2, 1971.

An answer to such claims is important, not only with respect to occupational TLV, but also because our attitude toward potential health hazards posed by asbestos fibers existing in the community air must at this time be based primarily on the experiences that have been gleaned from observations of individuals exposed for many years in occupational settings. The insulation worker group comprises only one of a number of such occupational settings.

There is, as you know, a rather striking difference in the health experiences of the insulation workers as compared to a number of other occupational groups in that they appear to have had a much more severe experience than have those who mine and mill asbestos or utilize it in manufacturing processes with perhaps the exception of the production of asbestos textiles. Just why the striking difference exists has never been satisfactorily answered.

When one reviews the published data, as for example those published by Newhouse, McDonald or Knox, which bear upon the health experiences of persons in various occupational settings to asbestos fiber, it is clear that the three major health effects, asbestosis, bronchogenic cancer and mesothelioma are dose related, and that mesothelioma may be related to type of asbestos as well as possibly developing at lower intensities of exposure than do the other biological manifestations.

The insulation workers studied by Dr. Selikoff have experienced a striking excess of all three health effects - as severe or perhaps more so than any other



Samuel Jenner
September 17, 1971

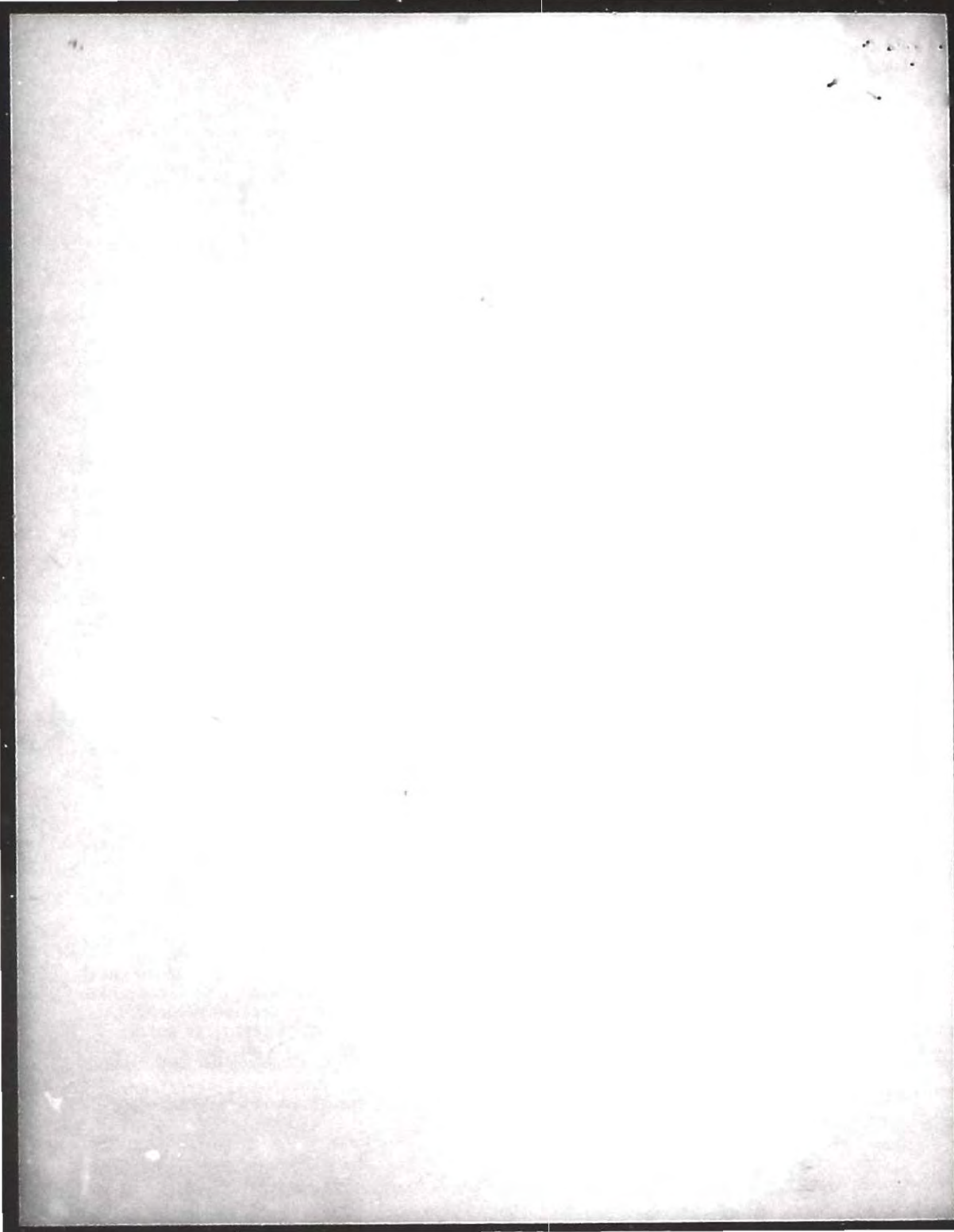
This important lack of information, since insulation workers are

group reported. Knowledge of the actual exposures experienced by this group, and in addition a contrast of the exposure of those who did to those who did not develop cancer, could be of great value. Unfortunately, Dr. Selikoff has never published detailed exposure data, nor has he ranked his group in such broad terms as light, moderate, or heavy exposure as related to their health experiences. He has not contrasted the exposure experience of those insulation workers who did, with those who did not develop cancer. He has not set forth the various other agents to which insulation workers may be exposed simultaneously with asbestos. In other words, we have essentially no information from the Mt. Sinai group as to the kind of exposures experienced by the insulation workers they have under study which would be meaningful in terms of severity of exposure or the possibility that their experiences have been dose related or whether or not co-existing agents play a strong role. The only exception to this is the statement by them that cigarette smoking plays a very important role in the development of cancer in insulation workers. There may be extenuating circumstances to explain a mobile population, and their exposure patterns as to concentration of asbestos fibers and co-existing potentially harmful agents would be difficult to define. This would be especially true for exposures experienced in past years. It is entirely possible that Dr. Selikoff cannot describe the exposure patterns and hence, has not done so. Nevertheless, until such information becomes available, it is most difficult to evaluate the exposure pattern of his group of insulation workers as it may relate to the striking contrast of severity of biological manifestations noted between his insulation workers and other occupational groups evidencing far less severe reaction.

I don't understand how his group can attribute biological effects caused by exposures going back forty years to this scant information about exposure measured in recent times. Nicholson of the Mt. Sinai group has indicated without supporting detailed data, that contemporary environments of their group of insulation workers contained fiber counts varying from 0.1 to 100 fibers per cc. He has then averaged these exposures over an unstated period indicating that in doing so he has included substantial work periods of no exposure to asbestos, and arrives at an average exposure of 3 fibers per cc. The statement is then made that it is this level of exposure that is responsible for the severe health effects observed in their insulation working group. The validity of this kind of averaging with no attention to peaks or effect of prolonged periods of high levels of exposure is dubious to say the least, and cannot be accepted without serious reservations. Moreover, no date on exposure during removal of old insulation - a notoriously dusty job - was given.

It must be emphasized that the insulation working group they have under study has experienced exposures extending over the past forty or more years. While contemporary fiber counts have importance in terms of current conditions, they have little or no importance for evaluating, in terms of numbers of fibers per cc, the exposures of men who have worked in the trade for forty or more years. The only way this information can be used is by introducing a conversion factor such as total dust counts over the past forty years, or some other such indicator. Such conversion factors apparently are not available for this group of insulation workers. Hence we have no direct knowledge of the fiber count, in modern terms, to which these men have been exposed during their lifetime of work.

It is of considerable interest that others have reported higher fiber counts characterizing the contemporary environment of insulation workers than did



Mr. Edward Fenner
September 17, 1971

Nicholson. One wonders how the work habits and materials used could be so different leading to higher fiber counts during insulation work published from England and Finland.

Regardless of the fiber counts, alleged by Nicholson to represent exposure of the past twenty years, it is clear that biological effects at these levels could be operative only in recent years and would not describe the total exposure of men working prior to 1950. The Mt. Sinai group infer that the exposure since 1950 characterizes the conditions that caused the severe biological experiences of their insulation workers study group. Included in all of their reports, however, are men exposed at least forty years ago and the most severe effects to date appear to have occurred in men exposed more than twenty years ago. To fail to indicate this is a serious misrepresentation of their data.

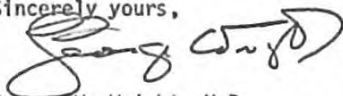
Nicholson appears to believe that contemporary dust counts reflect the exposure pattern over the past twenty years because insulating practices have not changed during that period. The real question is not whether they have changed over the past twenty years, but whether they have changed over the past forty to fifty years. Some months ago I asked Johns-Manville to describe to me the changes if any, in insulation materials over the past forty years. Mr. W.K. Hesse did this, and his document is available. Striking changes in materials alone that would lessen the chance for airborne asbestos dissemination are reported by him. In comparison to the asbestos content of insulation in use in the early 1930s, he shows that by 1960, high temperature magnesium insulation has 50% less asbestos, and others such as calcium silicate and "fibrous" insulation have 25% less asbestos. These materials constitute 95% or more of the asbestos containing insulation on the market. Of even greater importance is the fact that fibrous glass began replacing asbestos insulation beginning around 1945, and now constitutes 50% of all insulation used. These changes cannot fail to have reduced the concentration of asbestos in the environment of the insulation worker who has been exposed during the past forty years. It must also be recalled that the most severe type of exposures, such as ship repair and boiler work, were accelerated under adverse circumstances for the insulation worker during the second World War - prior to 1950. Unless there is something quite different about the exposure of insulation workers (always a possibility since their total environment has never been described) it would appear inescapable that their exposure to asbestos fiber has been considerably higher than those asserted to have existed by Nicholson. There are evidences that this is the case. In the Insulation Hygiene Progress Reports, Vol. No. 3 (1971), Selikoff states "today's disease is the result of practices twenty, thirty, or even forty years ago. Work conditions then, in terms of dust, were bad. On many jobs they are still bad". Table 1, referred to by him in that publication is of interest. It shows that 67% of the men exposed thirty to forty years ago have developed severe asbestosis, while only 21%, and 9% of those exposed twenty to thirty and ten to twenty years respectively, have developed this grade of asbestosis. These data are strong evidence that a reduction of asbestos in the environment of insulation workers occurred over the past forty years, especially since x-ray evidence begins to appear within ten years of moderate or severe asbestos exposures. Moreover, in other occupations showing a substantial frequency of asbestosis, the dust counts and more recently, the average fiber counts, have been far higher than 5 fibers per cc - often in the estimated range of 50 to 75 or more,

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and surely greater than 20 fibers per cc. Based upon the knowledge of exposure and biological reaction in other asbestos using occupations, it is inconceivable to me that two to three fibers per cc could be the intensity of exposure that has led to the biological changes manifested by the insulation workers under study by Selikoff and his co-workers.

On the basis of current scientifically gathered data, I am forced to believe either that the exposures evoking the severe biological response of the insulation worker studied by Selikoff, were substantially higher than they allege or that there are co-existing factors peculiar to insulation worker exposure which do not exist in other occupations. They have not presented evaluated scientific data bearing upon this latter question.

Sincerely yours,

A handwritten signature in dark ink, appearing to read "George Wright", with a stylized flourish at the end.

George W. Wright, M.D.
Head, Department of Medical Research

GWW/ep

PULMONARY FERRUGINOUS BODIES STUDIES ON THEIR ORIGIN

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Since the discovery by Thomson *et al.*¹ of structures indistinguishable from asbestos bodies in lungs of urban dwellers of Cape Town more than 6 years ago, his findings have been amply confirmed by investigators in large cities as well as some smaller communities in Europe and from North America.²⁻⁷ Although it has long been known that filamentous dust particles of diverse composition, when deposited in lungs, may cause the formation of bodies similar to asbestos bodies,⁸ these could generally be differentiated from asbestos bodies by their usually opaque and, at times, irregular contour of the central core, and they were then called pseudo-asbestos bodies. However, with the advent of industrial production of transparent, thin fibers approximating in size and optical microscopic appearance to some of the respirable asbestos fibers, the possibility arose that these man-made fibers could also cause the production of pseudo-asbestos bodies when inhaled. Recently it was demonstrated that these man-made fibers, when injected intratracheally into hamsters and guinea pigs, actually did cause the production of structures indistinguishable from asbestos bodies. With proof at hand that these golden to red-brown, elongated, bipolar-clubbed, but not necessarily segmented, iron-containing structures are not specific indications of the presence of asbestos fibers, these structures were given the generic name of 'ferruginous' bodies, reserving the term 'asbestos' body for those ferruginous bodies in which the central core is known to be an asbestos fiber.⁹

As Thomson has pointed out, the presence of ferruginous bodies in the lungs of urban dwellers suggest contamination of urban air with a filamentous dust resembling asbestos. Whether or not this dust actually is asbestos has not been established.

Because, at one time, the pathogenicity of asbestos dust was believed to be linked with

the production of innumerable traumatic punctures of tissue by the pointed ends of the dust fibers, in the minds of some all fibrous dusts were assumed to be endowed with a similarly high pathogenic potential. However, more than 10 years ago, a dust composed of filamentous ceramic aluminium silicate was shown to belong in that category of dusts designated as biologically 'inert'.¹⁰ This type of dust produces a pulmonary response having the following characteristics:

1. Collagenization is absent or insignificant;
2. The anatomic integrity of the air spaces is preserved;
3. The changes are potentially reversible.

This finding, of itself, should have laid the mechanical theory of the pathogenesis of asbestos dust to rest just as the biologic 'inertness' of diamond dust laid the mechanical theory of the pathogenesis of quartz dust to rest. Nonetheless, the dust of filamentous glass has become suspected of being fibrogenic, apparently on the basis of assumed mechanical trauma. Yet, glass dust derived from flakes of gossamer-thin glass, possessing sharp cutting edges, when deposited in air spaces, has been shown to be biologically 'inert'.¹¹

Rats and hamsters were exposed to 3 kinds of fibrous glass dusts; uncoated, coated with a phenol-formaldehyde type of resin, and coated with a starch binder. The concentration of the dusts in the inhalation chambers averaged 100 mg./m.³. About 60% of the dust particles were fibrous and their average diameter was 1 μ . After one year of exposure to this heavy concentration of all 3 types of dust, the pulmonary reaction consisted merely of focal accumulations of dust-filled macrophages localized largely to alveoli in the proximal portion of primary lobules. The reactive stroma associated with these cells consisted of a few delicate argyrophilic fibers. There was no destruction of alveoli and no

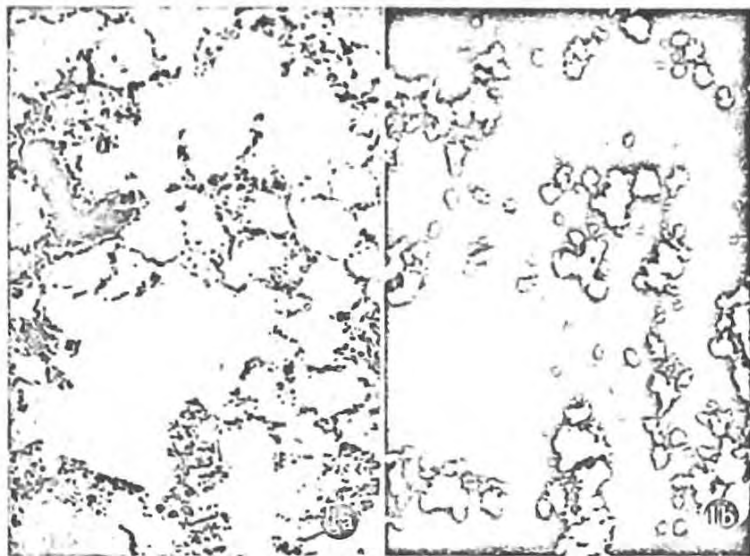


Fig. 1. The pulmonary response to inhaled fibrous glass dust consists only of macrophages which are filled with this dust. The macrophages are well preserved and show no evidence of degeneration. There is no fibrosis and alveolar walls are thin. From a rat exposed to fibrous glass dust for twelve months at 100 mg/m³. Hematoxylin and eosin. $\times 110$.

Fig. 2. Ferruginous body washed from the lungs of a hamster exposed to fibrous glass dust for twelve months at 100 mg/m³ concentration. The ferruginous material appears as though fractured. Nevertheless, the central glass core appears to be continuous. Unstained. $\times 960$.



collagen formation (Fig. 1). Abundant ferruginous bodies were found in the hamster lungs (Fig. 2). No differences in the reaction to the 3 different kinds of glass dust could be detected.

It has been suggested that one source of the ferruginous bodies in urban dwellers could very well be the asbestos that is comminuted by the friction of the asbestos-containing brake lining against the steel brake drum of automobiles. This possibility could

dust was rendered iron-free by magnetic separation. Although it contains a few scattered transparent fibers 50 to 75 μ long and about 1 μ in thickness, the X-ray diffraction pattern was negative for chrysotile. The dust is essentially non-fibrous. As this was intended to be a preliminary type study, only a few rats and hamsters were employed (10 of each). These animals were injected intratracheally with aqueous suspensions of the dust. The dosage was 2×10 mg. for rats

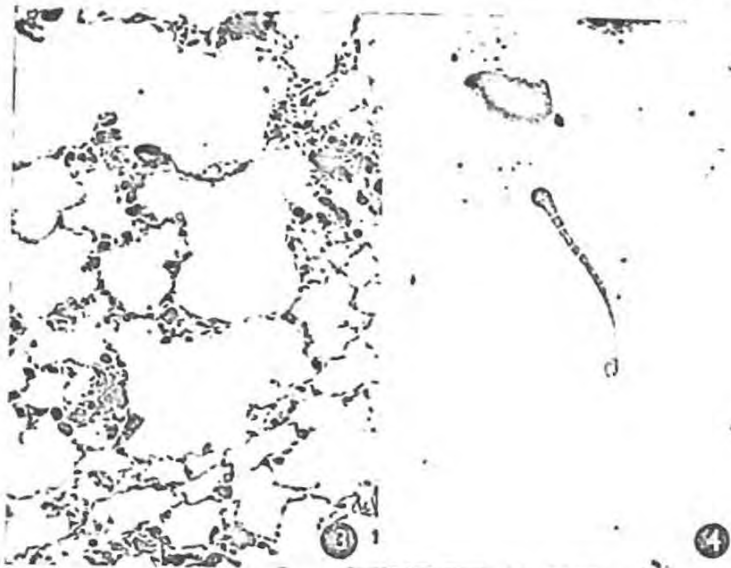


Fig. 3. This rat was injected intratracheally with 20 mg. of iron-free brake-drum dust and killed one year later. Similar to the reaction to fibrous glass, there is no fibrosis and alveolar walls remain normal. Air spaces contain collections of macrophages which stain darkly because of the ingested dust. Hematoxylin and eosin, $\times 110$.

Fig. 4. Ferruginous body from the lung of a hamster injected with brake-drum dust and killed 6 months later. This ferruginous body is an asbestos body. It was found in a smear of the sediment from saline injected into the lung. Perl's stain, $\times 900$.

be evaluated by animal experiments and was considered worthy of a pilot study before proceeding with a full-scale investigation.

Brake drum dust consists mainly of iron, a phenol-formaldehyde type resin, and forsterite. The latter is a mineral which is formed when chrysotile is heated so that it loses its water of crystallization. Brake drum

and 3×3.5 mg. for hamsters. The animals tolerated the dust well and they were killed one year later. The tissue reaction to this dust was very similar to that seen in the animals injected with fibrous glass, consisting of collections of dust-filled macrophages situated in alveoli clustered in the proximal portion of primary lobules. There was no

indication of collagenous fibrosis (Fig. 3). The hamster lungs contained many ferruginous bodies (Fig. 4).

In order to determine the nature of the dust causing the production of ferruginous bodies in the lungs of urban dwellers, it has become necessary to identify the material constituting the central core of the ferruginous bodies isolated from the lungs of these people. The method of isolation has been published¹² and involves the digestion of small pieces of lung tissue (about 300 gm. wet weight) with 5% sodium hypochlorite (household bleach) for 24 hours, removal of the remaining undigested lung tissue, and resuspension of the centrifuged sediment in a mixture of 50% alcohol and chloroform. Ferruginous bodies, because of their high iron content, have a high specific gravity and will be found in the sediment at the bottom of the tube. Carbonaceous material, if not dehydrated by excessive strength of alcohol, will be found at the interphase between the chloroform and the aqueous alcohol. The sediment containing the ferruginous bodies is washed and resuspended in water. Ferruginous bodies are transferred to electron microscope grids from such suspensions by means of micro-manipulators. It may be of interest to observe that the sediment from the lungs consists mostly of angulated colorless, glass-like particles and often contains many more naked fibers of different sizes than ferruginous bodies. Some of these naked fibers are obviously man-made, since the ends show evidence of heat-softening as indicated by the formation of loops, shepherd crooks, or merely bulbous enlargement.

Twenty-eight ferruginous bodies from as many lungs of city dwellers from Pittsburgh that had been transferred individually to electron microscope (E-M) grids were examined by electron diffraction at the Mellon Institute (Carnegie-Mellon University), along with 6 ferruginous bodies similarly isolated from lungs of hamsters injected intratracheally with iron-free brake drum dust and then transferred to E-M grids.¹³

Inasmuch as the iron encrustation in the ferruginous body interferes with the production of a satisfactory electron diffraction pattern, the central filament had to be scanned from end to end to find a region, at least $500 \times 500 \text{ \AA}$, which was free of iron deposits. Such an area was found on each of the ferruginous bodies examined and provided a satisfactory electron diffraction pattern.

Chrysotile, which constitutes about 90-95% of all asbestos used in the United States, is different from other types of asbestos in that it is composed of tubular crystals which give it a unique electron diffraction pattern. The other types of asbestos, composed of solid filaments, require more exact orientation of the fiber in order to obtain the same electron diffraction pattern as that of a standard sample.

Not one of the consecutive 28 ferruginous bodies isolated and analyzed yielded an electron diffraction pattern which could be identified as that of chrysotile (Fig. 5). On the other hand, of the 6 ferruginous bodies isolated from hamster lungs injected with brake drum dust, all gave an electron diffraction pattern characteristic of chrysotile (Figs. 6 and 7); this, in spite of the relative paucity of fibers in the dust by optical microscopy and the negative X-ray diffraction pattern of the powder. These 6 ferruginous bodies were, therefore, asbestos bodies.

Since the ferruginous bodies from the human lungs failed to give the electron diffraction pattern of chrysotile, the latter can be excluded as the central fiber in all bodies examined to date. However, because a diffraction pattern was demonstrated, the fibers must necessarily have a crystalline structure. This excludes such vitreous materials as glass, mineral or rock wool, and other ceramic fibers. At the same time, the diffraction pattern is not sufficiently similar to patterns of known samples of amosite or crocidolite to allow positive identification of either, but neither is it possible to exclude these or possibly some of the other forms of asbestotic minerals.

A recent report of an analysis of ferruginous bodies isolated from the lungs of shipyard workers in Flushing, Netherlands, has shown that these were indeed asbestos bodies, inasmuch as the central cores consisted of amphibole asbestos.¹⁴ Such findings were to have been expected since shipyard workers are exposed to amosite and crocidolite asbestos. It should be noted, however, that the above results do not necessarily apply to the ferruginous bodies of urban dwellers who are not occupationally exposed to asbestos.

Since 90-95% of the asbestos used in the United States is chrysotile, one would expect chrysotile to constitute the central core of at least some of the ferruginous bodies isolated from the lungs of city dwellers, i.e. if these people were actually exposed to asbestos dust.

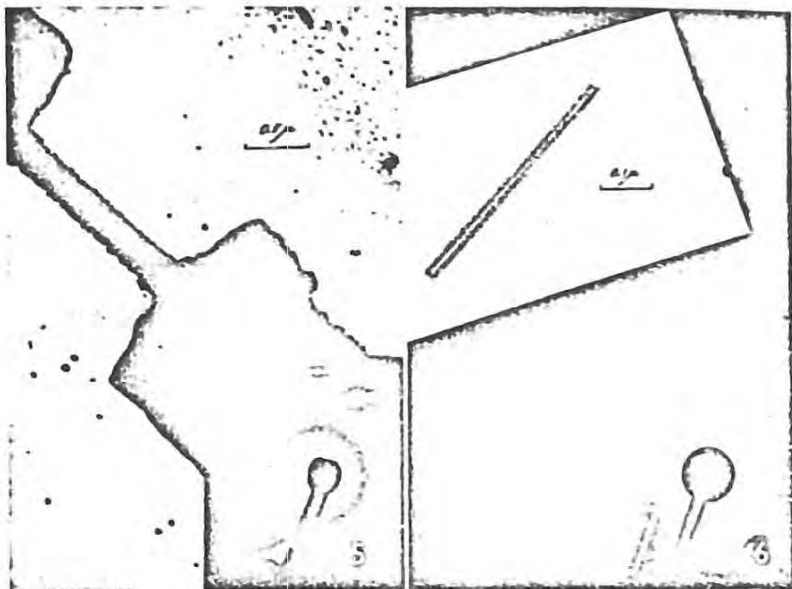
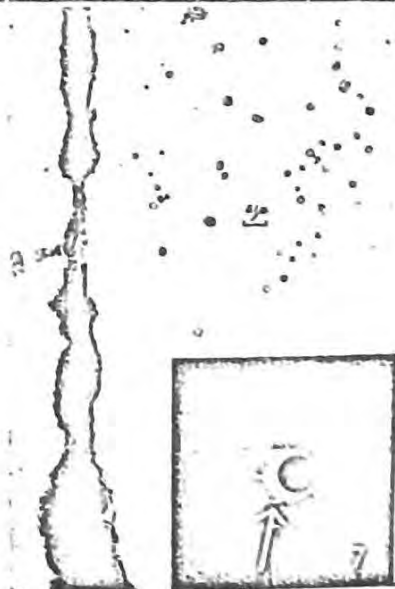


Fig. 5. Electron photomicrograph of a portion of a ferruginous body from a human lung. The region photographed was selected because it is not covered by ferruginous material. It measures about $0.3 \times 1.6 \mu$ and is from where the electron diffraction pattern was obtained (lower right corner). This pattern is unlike that obtained with chrysotile (compared with Fig. 6).

Fig. 6. Electron photomicrograph to show the central cavity of a tubular crystal of chrysotile. The electron diffraction pattern of chrysotile is shown in lower right corner. The characteristic and unique feature of this electron diffraction pattern is the exclamation-mark or tear-drop shape of the pips on the layer lines (arrow).

Fig. 7. Portion of a ferruginous body, as seen under an electron microscope, from the lung of a hamster infected with brake-drum dust intratracheally and killed one year later. The naked portion of the central core measures about $0.06 \times 0.3 \mu$. The electron diffraction pattern from this region of the core (lower right corner) resembles that of chrysotile (compared with Fig. 6). This ferruginous body is, therefore, an asbestos body.



It is, of course, possible that in continuing this investigation and isolating ferruginous bodies from the lungs of many more people and examining more than one body per lung, asbestos fibers may be identified in their cores. Nevertheless, the failure to find a chrysotile core in any of the 28 consecutive ferruginous bodies examined is considered significant.

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Experimental Pulmonary Carcinogenesis with Asbestos

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Three methods have been used to test the carcinogenic response of the lung and pleura of the rat to asbestos. No carcinogenic response of pulmonary epithelium or pleura was noted to either healing infarcts or implanted wax pellets that contained asbestos. Sarcomas of the pleura and pericardium developed in 74% of rats in which the pleura was covered with an asbestos-impregnated fibrous glass coat. The fibrous glass alone caused only slight reaction, with complete healing. The last method may serve as an accurate and convenient means of quantitating the neoplastic response of the pleura to asbestos.

Introduction

IN MAN, A CAUSAL relationship between pulmonary cancer and asbestosis has been suspected since 1935.^{1,2} Recent epidemiological studies have supported this idea and have indicated further that exposure to asbestos without the development of asbestosis may be sufficient to cause carcinomas of the lung, carcinomas of the gastrointestinal tract, and particularly mesotheliomas of the pleura and peritoneum.³⁻⁸ While it is understandable that epidemiological data might require a number of years for collection, it is unfortunate that some 30 years elapsed between the association of asbestosis and cancer noted by Gloyne and by Lynch and Smith, and the experimental demonstration that asbestos could indeed cause cancer. Recent experimental studies have adequately demonstrated that pleural mesotheliomas can be induced by several types of asbestos in both rats and hamsters.⁹⁻¹¹ Efforts to induce cancers of the respiratory tract have been less successful, but a recent report indicates that carcinomas of the lung occasionally develop in very old rats that for 62 weeks have inhaled chrysotile asbestos particles presumably contaminated with traces of nickel, chromium, and cobalt.¹² Thus, both epidemiological and experimental studies of the relationship of pulmonary can-

cer to asbestos exposure have a strong beginning, and more refined methods are now being sought to clarify this relationship.

One of the prerequisites for a quantitative study of carcinogenesis is a method that induces cancers rapidly and consistently. The method need not mimic exactly the conditions thought to induce cancer in man as long as the suspected carcinogen induces cancers of the appropriate type in the appropriate organ. The purpose of the three experiments described in this report was to induce cancers of the pulmonary epithelium or pleura in the most effective manner possible. Consequently, somewhat unconventional methods were used.

Methods

In all three experiments, 8- to 12-week-old female Osborne-Mendel rats were used. Each experimental group had 20 to 50 rats exclusive of rats that died in the first 30 days. Observation periods extended from 12 to 24 months, depending on mortality during the experiment. In old age, this strain has a relatively high incidence of chronic pneumonitis and neoplasms of the mammary gland, adrenal cortex, and genital tract. All of these tumors were taken into account by parallel untreated groups and the continuing patho-

TABLE I
Pulmonary Response to Chrysotile in Infarcts

Material	Dose per Rat	Duration of Experiment (months)	Deaths* (%)	Infarct Scar Characteristics	Neoplasms		
					Pulmonary	Mammary %	Other
Fluorocarbon (alone)	0.05 ml	20	22	Simple fibrous	None	24	2 Adrenal CA
Asbestos + Fluorocarbon	(1 mg/0.05 ml) X 2	18	17	Extensive pleural fibrosis	None	13	1 Small intestine leiomyosarcoma
3-MCA ^b + Fluorocarbon	(1 mg/0.05 ml)	16	30	Keratinizing epithelial metaplasia	25% Epidermoid CA	8	1 Uterine sarcoma 1 Subcutaneous sarcoma

*% deaths during experiment. Each group, exclusive of deaths within the first month of the experiment, consisted of 25 to 30 rats (8- to 12-week-old female, strain OM N).

^b3-Methylcholanthrene

logical analysis of several thousand rats of the same sex and strain that have been necropsied in the past ten years. During this time no tumors of the lung have been observed in untreated rats.

All rats that died or were killed were necropsied. Routinely, histologic sections stained with hematoxylin and eosin were studied from lungs fixed by intratracheal instillation of formalin, and from all other grossly apparent lesions. Selected tissues were stained with Masson's trichrome, Wilder's reticulum stain, periodic acid-Schiff stain, and the Rinehart and Abul-Haj method for acid mucopolysaccharides.

The asbestos used in the first experiment was an ore sample of soft-fibred Canadian chrysotile that was pulverized in this laboratory using only a pyrex glass chamber as the pulverizer. Pulverization proceeded until all clumps and particles were less than 5 microns in least diameter. Direct microscopic examination indicated that the bulk of the particles were asbestos spicules that measured up to 100 microns in length and less than 1 micron in diameter.

In the last two experiments, the asbestos was samples received from Dr. J. C. Wagner that had been shown to be carcinogenic for the pleura of the rat.^{9,10} Data regarding particle size and hydrocarbon content of the pulverized asbestos from which the samples came are given in the literature.^{10,12} Dr. Lewis J. Cralley and his associates of the Bureau of Occupational Safety and Health, Cincinnati, have recently analyzed by atomic

absorption similar samples from Dr. Wagner and have found appreciable amounts of nickel, chromium, cobalt, and manganese in these samples.^{14,15}

Pulmonary Response in Infarcts

Our first experiment was based on the premise that regenerating tissues at the site of infarction might be more susceptible to carcinogens than normal tissues. Previous work indicated that 0.05 ml of an inert fluorinated hydrocarbon (hexachlorotetrafluorobutane), when injected intravenously in 250-gm rats, occludes segments of peripheral pulmonary capillaries with resultant ischemic infarction of approximately 20% of lung tissue.¹⁶ Known chemical carcinogens added to the injected fluorocarbon are trapped at the sites of

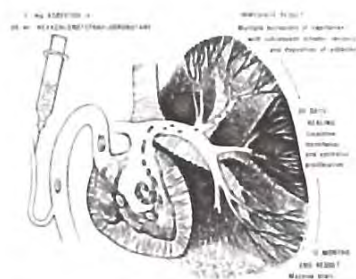


FIGURE 1. Method of depositing asbestos in pulmonary infarcts (2 mg asbestos in 0.05 ml hexachlorotetrafluorobutane).

TABLE II
Pulmonary Response to Asbestos in Beeswax Pellets

Material	Dose ^a per Rat	Duration of Experiment (months)	Deaths ^b (%)	Cyst Types ^c (%)				Neoplasms	
				GR → KEC	→ KECI	→ CA	Mammary %	Other	
Beeswax (alone)	—	24	21	100	—	—	32	1 Adrenal CA	
3-Methyl- cholanthrene	50 µg	12	27	17	63	11	9	18	None
3-Methyl- cholanthrene	1 mg	12	64	—	18	18	64	17	1 Abdominal sarcoma
<i>Asbestos:</i>									
Canadian chrysotile	5mg × 2	24	53	All pellet lesions granulomatous				47	2 Adrenal CA 1 Ovarian CA 1 Leukemia
Rhodesian chrysotile	5mg × 2	24	35					53	2 Adrenal CA 1 Uterine CA
Amosite	5mg × 2	24	60					35	1 Adrenal CA 1 Uterine sarcoma 1 Subcutaneous sarcoma
Crocidolite	5mg × 2	24	35					37	1 Adrenal CA 1 Ovarian sarcoma

^aEach pellet contained 0.05 ml of a 1:1 mixture of purified USP beeswax-tricaprylin to which test material was added. Each rat received one pellet upper half and one pellet lower half left lung.

^b% deaths during the experiment. Each group, exclusive of deaths within first month, consisted of 20 to 28 rats (12-week-old, female, strain OM/N).

^cGR = granulomatous cyst wall; KEC = keratinizing epidermoid cyst wall; KECI = keratinizing cyst with invasion; CA = epidermoid carcinoma.

recognizable by their destructiveness, gross invasion, and metastases. Frequently the carcinomas are poorly differentiated, but consistently they possess keratinizing epithelial cells indicating that conversion to squamous epithelium may play a role in their initial development. Of particular importance is the fact that, with a known carcinogen, the degree of response varies with the dose. Two examples of this variation with different doses of 3-MCA are shown in Table II. With 1 mg of 3-MCA in the pellet, more than half the lesions are carcinomas, while the remaining lesions are divided equally between those showing simple keratinization and those with keratinization and local invasion. In contrast, 50 µg of 3-MCA induces only a few carcinomas, but the wall of most pellets still shows conversion to keratinizing squamous epithelium, and at even lower doses keratinization of the wall occurs even though no carcinomas are induced. If one assumes the three types of lesion to represent degrees of neoplastic conversion, then keratinizing squamous metaplasia in itself is probably a sensi-

tive indicator of minute amounts of carcinogen.

Table II indicates the result of adding four different types of asbestos in 5-mg amounts to the pellets and depositing them into the pulmonary parenchyma of both the upper and the lower half of the left lung of 12-week-old female Osborne-Mendel rats. From 20 to 28 of 30 rats survived the initial operative procedure. In each experiment more than 90% of the survivors lived more than one year following the operation. The table indicates the percentage surviving two years. No neoplasms were found in the lungs of any of these rats, nor was there evidence of keratinizing metaplasia, the harbinger of carcinogenicity that one might have expected if the carcinogenic potential of asbestos for respiratory epithelium was low.

The number of neoplasms at other sites was carefully analyzed also, and although a high incidence of mammary tumors and a good scattering of other types of neoplasms are indicated in Table II, it should be remembered that most of these rats were 27 months



FIGURE 3. Method of exposing the pleura to a fibrous glass pledget saturated with asbestos.

old and controls of comparable age have comparable numbers of background tumors.

Pleural Response: Asbestos—Fibrous Glass Pads

The third series of experiments was designed to evaluate the response of the pleura to asbestos. Many of these experiments are still in progress, since it has become apparent that at least 2 years of observation are necessary for results, but the first experiments are completed. Two factors determined the method outlined in Figure 3. First, we wanted to fix asbestos to a large area of the mesothelial surface. Second, we wanted to learn whether nonspecific irritation of the pleura might play a role in carcinogenesis, since it has been suggested that pleural effusion and mesothelial cell proliferation in response to injury is in effect an *in vivo* cell culture that might simply follow the tendency of *in vitro* cell cultures to become neoplastic.¹⁹

A thin coat of fibrous glass was a simple means of inducing nonspecific irritation of the pleura, and it also served as a scaffold for retaining asbestos in contact with a wide area of the pleural surface. Autoclaved fibrous glass of the type commonly used as insulating material was used.

The fibrous glass, as manufactured, consists of a meshwork of long, intertwined, flexible strands of glass that are coated with a heat-cured phenol formaldehyde resin. The fibers average 6.5 microns in diameter, with a few fibers as great as 25 microns in di-



FIGURE 4. Fibrous glass pledget after infiltrating with crocidolite asbestos. Note adherence of asbestos fibers to surface of glass fibers. 850 X.

ameter, and less than 3% in the approximate range of asbestos fibers with diameters of 1 micron or less.

From this material, 30 × 20 × 3-mm pledgets were cut and trimmed to weigh 45 mg. Then, for convenience and safety in handling, 40 mg of crocidolite asbestos was suspended in 2 ml of 10% solution of gelatin, and each pledget was saturated with this mixture. Control pledgets were treated similarly with gelatin alone, and the gelatin was allowed to harden. It was of particular interest that the asbestos fibers in the pledgets tended to adhere and line up as barlike projections along the glass fibers (Figure 4). This illustration also emphasizes the tenfold difference in the size of most of the glass fibers and the asbestos particles. The crocidolite asbestos was the same as that used in the previous experiment and was composed of iron-containing amorphous granules as well as spicules



FIGURE 5. Reaction within and around asbestos-saturated fibrous glass pledget.
65 $\times$

that measured less than 1 micron in diameter and 10 to 100 microns in length.

By open thoracotomy, the fibrous glass pledgets were placed over the surface of the left lung and pericardium of seventy 12-week-old female Osborne-Mendel rats that were randomly distributed into equal control and asbestos groups. Six of the rats died with pneumonia in the first month. In these, both control and asbestos-saturated pledgets were firmly attached to the pericardium and visceral pleura by granulation tissue. However, in rats that died subsequently, a marked difference in reactivity of the pleural and pericardial surfaces was noted. Pledgets without asbestos were gradually absorbed. The resin coating on the glass was rapidly sequestered in phagocytes. The glass fibers were broken down more slowly, but in those rats that survived 23 months only a thin layer of scar tissue remained over the lung, and no local neoplasms occurred.

In contrast, development of fibrous tissue seemed to be progressive in and about the asbestos-laden glass fibers, and thick fibrous

coats invested the left lung and pericardium of all rats in the experimental group (Figure 5). Grossly, this fibrous tissue was sufficiently extensive to appear as tumor in every rat, but on histological examination the distinction between neoplasm and reactive fibrous tissue could be made without difficulty.

After 23 months, 22 of the 30 rats surviving more than one month had mesothelial sarcomas at the site of the crocidolite-saturated pledgets (Table III). The first death from this type of tumor occurred 16 months after treatment, and 5 of 6 survivors at 23 months had sarcomas. More than half the neoplasms seemed to be derived from the pericardial mesothelium, and compression of the heart was the most common cause of death (Figure 6). The pattern of spread was over serosal surfaces to the thorax and opposite lung. In one rat, direct extension to abdominal viscera occurred. Hilar lymph nodes consistently contained asbestos fibers and other pigmented particles, but no lymphatic or vascular metastases were found. All neoplasms were of mesenchymal origin, with

TABLE III
Response of Pleura and Pericardium to Crocidolite Asbestos in Fibrous Glass Pledgets

Material	Dose per Rat	Duration of Experiment (months)	Deaths (%)	Mesothelial Sarcomas ^a (%)	Neoplasms: Mesothelioma (%)	Other
Fibrous glass (alone)	45mg/pul	25	50	None	45	1 Adrenal CA 1 Liver CA
Fibrous glass + crocidolite	glass + 45mg	25	80	5	5	1 Mesenteric granuloma

^aeg. deaths during the experiment. Each group consisted of death within first month increased of 25 to 30 rats (12-week-old, female, strain OM/N).

^bFirst mesothelial sarcoma was lethal at 16 months. 12 of 22 mesothelial sarcomas were apparently derived from pericardium.

Tumor types: Fibrogenic spindle-cell type 5/22
Fibrogenic spindle with tubular component 6/22
Fibrogenic spindle with sarcomatous component 1/22
Polymorphous, small cell type 4/22

no evidence of neoplastic transformation of adjacent pulmonary epithelium. Variation in the type of sarcoma did occur, as indicated in Table III and Figures 7 and 8. No increase

in tumors outside the lung occurred. The single exceptional lesion was a mesenteric granuloma that contained asbestos particles.

Discussion

The first two experiments designed to test the response of pulmonary epithelium are worth little comment, since no neoplasms resulted. The pellet experiment has been effective with other carcinogens, and since Gross and associates¹⁵ have shown that the pulmonary epithelium of the rat will respond to the types of asbestos that we used, it is probable that the carcinogenicity of asbestos is not the result of a soluble or easily mobilized component of the fiber—a conclusion in general agreement with the findings of others, but one that still requires a complete study of the natural-occurring and processing oils and metals that contaminate asbestos fibers.^{15,20} The deposition of asbestos in infarct sites suggests that reactivity to asbestos is greater in the pleura than in the pulmonary parenchyma. The failure to induce mesotheliomas most probably was related to the small amount of asbestos present. Covering the pleura and pericardium with an asbestos-saturated coat of fibrous glass confirmed previous observations that asbestos is a strong carcinogen in these sites. Not only is the method convenient, but the high incidence of neoplasms makes it effective for quantitative studies. We have not hastened the development of neoplasms,²⁰⁻²² and an observation



FIGURE 6. Neoplasm occupying the anterior mediastinum and completely enveloping the heart. 1.2 X.

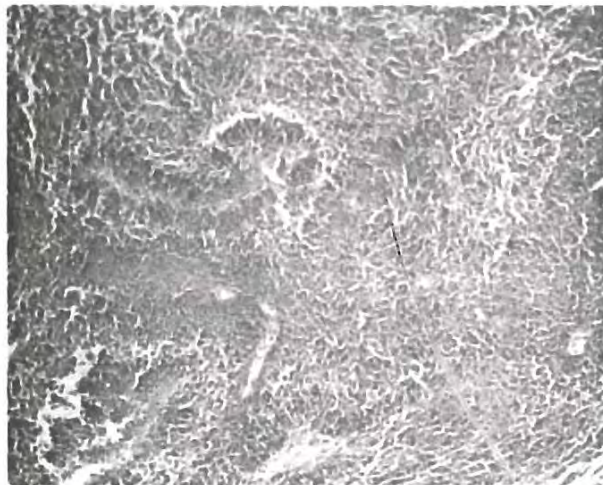


FIGURE 7. Growth pattern commonly noted in the mesothelial sarcoma (H&E).

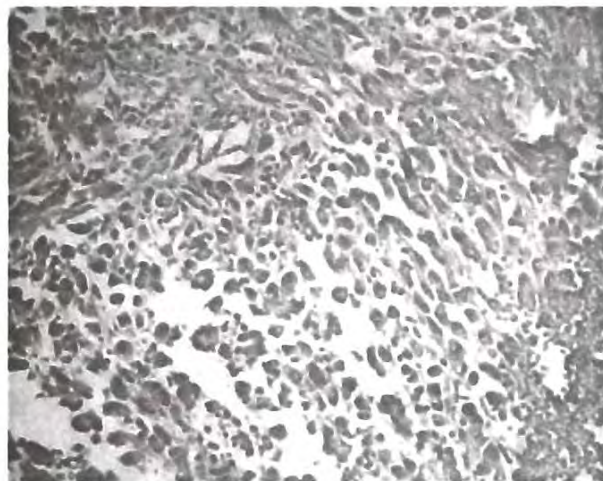


FIGURE 8. Growth pattern of the small polymorphous cell type of mesothelial sarcoma. This type is sometimes considered "epithelial-like" (H&E).

period of 24 months must be considered essential. Variation in cell type in the neoplasms is like that seen in the mesothelial tumors in man^{22,23} and is perhaps more attributable to selective modulation than to variation in the type of cell that responds. In relation to pathogenesis, the asbestos-saturated pledgets caused extensive and progressive fibrosis in all rats, and the neoplasms always appeared to arise from this overt mesothelial response, suggesting that reactive proliferation may be a primary step in neoplastic development. We had expected the fibrous glass to call forth a healing response nearly as vigorous as that of asbestos. Clearly this was not the case, the fibrous glass showing only negligible reactivity at the end of 23 months. The explanation for this difference in reactivity is not clear, since Wagner has been able to induce a vigorous pleural fibrosis and neoplasms with intrapleural injections of silica.¹⁹ It is perhaps important that the fibrous glass differed from the silica used by Wagner—first, in that the glass fibers were much larger than the silica particles, and second, in that they were coated with a phenol-formaldehyde resin. It would be interesting to learn what influence a similar resin coating would have on the fibrogenic and carcinogenic potential of asbestos.

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Smoke

According to the Washington, D. C., Inter-agency Council on Smoking, these are some of the gases or chemical compounds in smoke from cigarettes: acetone, acetaldehyde, acetylene, acrolein, ammonia, butane, carbon dioxide, carbon monoxide, ethane, formaldehyde, hydrogen cyanide, hydrogen sulphide, methane, methanol, methyl ethyl ketone, methyl chloride, methyl nitrate, nitrogen dioxide, propane, and propylene.

MORTALITY EXPERIENCES OF ASBESTOS INSULATION WORKERS

1943 - 1968*

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On December 31, 1942, there were 632 members of the Asbestos Workers Union in the New York area (New York and Newark, New Jersey locals of the International Association of Heat and Frost Insulators and Asbestos Workers, I.A.H.F.I.A.W.). From January 1, 1943 to December 31, 1962 another 890 men joined the union. We have followed each of these 1,522 men prospectively to the present. Four hundred and four died by December 31, 1968. Analyses of these deaths demonstrate that serious risks have been associated with work in this trade.

own observations and those of others^{1,2} indicate that trade practices are essentially uniform through the country, the data which follow may be taken to represent mortality experience in this trade for the United States and Canada, direct study of all United States union insulation worker deaths, 1967-1968, supports this.³

Exposure: There is scattered information recorded concerning variation in insulation materials used in the United States, 1870-1969^{1,2}; it would appear that some materials (as, hair-felt, cork, mineral wool) have sharply decreased while others (as, fibrous glass, plastics) have increased.⁴ Table 1 lists material currently used.

TABLE 1: INSULATION MATERIALS (U.S.)-1969*

Product	Asbestos Content
Blankets	100% - amosite
Moulded	95% - amosite (5% filler)
Insulation block	(a) 85% magnesite: 10-15% amosite + chrysotile
	(b) 85% cal. silicate: 10-15% amosite
	(c) 85% cal. silicate: 10-15% amosite + chrysotile
	(d) 85% calcined diatomaceous silica
	10-15% amosite + chrysotile
	(e) 85% calcium silicate: 10-15% chrysotile
Finishing 'cements'	(a) 100% chrysotile
	(b) 50% chrysotile, 50% cement
Sprayed asbestos	10-15% asbestos-variable
2. Fibrous Glass and/or Mineral Wool	
Block, rolls, prefabricates, high temperature insulation, finishing cements, mineral wool and cement.	
3. Plastics	
Polystyrenes and polyurethanes: prefabricated, powdered and foamed in place.	
4. Other	
Cork, glass flakes, adhesives (epoxy and solvents; asphalt base and solvent, wheat paste, silicones, magnesia, portland cement).	

MATERIALS AND METHODS

The procedures followed in this study have been described.^{1,2} Briefly, these men install insulation in the construction and (to a limited extent) in the shipbuilding industries. Approximately 10% of United States union insulation workers are in the New York-New Jersey locals and since our

The quantity of asbestos used annually in insulation materials has not changed very much in the past fifty years—12,500 tons in 1920 and 16,000 tons in 1965 (Table 2).⁴ There are currently, however, more insulation workers than previously; pre-

*This study was supported by the Health Research Council of the City of New York.

sumably, less asbestos per workman is now the case. Asbestos exposure varies by type of installation (Table 3); about 40% of an insulation

TABLE 2: ASBESTOS USED IN INSULATION MATERIALS IN THE U.S., 1920-1965

Year	Short Tons
1920	12,300
1925	19,200
1930	16,600
1935	7,400
1940	13,500
1945	14,800
1950	12,700
1955	12,200
1960	13,000
1965	16,100

TABLE 3: USE OF ASBESTOS INSULATION (%) VARIATION BY CONSTRUCTION TYPE 1969

Construction	Fibrous Glass	Asbestos	Other
Commercial (Schools, factories)	75	10	15
Power process (Power plants, boiler rooms)	20	60	20
Marine	20	70	10

workers' time is spent in work using asbestos materials (Table 4).

TABLE 4: USE OF ASBESTOS BY INSULATION WORKMEN IN THE U.S., 1969

Material	Volume % of Total	Application % of Time
Asbestos	35	43
Fibrous glass	45	40
Other	20	17

Exposure of insulation workers in the United States cannot necessarily be equated with that of similar workmen in other countries. For one thing, besides variations in working conditions, the type of fiber and materials used may be different. Until 1930, chrysotile was almost the only asbestos used, amosite making its appearance later and crocidolite practically not at all (see below). Second, spraying of insulation has not been, until recently, widely used in this country, in contrast to some other areas and, insofar as this effects exposure, would not be reflected in our mortality observations. Finally, because of union jurisdictional agreements, the men we have studied are restricted to insulation against temperature changes; acoustical insulation and fireproofing are the work of men in other trades and variables peculiar to such work would not be demonstrated by our data.

OBSERVATIONS

632 MEMBERS OF THE UNION ON DECEMBER 31, 1942

Three hundred and eighty of these men were dead by December 31, 1968. Age, year and sex specific expected death rates were available to mid-1967;^{1, 10} observed rates by cause are compared in Table 5. All deaths to December 31, 1968 are tabulated in Table 6, by cause.

TABLE 5: EXPECTED AND OBSERVED DEATHS AMONG 632 NY-NJ INSULATION WORKERS, JANUARY 1, 1943 TO APRIL 30, 1967*

	Expected**	Observed
All causes	251.0	349
Lung Cancer	8.9	66
G.I. Cancer	11.2	37
Pleural Mesothelioma	***	6
Peritoneal Mesothelioma	***	14
All other Neoplasms	25.0	21
Asbestosis	***	27

* Of workmen reaching 20 years from first exposure. In addition seven men died before reaching this point.

** Based on United States Mortality data.^{1, 10}

*** United States data not available, but these are rare causes of death in general population.

TABLE 6: CAUSES OF 380 CONSECUTIVE DEATHS AMONG 632 INSULATION WORKERS JANUARY 1, 1943 TO DECEMBER 31, 1968

	Before 20 Years from First Exposure	After 20 Years from First Exposure
Lung cancer	0	72
Pleural Mesothelioma	0	6
Peritoneal Mesothelioma	0	16
Gastrointestinal cancer	0	37
Oropharynx Larynx Cancer	1	5
Cancer of Pancreas	0	3
All other Neoplasms	0	19
Asbestosis	0	30
All other causes	6	185
	7	373

LUNG CANCER

One in five deaths was caused by lung cancer. Despite the recent rise in prominence of mesothelioma, this remains the most important asbestos-associated neoplasm among insulation workers. It is of interest, therefore, that there appears to be a special relationship to

TABLE 7: PLEURAL AND PERITONEAL MESOTHELIOMA AMONG NYC ASBESTOS INSULATION WORKERS,* JANUARY 1, 1943 TO DECEMBER 31, 1968

		Mesothelioma			
		Pleural 6	Peritoneal 16	Bronchogenic Carcinoma 72	Asbestos 30
Number					
<i>Age at Onset (Years)</i>					
Mean	.	25.2	20.0	25.4	24
Median	.	21	19	25	24.5
S.D.	.	±5.4	±4.3	±6.36	±5.6
Range	.	17-25	10-30	16-54	14-36
<i>Age at Death (Years)</i>					
Mean	.	56.0	62.7	63.8	66.7
Median	.	55	60	64	66
S.D.	.	±2.4	±10.2	±7.25	±8.02
Range	.	54-60	50-83	39-83	53-79
<i>Lapsed Period (Years)</i>					
Mean	.	34.8	43.0	38.7	42.8
Median	.	34.5	40.5	39	44
S.D.	.	±2.3	±8.9	±8.5	±7.69
Range	.	32-38	32-64	22-60	30-59

*In addition, we have observed 2 peritoneal mesotheliomas found at autopsy, in men who had died of bronchogenic carcinoma.

cigarette smoking among asbestos insulation workers¹⁰ (see below). Overall lung cancer death rates were seven times expected.¹¹

MESOTHELIOMA

Twenty-two of the 380 deaths were due to mesothelioma, 6 pleural and 16 peritoneal. Two occurred 1943-1954; 20, 1955-1968; average lapsed period from onset of exposure was longer and average age at death was greater for peritoneal than for pleural mesothelioma.

Peritoneal mesothelioma does not seem to be related to cigarette smoking—we have seen 4 such cases in men who never smoked cigarettes. In the 3 cases of pleural mesothelioma for which we personally recorded smoking histories, all had smoked cigarettes.

The preponderance of peritoneal mesothelioma among our cases, as in the series of Enticknap and Smither,¹² is not necessarily contradictory to the observations of Wagner,¹³ Hourihane¹⁴ and others in which pleural neoplasms were largely seen. It may, rather, reflect the derivation of the cases—consecutive series with regular occupational exposure on the one hand, against random cases, many with non-occupational environmental ex-

posure, on the other. It may be that in those people who develop mesothelioma in association with asbestos exposure, the site of the mesothelioma tends to vary to some extent with the intimacy and duration of exposure—pleural with lesser exposure and peritoneal with greater.

GASTROINTESTINAL CANCER

As in our first observations,¹ we have continued to see more deaths due to cancer of the stomach and colon than expected. However, the ratio is only 3 times expected and, with so few deaths, we are not yet certain of the relationship.

CANCER OF PANCREAS; OROPHARYNGEAL CANCER

The same insecurity noted with gastrointestinal cancer is associated with these as well. We are, however, concerned that the incidence of both of these neoplasms will be increased among asbestos workers. As a practical measure, we now inspect the oral cavity during all clinical examinations of asbestos workers; one cancer of the lip has been discovered and successfully excised.

It is of interest that 2 of the 3 recent deaths of tongue cancer, were in men who never smoked cigarettes.

*There are, in addition, three men found to have lung cancer during our survey, and who are now alive following therapy.

ASBESTOSIS

Thirty deaths of the 380 were due to respiratory insufficiency with cor pulmonale.

890 MEN ENTERING UNION AFTER DECEMBER 31, 1942

Fifty-four of these men had prior insulation work experience before entering this union in 1943 or later; 836 did not. By and large, therefore, the men in this group are younger and would not be expected to have yet achieved a lapsed period from onset of exposure sufficient to have brought them to a period of serious risk of neoplastic death. This theoretical consideration has so far been confirmed by our experience (Table 8).

TABLE 8: ONSET OF EXPOSURE AND DEATHS OF NEOPLASM AMONG 890 MEN WHO JOINED INSULATION WORKERS' UNION, 1943-1962

Year Onset of Asbestos Insulation Exposure	Number	Deaths 1943-1968	
		Total	Neoplasm
1 ≤1929 . . .	8	3	3
2 1930-1942 . .	46	2	1
3 1943-1947 . .	164	9	4
4 1948-1952 . .	250	5	0
5 1953-1957 . .	186	5	0
6 1958-1962 . .	236	0	0
	890	24	8

1: CA Lung (32), CA Lung (41), Leukemia (31).

2: CA Lung (31), Pancreatitis.

3: CA Lung (21), Asbestosis, Peptic Ulcer, CA Pancreas (20), CA Tongue (20), CA Colon (17), Palm. embolism, Cirrhosis, Coronary.

4: Accident, Coronary, Cong. Heart Failure, Coronary, Coronary.

5: Pneumonia, Rheumatic, H.D., Coronary, Cirrhosis, S.B.E.

6:

() = years from onset of exposure.

MULTIPLE NEOPLASMS

We have seen several instances of more than one primary neoplasm in the same individual (lung-lung; lung-larynx, etc.). These have been too infrequent to warrant more than brief recording except perhaps for two men who died of bronchogenic carcinoma and were found, at autopsy, to also have peritoneal mesothelioma.

EFFECTS OF CIGARETTE SMOKING

There appears to be an important influence of cigarette smoking on the incidence of lung

cancer among asbestos insulation workers.*

On January 1, 1963, there were 370 men still alive of our original 632 cohort (all now with long-lapsed periods from onset of exposure and very much at risk). Eighty-seven never smoked cigarettes: one died of lung cancer by December 31, 1968. Two hundred and eighty-three had a history of cigarette smoking—27 have died of lung cancer (Table 9).

TABLE 9: CIGARETTE SMOKING AND LUNG CANCER IN ASBESTOS INSULATION WORKERS. EXPERIENCE OF 370 MEN FOLLOWED PROSPECTIVELY JANUARY 1, 1963 TO DECEMBER 31, 1968

	No History of Cigarette Smoking	History of Cigarette Smoking
Number of men . . .	87	283
Expected lung cancer deaths to 4.30.67 . . .	0.15	2.83
Observed deaths to 4.30.67 . . .	0	24
Observed deaths to 12.31.68 . . .	1	27
Total cases to 12.31.68 . . .	1	30*

*3 men still alive (all cigarette smokers).

Computations suggest that an asbestos worker who smokes cigarettes has 90 times the risk of dying of lung cancer compared with a man who neither works with asbestos nor smokes cigarettes.

AGE AT ONSET OF EXPOSURE

Table 10 records the mortality experience of the 632 men, by age category at onset of exposure.

FIBER VARIETY AND MESOTHELIOMA; EVIDENCE FROM PRESENT OBSERVATIONS

Crocidolite seemed initially to be unique among asbestos varieties in having a special relationship to mesotheliomas. It has been considered desirable to obtain further evidence on this question, by study of populations exposed to other fiber varieties.^{11,12} It may be useful, therefore, to evaluate the experiences of N.Y.-N.J. insulation workers in this regard.

CROCIDOLITE EXPOSURE OF INSULATION WORKERS IN THE UNITED STATES

There is no crocidolite mined in the United States: all must be imported either from South Africa, Bolivia or Australia. It has been possible, by review of import data, to accurately calculate crocidolite use.

TABLE 10: MORTALITY EXPERIENCE OF 632 NYC INSULATION WORKERS, BY AGE AT ONSET OF EXPOSURE

	Age at Onset of Exposure			Total
	≤20	21-30	30+	
Cancer of Lung	14 (5.7%)	52 (17.3%)	6 (7.1%)	72
Pleural Mesothelioma	3 (5.2%)	3 (4.7%)	0 (1.2%)	6
Peritoneal Mesothelioma	10 (4.9%)	5 (5.7%)	1 (9.4%)	16
Gastrointestinal Cancer	12 (4.9%)	17 (5.7%)	8 (9.4%)	37
All other neoplasms	6	15	7	28
Asbestosis	11 (4.5%)	15 (5.0%)	4 (4.7%)	30
All other causes	45	92	54	191
Deceased 12.31.68	101	199	80	380
Alive 12.31.68	145	102	5	252
Total	246	301	85	632

TABLE 11: ESTIMATED U.S. ASBESTOS FIBER CONSUMPTION¹⁴

Year	Crocidolite	Amosite	Canadian Chrysotile	Other	Total
1920	No data	No data	152,000	4,000	156,000
1925	No data	No data	300,700	4,100	201,800
1930		1,997	198,200	8,500	208,497
1935		501	154,200	20,500	175,201
1940		8,068	225,900	36,300	270,268
1945	8,700	4,500	355,800	9,000	378,000
1950	8,500	5,400	687,400	27,400	728,700
1955	11,700	18,000	695,100	11,600	740,400
1960	19,600	19,500	605,800	24,600	669,500
1965	17,100	21,400	661,100	20,100	719,700

Total Amosite and Crocidolite¹⁵

1930	1,997
1931	823
1932	212
1933	233
1934	152
1935	501
1936	1,432
1937	2,928
1938	3,282
1939	6,129

¹⁴Source: U.S. Department of Commerce.

Very little crocidolite entered this country until World War II (Table 11). Asbestos used here was almost entirely chrysotile. The largest asbestos manufacturer in the United States first used crocidolite in 1929 (for sheet packing, not insulation). This company never used crocidolite for the manufacture of insulation at any time (1929-1968), employing it only in two product lines (asbestos cement pipe, and packings).¹⁶ These data are consistent with an analysis of insulation materials used in past decades on the United States west

coast, where only chrysotile and amosite were found.¹⁷ During these same years, 1920-1940, more than 267,000 tons of chrysotile were used for insulation purposes.

This is not to say that it can be proven that crocidolite was never used before 1930 for insulation in the United States. Rather, if any was so utilized, it could only have been in very small amounts. Therefore, crocidolite exposure of United States insulation workers before 1930 and probably before 1950 is unlikely to have occurred to any significant

extent and, as seen in Table 1, is still a very minor affair.

MESOTHELIOMA AMONG UNITED STATES INSULATION WORKERS

The first mesothelioma reported in which occupational asbestos exposure was described, occurred in an insulation worker, a member of the I.A.H.F.I.A.W., in 1946.¹¹ It is not known whether cases occurred before then, but they have been noted since.¹² We have seen 22 cases, 1947-1968, in our group of men, as detailed above. Every one has occurred in an insulation worker who began work before 1930 (Table 12). The mean lapsed period from onset of exposure to death

of mesotheliomas among the 836 men in our study who began work in 1943 or later (Table 8).

These considerations are relevant only to conditions in the United States, among insulation workmen. They do not disprove a special role for crocidolite elsewhere, in other circumstances. They should, however, engender caution in relying on other asbestos varieties always to be significantly less likely to cause mesothelioma. The relative mesothelioma hazard for each fiber variety is still to be defined in humans. At this time our data suggest that it would be prudent to regard all fiber varieties as potentially hazardous, and to utilize appropriate industrial hygiene measures for all.

TABLE 12: Lapsed Period from Onset of Exposure to Death of 22 Cases of Mesothelioma, 1947-1968, Among 632 Insulation Workers Followed Prospectively from January 1, 1943

Patient	Site	Year Onset Employment	Year of Death	Age at Death	Years from Onset
1. A.C.	Pleural	1922	1947	60	25
2. A.M.	Pleural	1923	1950	58	27
3. A.S.	Pleural	1929	1964	54	35
4. J.B.	Pleural	1929	1963	54	34
5. E.C.	Pleural	1927	1964	53	37
6. T.C.	Pleural	1928	1966	56	38
7. E.M.	Peritoneal	1910	1949	61	39
8. F.S.	Peritoneal	1923	1956	50	33
9. M.C.	Peritoneal	1924	1956	52	32
10. H.H.	Peritoneal	1918	1959	60	41
11. J.B.	Peritoneal	1905	1963	86	58
12. H.D.	Peritoneal	1925	1963	66	38
13. W.N.	Peritoneal	1919	1964	63	45
14. A.C.	Peritoneal	1918	1964	68	46
15. J.D.	Peritoneal	1924	1965	58	41
16. J.L.G.	Peritoneal	1929	1965	53	36
17. J.O.C.	Peritoneal	1917	1966	79	49
18. G.V.	Peritoneal	1924	1967	59	43
19. H.C.	Peritoneal	1929	1967	57	38
20. J.P.	Peritoneal	1929	1968	58	39
21. R.B.	Peritoneal	1907	1968	83	61
22. H.T.	Peritoneal	1929	1968	56	39

was 34.8 years for pleural mesothelioma and 43.0 years for peritoneal.

As detailed, crocidolite exposure of United States insulation workers, to the extent that it occurred, could not have been very extensive before 1940. Therefore, if a long lapsed period is required for asbestos-induced mesothelioma, crocidolite exposure alone cannot easily explain the mesotheliomas seen among insulation workers in the United States. Of course, it may be that crocidolite's virulence makes for shorter lapsed periods between onset of exposure and death. But if this is so, it is difficult to explain the absence

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**Recommended
Safety
Practices
for
Handling
Asbestos
Fiber**



Johns-Manville

Safety Practices for Handling Asbestos Fiber

- 1** In all stages of handling—packaging, transporting, unloading, storing and in-plant moving—asbestos fiber bags should be handled carefully and in such a manner as to prevent the generation of dust.
- 2** Unitized loads should be lifted intact to and from holds of ships, and off and on land transport by fork lift trucks or other suitable handling equipment.
- 3** Care should be taken to avoid puncturing the asbestos fiber bags during unloading from ships, railroad cars, trucks or other modes of transportation. No hooks or other sharp instruments should be used. Loose fiber accumulated during transit should be picked up by vacuum cleaner before unloading.
- 4** Damaged bags should be immediately repaired, resealed or put into suitably closed receptacles. Any spillage of asbestos fiber should be picked up either by vacuum equipment or after thoroughly wetting, by sweeping, and should be deposited in suitable closed receptacles.
- 5** For storage, fiber bags should be stacked and moved with care to avoid breakage, protected from puncture by moving vehicles and, if possible, should be isolated from traffic in the warehouse and covered to prevent dust generation. To prevent deterioration of stored bags, the principle of first-in, first-out should be followed.
- 6** Maximum possible enclosure and fully effective dust control hoods should be provided at

all stages of in-plant handling where dust is unavoidably created, such as at bag opening, fiber dumping and bag disposal operations. Loose fiber should be cleaned from work areas as described in 4, above, to prevent dust regeneration.

- 7** The wearing of respiratory protective equipment approved for protection against asbestos dust should be required at all stages of asbestos fiber handling where quantities of dust are generated which cannot be kept within the Threshold Limit Value by dust control equipment or other methods.
- 8** Waste materials, such as collected dust and empty fiber bags, should be placed in special closed receptacles and disposed of in such a way as to prevent the release of dust. Methods applicable under 6 and 7, above, should be used during these operations.
- 9** Where the use of asbestos requires the transport of loose fiber in open carts or buggies, tight covers should be used to prevent spilling and dust generation.
- 10** All dust from exhaust systems should be collected and safely disposed of. It should not be released into the outside air. Particular attention should be paid to the protection of the employees whose job it is to dispose of the collected dust.
- 11** Local codes, rules or regulations governing the safe handling and use of asbestos fiber should be consulted and observed.

additional copies

For additional copies of this brochure, contact your local Johns-Manville office, or write to:

Johns-Manville
Environmental Control Department
P.O. Box 159
Manville, New Jersey 08835
U.S.A.

additional information

For additional information on the safe handling of asbestos fiber, we suggest that you write to:

CANADA AND U.S.A.

Johns-Manville
Environmental Control Department
P.O. Box 159
Manville, New Jersey 08835
U.S.A.

UNITED KINGDOM

The Asbestos Information Committee
Secretary
10 Wardour Street
London, W1V 3HG
England

OTHER COUNTRIES

Asbestos Industry Association or Government unit that deals with occupational health.

Johns-Manville

22 East 40th Street • New York, N.Y. 10016 U.S.A.



**RECOMMENDED
HEALTH SAFETY PRACTICES
for HANDLING
and FABRICATING
ASBESTOS TEXTILE PRODUCTS**



ASBESTOS TEXTILE INSTITUTE
founded 1944

INTRODUCTION

Asbestos textile products manufactured from chrysotile asbestos fibers are a growing and indispensable class of materials in industry today. Fabricated products of asbestos textiles are used in many and diverse applications. The strength, chemical resistance, incombustibility, and reinforcement characteristics make asbestos textiles a unique product.

Recently there have been numerous articles in the scientific press, as well as other news media, concerning the potential health risk for workers involved in handling asbestos. Manufacturers of asbestos textile products have for years been taking protective measures to eliminate exposure of their workers to the inhalation of dust.

The Asbestos Textile Institute recognizes that extensive exposure to the inhalation of asbestos dust over a long period of time may be injurious. The following general recommendations for handling and fabricating asbestos textile products are based on the current scientific assessment of the potential health risk and the level of the art in reducing the risk. Particular circumstances, of course, may require different handling or fabricating procedures; obviously, these general recommendations cannot be considered adequate to cover all possible situations.

HEALTH SAFETY PRACTICES

- 1.** An organized housekeeping program should be maintained in all work areas in order to prevent the generation and accumulation of dust. Vacuum cleaning equipment is recommended rather than sweeping or brushing loose material. If sweeping or brushing is essential, wetting down is recommended.
- 2.** Effective mechanical dust collecting systems, designed to collect dust at the source, should be used where dust above the Federal, State, or Provincial allowable threshold limit is unavoidably created.
- 3.** Where mechanical dust collection systems are impractical, and dust is unavoidably created above the allowable threshold limit, U.S. Bureau of Mines approved respiratory equipment* should be worn.
- 4.** During the fabricating or processing operations dust may be effectively minimized by dampening or wetting the material.
- 5.** Asbestos textiles which are manufactured by a damp process, or which are treated, minimize dust in subsequent handling and fabricating and are therefore recommended.
- 6.** Isolation and frequent collection of waste and dust is important. This waste material should be disposed of in closed containers.

* Bureau of Mines
U.S. Dept. of the Interior
Nineteenth and E Streets, N.W.
Washington, D.C. 20240

Prepared by
The Asbestos Advisory Committee
Edited by
The Environmental Health Committee
of the
ASBESTOS TEXTILE INSTITUTE

**RECOMMENDED
LTH SAFETY PRACTICES
for HANDLING
and FABRICATING
TEXTILE PRODUCTS**

For additional copies, write
Executive Secretary
Asbestos Textile Institute
P.O. Box 239
Pompton Lakes, N.J. 07442 U.S.A.



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STITUTE

**RECOMMENDED
HEALTH SAFETY
PRACTICES**

*For Handling
And Applying
Thermal
Insulation Products
Containing
Asbestos*

nima

NATIONAL

INSULATION

MANUFACTURERS ASSOCIATION

HOUSEKEEPING

An organized housekeeping program should be maintained in all areas and on all operations at all times.

A major source of excessive airborne dust is from loose materials, scrap and debris distributed throughout the job area and disintegrated by trampling on, running over, etc. Much of the airborne contamination can be eliminated by exercising simple good housekeeping practices.

Specific standards and practices designed for providing safe, clean and orderly working conditions should be defined.

Efficient supervisory control, planning of systematic cleaning plus day to day follow-up are the basic requirements for dust control and injury prevention.

PIPE AND BLOCK INSULATIONS

Warehousing. Storage and Handling

Under normal conditions, the handling, storage and warehousing of undamaged cartons of pipe and block insulations, containing asbestos, present no health safety problems. If cartons are broken or materials damaged while insulation is being handled, the following steps should be taken:

- 1 If spillage occurs, debris should be cleaned up immediately. Repack usable materials.
- 2 Care should be taken to avoid generating dust when piling, stacking or placing pieces into containers.
- 3 When cleaning spillage, vacuum equipment is recommended for picking up dust and small particles.
- 4 If large quantities of spillage occurs and cleaning involves sweeping and/or shoveling that could create excessive airborne dust, *Respirators should be worn.
- 5 Only usable material should be returned from the job site to the warehouse.
- 6 When returning material from the job site, clean dust and debris off cartons. Repack odd lot pieces. Close cartons.

*Bureau of Mines-approved respirators. See page 18.

***Fabrication in
Warehouse and
Job Site Shops***

- 1 Open and remove the material from cartons with care.
- 2 Handle and stack pieces of material in a manner that will avoid generating dust and breakage.
- 3 Mechanical dust collecting systems adequately designed to remove dust at the source should be installed on power-operated, fixed and portable equipment in the warehouse shop and /or in the job site shop.
- 4 All machines should be furnished with suitable waste containers for the collection of scrap and waste to prevent the material from falling on the floor and generating dust.
- 5 Shop-fabricated pieces of Thermal Insulation such as fittings, segments, etc., should be placed in containers. Care should be taken when handling these pieces during piling, stacking and packing.
- 6 Containers should be closed after packing and remain closed until the material is used.

Application Procedures

- 1 Unpacking and application of the material should be done in a manner that will minimize airborne dust.
- 2 Avoid dropping, throwing or unnecessary rough handling of insulation materials.
- 3 Keep floors and surfaces clean of scrap, shavings and debris. Put unusable material into waste containers immediately.
- 4 Hand sawing, cutting, jacket stripping, etc. should be done in a manner that will create minimal airborne dust. Use of proper methods and tools will help considerably towards dust abatement.
- 5 When working in confined spaces, portable dust collectors or exhaust blowers should be provided to remove dust from the source.

Application Procedures (Cont'd)

- 6 If conditions prevent collecting or exhausting dust from the source, use exhaust blowers to provide general room air changes.
- 7 Avoid exhausting into other working areas.
- 8 In confined spaces, if mechanical exhaust equipment cannot be used, *Respirators should be worn.
- 9 When wiring or banding insulation and applying jackets or facings, care should be taken to avoid generating excessive dust, using the precautionary measures described above.
- 10 Surplus usable materials should be placed in cartons, which should be closed and returned to the warehouse.

CEMENTS

Warehousing, Storage and Handling

Handling and storage of closed bags containing cements do not present health safety hazards provided bags are unopened and not broken.

- 1 When unloading railroad cars and trucks, if there is excessive loose material resulting from opened or broken bags, *Respirators should be worn during clean-up and unloading.
- 2 Spillage should be cleaned up and open or broken bags repacked, resealed or discarded immediately to prevent distribution of dust into other areas.
- 3 If bags are damaged and spillage occurs in the warehouse, material should be cleaned up immediately to prevent distribution into other areas. Vacuum equipment is recommended for this cleaning.
- 4 If large quantities of spillage occurs and cleaning involves sweeping and/or shoveling that could create excessive dust, *Respirators should be worn.

Application of Cements

Opening, dumping and discarding of bags should be done with the utmost care to avoid generating excessive dust.

- 1 Open the bag wide enough to empty without shaking.
- 2 When dumping, keep bag on the mortar box mixing level to avoid dropping.
- 3 Do not shake bags.
- 4 Place empty bags in a container, wet down contents, and discard.
- 5 Mixing area should be well ventilated but free from heavy drafts that will cause dry material to become airborne.
- 6 Material should be mixed in a mortar box, pail or tub and not on the floor.
- 7 Wet the material and mix into a slurry as quickly as possible.
- 8 When mixing cement in a confined space where proper ventilation is inadequate, *Respirators should be worn.

Cleaning

- 1 Bag breakage, cleaning up dry cement and discarding bags at the mixing area should be done in the same manner as recommended under *Warehousing, Storage and Handling*.
- 2 Wet down tools, mortar boxes, wheelbarrows, mixing containers, and any spillage before scraping and cleaning.
- 3 Cements should be handled wet whenever possible. Wet material will create no dust problem.

*Bureau of Mines-approved respirators. See page 15.

SPRAYING OF THERMAL INSULATION

Spraying Areas

Spraying of Thermal Insulation Products containing asbestos requires special precautionary measures. Refer to the Sprayed Mineral Fiber Manufacturers Association for Sprayed Fiber Application Practices.¹

- 1 The equipment and machinery to be sprayed should be shielded by adequate wind breaks and, where practicable, drop cloths to prevent excessive dust and contamination in adjacent areas.
- 2 Workers not engaged in the spraying operations should wear *Respirators when entering an area within 15 feet of the spraying machine, nozzle or equipment.
- 3 Where Sprayed Fiber Application Practices cannot be followed and excessive airborne dust is generated, it is recommended that portable mechanical exhaust ventilation be used to remove airborne dust. Exhaust must be located to avoid contamination of other occupied areas.

Application

- 1 All personnel in the immediate area engaged in the machine feeding and spraying operation should wear *Respirators, whether in confined or open areas, ventilated or not ventilated.
- 2 *Respirators should be worn after the spraying operation has been concluded and until the generated dust concentration has cleared.

¹For copy of Practices, write to Executive Secretary, Sprayed Mineral Fiber Manufacturers Association, P.O. Box 137, Westfield, New Jersey 07090

*Bureau of Mines-approved respirators. See page 15.

Other Workmen

All personnel not involved with the machine feeding and spraying operation should be kept from the spraying area until the generated dust concentration has cleared.

Cleaning

- 1 Spillage and excess spraying materials should be cleaned up immediately after spraying operations have been completed.
- 2 Settled dust and fly material should be removed from all surfaces to avoid airborne contamination. Vacuum cleaning is recommended for this purpose.
- 3 Scrap spray material should be wet down and placed in closed containers for disposal.
- 4 Tools, ladders, etc. should be cleaned wet.
- 5 Avoid brushing, sweeping and shoveling dry material whenever possible.
- 6 *Respirators should be worn during clean-up.

*Bureau of Mines-approved respirators. See page 15.

STRIPPING OF THERMAL INSULATION

Stripping Area

Careful work methods, use of proper tools, and wetting down will minimize excessive dusts in stripping operations.

- 1 The area in which stripping takes place should be confined by means of curtains, portable partitions, etc., to prevent excessive dust contamination in adjacent areas.
- 2 If area cannot be confined, only workmen engaged in stripping should be permitted in the work location.

Stripping and Tearing Off

- 1 All personnel in the stripping area engaged in tearing off, handling, cleaning and disposing of material should wear *Respirators.
- 2 Where circumstances permit, materials should be wetted down prior to and during removal.
- 3 Where applicable, portable mechanical exhaust ventilation should be used to remove airborne dust.
- 4 Avoid unnecessary re-handling of scrap.

(Continued on Next Page)

*Bureau of Mines-approved respirators. See page 15.

Stripping and Tearing Off (cont'd)

- 5 Locate truck, dumpster bucket or final disposal equipment as close to stripping site as possible.
- 6 Wet down scrap material before shoveling, hauling or dumping.
- 7 Do not permit accumulation of scrap and debris on floor and other surfaces.
- 8 Vacuum equipment is recommended for cleaning up during and after stripping.

Other Workmen

- 1 Personnel not involved in the stripping and tearing off operation should not be permitted in the stripping area.
- 2 If it is essential for other workmen to be in the work area, *Respirators should be worn by them.

*Bureau of Mines-approved respirators. See page 18.

RESPIRATORY EQUIPMENT

Only U.S. Bureau of Mines-approved respiratory equipment is recommended for use with asbestos, silica and other pneumoconiosis-producing dusts. Approved respiratory equipment must be used at all operations where dust control is not sufficient to maintain airborne dust concentration within allowable limits for asbestos and other pneumoconiosis-producing dusts, as established by governmental and professional agencies.

The efficiency and effectiveness of respiratory equipment is dependent upon proper care, cleaning and maintenance. A systematic procedure should be established to assure that this is done.

Lists of currently approved respiratory equipment can be obtained from:

Bureau of Mines
United States Department of the Interior
Nineteenth and E Streets, N.W.
Washington, D.C. 20240

American Industrial Hygiene Association
14125 Prevest
Detroit, Michigan 48227

for additional copies, write

Executive Secretary
National Insulation Manufacturers Association, Inc.
441 Lexington Avenue, New York, N.Y. 10017

Recommended Practices

For Fabricating,
Handling and
Applying
Asbestos-Cement
Products in
the Building
and Construction
Industries

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Asbestos-cement products, in place and under normal use, present no known health risk to the user or general public.

Introduction

The discovery and production of hundreds of natural and synthetic materials including asbestos-cement products has in some instances prompted the need for special protective equipment and practices that should be employed by all in the industry where dust is created.

Manufacturers of asbestos-cement products have for years been taking protective measures to eliminate exposure of their workers to the inhalation of dust.

The fabricating, handling and applying of asbestos-cement products are diverse and range from the installation of a single piece to extensive operations involving cutting and machining by mechanical equipment. While care should always be exercised, special precautions are necessary when dusts in excess of federal, state or provincial allowable threshold limits are generated as the result of sawing, drilling or other operations. Respirators should be worn.

The following practices are recommended for fabricating, handling and applying asbestos-cement products in the building and construction industries. Contractors and applicators are urged to study these recommended practices carefully and to see that they are observed by all workers. It is in the best interest of both employer and employee to see that these recommended practices are strictly observed.

Fabrication and Application

1. When moving asbestos-cement products to and from the fabrication site, individual pieces of materials should be handled with care to avoid breakage and dust.
2. Fabrication of asbestos-cement products involving cutting, drilling, sawing, etc., should be done in a manner that will minimize dust. The use of proper methods, tools and equipment is essential.
3. When cutting, drilling, sawing, etc., on a repeated basis with power equipment, mechanical dust collecting systems adequately designed to remove dust from the source should be used.
4. Fabrication of asbestos-cement products in confined spaces requires adequate mechanical dust collecting systems designed to collect dust at the source. Where these systems are impracticable, approved respirators should be worn.
5. Provide waste containers at all fabrication and application areas. Dispose of unusable material. Keep floors and surfaces clean. Vacuum cleaning equipment is recommended to remove dust and small particles. If vacuum equipment is impracticable, wet down waste material before sweeping and shoveling. Respirators should be worn during clean-up.

Respiratory Equipment

Only U. S. Bureau of Mines approved respiratory equipment is recommended for use. The efficiency and effectiveness of this equipment is dependent upon proper care, cleaning and maintenance. A systematic procedure should be established to insure that this is done.

A list of approved respiratory equipment can be obtained from

American Industrial Hygiene Association
14125 Prevost
Detroit, Michigan 48227

U. S. Department of the Interior
Bureau of Mines
Nineteenth and E Streets, N.W.
Washington, D. C. 20240

Summary

Three major points to keep in mind in the fabricating, handling and applying of asbestos-cement products.

1. Keep dust to a minimum.
2. Wear U. S. Bureau of Mines approved respirators when exposed to dusts above federal, state or provincial allowable threshold limits.
3. Maintain good housekeeping.

Prepared by

Health and Safety Council
Asbestos Cement Products Association
521 Fifth Avenue - 20th Floor
New York, New York 10017

— R71-i6 —

EXHIBIT 22

SPRAYED FIREPROOFING - AN ENVIRONMENTAL PRIORITY

BY: HAROLD ROMER, P.E.
ASSISTANT COMMISSIONER
ENGINEERING RESEARCH AND DEVELOPMENT
NEW YORK CITY DEPARTMENT OF AIR RESOURCES

October 15, 1971

Public Hearing on Proposed Asbestos Emissions Controls
Illinois Pollution Control Board
Chicago, Illinois

Epidemiology of Asbestos:

It is essentially because of the very disturbing medical statistics that The New York City Environmental Protection Administration and the Department of Air Resources have been so concerned and have become, literally and figuratively, so completely immersed in the sprayed asbestos problem.

With your permission, I will quote one or two references which are not only authoritative but also provide the punch merited by the facts:

1. The following material is abstracted from a 1969 U. S. Public Health Service report, "Preliminary Air Pollution Survey of Asbestos - A Literature Review":

"- - a large fraction of the people in the U. S. have been exposed to asbestos, including the following:

- a) approximately 100,000 workers using asbestos in their occupations.
- b) approximately 3,500,000 construction workers exposed indirectly to asbestos as they work.

(continued)

with asbestos products or near those who handle asbestos products.

- c) approximately 50,000,000 Americans who possibly have "asbestos bodies" in their lungs.

2. In November 1970, in a court case against the City of New York, we called upon Dr. Irving Selikoff to provide expert medical testimony. Dr. Selikoff, a consultant to our agency, is a renowned researcher and pioneer in the field of asbestos diseases; he is also Director of Environmental Science Laboratories at the Mt. Sinai School of Medicine in New York City. The following quotes are abstracted from his testimony:

"In New York, in 3,000 consecutive autopsies at Elmhurst Hospital in Queens, the V.A. Hospital in the Bronx, and Mt. Sinai Hospital in Manhattan, examination of ----lung tissues has shown that asbestos bodies exist in 45% of all cases. Smaller particles of asbestos, seen only by electron microscopy, were found to be commonly present in almost all cases examined. It now appears that virtually all New Yorkers' lungs contain asbestos."

"Among asbestos workers, for example, the incidence of death from lung cancer among smokers is about 20 times as high as for non-smokers. If a person is both an asbestos worker and a

cigarette smoker, his chance of dying from lung cancer is 92 times the chance of a man who neither smokes cigarettes nor works with asbestos."

"Recently, I conducted an investigation with respect to asbestos-related conditions among the members of Local 12 (New York City) and Local 32 (Newark) of the International Association of Heat & Frost Insulators and Asbestos Workers, one of the building trade unions."

"As of April 30, 1970, 403 of the original 632 union members in our study were dead, 72 of lung cancer. Currently

(continued)

History and Characteristics of Asbestos:

The consumption of asbestos in the United States has assumed large proportions and its use is increasing at a rapid rate. The 1968 consumption in the U. S. was equal to 25% of the total world production. Annual world production in 1930 was 500,000 tons; in 1967, four million tons of asbestos were produced.

During the past 10 years, asbestos-containing material has been used extensively in sprayed form at an increasing rate, to protect steel structures from fire. In 1970, approximately three million pounds of asbestos were used to fireproof structures. It is particularly with reference to this use of sprayed asbestos that I wish to address my remarks.

In the United States, almost 90% of the asbestos used is of the chrysotile fiber type that easily fragments into fibrils 200 to 400 angstrom units in diameter and often 2,000 angstrom units or smaller in length (200 angstrom units equals approximately a millionth of an inch). Thus 10^{-9} grams (one billionth of a gram) of chrysotile asbestos could represent a million fibrils. Chrysotile asbestos fibrils can only be identified by use of the electron microscope.

Seventy percent of the asbestos in this country is used in the construction industry.

(continued)

From the published medical reports and epidemiological studies it would be fair to state that the individual, invisible asbestos fibril represents a built-in "time bomb" set eons ago when the fibrous silicate crystals cooled and began to grow within the fractures of the serpentine rock. The mechanism can be activated when these fibrils are inhaled or ingested - albeit it may take 20-25 years to wreak its havoc in the form of excessive cancer incidence rates, including the invariably fatal pleural and peritoneal mesotheliomas.

Action by the Dept. of Air Resources:

It is because of the relative indestructibility of the asbestos fibril, and its inherent potential for harm, as well as the unsatisfactory experience of the N.Y.C. Dept. of Air Resources in its efforts to control asbestos spraying operations, that the City of New York urges the banning of the spraying of any substance containing asbestos in or upon a building or other structure during its construction, alteration or repair.

Because of our recognition of the intense health hazard of asbestos exposure, the Dept. of Air Resources, on April 13, 1970, issued interim regulations, known as Commissioner's Orders, prescribing containment and clean-up methodologies. This was the first such set of rules anywhere pertaining to the regulation of sprayed asbestos insulation. This was the catalyst that triggered

(continued)

regulatory agencies throughout the country to adopt similar procedures and the subsequent banning of sprayed asbestos by several cities. We are pleased that both the States of Illinois and New York have proposed the prohibition of this dangerous practice throughout these States. I understand that the Federal EPA is also considering the banning of sprayed asbestos.

A challenge to the power of the Commissioner of the Department of Air Resources to regulate spraying operations using asbestos-containing materials for fireproofing and insulation at construction sites arose in the November, 1970 case of La Monica vs Jerome Kretchmer, Administrator of the Environmental Protection Administration. The State Supreme Court ruled in favor of the City and denied the injunction requested by the plaintiff, and declared the Commissioner's Orders and the Air Pollution Control Code to be constitutional.

Dr. Selikoff, in testimony during the La Monica-Kretchmer case, although the bulk of his remarks centered on the medical effects of asbestos inhalation, stated, "In Manhattan ambient levels far removed from construction sites are now $20-60 \times 10^{-9}$ grams per cubic meter of air, up to 6 times more than in, say, Staten Island, or Ridgewood, N.J. Indeed, ambient air levels about construction sites are even several times higher than these background levels. At these levels the average person might inhale 10 to 15 million asbestos fibrils in one 24 hour day."

The April 1, 1971 issue of the New York Times reported that

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Federal EPA began publishing a list of air pollutants, which in the opinion of Administrator William D. Ruckelshaus "may cause, or contribute to, an increase in mortality or an increase in serious irreversible, or incapacitating reversible illness." Mr. Ruckelshaus "named three such pollutants - asbestos, beryllium and mercury."

Our efforts at controlling the emission of asbestos from spray operations have not been successful. On numerous occasions, emissions were such that heavy asbestos snowfalls were observed and accumulations of asbestos as deep as 3 inches at curbs and at other vertical rises occurred within a radius of several blocks of spraying site.

Since there are 25 to 40 skyscraper asbestos insulation-spray operations in progress at any one time, it would take virtually our entire field staff to provide adequate surveillance for this one function. Even if this were feasible, which it is not, I strongly question the ability of the industry to prevent the emission of asbestos into the outside air under actual, day-to-day conditions of construction.

As usual, court actions resulting from the issuing of many summonses, have had but limited deterrent value; the adjournments were endless and the fines, when finally imposed, were only a minor overhead to the contractor. The administrative hearings held by the Dept. of Air Resources to show cause why an asbestos-spraying

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operation should not be halted, have been very time-consuming though, temporarily, of some value.

It should be made clear that the Commissioner's Orders, promulgated on April 13, 1970, were intended to provide a workable methodology which could be implemented immediately by the construction industry. To provide for a methodology which would assure positive containment of this carcinogenic material, would have resulted in the complete cessation of the construction on all skyscrapers in our City.

Our experience clearly indicates that, with the present methodology, even with conscientious follow-through by the contractor, asbestos dust, in lesser or greater quantities, can be found in almost any part of the building under construction. In industry, where asbestos products are manufactured, "clean-room" procedures often are employed, including an exhaust system and filter baghouse placed directly above or below such processes as cutting operations to preclude the likelihood of asbestos dust reaching the face of the worker or entering the workroom or outside air. This positive type of control is required when handling asbestos, if confidence in the methodology and protection of the public and worker are to be assured.

Visible emissions, prohibited by our Code, are difficult to observe, especially when spraying occurs on the upper floors of

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skyscrapers, unless there is a veritable snowstorm of asbestos into the outside air. Perhaps more important than the visible emissions, however, are the fine asbestos particles which are not visible to the naked eye, but which are more hazardous because they can penetrate the lungs' smallest alveoli. Again, I note that a billionth of a gram of chrysotile asbestos may contain a million fibrils of asbestos. Sampling for such particulates is not only extremely difficult at upper stories, but also time-consuming and expensive. By the time laboratory results would become available, the spraying procedure of the specific area sampled would be long completed.

Demolition:

Even if a methodology were developed which could protect the ambient air from sprayed-asbestos particulates, a problem, for which no solutions are currently known, would dictate the banning of sprayed asbestos. I am talking about demolition dust. It is difficult to visualize the demolition of a magnificent 30, 40 or 50 story structure. Nevertheless, based upon the construction and demolition history of buildings in New York City, we can anticipate that a significant percentage of these "old fashioned" structures will be demolished 30,40 or 50 years hence.

The present state of the art is such that there is reasonable assurance that the City of New York will be coated with asbestos

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when these buildings are demolished. We hope to develop ways and means to cope with demolition dust problems, however, there is no certainty of success, particularly because of the inherent difficulties and high costs.

Hazard to Workers:

Last, and certainly not least, is the unbelievable hazard to workers employed in spraying asbestos. The tremendously high incidence of lung cancer, the 100% fatal mesotheliomas and other carcinogenic causes of death experienced by asbestos-insulation workers cry out against the use of sprayed-asbestos.

With the knowledge that it was, at best, a most difficult task adequately to supervise all construction sites using sprayed asbestos insulation; and the time-consuming administrative hearings; and with the problem of multiple adjournments and meaningless fines in court; as well as the preponderance of medical evidence as to the pathogenicity of asbestos, the Department of Air Resources embarked on a campaign to encourage manufacturers to eliminate asbestos and develop suitable substitute fireproof materials.

At present, I can report that our Buildings Department has approved of 3 sprayed non-asbestos fireproofing materials for specific assemblies and a fourth material is well on its way to probable approval. Furthermore, at the request of the Department of Air Resources, the Health Research Council of the City Department of Health is evaluating the health aspects of the substitute

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materials.

On August 25, 1971, our City Council, at the recommendation of the NYC Environmental Protection Administration and Department of Air Resources, enacted into law the prohibition, "--no person shall cause or permit the spraying of any substance containing asbestos in or upon a building or other structure during its construction, alteration or repair--." The cities of Chicago, Philadelphia and Boston also have banned the use of sprayed asbestos insulation.

The NYC Environmental Protection Administration and the Department of Air Resources have great concern, not only with sprayed asbestos, but also with all sprayed insulating materials. On August 25, 1971, our City Council enacted into law provisions regulating the methods of enclosure and clean-up when spraying any insulating material.

The same problems of emissions into the open air we experienced with sprayed asbestos-containing insulation are being and, doubtlessly, will continue to be experienced with non-asbestos sprays, as long as the present methodology of temporary enclosure is practiced. The use of tarpaulins for enclosure is not satisfactory on a continuing basis.

I have proposed several alternatives:

1. Fireproofing of peripheral columns, spandrels, etc.
by non-spray means, such as boxed beam with lathe and plaster,

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drywall encasement, concrete encasement, etc. Spraying of perhiperal steel is a major source of visible emissions into outer air.

2. Design structure so that skin and windows can be installed before any spraying is done.
3. In any insulation spraying operation, install exhaust system and baghouse to ameliorate the astonishingly bad conditions under which sprayworkers now operate and to reduce emissions outside the building.

In my many consultations with architects, engineers, builders and their associations, there is general agreement that there are virtually insurmountable problems associated with insulation spraying.

There are many available alternatives to spraying, however, each is more costly than spraying. Reported costs for the various alternatives differ widely, depending upon the builder one speaks with. Some average numbers indicate that for high rise structures the cost of spray insulation is 3/4 of 1% to 1% of the total cost. What does "total cost" mean? Some mean "bricks and mortar"; others include bricks and mortar, foundation, land costs , architectural fees, financing charges, profit, etc. Costs for drywall encasement vary from 2-3% ot "total cost"; boxed beam with lathe and plaster from 2-3% of "total cost"; concrete encasement as high as 5%.

If one of the less costly, approved alternatives were selected, the increase in cost would be in the area of 13/4%. No one wants to spend more than he is required, however, this additional cost does not appear prohibitive and, above all, will protect both public and worker.

We have been encouraging and working closely with product manufacturers to develop competitive non-asbestos insulating materials and methodologies. Among these are application of insulation at the steel fabrication mill; insulation in rolls and strips which can be applied to the steel with adhesives; application by trowelling; intumescent mastics; insulation blankets* wrapped around steel members on the site, etc. For reasons of confidentiality, I cannot go into additional details at this time. Some of these alternatives should be ready within 6-12 months. We welcome any new ideas and stand ready to be of assistance in bringing new products to fruition.

"With all the current emphasis on the systems approach, it is curious that a practical factory assembled enclosure has not yet been developed for the fire protection of steel beams. In addition to obtaining the usual advantages of factory production, the problem of air pollution would be avoided. The prefabricated units could consist of two sides and a bottom that enclose the beam and are attached to the floor slab by stud welds or powder-actuated fasteners. Or the units could be hung from studs welded to the bottom flange.

*On October 1, 1971, mineral wool blankets were approved in NYC for 4 hr. protection of columns.

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while the sides are braced by studs welded to the web." (Ira Hooper, Modern Steel Construction, 1971).

The Problem of Standards:

State and federal labor departments have set up the working atmosphere dust standard of not more than 5 fibers per c.c., 5 microns or larger. What does this mean in view of our recent experience? A study in which we are now engaged produced air analyses yielding no fibers 5 microns or larger; all particulates, asbestos fibrils, were submicron. The concentration of the submicron fibrils was approximately 200 per c.c. This is 40 times greater than the industrial standard for fibers. By definition, the air met industrial quality standards since there were no fibers 5 microns or larger. Normally, industrial air analyses do not include examination for submicron particulates. A sample of air having a zero count of fibers, 5 microns or larger, would be considered excellent. Obviously, the standard must be re-examined, especially since the submicron particulates are highly respirable and present a greater hazard to health.

The practical problems of performing electron microscopy, X-Ray diffraction, etc., examination of air samples for the positive identification of asbestos fibers and for the identification of other insulation fibers less than 5 microns in length are recognized. Accordingly, it is suggested that the method proposed by the Illinois Pollution Control Board (phase contrast microscopy at 430 X magnification to identify fibers 5 microns or larger) be utilized routinely provided that electron microscopy, X-Ray diffraction, etc., be employed at regular intervals of time to determine whether the ratio of the less than 5 micron fibers to fibers 5 microns or larger has increased over previous experience.

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Since medical evidence indicates that very small fibers may be the most dangerous, it is further recommended that consideration be given to revision of the air quality standard wherein fibers of any length be counted rather than only fibers 5 microns or larger in length.

A Caveat:

There are more than 3200 manufactured products in this country which contain asbestos. In my judgement, control of the ubiquitous asbestos fibre should be given a high order of priority if the public's health is to be protected. The scope of the asbestos problem can be recognized by enumerating a few of its common uses:

1. Textiles (recently, women's coat fabric was found to contain asbestos, emitting high concentration of asbestos fibre when shaken);
2. Yarns, cord and braiding;
3. Sealing and packing material;
4. Gaskets;
5. Filter-paper (used to filter injectable drugs);
6. Coverings, coatings and jacketings;
7. Auto parts;
8. Protective walls and curtains;
9. Furnace and drying oven linings;
10. Asbestos cement such as water and sewer pipes, corrugated sheets, interior walls, ventilating shafts, air conditioning assemblies;
11. Thermal insulation;
12. Road surfaces;
13. Friction materials such as brake linings and clutch lining;

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14. Asbestos plastics such as floor tiles and molded insulation; also used in aircraft industry;
15. Synthetic snow sprayed by small children on artificial Christmas trees from aerosol cans;
16. And finally, asbestos can be found in talcum powder dusted liberally on baby bottoms and inhaled with the nursery air.

Recommendations:

I urge the Illinois Pollution Control Board to ban immediately the spraying of asbestos-containing insulation and fireproofing in or upon a building or other structure during its construction, alteration or repair; also prohibit the sale of motor vehicles manufactured after January 1, 1975, if brake linings contain asbestos.

I urge the Illinois Pollution Control Board to closely control other commercial uses of asbestos in construction work; the demolition of asbestos-containing structures; the manufacture and processing of asbestos-containing products; the transportation, storage and handling of asbestos-containing products; and the disposal of asbestos-containing wastes.

I further urge the Illinois Pollution Control Board to control the spraying of any insulating material, not otherwise prohibited, in or upon a building or other structure during its construction, alteration or repair.

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— R71-16

EXHIBIT 26

air and water news

McGraw-Hill's weekly report on environmental pollution: the law, the markets, the technology

Ex 26

Vol. 5, No. 39

October 4, 1971

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- * *Efforts to Bar Strip Mining Wane in Congress: page 3*
- * *Ford Likely to Turn to Englehard for Platinum Catalytic Converters: page 4*
- * *California Court Bars Industry-Controlled Forest Board: page 6*

ZERO-DISCHARGE PROVISION WEIGHED BY SENATE PANEL

The Senate Public Works Committee will continue deliberation this week on water pollution legislation, with a decision still to be made on what sort of national standard to set. In addition to the national minimum standard proposal to achieve waters fit for swimming and fish life by 1980--contained in a bill by the subcommittee on air and water pollution--the committee now is considering a proposal which eventually would impose a zero discharge provision.

If this were done, it probably would provide for effluent reduction in several stages. If a fixed date is set for the zero discharge it probably would be later than 1980. The current working definition of a pollutant is "matter of any kind or description including, but not limited to dredged spoil, solid waste, incinerator residue, sewage, garbage, sewage sludge, munitions, chemical materials, biological materials, radioactive materials, heat, wrecked or discarded equipment, rock, sand, cellar dirt and industrial waste."

The committee now is expected to retain the funding formula in the original bill--maximum 75% federal share. The House Public Works Committee began work last week on drafting its own version of the bill. It is not yet clear, however, what will emerge on funding or standards. For more information on the "clean streams" proposal, see AWN, Sept. 13, p. 1. Alternative funding plans were discussed last week (p. 3).

OMB REVIEWING PROPOSED EMISSION LIMITS FOR MERCURY, ASBESTOS AND BERYLLIUM

The Office of Management and Budget (OMB) is reviewing proposed emission standards for mercury, beryllium and asbestos under the "hazardous air pollutants" section of the 1970 Clean Air Act. The Environmental Protection Agency said late last March that it would set emission standards for these three pollutants from stationary sources (AWN, April 5, p. 1). The September 27 deadline for doing so passed last week, with comments still being received by OMB from other federal agencies. Pending OMB review, here are the standards, as gleaned from EPA sources by AWN.

* Mercury: 5 pounds per day, not to be exceeded in any one day. The standard will be a problem only to smelters extracting mercury and to chloralkali plants, EPA thinks. Mercury emissions from powerplants will be ignored, at least for the time being. EPA



concedes that powerplants, especially those burning coal, are responsible for considerable mercury emissions. But agency sources say that current emissions data and monitoring techniques are not equal to the task of enforcing a mercury limit on powerplants, and that powerplant stacks are quite high generally, reducing the ambient air concentration of mercury near the plant. EPA sources say the flat 5-pound limit would give an ambient air concentration below 1 microgram per cubic meter, 30-day average, and that this is the level below which they have determined no clear health effect will occur.

* **Beryllium:** 10 grams per day, not to be exceeded on any day. This will produce an ambient air level below the EPA-determined danger level of 0.01 microgram per cubic meter, EPA sources say. Major plants to be affected are the four U.S. facilities for extracting beryllium from ore. These four (two in Pennsylvania, one in Ohio and one in Utah) are already under similar restrictions by the Atomic Energy Commission. Machine shops working on beryllium and its alloys may also be affected.

* **Asbestos:** EPA has suggested a ban on visible emissions, as well as a ban on spraying asbestos insulation in construction and in using asbestos mine tailings in road construction. EPA sources say the difficulty of measuring asbestos--there's no stack to put a probe on--makes assignment of an emissions number almost meaningless.

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REFUSE ACT GUIDELINES:

THEY'RE COMING--SLOWLY

Still waiting for some numbers--even rough numbers--for use as guides for what is acceptable effluent under the Refuse Act? The first of the guides was originally promised for last week. Now they will be held up for a few weeks more. In fact, the bulk of the 20 guides will not be published until late November, an Environmental Protection Agency source told AWW. The guidelines will be available for purchase by affected companies once they are published.

Guides are being prepared for: pulp and paper-products, petrochemicals and organic chemicals, steam generating, beverages, beet sugar, grain milling, inorganic chemicals, fertilizer, petroleum refining, basic steel, basic nonferrous metals, canned and preserved produce, dairy products, meat products, textile mills, plating and finishing of motor vehicles and aircraft, flat glass, cement, asbestos and leather tanning and finishing and--surprisingly, since few qualify under the Refuse Act Permit Program--livestock feedlots.

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U.S. STEEL BACKS DOWN

ON REFUSE ACT PERMIT

The first major challenge of the 1899 Refuse Act's permit program has collapsed, apparently leaving the much-disputed antipollution tool stronger than ever and the challenger, U.S. Steel, a bit bruised. Late last week, the steelmaker did an about-face and announced that it would supply in full, and with no restrictions, information for its permit applications. In addition, the steel company said that a top corporate official would now sign the application. U.S. Steel had been recommended by EPA for prosecution over three improperly signed applications (AWW, Sept. 27, p. 1).

The sudden capitulation is more a public relations victory than a legal one. But this was enough to make EPA officials in Washington and New York jubilant. Since this summer, U.S. Steel has been trying to line up allies in its battle with the EPA. It had contended that the permit program was unfair and, among other things, required it to document information that it considered secret. But it's understood that no other major company was willing to join U.S. Steel in its battle.

A U.S. Steel insider, however, said that the capitulation does not mean that the company has lowered its opposition to the permit program and he hinted that at some future time a protest might be waged. But he declined to predict the form of such a protest.

One legal observer says there are at least two unexplored legal grounds on which a firm could protest the permit program: The 1899 act made no provision for a permit

program as such, and President Nixon had no authority to have EPA share the permit-review power that the 1899 act gave to the Army Corps of Engineers. But both may be moot points since the new water bill may replace the 1899 law before such a case reaches trial.

The move by U.S. Steel leaves unclear the disposition of the legal action sought by EPA. The Justice Department has a total of 35 such cases under advisement. EPA has recommended that all 35 plants, including three of U.S. Steel's, be sued for failing to file permits.

Last week, *Newsweek* magazine reported that the move by EPA was an embarrassment to the White House because Attorney General John Mitchell was overheard to have promised some big-business men that he would never sue them for failure to apply for the permits. Although AWH has been unable to confirm the statement, it was learned from a government insider that the Justice Department is not happy with the prospect of having to sue over the permit application issue. The source said the lawyers in Justice feel they would have a stronger case if they took a firm to court for an actual discharge instead.

ABOLITION OF STRIP MINING NOT CONSIDERED LIKELY

Backers of legislation to outlaw strip mining believe chances for such tough legislation are very slim as a House Interior subcommittee proceeds with hearings on the subject. Further hearings on strip mining are scheduled Oct. 21 and 26. In hearings so far, the subcommittee has been very hostile to the abolition proposal supported by Rep. Ken Hechler (D., W.Va.). Observers believe the measure most likely to be reported to the House floor will be similar to that introduced by the powerful Interior Committee chairman, Rep. Wayne Aspinall (D., Colo.). The Aspinall bill would require mining firms to post bonds to guarantee reclamation of the stripped land. But Hechler and conservation groups such as Friends of the Earth object to the large role in regulation that would be left to the states, which have shown themselves to be not very tough toward strip miners. They also think the measure would be tough to enforce.

More acceptable, although still not satisfactory to those who want to ban strip mining, is a measure supported by Rep. Wayne Hays (D., Oh.). It would set up an independent board to administer strip mining and would take it out of state hands. It would call for permits to be issued to strip miners and would provide for certain areas to be declared out of bounds for stripping. Hays, who also is powerful as chairman of the Committee on House Administration, may take the fight to the floor of the House where he is likely to get considerable support.

HALE NAMED AS SOLID WASTE CHIEF OF EPA, SUCCEEDING VAUGHAN

The Environmental Protection Agency last week named Samuel Hale Jr. as deputy assistant director for solid waste programs. The agency is expected to announce shortly that Hale's present job--director of special projects, specializing in the economic impact of pollution control laws--will be taken by Robert Sansom, 50, a staff assistant to Henry Kissinger. Sansom's most recent job has been an evaluation of U.S. policy in Vietnam and Eastern Europe.

Hale, 29, helped establish EPA's noise abatement program and environmental impact statement office. Prior to joining EPA, Hale worked in the management planning group, office of the undersecretary, Department of Health, Education and Welfare. A political science graduate from Stanford, Hale also has a master's degree in business administration from Harvard and has done work on a doctorate in economics at U.C.L.A. He replaces Richard Vaughan, a sanitary engineer, who resigned recently to take a job with a subsidiary of Zurn Environmental Engineering (AWN, Aug. 2, p. 3). Sansom was the top graduate from the Air Force Academy in 1964, has a master's in economics from Georgetown and a Ph.D. from Oxford. He has been a Rhodes and Fulbright scholar and a White House fellow.

The EPA has named lawyer and city planner Edward Furia as administrator of its Region Three office in Philadelphia. Furia, who has headed a consulting firm in Phila-

delphia specializing in the environment, served as director of 1970 Earth Week there. Purie, 30, is a cum laude graduate of the University of Pennsylvania law school and holds a master's in city and regional planning. He replaces acting director Lloyd Gebhard in overseeing EPA activities in Pennsylvania, Delaware, Maryland, Virginia, West Virginia and the District of Columbia.

THOMAN TO WORK IN ATLANTA FOR ZURN ENVIRONMENTAL

John R. Thoman, recently deposed head of the Environmental Protection Agency's Atlanta office, will open an office there for a subsidiary of Zurn Environmental Engineering. The disclosure came in a letter sent last week to all water quality control boards of states within EPA's Southeast region. Thoman is the second top EPA official given a job by Zurn in the past few months. Former Solid Waste Office head Richard Vaughan is working for Zurn in Washington, D.C.

EPA's TURNKEY RULING

UPSETS CONSULTING ENGINEERS

The Environmental Protection Agency decision to encourage municipalities to enter into turnkey contracts for sewage treatment plants (AWN, Sept. 20, p. 1) is being protested by consulting engineers. Officials of the Consulting Engineers Council say they fear that their members' work in sewage treatment construction will be cut back sharply. And they are charging that the turnkey system, where one contractor guarantees design, construction and performance, will not meet the communities' needs.

"Turnkey firms frequently operate with the engineer as subordinate to either the contractor or the manufacturer and as such, are locked into a single process which may or may not be appropriate for a specific community's needs," said council president William A. Sowers. But Eugene Jensen, head of EPA's Water Programs Office, counters that the agency is not requiring turnkey, merely permitting it where it has been forbidden before. He says that no one firm is likely to do all the work itself, but would still hire consulting engineers.

Jensen added, however, that consulting engineers might be affected in cases where a new technology, unfamiliar to an engineer, is sought by a community. In that case, the community would turn completely to the firm specializing in that technology. EPA, in revising regulations on approved technology and financing procedures, has been trying in the last year to encourage adoption of improved technology, he noted. Read that to mean "physical-chemical treatment" * * *

TECHNOLOGY

FORD GOES ENGELHARD: Sources in Detroit last week told AWN that Ford was close to an agreement with Engelhard for that firm to supply platinum catalytic converters for all 1975-model cars. AWN has been unable to confirm the report. But Ford did choose platinum catalysts from Engelhard for 1974-model cars slated for the California market, and announced that it would go platinum in 1975 as well, to meet national hydrocarbon and carbon monoxide standards (AWN, June 7, p. 1).

One source noted that to meet the 1974 order, Engelhard had to ask its suppliers to open new facilities. It would be uneconomical for them to do so without

prospects for continued orders. Most likely alternative supplier: Universal Oil Products.

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UNOX FOR PULP MILL: Union Carbide's Unox system--substituting oxygen for air in biological treatment of organic wastes--will be piloted in a pulp mill for the first time. A \$73,274 grant has been made by Canada's Department of the Environment to conduct a study at Nova Scotia Pulp's Port Hawkesbury, N.S., mill. The mill currently uses a settling lagoon. Unox systems are now pending on nearly two dozen municipal waste treatment plants, mainly in the United States.

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expected in mid-October, will likely be delayed again to late November. Interior sources discounted an interpretation of a recent interview with Secretary Rogers C.B. Morton, who some stories in the national press inferred was seriously considering a Canadian pipeline as an alternative. The Canadian line is being investigated as part of Interior's environmental impact statement, these sources told AWW, but the betting is that the Alaska route will eventually be approved.

LAW TO LET AUTO FIRMS COOPERATE STALLED IN SENATE

A proposal by Sen. Robert Griffin (R., Mich.) to exempt auto companies from antitrust regulations in order to cooperate on pollution control work appears stalled, at least for the moment. Michigan's other senator, Democrat Philip Hart, has refused to hold hearings before his Judiciary Antitrust Subcommittee until the auto companies answer questionnaires he sent out. Among other questions, he asked them to specify their expenditures, with details, for pollution control work. The companies, which have refused in the past to give such detailed information, have not responded to the questionnaires. They reportedly also want to avoid the publicity that the hearings would generate.

Meanwhile, American Motors--one of the firms to be most helped by such an arrangement--is trying to achieve the same result through other methods. It is trying to convince the Justice Department to approve such cooperation without further legislation. American Motors is arguing that a 1969 consent decree the companies signed in settlement of a legal action against them does not preclude valid cooperation. The original legal action had charged the companies with collusion to prevent the development of emission control devices. It was settled through the consent decree. Justice's position on the American Motors plea is not known, but antitrust officials in the past have been against interpreting the consent order loosely.

THE STATES: NEWS IN BRIEF

ALABAMA: Gov. George Wallace last week signed into law a water quality bill closely paralleling the strong air code he signed last month (AWW, Sept. 6, p. 7). The new water law creates a seven-man governing board, none of whose members can be an employee of a firm holding a state water permit. The old board had 14 members, eight of whom were either employees of such firms or state officials with a pro-business slant.

Under the new law, the board may move for immediate injunctions against polluters. Penalties include fines of up to \$10,000 a day for continuing violations. The law also contains provisions for civil actions to recover damages for loss of fish and wildlife and permits the state to sue to recover cleanup costs associated with any act of pollution. No court action was ever brought under the old law. Alabama for the past two years has led the nation in the number of fish kills.

CALIFORNIA: The state court of appeals has declared the State Forest Practice Act in violation of the state and federal constitutions because it leaves rules for forest use "to the uncontrolled discretion of the (forest practice committees) and the timber ownership." Because these committees are industry-dominated, Justice Norman Elkington ruled that the state legislature cannot delegate its authority over forest practices to them. The ruling could give environmental groups a good precedent in trying to overturn the industry-dominated pollution control boards that still exist in many states. Elkington's ruling came in a suit brought by the Sierra Club. Michael McCloskey, the club's executive director, said his group would lobby in the state legislature for a board free of domination by interested parties.

* Four oil companies denied permission to build a drilling platform in the Santa Barbara Channel (AWW, Sept. 27, p. 4) have sued in federal district court in an attempt to upset the decision. The four--Mobil, Gulf, Union Oil of California and Texaco--jointly hold a lease in the channel where Union was planning the platform. That platform and another by Sun Oil were blocked by Interior Secretary Rogers C.B. Morton after what observers feel was political pressure from California Congressmen. In a twist of the normal state of affairs, which has environmental groups citing impact statements to

prove degradation, a spokesman for Mobil noted that "Secretary Morton has elected to ignore the findings of his own (U.S. Geological Survey)...environmental impact statement, which concluded that the platform should be installed to reduce oil pressure in the oil reservoir" under the channel.

HAWAII: The Environmental Protection Agency has given civilian and military officials three years to stop pollution of Pearl Harbor and adjacent areas. The action is the first to be completed successfully under a section of the 1965 Water Pollution Control Act that allows federal action when pollution of navigable waters adjacent to a state endangers public health or the shellfish industry--even if interstate waters are not involved. EPA is involved in similar action in the Galveston Bay area (AWN, June 14, p. 8). Among the restrictions agreed to after a three-day enforcement conference: Erosion controls must be imposed on all state projects, and the Navy was given until the end of this year to develop a waste-control program for its ships in the harbor.

MAINE: The federal district court in Portland has dismissed a civil suit brought in July, 1970, by the Justice Department against Oxford Paper in Rumford. The suit was one of the first 10 brought for alleged mercury pollution under the 1899 Refuse Act. Oxford voluntarily shut down its offending chloralkali facility soon after the suit was filed, but mercury still on the bottom was at issue. Judge Edward Gignoux said, however, that "the necessity for further tests in order to determine the extent of mercury-laden bottom sediments...and the available methods for eliminating such contaminating substances" meant that the government was not prepared for trial.

PENNSYLVANIA: The applicability of the state's pioneering Clean Streams Law, which according to officials holds miners responsible for drainage even from abandoned mines, could be settled by this December. Barnes & Tucker, former operator of a coal mine whose acid drainage last year was responsible for poisoning 200 miles of streams, is fighting a state charge that it is responsible for \$1.5 million in cleanup costs and a \$48,000 monthly treatment bill. Treatment costs are now being shared equally by the company and the state under an interim order. The state last week filed its proposed findings of fact and conclusions. Barnes & Tucker is scheduled to file on Nov. 1.

In 1965, the law was amended to require mine operators to prevent pollution from their mines even after mining ceases. The company says it was issued extensions of a permit granted originally under the old law. Pennsylvania says the extensions were issued with the condition that Barnes & Tucker comply with the amended law. The state also claims that Barnes & Tucker is responsible for acid discharges from adjacent workings, since those discharges enter the river through Barnes & Tucker's mine.

Another key issue: The state contends that Barnes & Tucker failed to depict clearly the exact location of the mine in applying for a permit extension. The company has produced in evidence maps that accurately portrayed the mine as being along the river. The maps had been in the possession of the state.

BOTTLE DEPOSIT LAW

GAINS IN ILLINOIS

In Illinois a proposal mandating a 5-cent deposit on soft-drink and beer containers was passed by the state's Institute of Environmental Quality. The vote was 14 to 7, with public officials and educators supporting the measure and industry members lining up against it. The proposal now goes to the Pollution Control Board, which will begin hearings before the end of the year. Based on recommendations by the state's solid waste management task force, the proposal is modeled after a law passed by Oregon earlier this year. To date, New Jersey is the only other industrial state to consider the Oregon law, but others may follow suit shortly.

Meanwhile, New York City's new packaging law is heading for a showdown. After scheduling a trial to test the constitutionality of a tax on plastics packaging, the state's supreme court (a county court), granted a temporary injunction barring the city, which was not prepared for trial, from collecting the tax. The trial was scheduled to begin Oct. 1, but indications are that it may be further delayed to this week. The court seems sure that the issue can be settled before Oct. 20, when the first payments are due.

PUBLICATIONS

TRANSIENT CLIMATE MODIFICATION is the report of the Study of Man's Impact on Climate sponsored by MIT near Stockholm last summer. The study group, 30 scientists from 14 countries, concluded that "an examination of the critical questions associated with the climatic effects of man's activities should be conducted by scientists from many nations at the earliest possible date." Recommendations were made that global monitoring systems be approved by the United Nations conference on the environment scheduled for Stockholm next June.

This latest report is noteworthy in that it spends some time discussing the effects of man's actions other than industrialization. For instance, 20% of the world's land area has been modified over the past few thousand years by agricultural activities. These modifications have changed the land's ability to absorb and release heat and water. MIT Press, 28 Carleton St., Cambridge, Mass. 02142. 291 pages. \$2.95, paper; \$15 cloth.

Although the book was written mainly by scientists for the U.N. committees working on the Stockholm meeting, it can be profitably absorbed by the lay reader. The first half of the book is an overview of the field. The second comprises a detailed assessment of our present knowledge (for lack of it) and pinpoints some specific problem areas. The reader can be sure he is getting an accurate, politically detached view of the possible effects of man's activities on climate, with areas of uncertainty clearly mapped out. As such, this report updates and expands the discussion of climate modification in *Man's Impact on the Global Environment*, published by M.I. last year (reviewed in *AMN*, Dec. 7, p. 8).

EFFLUENT TESTING: "What do the numbers mean?" is one of the first questions environmental engineers ask when confronted with new standards. The test method—and the way it is executed—all too often make the difference between a tight standard and a lax one. The distinction is important not only to the men who do the testing, but to the government and corporate officials who depend on their data. *Instrumental Analysis for Water Pollution Control* can be used by those involved in test procedures every day. But this presupposes little knowledge of chemistry, making it ideal for those in management who must supervise such testing.

The book is also well-suited for students in environmental science courses. In fact, it was prepared for use in World Health Organization educational programs. If there is one phrase to describe the book, it is "up to the minute" in discussing the application of measurement techniques—from advanced gas chromatography and continuous monitoring to anodic stripping voltammetry. Another description would be "accurate." Khalil Mance of the University of Michigan has done a fine editing job. However, for use as a text one would have wished more numerical examples than are given in the 20 pages of chapter XIV. An accompanying workbook would do the trick nicely. Ann Arbor Publishers, P. O. Box 1425, Ann Arbor, Mich. 48106. 331 pages. \$16.50.

ELECTRIC POWER: The electric utilities, their suppliers and their regulating agencies are being flayed for a number of practices which help lead to unnecessary environmental degradation. Yes, most utilities continue to bemoan impending power shortages while promoting increased electricity use. Yes, the price of electricity does not even begin to pay the social costs of environmental degradation incurred in, for instance, attendant strip mining and oil exploration. Yes, American utilities this year are spending only \$41 million for R&D—three-fourths of which is for plant beautification and environmental impact statements—and nearly \$400 million for promoting increased use. These dreary facts—and some equally dreary stuff on nuclear power—are rehearsed with an emphasis on New York State's problems in *Second National River Polls* (Neil Fabricant and Robert Marshall Hallman, George Braziller, One Park Ave., New York, N.Y. 10016. \$8.95, cloth; \$3.95, paper).

Is there an easy answer to the nation's power problems, one that can be applied soon? The book offers the standard recommendations for environmental controls. Helpfully (certainly). A final solution? Not likely. Consider this: "Even assuming near zero population growth [and] a...50% reduction in the current rate of increase in power use in the next decade, U.S. consumption of electricity will still triple by 1990." The quote is from *People, Power, Pollution*, a study of the environmental and public interest aspects of electric powerplants sitting by California Institute of Technology's Environmental Quality Lab., 1201 E. California Blvd., Pasadena, Calif. 91106. \$1.00 plus handling. Both books are fairly well documented and both recommend improved methods and institutions for balancing environmental risks against public benefits.

Much has happened and much has been learned since the December, 1969, American Association for the Advancement of Science session on environmental alteration took place. Nonetheless, the proceedings of that meeting, just published, command the attention of those who wish the "insight gained by tracing the origins of current arguments." Here is a landmark speech on the possible dangers of nuclear power by Arthur Tamplin—along with comments by Merrill Eisenbud and others. Here, too, is a warning on possible climatic changes from increased atmospheric carbon dioxide by Gordon MacDonald. Edited by David Berkowitz and Arthur Squires. MIT Press, 28 Carleton St., Cambridge, Mass. 02142. 440 pages. \$15.95.

NAM TELECONFERENCE: On May 26 of this year, the National Association of Manufacturers held a nationwide teleconference in which environmental officials explained new air and water legislation to 7,000 businessmen (*AMN*, May 31, p. 2). NAM has now published the transcript of the conference, including 300 representative questions asked by the conferees and audience. Ratifications of the 1899 Refuse Act permit program were especially well explored. Available from NAM, 277 Park Ave., New York, N.Y. 10017. 138 pages, paper. \$4 for NAM members, \$8 for nonmembers.

Another useful tool from NAM: proceedings of the National Conference on Depreciation, Reform and Capital Recovery Policy, held in Washington last April 21st. 39 pages, paper. \$2.

TEST LABS: Need a stack or outfall checked? The American Society for Testing and Materials has published a 39-page directory of commercial and institutional testing laboratories. Ask for ASTM publication 3338, ASTM, 1916 Race St., Philadelphia, Pa. 19103. \$3.50.

CADMIUM IN THE ENVIRONMENT: an annotated compilation of almost every significant piece of research that has been done on this ubiquitous metal, is now available from the National Technical Information Service, Springfield, Va. 22151. This report, done at Stockholm's Karolinska Institute under contract to EPA, was first presented at the University of Rochester in June (*AMN*, June 21, p. 3).

While no specific recommendations for industrial cadmium emissions or human intake were made, the report is likely to form the cornerstone of standards EPA may eventually set for cadmium. One thing appears certain at this point: current cadmium levels will be lowered. In the United States, the only cadmium standard is for drinking water—0.01 milligram per liter. As a guide, consider standards set in Japan, where cadmium has been blamed for causing deformities and death. The limit for mine drainage is 0.1 mg/l. The drinking water standard is the same as in the U.S. Maximum ambient atmospheric levels range from 0.88 to 2.93 micrograms per cubic meter. Means are limited to 0.1 microgram per cubic meter. Ask for Report PB-199 795. 353 pages. \$6, paper. 95 cents in microfiche.

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HEALTH ASPECTS OF FIBROUS GLASS

*In
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Prepared By
HEALTH AND SAFETY COMMITTEE



INTRODUCTION

CERTAIN QUESTIONS have been raised about the health aspects of fibrous glass insulation in air-transmission systems for building construction.

Specifically, these questions relate to

1. whether or not fibrous glass insulation erodes from the system and enters the air stream, and
2. whether or not fibrous glass insulation presents a health risk.

This booklet summarizes the important facts about fibrous glass insulation in air-transmission systems. These include:

- * Fibrous glass air-transmission insulation materials meet the requirements of nationally recognized codes and standards and are approved for use by governmental agencies.
- * Extensive industrial hygiene engineering research studies have been carried out by highly qualified university-affiliated medical scientists and industrial hygiene engineers. Studies both in the laboratory and in actual installations demonstrate no significant erosion from the standpoint of either health or comfort.
- * Published authoritative medical scientific research studies over the past 30 years show fibrous glass to be a biologically inert substance which presents no health risk greater than any other nuisance-type dust.
- * The American Conference of Governmental and Industrial Hygienists has scrutinized the scientific research studies concerning the pulmonary effects of fibrous glass and has classified this material as an inert or nuisance-type dust.

BASIC INFORMATION ABOUT FIBROUS GLASS

Types of fibrous glass

As the name implies, fibrous glass is fiber made of glass. It is supplied in two primary forms as a wool-type product principally for thermal and acoustical insulation and as textile yarn for fabrics and strength-giving reinforcements.

Glass fibers are inert, inorganic, dimensionally stable and have high tensile strength. In addition, glass fibers will not burn, rot, absorb moisture or odors.

Fibrous glass cannot be characterized with other fibrous types of insulating materials. Like many substances, while the appearance may be generally similar, the chemical composition, physical characteristics and fiber size are not comparable.

Uses of fibrous glass

Since its development in the early 1930's, fibrous glass has become one of man's most useful basic materials. In its wool-like form, fibrous glass is a widely known insulation for controlling temperature and sound. Among its applications are home insulation, acoustical ceilings, pipe insulation, air-handling ducts and insulation for refrigerators, ranges, automobiles, airplanes and mobile homes. Major markets for textile-type fibrous glass include: curtains and draperies, upholstery, fire-protective clothing plus reinforcements for plastics, paper, rubber products, tires, etc.

FIBROUS GLASS INSULATION IN AIR-TRANSMISSION SYSTEMS

How it is used

When applied to the **exterior** of metal ducts, fibrous glass wool insulation reduces heat loss.

When applied to the **interior** of metal ducts, fibrous glass wool insulation primarily provides noise control in addition to thermal control.

Air ducts made **entirely** of fibrous glass wool insulation provides acoustical and thermal benefits plus certain economic and engineering advantages in specific applications.

Why it is used

Noise control - Fibrous glass insulation cushions and absorbs noise generated by mechanical equipment and air movement.

Temperature control - Fibrous glass insulation materials control heat loss or gain.

Condensation control - Fibrous glass helps control condensation on the inside and, with vapor barrier, on the outside of air-conditioning duct systems.

Safety and Cleanliness - Fibrous glass insulation is fire resistant and does not support mildew, mold or bacteria.

Where it is used

Fibrous glass wool insulation is used as a component of most air-duct systems installed today in residential, industrial, commercial and public buildings.

COMPLIANCE WITH NATIONALLY RECOGNIZED PERFORMANCE CODES AND STANDARDS

Fibrous glass duct materials meet stringent requirements of Underwriters' Laboratories, Inc., and the National Fire Protection Association. These groups determine performance criteria for construction, installation and safety, which are adopted as building codes at federal, state and local levels.

Standards criteria

Underwriters' Laboratories Standards for Safety, U.L. 181-Air Ducts, Test for Erosion Section, states: "Material for duct and connectors shall not break away, flake off or show evidence of delamination or continued erosion when air is passed through typical sections at a velocity of two and one-half times the manufacturer's rated velocity, but not less than 2,500 feet per minute."

Recognized standards developed by the Underwriters' Laboratories and the National Fire Protection Association also evaluate fire-resistance characteristics, strength characteristics, resistance to mildew and mold or bacteria attack and other qualities that an approved material should exhibit.

Fibrous glass air-transmission insulation products are designed and manufactured by the National Insulation Manufacturers Association member companies to meet the requirements of these standards. These products are accepted by all major code-making bodies. In addition, fibrous glass duct insulation materials manufactured for air-conditioning systems by NIMA member companies carry the Underwriters' Laboratories label. The followup inspection and testing procedures of Underwriters' Laboratories assures that the materials comply with these standards.

Standards recognition and application

Because fibrous glass insulation meets the requirements of recognized standard-making bodies, it is used in schools, hospitals, hotels, motels, apartments, factories, theaters, shopping centers, office buildings, homes, etc.

The Federal Government is one of the largest single consumers of fibrous glass air-handling products. The Corps of Engineers, Veterans Administration, General Services Administration, United States Public Health Service (Hill-Burton Program), and other governmental agencies specify and use these products. Other organizations such as the International Conference of Building Officials, Southern Building Code Congress and the Building Officials Conference of America also approve the use of

MEDICAL-SCIENTIFIC RESEARCH—TOXICOLOGY

Published authoritative medical-scientific studies over the past 30 years show fibrous glass to be a biologically inert substance. These studies have been conducted by highly qualified scientists affiliated with independent, university and governmental environmental health groups.

Some of the major studies and a brief summary of their conclusions are as follows:

1. American Conference of Governmental Industrial Hygienists—Adopted Threshold Limit Values and Intended Changes

At the May 1969 annual meeting, this organization changed the tentative threshold limit value of fibrous glass from 5 milligrams-cubic meter of air to 15 milligrams/cubic meter of air (same as nuisance dust value) with regard to airborne fibrous glass which may be inhaled.

2. A report on Health Aspects of Fibrous Glass, Medical Series, Bulletin No. 13-68 by Dr. Robert de Treville, President, Industrial Hygiene Foundation

Medical investigation and scientific medical studies conducted by highly qualified specialists in skin and lung diseases have shown fibrous glass to be a biologically inert substance that produces no permanent injury to individuals exposed to it.

"The documented evidence substantiating this conclusion included investigation of health of hundreds of fibrous glass workers who have been exposed to the material on a regular basis for as long as 25 to 30 years."

3. Airborne Fibrous Glass Particles, by Dr. George W. Wright, A.M.A. Archives of Environmental Health, February, 1968

"The effect, as revealed in the chest roentgenogram, of ten to twenty-five years of exposure in an environment containing airborne particles of fibrous glass has been studied. No unusual pattern of radiologic densities was observed.

"The frequency of various radiologic appearances known to occur in the general population was no higher in those with greatest exposure than in those with the least."

4. Glass Dust: A Study of Its Biological Effects, Dr. Paul Gross, A.M.A. Archives of Industrial Health, January, 1960 —

"No effect of glass dust and glass flakes was demonstrable upon the cornea or the conjunctiva of the eyes."

"No toxic or traumatic effect was demonstrable upon the gastrointestinal tract. There was only slight depression of the growth rate when rats ingested a diet containing as much ground glass (50%) as food. This depression was due entirely to a lower food intake."

"The pulmonary changes in guinea pigs exposed to fine glass dust for one year and those of rats either exposed to the same dust or given injections intratracheally with it were minimal in character and consisted only of small focal alveolar dust cell collections without fibrosis. Coarse glass dust injected into the lungs of rats behaved like inert foreign material. . . ."

Moreover, as recently as April, 1968, Dr. Gross reaffirmed the conclusions as follows:

"Based on all completed and in progress experimental and human data of which we have knowledge, fibrous glass dust should be classified as 'inert', i.e., without demonstrable pathologic effect."

"There should be no adverse effects to health from inhalation of glass dust of respirable size whether in pulverized or filamentous forms."

5. A 1969 letter from E.E. Merrill, Jr., Industrial Hygienist Consumers Protection and Environment Health Service, U.S. Department of Health, Education and Welfare written to Forsythe, Bergmann, Vanek, Architects/Engineers, Canton, Ohio —

"Fibrous glass and rock wool are practically inert materials which do not have any significant toxic effects. Studies of the inhalation of dust from fibrous glass materials have not shown this dust to produce any toxic manifestations beyond the skin irritant properties, which are mechanical in nature."

6. Fiber Glass: Chemistry and Technology, Dr. Carey P. McCord, Journal of Occupational Medicine, July, 1967 —

"Fiber glass, like all common forms of glass, is a chemically inert substance. It possesses no known toxic properties. On a mechanical basis, spicules may abrade the skin leading to superficial and transitory irritation. Among fiber glass makers, particularly new workers who are most exposed, skin irritation occurs, but is not remarkable. Although free silica is a major primary

ingredient of the batch before melting, no free silica occurs in the glass fibers or in the ultimate products. Silicosis is not a fiber glass hazard."

"The present consensus is that, because of the particle size, fiber glass is not respirable, and thus is unlikely to reach the alveoli of the lungs, but it may reach the tissues of the upper respiratory tract. In animal experiments the introduction of fiber glass particles into the respiratory tract has failed to provoke pneumoconiosis. However, some minor tissue changes have been observed, chiefly reflecting a foreign-body reaction. Fiber glass is not a known carcinogen. Animal experimental work with fiber glass particles introduced into the lungs has failed to provide lung cancer. No clinical cases of lung cancer have been traced to fiber glass as the source."

7. Pulmonary Hazards from Exposure to Glass Fibers, Dr. Ahmed N.M. Nasr, Journal of Occupational Medicine, July, 1967

"As far as can be ascertained from a search of the American and European literature, available evidence indicates that fibrous glass is a relatively inert material with no fibrogenic or other significant toxic properties."

8. Fiber Glass Health Hazard Investigation, Dr. W.J. Siebert, Industrial Medicine, January, 1962 —

"In submitting his best judgment, based on his professional interpretation of all facts assembled in the course of the investigation, the author finds that there is no obvious fibrosis or dust hazard connected with working with Fiberglas, and that the manufacture, fabrication and application of Fiberglas products is attended by no unusual occupational hazards."

9. The Pulmonary Reaction to High Concentrations of Fibrous Glass Dust, Dr. P. Gross, Marianne Kaschak, Ethel B. Tolker, Mary Ann Babyak, and Dr. Robert T.P. de Treville, A.M.A. Archives of Environmental Health, June, 1970 —

"The pulmonary response, in both rats and hamsters, to the presence of fibrous glass dust, is characterized by relatively small accumulations of macrophages without significant stromal change. Whether the fibrous glass dust used was coated (either with a phenol-formaldehyde-type resin or with a textile-type binder) or not, there was no demonstrable difference in reaction. This biologic 'inertness' of fibrous glass dust persisted in spite of an extremely high total exposure — 24 months to dust concentrations which averaged somewhat in excess of 100 mg/cu m."

A more detailed summary of these studies is available from NIMA.

INDUSTRIAL HYGIENE ENGINEERING RESEARCH— AIR VELOCITY AND EROSION

Industrial hygiene engineering studies have been conducted by highly qualified scientists affiliated with university and governmental environmental health groups.

Some of the major studies and brief summaries of their conclusions are as follows:

1. Exposures to Fibers in the Manufacture of Fibrous Glass, D.L. Johnson, J.J. Healy, H.E. Ayer, J.R. Lynch, Bureau of Occupational Safety and Health, U.S. Department of Health, Education and Welfare, American Industrial Hygiene Foundation Journal, December, 1969

"In this paper the results of environmental studies of four plants manufacturing fibrous glass insulation and one plant manufacturing fibrous glass textile products are presented. The results in terms of airborne concentration of glass fibers and total dust would indicate that the workman's exposure to these materials is negligible. The airborne concentrations found in fibrous glass industry are contrasted to those found in similar environmental samples taken in a study of the asbestos textile industry. Exposures in the asbestos textile industry to fibers are of the order of twenty times those in the fibrous glass industry."

"Animal experiments and human experience reported in the literature seem to indicate that fibrous glass is a relatively inert material with no fibrogenic or other significant toxic properties."

"From the environmental results found in this survey and data from the previous toxicologic and worker studies, there appears to be no reason to class the manufacture of fibrous glass products as a hazardous occupation."

Industrial Hygiene Engineering Research— Air Velocity and Erosion (cont'd)

2. Fibrous Glass Lined Air Transmission Systems, Dr. W.C. Cooper, LeRoy J. Balzer, D.P. Fowler, June 1, 1970

"In 13 air transmission systems lined with fibrous glass there was a decrease in concentrations of glass fibers in air after the air had passed through the systems; both in number of glass fibers and in the relative proportion of total fibers which were identifiable as glass."

"Data did not preclude the occurrence of erosion, but any which occurred apparently was more than offset by deposition and entrapment of fibers in filters and elsewhere within the systems."

"Glass fibers within building correlated more closely with fiber concentrations in ambient air than with air exiting from the air supply systems lined with fibrous glass."

"The concentrations of total fibers and glass fibers in ambient air, in air exiting air supply systems, and in building areas were extremely low and do not appear biologically significant."

3. The Evaluation of the Effect of Air Velocity on Fibrous Glass Duct and Duct Liner Systems, J. Cholak, Ch. B., P.E., L.J. Schafer, Ch. E., P.E. Kettering Laboratory, University of Cincinnati, September, 1967

"A study of the effects of air velocity on fibrous glass materials in contact with the moving air stream in typical heating and air conditioning ducts was made to determine if fibers were eroded from the fibrous glass duct materials and entrained in the air stream."

"No fibers were found in the air stream exiting seven separate duct systems constructed from commercially available fibrous glass duct materials exposed to air traveling at 3000 fpm continuously for 136 hours."

"It may be concluded that during this test period fibrous duct systems and ducts lined with fibrous glass liner showed no evidence of erosion of fibers."

The Kettering Laboratory made further tests in buildings at Cape Kennedy, Florida, where fibrous glass ducts had been in service for seven years and at Milwaukee, Wisconsin, where fibrous glass ducts had been in service for 10 years. Tests at both sites showed no evidence of fibrous glass insulation erosion."

Industrial Hygiene Engineering Research— Air Velocity and Erosion (cont'd)

4. Exposures of Insulation Workers to Airborne Fibrous Glass, Dr. W. C. Cooper, Leroy J. Balzar, D.P. Fowler, May, 1970.

"Insulation workers, during the actual application of fibrous glass insulation products were exposed to airborne concentrations of glass fibers ranging from 0.5 to 8 fibers/ml, with a median of 1.3 fibers/ml and mean of 1.8 fibers/ml.

"The computed gravimetric concentrations of airborne glass fibers less than 7 μ m in diameter did not exceed the tentative threshold limit value of 10 mg/m³ during any peak period. The majority of these measurements were less than 1.0 mg/m³.

"No time-weighted average could be computed which would be representative of a work day exposure. In any case, it would be considerably less than the peak concentrations measured.

"Insulators work with fibrous glass containing materials from less than 10% to 100% of their time depending on employer and type of construction site.

"It is doubtful that the concentrations of airborne fibrous glass found in our study are of long-term biological significance."

5. The Erosion of Fibers from Installed Fibrous Glass Ducts, J. Cholak, Ch. E., P.E., L.J. Schafer, Ch. E., P.E., Kettering Laboratory, University of Cincinnati, 1970.

"A field study of installed fibrous glass duct systems which had been in service for some time at six different sites was undertaken as a follow-up of a study of new fibrous glass material which had been tested in accordance with specifications by the Underwriters' Laboratories, Inc. As was the case with the earlier test, air samples collected at the outlets of the installed duct systems showed no evidence of the erosion of fibers. Dust which had settled on superstructures or which had been removed by wiping walls near the sampling sites generally contained some glass fibers. The presence of glass fibers in these ducts is attributed to dust which had accumulated over several years or more from many possible sources."

A more detailed summary of these studies is available from the National Insulation Manufacturers Association.

LABOR UNIONS JURISDICTIONAL DISPUTE CONCERNING INSTALLATION

Effective March 12, 1968, national agreements were abrogated between the International Association of Heat and Frost Insulators and Asbestos Workers Union and the Sheet Metal Workers International Association Union.

This problem between two major construction unions arose as the result of work assignment disputes concerning the installation of materials.

The International Association of Heat and Frost Insulators and Asbestos Workers Union generally has the work assignment for placing insulation on the outside of sheet metal ducts for thermal insulation purposes.

About 1950, architects and engineers recognized that fibrous glass duct liner insulation placed on the inside of the metal ducts provides a system that controls noise as well as temperature.

The more practical and economical method for installing fibrous glass duct liner on the inside of the sheet metal duct was to line the inside during fabrication in the sheet metal shop. The Sheet Metal Workers' International Association therefore obtained the work assignment for installing fibrous glass duct liner on the inside of sheet metal ducts during fabrication.

About 1960, engineering specification factors stimulated the need for ducts fabricated entirely of fibrous glass with an external air/vapor barrier. The work assignment for installation of these ducts also was obtained by the Sheet Metal Workers' International Association.

In today's modern heating and air-conditioning systems architects and engineers utilize all the available types of air-transmission systems. However, there has been an increasing trend towards use of fibrous glass internally lined metal ducts and air ducts made entirely of fibrous glass because of benefits to building owners and occupants.

The Heat and Frost Insulators and Asbestos Worker's have recognized the loss of job opportunity because of this new technology and the improved

Labor Unions Jurisdictional Dispute Concerning Installation (cont'd)

design trend. They have attempted, by various means, to reverse this trend and revert design back to thermal insulation on the outside of metal ducts thus preserving their volume of work assignments.

The subject of safety and health has been utilized extensively for the purpose of eliminating fibrous glass thermal and acoustical insulation from the **inside** of ducts and next to the air stream. This strategy and campaign has been pursued unsuccessfully. The preponderance of medical and engineering scientific research to date, shows fibrous glass insulation to be a biologically inert, safe, effective and efficient material.

RESULTS OF PROPOSED LEGISLATION CONTROL

Legislation and codes that would eliminate fibrous glass insulating materials inside of air-transmission systems of buildings have been considered or introduced by various states, counties and municipalities.

The National Insulation Manufacturers Association, along with other national groups, have been active in providing accurate and factual information on this subject to the responsible legislators and code authorities.

As a result, there have been no adverse legislation or code restrictions regarding the use of fibrous glass insulation in air-transmission systems.

The NIMA Health and Safety Committee is available to provide assistance in these matters.

SUMMARY

1. Fibrous glass is an extremely valuable basic material which benefits everyone every day.
2. In the construction industry, fibrous glass is broadly accepted and specified for thermal and acoustical insulation requirements.
3. Fibrous glass duct insulation materials meet the requirements of nationally recognized standard-making bodies and are approved for use by key governmental agencies.
4. Extensive industrial hygiene engineering research studies have been carried out by highly qualified university affiliated industrial hygiene engineers and physicians. Studies both in the laboratory and in actual installations demonstrate no significant erosion from the standpoint of either health or comfort.
5. The subject of safety and health has been utilized extensively by a certain few individuals and groups for the purpose of eliminating fibrous glass thermal and acoustical insulation from the **inside** of ducts. This strategy and campaign has been pursued unsuccessfully. The preponderance of medical and engineering scientific research to date shows fibrous glass insulation to be a biologically inert, safe, effective material.
6. Published authoritative medical scientific research studies over the past 30 years show fibrous glass to be a biologically inert substance which presents no health risk greater than any other innocuous dust.
7. The American Conference of Governmental Industrial Hygienists has scrutinized the scientific research studies concerning the pulmonary effects of fibrous glass and has classified this material as an inert or nuisance-type dust.

Threshold Limit Values
of Airborne Contaminants
AND INTENDED CHANGES
Adopted By ACGIH
For 1970



PREFACE
THRESHOLD LIMIT VALUES FOR 1970

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Documentation of Threshold Limit Values. A separate companion piece to the TLVs is issued by ACGIH under this title. This publication gives the pertinent scientific information and data with reference to literature sources that were used to base each limit. Each documentation also contains a statement defining the type of response against which the limit is safeguarding the worker. For a better understanding of the TLVs it is essential that the Documentation be consulted when the TLVs are being used.

Information concerning the availability of copies of the Documentation of Threshold Limit Values should be directed to the Secretary-Treasurer.

Threshold limit values refer to airborne concentrations of substances and represent conditions under which it is believed that nearly all workers may be repeatedly exposed day after day without adverse effect. Because of wide variation in individual susceptibility, however, a small percentage of workers may experience discomfort from some substances at concentrations at or below the threshold limit, a smaller percentage may be affected more seriously by aggravation of a pre-existing condition or by development of an occupational illness.

Simple tests are now available (J. Occup. Med. 9, 537, 1967; Ann. N.Y. Acad. Sci., 151 Art. 2, p. 968, 1968) that may be used to detect those individuals hypersusceptible to a variety of industrial chemicals (respiratory irritants, hemolytic chemicals, organic isocyanates, carbon disulfide). These tests may be used to screen out by appropriate job placement the hyperreactive worker and thus in effect improve the "coverage" of the TLVs.

Threshold limit values refer to time-weighted concentrations for a 7 or 8-hour workday and 40 hour workweek. They should be used as guides in the control of health hazards and should not be used as fine lines between safe and dangerous concentrations. (Exceptions are the substances listed in Appendices A and E and those substances designated with a "C" or Ceiling value, Appendix C.)

Time-weighted averages permit excursions above the limit provided they are compensated by equivalent excursions below the limit during the workday. In some instances it may be permissible to calculate the average concentration for a workweek rather than for a workday. The degree of permissible excursion is related to the magnitude of the threshold limit value of a particular substance as given in Appendix C. The relationship between threshold limit and permissible excursion is a rule of thumb and in certain cases may not apply. The amount by which threshold limits may be exceeded for short periods without injury to health depends upon a number of factors such as the nature of the contaminant, whether very high concentrations - even for short periods - produce acute poisoning, whether the effects are cumulative, the frequency with which high concentrations occur, and the duration of such periods. All factors must be taken into consideration in arriving at decision as to whether a hazardous condition exists.

A threshold limit of $10\text{mg}/\text{M}^3$, or 30 mppcf, of total dust $\leq 1\%$ SiO_2 , whichever is less, is recommended for substances in these categories and for which no specific threshold limits have been assigned. This limit, for a normal workday, does not apply to brief exposures at higher concentrations. Neither does it apply to those substances which may cause physiologic impairment at lower concentrations but for which a threshold limit has not yet been adopted. Some "inert" particulates are given in Appendix D.

Simple Asphyxiants - "Inert" Gases or Vapors. A number of gases and vapors, when present in high concentrations in air, act primarily as simple asphyxiants without other significant physiologic effects. A TLV may not be recommended for each simple asphyxiant because the limiting factor is the available oxygen. The minimal oxygen content should be 18 percent by volume under normal atmospheric pressure (equivalent to a partial pressure, p_{O_2} of 135 mm Hg). Atmospheres deficient in O_2 do not provide adequate warning and most simple asphyxiants are odorless. Several simple asphyxiants present an explosion hazard. Account should be taken of this factor in limiting the concentration of the asphyxiant. Specific examples are listed in Appendix E.

Physical Factors. It is recognized that such physical factors as heat, ultraviolet and ionizing radiation, humidity, abnormal pressure (altitude) and the like may place added stress on the body so that the effects from exposure at a threshold limit may be altered. Most of these stresses act adversely to increase the toxic response of a substance. Although most threshold limits have built-in safety factors to guard against adverse effects to moderate deviations from normal environments, the safety factors of most substances are not of such a magnitude as to take care of gross deviations. For example, continuous work at temperatures above 90°F or over-time extending the workweek more than 25%, might be considered gross deviations. In such instances judgment must be exercised in the proper adjustments of the threshold limit values.

"Notice of Intent." At the beginning of each year, proposed actions of the Committee for the forthcoming year are issued in the form of a "Notice of Intent." This Notice provides not only an opportunity for comment, but solicits suggestions of substances to be added to the list. The suggestions should be accompanied by substantiating evidence. The list of Intended Changes follows the Adopted Values in the TLV booklet.

As Legislative Code. The Conference recognizes that the TLVs may be adopted in legislative codes and regulations. If so used, the intent of the concepts contained in the Preface

should be maintained and provisions should be made to keep the list current. These values are reviewed annually by the Committee on Threshold Limits for revision or additions, as further information becomes available.

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Substance	ppm ^{a1}	mg/M ³ b)
Cobalt, metal fume & dust	--	0.1
Copper fume	--	0.1
Dusts and Mists	--	1
Corundum (Al ₂ O ₃)	--	1
Cotton dust (raw)	--	1
Crag [®] herbicide	--	15
Cresol (all isomers) - Skin	5	22
Crotonaldehyde	2	6
Cumene-Skin	50	245
Cyanide (as CN)-Skin	--	5
* Cyanogen	10	--
Cyclohexane	300	1,050
Cyclohexanol	50	200
Cyclohexanone	50	200
Cyclohexene	300	1,015
Cyclopentadiene	75	200
2, 4-D	--	10
DDT-Skin	--	1
DDVP, see Dichlorvos	--	--
Decaborane-Skin	0.05	0.3
Demeton [®] -Skin	--	0.1
Diacetone alcohol (4-hydroxy-4-methyl-2-pentanone)	50	240
1, 2-Diaminoethane, see Ethylene-diamine	--	--
Diazomethane	0.2	0.4
Diborane	0.1	0.1
C 1, 2-Dibromoethane (ethylene dibromide)-Skin	25	190
Dibutyl phosphate	1	5
Dibutylphthalate	--	5
* C Dichloroacetylene	0.1	0.4
C o-Dichlorobenzene	50	300
p-Dichlorobenzene	75	450
Dichlorodifluoromethane	1,000	4,950
1, 3-Dichloro-5, 5-dimethyl hydantoin	--	0.2
1, 1-Dichloroethane	100	400
1, 2-Dichloroethane	50	200
1, 2-Dichloroethylene	200	790
C Dichloroethyl ether-Skin	15	90
Dichloromethane, see Methylene-chloride	--	--
Dichloromonofluoromethane	1,000	4,200
C 1, 1-Dichloro-1-nitroethane	10	60
1, 2-Dichloropropane, see Propylene-dichloride	--	--
Dichlorotetrafluoroethane	1,000	7,000
Dichlorvos (DDVP)-Skin	--	1
Dieldrin-Skin	--	0.25
Diethylamine	25	75
Diethylamino ethanol-Skin	10	50
** C Diethylene triamine-Skin	10	42
Diethylether, see Ethyl ether	--	--
Difluorodibromomethane	100	860
C Diglycidyl ether (DGE)	0.5	2.8
Dihydroxybenzene, see Hydroquinone	--	--
Diisobutyl ketone	50	290
Diisopropylamine-Skin	5	20

* 1970 Addition

** See Notice of Intended Changes

Substance	ppm ^{a1}	mg/M ³ b)
Dimethoxymethane, see Methylal	--	--
Dimethyl acetamide-Skin	10	35
Dimethylamine	10	18
Dimethylaminobenzene, see Xylidene	--	--
Dimethylaniline (N-dimethylaniline)-Skin	5	25
Dimethylbenzene, see Xylene	--	--
Dimethyl 1, 2-dibromo-2, 2-dichloroethyl phosphate, (Dibrom)	--	3
Dimethylformamide-Skin	10	30
2, 6-Dimethylheptanone, see Diisobutyl ketone	--	--
1, 1-Dimethylhydrazine-Skin	0.5	1
Dimethylphthalate	--	5
Dimethylsulfate-Skin	1	5
Dinitrobenzene (all isomers)-Skin	--	1
Dinitro-o-cresol-Skin	--	0.2
Dinitrotoluene-Skin	--	1.5
Dioxane (Diethylene dioxide)-Skin	100	360
Diphenyl	0.2	1
Diphenyl amine	--	10
Diphenylmethane diisocyanate (see Methylene bisphenyl isocyanate (MDI))	--	--
Dipropylene glycol methyl ether-Skin	100	600
Di-sec, octyl phthalate (Di-2-ethylhexylphthalate)	--	5
Emery	--	0
* Endosulfan (Thiodan [®]) -Skin	--	0.1
Endrin-Skin	--	0.1
Epichlorohydrin-Skin	5	19
EPN-Skin	--	0.5
1, 2-Epoxypropane, see Propylene-oxide	--	--
2, 3-Epoxy-1-propanol, see Glycidol	--	--
Ethane	E	--
Ethanethiol, see Ethylmercaptan	--	--
Ethanolamine	3	6
2-Ethoxyethanol-Skin	200	740
2-Ethoxyethylacetate (Cellulosolve acetate)-Skin	100	540
Ethyl acetate	400	1,400
Ethyl acrylate-Skin	25	100
Ethyl alcohol (ethanol)	1,000	1,900
Ethylamine	10	18
Ethyl sec-amylic ketone (5-methyl-3-heptanone)	25	130
Ethyl benzene	100	435
Ethyl bromide	200	890
Ethyl butyl ketone (3-Heptanone)	50	230
Ethyl chloride	1,000	2,600
Ethyl ether	400	1,200
Ethyl formate	100	300
Ethyl mercaptan	0.5	1
Ethyl silicate	100	850
Ethylene	E	--
Ethylene chlorohydrin-Skin	5	18

* 1970 Addition

** See Notice of Intended Changes

Substance	ppm ¹⁾	mg/M ² b)
1,4-Cyclohexanone-Skin	100	460
1-Ethyl ketone (MEK), see Hexanone	--	--
1-Propanol	--	--
1-Propanol formate	100	250
1-Propanol iodide - Skin	5	28
1-Propanol isobutyl carbinol-Skin	25	100
1-Propanol isobutyl ketone, see Hexanone	--	--
1-Propanol isocyanate - Skin	0.02	0.05
1-Propanol mercaptan	0.5	1
1-Propanol methacrylate	100	410
1-Propanol propyl ketone, see 2-Pentanone	--	--
1-Propanol silicate	5	30
1-Propanol styrene	100	480
1-Propanol bisphenyl isocyanate (MDI)	0.02	0.2
1-Propanol chloride (dichloromethane)	500	1,740
1-Propanol (soluble compounds)	--	5
1-Propanol (insoluble compounds)	--	15
1-Propanol aniline-Skin	2	9
1-Propanol hydrazine-Skin	0.2	0.35
1-Propanol isocyanate-Skin	20	70
1-Propanol (coal tar)	100	400
1-Propanol	10	50
1-Propanolamine	--	A ¹
1-Propanol carbonyl	E	--
1-Propanol, metal and soluble compds, as Ni	0.001	0.007
1-Propanol-Skin	--	1
1-Propanol acid	2	8
1-Propanol oxide	25	30
1-Propanol-Skin	1	6
1-Propanol-Skin	1	5
1-Propanol benzene-Skin	--	1
1-Propanol	100	310
1-Propanol iodide	E	--
1-Propanol diiodide	5	9
1-Propanol trifluoride	10	29
1-Propanol-Skin	0.2	2
1-Propanol	100	250
1-Propanol	25	90
1-Propanol	25	90
1-Propanol dimethylamine (dimethyl-amine)-Skin	--	A ¹
1-Propanol-Skin	5	30
1-Propanol chloromethane, see Chloromethane	--	--
1-Propanol iodide	E	--
1-Propanol naphthalene-Skin	--	0.1
1-Propanol particulate	400	1,900
1-Propanol vapor	0.1 A ³	sh
1-Propanol iodide	--	0.002
1-Propanol iodide	--	1
1-Propanol iodide	0.05	0.1
1-Propanol iodide	0.1	0.2
1-Propanol-Skin	--	1.5

Substance	ppm ¹⁾	mg/M ² b)
Parathion-Skin	--	0.1
Pentaborane	0.005	0.01
Pentachloronaphthalene-Skin	--	0.5
Pentachloronaphthalene-Skin	--	0.5
Pentacrythritol	500	1,500
* Pentane	200	700
2-Pentanone	100	670
Perchloroethylene	0.1	0.8
Perchloromethyl mercaptan	3	13.5
Perchloryl fluoride	3	13.5
* Petroleum Distillates (naphtha)	1) A ³	--
Phenol-Skin	5	19
p-Phenylene diamine-Skin	--	0.1
Phenyl ether (vapor)	1	7
Phenyl ether-Biphenyl mixture (vapor)	1	7
Phenylethylene, see Styrene	--	--
Phenyl glycidyl ether (PGE)	10	60
Phenylhydrazine-Skin	5	22
Phosdrin (Mevaphos * 3-Skin)	--	0.1
Phosgene (carbonyl chloride)	0.1	0.4
Phosgene	0.3	0.4
Phosphine	--	1
Phosphoric acid	--	0.1
Phosphorus (yellow)	--	1
Phosphorus pentachloride	--	1
Phosphorus pentasulfide	--	1
Phosphorus trichloride	0.5	3
Phthalic anhydride	2	12
Picric acid-Skin	--	0.1
Pival * (2-Pivalyl-1, 3-indandione)	--	0.1
Pival	--	D
Plaster of Paris	--	0.002
Platinum (Soluble Salts) as Pt	--	0.002
Polytetrafluoroethylene decomposition products	--	A ²
Propane	E	--
Propyl acetate	1	A ¹
Propargyl alcohol-Skin	200	840
n-Propyl acetate	200	500
Propyl alcohol	25	110
n-Propyl nitrate	75	350
Propylene dichloride	2	5
Propylene imine-Skin	100	240
Propylene oxide	--	--
Propyne, see Methylacetylene	--	5
Pyrethrum	5	15
Pyridine	0.1	0.4
Quinone	--	1.5
RDX-Skin	--	0.1
Rhodium, Metal fume and dusts, as Rh	--	0.001
Soluble salts	--	10
Ronnel	--	5
Rotenone (commercial)	--	D
Rouge	--	0.2
Selenium compounds (as Se)	0.05	0.4
Selenium hexafluoride	--	D
Silicon carbide	--	D

1) According to analytically determined composition.

Capital letters refer to Appendixes.

* 1970 Addition

** See Notice of Intended Changes

Substance	ppm ¹	mg/m ³ bi
Silver, metal and soluble compounds	--	0.01
Sodium fluoroacetate (1080)-Skin	--	0.05
Sodium hydroxide	--	2
Stibine	0.1	0.5
Starch	--	D
* Stoddard solvent	200	1,150
Strychnine	--	0.15
**C Styrene monomer (phenylethylene)	100	420
Sucrose	--	D
Sulfur dioxide	5	13
Sulfur hexafluoride	1,000	6,000
Sulfuric acid	--	1
Sulfur monochloride	1	5
Sulfur pentafluoride	0.025	0.25
Sulfuryl fluoride	5	20
Systox, see Demeton *	--	--
2, 4, 5 T	--	10
Tantalum	--	5
TEDP-Skin	--	0.2
Teflon * decomposition products	--	A ²
Tellurium	--	0.1
Tellurium hexafluoride	0.02	0.2
TEPP-Skin	--	0.05
C Terphenyls	1	9
1, 1, 1, 2-Tetrachloro-2, 2-di-fluoroethane	500	4,170
1, 1, 2, 2-Tetrachloro-1, 2-di-fluoroethane	500	4,170
1, 1, 2, 2-Tetrachloroethane-Skin	5	35
Tetrachloroethylene, see Per-chloroethylene	--	--
Tetrachloromethane, see Carbon tetrachloride	--	--
Tetrachloronaphthalene-Skin	--	2
Tetraethyl lead (as Pb)-Skin	--	0.100 (1)
Tetrahydrofuran	200	590
Tetramethyl lead (as Pb)-Skin	--	0.150 (1)
Tetramethyl succinonitrile-Skin	0.5	3
Tetranitromethane	1	8
Tetryl (2, 4, 6-trinitrophenyl-methyl)nitramine-Skin	--	1.5
Thallium soluble compounds-Skin as Tl	--	0.1
Thiram	--	5
Tin (inorganic cmpds, except SnH ₄ and SnO ₂)	--	2
Tin (organic cmpds)	--	0.1
Tin oxide	--	D
Titanium dioxide	--	D
Toluene (toluol)	200	750
C Toluene-2, 4-dithiocyanate	0.02	0.14
o-Toluidine-Skin	5	22
Toxaphene, see Chlorinated camphene	--	--
Tributyl phosphate	--	5
1, 1, 1-Trichloroethane, see Methyl chloroform	--	--
1, 1, 2-Trichloroethane-Skin	10	45

Substance	ppm ¹	mg/m ³ bi
Trichloroethylene	100	335
Trichloromethane, see Chloroform	--	--
Trichloronaphthalene-Skin	--	5
1, 2, 3-Trichloropropane	50	300
1, 1, 2-Trichloro 1, 2, 2-tri-fluoroethane	1,000	7,600
Triethylamine	25	100
Trifluoromonomomomethane	1,000	6,100
* Trimethyl benzene	25	120
2, 4, 6-Trinitrophenol, see Picric acid	--	--
2, 4, 6-Trinitrophenylmethyl nitramine, see Tetryl	--	--
Trinitrotoluene-Skin	--	1.5
Triorthocresyl phosphate	--	0.1
Triphenyl phosphate	--	3
Tungsten & compounds, as W	--	--
Soluble	--	1
Insoluble	--	5
Turpentine	100	560
Uranium (natural) sol. & insol. compounds as U	--	0.2
C Vanadium (V ₂ O ₅ dust)	--	0.5
(V ₂ O ₅ fume)	--	0.1
Vinyl benzene, see Styrene	--	--
**C Vinyl chloride	500	1,300
Vinylcyanide, see Acrylonitrile	--	--
Vinyl toluene	100	480
Warfarin	--	0.1
Xylene (xylo)	100	435
Xylidine-Skin	5	25
Yttrium	--	1
Zinc chloride fume	--	1
Zinc oxide fume	--	5
Zirconium compounds (as Zr)	--	5

Radioactivity For permissible concentrations of radionuclides in air, see U.S. Department of Commerce, National Bureau of Standards, Handbook 69, "Maximum Permissible Body Burdens and Maximum Permissible Concentrations of Radionuclides in Air and in Water for Occupational Exposure," June 5, 1959. Also, see U.S. Department of Commerce National Bureau of Standards, Handbook 59, "Permissible Dose from External Sources of Ionizing Radiation," September 24, 1954, and addendum of April 15, 1958.

¹ For control of general room air, biologic monitoring is essential for personnel control.

* 1970 Addition

** See Notice of Intended Changes

* 1970 Addition

** See Notice of Intended Changes

MINERAL DUSTS

Substance	mg/m ³ f ¹
SILICA	
Crystalline	
** Quartz, Threshold Limit calculated from the formula	250 f ¹ %SiO ₂ x 5
** Cristobalite " " "	
Amorphous, including natural diatomaceous earth	20
SILICATES (less than 1% crystalline silica)	
** Asbestos, all types	5
Mica	20
Portland Cement	50
Soapstone	20
Talc (non-asbestiform)	20
Talc (fibrous), use asbestos limit	--
Tremolite, see asbestos	--
Graphite (natural)	15
** "Inert" or Nuisance Particulates	50 (or 15 mg/M ³ whichever is the smaller) of total dust < 1% SiO ₂
see Appendix D	
Conversion factors	
mg/m ³ x 35.3 = million particles per cubic meter	
= particles per c.c.	

- a) Millions of particles per cubic foot of air, based on impinger samples counted by light-field technique.
- b) The percentage of crystalline silica in the formula is the amount determined from x-ray fluorescence samples, except in those instances in which other methods have been shown to be applicable.

** See Notice of Intended Changes for Mineral Dusts.

NOTICE OF INTENDED CHANGES (for 1970)

These substances, with their corresponding values, comprise those for which either a limit has been proposed for the first time, or for which a change in the "Adopted" listing has been proposed. In both cases, the proposed limits should be considered trial limits that will remain in the listing for a period of at least two years. During this time, the previously Adopted Limit will remain in effect. If, after two years no evidence comes to light that questions the appropriateness of the values herein, the values will be placed in the "Adopted" list. Documentation is available for each of these substances.

MERCURY (all forms except alkyl) 0.05 mg/M³

Substance	ppm	mg/M ³
2-Acetylaminofluorene-Skin	--	A ¹
Allyl glycidyl ether	5	22
4-Aminodiphenyl-Skin	--	A ¹
Ammonia	25	18
Ammonium chloride fume	--	10
Asphalt (petroleum) fumes	--	5
Butyl lactate	1	5
Camphor (synthetic)	2	12
Diazinon-Skin	--	0.1
2-N Dibutylamino ethanol-Skin	2	14
Dichlorobenzidine-Skin	--	A ¹
Diethylene triamine-Skin	1	4
4-Dimethylaminoazobenzene	--	A ¹
Fibrous glass	g ¹ --	D
C Formaldehyde	2	3
Iron pentacarbonyl	0.01	0.08
Mercury (Alkyl compounds) - Skin	--	0.01
Methyl chloride	100	210
Methyl 2-cyanoacrylate	2	8
Methylcyclopentadienyl manganese tricarbonyl (as Mn)	0.1	0.2
Methyl demeton-Skin	--	0.5
Methyl parathion-Skin	--	0.2
Phenothiazine-Skin	--	5
Rosin Core Solder, pyrolysis products	--	0.1 (as aldehyde)
Styrene	100	420
C Subtilisin (Proteolytic enzymes)	--	0.0003 (as 100% pure crystalline enzyme)
Vanadium (V ₂ O ₅ Fume) as V	--	0.05
Vinyl acetate	10	30
Vinyl chloride	200	770
Wood dust (non allergenic)	--	5

- a) 1970 Revision or Addition
- Capital letters refer to Appendices
- g) 3-7 μ Diameter. No TLV for coarse fibrous glass has yet been set.

NOTICE OF INTENDED CHANGES (Cont'd) MINERAL DUSTS

SUBSTANCE

TLV

- Asbestos (All types)	5 fibers/ml - 5 μ in length ^k
- Coal dust (bituminous)	2 mg/m ³ (respirable dust) ^m
- Cristobalite	Use one-half the value calculated from the count or mass formulae for quartz.
- 'Inert' or Nuisance Particulates	10 mg/M ³ or 30 mppcf (whichever is the smaller) of total dust - 1% SiO ₂ ⁿ
- Quartz	TLV in mppcf = $\frac{300}{\%SiO_2 \cdot 10}$
	TLV for respirable dust in mg/m ³ = $\frac{10 \text{ mg/M}^3}{\% \text{ Respirable quartz} \cdot 2}$
	TLV for 'total dust', respirable and nonrespirable = $\frac{30 \text{ mg/M}^3}{\% \text{ quartz} \cdot 3}$
Silica, fused	Use quartz formulae
Tridymite	Use one-half the value calculated from formulae for quartz.

- 1970 Revision or Addition

- k) As determined by the membrane filter method at 450 X magnification, phase contrast illumination. Concentrations - 5 fibers/ml, but not to exceed 10, may be permitted for 15-minute periods each hour up to five times daily.
- m) "Respirable" dust as defined by the British Medical Research Council Criteria (1) and as sampled by a device producing equivalent results (2).
- (1) Hatch, T.E. and Gross, P., Pulmonary Deposition and Retention of Inhaled Aerosols, p. 149, Academic Press, New York, New York, 1964.
- (2) Interim Guide for Respirable Mass Sampling, ABIA Aerosol Technology Committee, ABIA J. 31, 2, 1970, p. 132.
- n) This automatically reduces all particulate substances in Adopted list with TLV of 15 mg/M³ to 10 mg/M³.
- p) Both concentration and per cent quartz for the application of this limit are to be determined from the fraction passing a size-selector with the following characteristics:

Aerodynamic Diameter (μ) (unit density sphere)	% passing selector
≤ 2	90
2.5	75
3.5	50
5.0	25
10	0

APPENDIX A

- A¹ Because of the high incidence of cancer, either in man or in animals, no exposure or contact by any route, respiratory, oral or skin should be permitted for the compounds:

2-Acetylaminofluorene	beta-Naphthylamine
4-Aminodiphenyl	4-Nitrodiphenyl
Benidine & its salts	N-Nitrosodimethylamine
Dichlorobenzidine	beta-Propiolactone
4-Dimethylaminoazobenzene	

Because of the extremely high incidence of bladder tumors in workers handling beta-naphthylamine and the potential carcinogenic activity of the other compounds, the State of Pennsylvania prohibits the manufacture, use and other activities that involve human exposure without express approval by the Department of Health.

- A² **Polytetrafluoroethylene* decomposition products.** Thermal decomposition of the fluorocarbon chain in air leads to the formation of oxidized products containing carbon, fluorine and oxygen. Because these products decompose in part by hydrolysis in alkaline solution, they can be quantitatively determined in air as fluoride to provide an index of exposure. No TLV is recommended pending determination of the toxicity of the products, but air concentrations should be minimal.

- A³ **Gasoline and/or Petroleum Distillates.** The composition of these materials varies greatly and thus a single TLV for all types of these materials is no longer applicable. In general, the aromatic hydrocarbon content will determine what TLV applies. Consequently the content of benzene, other aromatics and additives should be determined to arrive at the appropriate TLV (Elkins, et al, A.I.H.A.J. 24, 99, 1963).

* Trade Names: Algidon, Fluon, Halon, Teflon, Tefron

APPENDIX B

B.1 THRESHOLD LIMIT VALUES FOR MIXTURES

When two or more hazardous substances are present, their combined effect, rather than that of either individually, should be given primary consideration. In the absence of

information to the contrary, the effects of the different hazards should be considered as additive. That is, if the sum of the following fractions,

$$\frac{C_1}{T_1} + \frac{C_2}{T_2} + \dots + \frac{C_n}{T_n}$$

exceeds unity, then the threshold limit of the mixture should be considered as being exceeded. C_1 indicates the observed atmospheric concentration, and T_1 the corresponding threshold limit, (See Example 1A.a.).

Exceptions to the above rule may be made when there is good reason to believe that the chief effects of the different harmful substances are not in fact additive, but independent as when purely local effects on different organs of the body are produced by the various components of the mixture. In such cases the threshold limit ordinarily is exceeded only when at least one member of the series ($\frac{C_1}{T_1}$ or $\frac{C_2}{T_2}$ etc.) itself has a value exceeding unity, (See Example 1A.b.).

Antagonistic action or potentiation may occur with some combinations of atmospheric contaminants. Such cases at present must be determined individually. Potentiating or antagonistic agents are not necessarily harmful by themselves. Potentiating effects of exposure to such agents by routes other than that of inhalation is also possible, e.g. imbibed alcohol and inhaled narcotic (trichloroethylene). Potentiation is characteristically exhibited at high concentrations, less probably at low.

When a given operation or process characteristically emits a number of harmful dusts, fumes, vapors or gases, it will frequently be only feasible to attempt to evaluate the hazard by measurement of a single substance. In such cases, the threshold limit used for this substance should be reduced by a suitable factor, the magnitude of which will depend on the number, toxicity and relative quantity of the other contaminants ordinarily present.

Examples of processes which are typically associated with two or more harmful atmospheric contaminants are welding, automobile repair, blasting, painting, lacquering, certain foundry operations, diesel exhausts, etc., (Example 2.)

THRESHOLD LIMIT VALUES FOR MIXTURES EXAMPLES

1A. General case, where air is analyzed for each component

- a. Additive effects. (note. It is essential that the atmosphere be analyzed both qualitatively and quantitatively for each component present, in order to evaluate compliance or noncompliance with this calculated TLV.)

$$\frac{C_1}{T_1} + \frac{C_2}{T_2} + \frac{C_3}{T_3} + \dots = 1$$

Example No. 1. Air contains 5 ppm of carbon tetrachloride (TLV = 10 ppm) 20 ppm of ethylene dichloride (TLV = 50 ppm) and 10 ppm of ethylene dibromide (TLV = 25 ppm)

Atmospheric concentration of mixture =

$$5 + 20 + 10 = 35 \text{ ppm of mixture}$$

$$\frac{5}{10} + \frac{20}{50} + \frac{10}{25} = \frac{25 + 20 + 20}{50} = 1.3$$

Threshold Limit is exceeded. Furthermore, the TLV of this mixture may be calculated by reducing the total fraction to 1.0, i.e.

$$\text{TLV of mixture} = \frac{35}{1.3} = 27 \text{ ppm}$$

Example No. 2. Air contains 200 ppm of hexane (TLV = 500 ppm) 100 ppm of methylene chloride (TLV = 500 ppm) and 20 ppm of perchlorethylene (TLV = 100 ppm)

Atmospheric concentration of mixture =

$$200 + 100 + 20 = 320 \text{ ppm of mixture}$$

$$\frac{200}{500} + \frac{100}{500} + \frac{20}{100} = \frac{200 + 100 + 100}{500} = \frac{400}{500} = 0.8$$

Threshold Limit is not exceeded. The TLV of this mixture = $\frac{320}{0.8} = 400 \text{ ppm}$

T = Threshold Limit Value in ppm.
 C = Vapor concentration in ppm.
 p = Vapor pressure of component in solution.
 p⁰ = Vapor pressure of pure component.
 F = Mol fraction of component in solution.
 a = A constant of proportionality.

Subscripts 1, 2, . . . n relate the above quantities to components 1, 2, . . . n, respectively.

Subscript i refers to an arbitrary component from 1 to n.

Absence of subscript relates the quantity to the mixture.

1B.c. Solution to be applied when there is a reservoir of the solvent mixture whose composition does not change appreciably by evaporation.

Exact Arithmetic Solution of Specific Mixture

	Mol. wt.	Density	TLV	p ⁰ at 25°C	Mol fraction in half-and-half solution by volume
Trichloroethylene (1)	131.4	1.46g/ml	100	73mmHg	0.527
Methylchloroform (2)	133.42	1.33g/ml	350	125mmHg	0.473

$$F_1 p_1^0 = (0.527) (73) = 38.2$$

$$F_2 p_2^0 = (0.473) (125) = 59.2$$

$$TLV = \frac{38.2 + 59.2}{\frac{38.2}{100} + \frac{59.2}{350}} = \frac{(97.4) (350)}{133.8 + 59.2} = \frac{(97.4) (350)}{193.0} = 177$$

TLV = 177 ppm (Note difference in TLV when account is taken of vapor pressure and mol fraction in comparison with above sample where such account is not taken.)

2. A mixture of one part of (1) parathion (TLV, 0.1) and two parts of (2) EPN (TLV, 0.5).

$$\frac{C_1}{0.1} + \frac{C_2}{0.5} = \frac{C_m}{T_m} \quad C_2 = 2C_1$$

$$C_m = 3C_1$$

$$\frac{C_1}{0.1} + \frac{2C_1}{0.5} = \frac{3C_1}{T_m}$$

$$\frac{7C_1}{0.5} = \frac{3C_1}{T_m}$$

$$T_m = \frac{1.5}{7} = 0.21 \text{ mg/M}^3$$

1C. TLV for Mixtures of Mineral Dusts.

For mixtures of biologically active mineral dusts the general formula for mixtures may be used.

For a mixture containing 80% talc and 20% quartz, the TLV for 100% of the mixture is given by:

$$TLV = \frac{1}{\frac{0.8}{20} + \frac{0.2}{2.5}} = 8.4 \text{ mppcf}$$

Essentially the same result will be obtained if the limit of the more (most) toxic component is used provided the effects are additive. In the above example the limit for 20% quartz is 10 mppcf.

For another mixture of 25% quartz, 25% amorphous silica and 50% talc:

$$TLV = \frac{1}{\frac{0.25}{2.5} + \frac{0.25}{20} + \frac{0.5}{20}} = 7.3 \text{ mppcf}$$

The limit for 25% quartz approximates 8 mppcf.

FOREWORD

From time to time, since publication of its first bulletin in 1935, the Industrial Hygiene Foundation of America, Inc. (IHF) has issued medical reports on subjects of topical interest to its members.

IHF is a nonprofit research association of industries for the advancement of healthful working conditions and human relations in industry, and serves a unique function as the focal point for exchange of information on health problems allegedly arising from industries' operations, products and wastes.

The Foundation's ability is vested in individuals with highly specialized scientific, technical and professional backgrounds and experience in its member organizations as well as in its full-time staff, its standing committees, and expert panel of advisors. Its work is therefore supported by numerous individuals in hundreds of companies deeply committed to the advancement and fulfillment of the Foundation's mission.

At the invitation of IHF, Dr. Jon L. Konsen, Medical Director of Owens-Corning Fiberglass Corporation, Toledo, Ohio, assisted in the preparation of this bulletin in association with Dr. Paul Gross, Director of the Foundation's Research Laboratory, based on an extensive review of all available experimental and human data, published or in progress. The information contained herein is presented for the guidance of the members of IHF and all those in the fields of environmental and occupational health who should benefit from this documentation which gives evidence that fibrous glass is inert and non-injurious to health.

Robert T. P. deTreville, M.D., Sc.D.,
President
Industrial Hygiene Foundation of America, Inc.

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INTRODUCTION

Fibrous glass is produced in two forms—as a wool-type product primarily for thermal and acoustical insulation and as a textile fiber for fabrics and strength-giving reinforcements.

Since its development in the early 1930's, fibrous glass has become one of society's most useful basic materials. Originally manufactured as a material for furnace filters and home insulation, fibrous glass now goes into more than 33,000 end products.

In addition to furnace filters and home insulation, major applications of wool-type fibrous glass today include acoustical ceilings, filters for purifying water and thermal insulation for pipe, air conditioning ducts, food cold storage systems and household refrigerators, ovens and other appliances. Major markets for textile fibrous glass are: curtains and draperies, bedspreads, table cloths, awnings, fire protective clothing and reinforcements for plastics, rubber and paper.

Interest in possible occupational and environmental health problems with fibrous glass goes back almost as far as the first development work on the material in 1931. In most cases, this interest has centered on possible effects from skin contact, inhalation and ingestion of small fibrous glass particles.

Results of medical research, including examinations of hundreds of persons who have worked in fibrous glass plants as long as 25 to 30 years, gives evidence that fibrous glass is inert and non-injurious to the individual's over-all health.

The purpose of this report is to review the findings of the most significant of these studies in an effort to clarify the health aspects of fibrous glass.

EFFECTS OF EXPOSURE TO FIBROUS GLASS

Obviously, continued exposure to excessive quantities of any dust or fumes should be avoided. However, dusts and fumes differ widely in their potential harm to human health. Some prove injurious in low atmospheric concentrations; others are harmless at high concentrations.

Skin irritation occasionally results from exposure to fibrous glass particles, but this irritation has proved to be superficial and transitory.

Fibrous glass manufacturers have always been interested in determining whether or not fibrous glass particles are injurious if inhaled or ingested. As a result, they have cooperated in many medical research studies conducted over the past three decades by medical scientists and physicians considered to be authorities in their medical specialties.

Based on existing medical evidence dating back to 1940 through to the present, airborne fibrous glass is not a hazard to general health. Hundreds of glass workers have been exposed to higher-than-average concentrations of airborne glass particles virtually every working day for as long as 25 to 30 years without exhibiting any pattern of lung disease that can be attributed to exposure to fibrous glass.

MEDICAL RESEARCH STUDIES

Among the most significant of medical studies conducted to date on fibrous glass were the investigations in 1963 by George W. Wright, M.D., of the Medical Research Department of St. Luke's Hospital in Cleveland.

The study¹ is a review of periodic chest x-rays, medical records and job exposure histories of 1,401 employees at the largest and oldest fibrous glass manufacturing plant. None of these workers had been employed less than 10 years. Many were employed at the plant upwards of 25 years.

The report gives the purpose of the research in its opening sentence

The question to be explored is whether or not prolonged exposure of workers to an environment containing airborne fibrous glass coated with phenolic resin... is associated with a recognizable pattern of roentgenographic abnormality.

The report's summary states as follows

The effect, as revealed in the chest roentgenogram, of ten to twenty-five years of exposure in an environment containing airborne particles of fibrous glass has been studied. No unusual pattern of radiologic densities was observed.

The frequency of various radiologic appearances known to occur in the general population was no higher in those with greatest exposure than in those with the least.

Another important study² made between 1956 and 1958 by Paul Gross, M.D., Research Professor, University of Pittsburgh, Graduate School of Public Health; Senior Fellow, Mellon Institute; and Director of the IHF Research Laboratory, Pittsburgh, Pa., was a laboratory animal study consisting of three parts.

1. determination of the pulmonary response resulting from exposure to high concentrations of respirable, airborne glass particles (hundreds of millions of particles per cubic foot) and the injection into the lungs of both coarse and fine glass particles to determine pulmonary response to the glass;^{*}

* Respirable means sufficiently small to gain access to the deepest lung tissues, ordinarily 3μ or less in diameter, i.e., microscopic and submicroscopic sized particles. Fine particles used in this study were approximately 1μ in diameter. Coarse particles, put through a 325 mesh sieve (U.S. Standard No. 40) were 50μ or less in diameter with a few particles measuring as much as 400μ or 0.4 mm.

2. to document the effects of the massive ingestion of ground glass of microscopic dimensions (laboratory animals were fed a diet containing 50% finely milled glass flakes for one year); and
3. to study the effects resulting from dusting the eyes of laboratory animals with finely milled glass dust and glass flakes.

Following are excerpts from Dr. Gross' summary and conclusions:

"No effect of glass dust and glass flakes was demonstrable upon the cornea or the conjunctiva of the eyes.

"No toxic or traumatic effect was demonstrable upon the gastrointestinal tract. There was only slight depression of the growth rate when rats ingested a diet containing as much ground glass (50%) as food. This depression was due entirely to a lower food intake.

"The pulmonary changes in guinea pigs exposed to fine glass dust for one year and those of rats either exposed to the same dust or given injections intratracheally with it were minimal in character and consisted only of small focal alveolar dust cell collections without fibrosis... Coarse glass dust injected into the lungs of rats behaved like inert foreign material...."

As recently as April, 1968, Dr. Gross reaffirmed the conclusions:

"Based on all completed and in-progress experimental and human data of which we have knowledge, fibrous glass dust should be classified as 'inert', i.e., without demonstrable pathologic effect.

"There should be no adverse effects to health from inhalation of glass dust of respirable size, whether in pulverized or filamentous forms."

Two of the more recent investigations were reported in the July, 1967,

Journal of Occupational Medicine. In one article⁴ Carey P.

M.D., Professor Emeritus of the Institute of Industrial Health at

a memorandum to Robert T. P. deTreville, M.D. from Paul Gross, M.D., April 11, 1968.

the University of Michigan, Ann Arbor, Michigan, summarized his investigation of fibrous glass manufacture and technology with this statement:

Fiber glass, like all common forms of glass, is a chemically inert substance. It possesses no known toxic properties. On a mechanical basis, spicules may abrade the skin leading to superficial and transitory irritation. Among fiber glass makers, particularly new workers who are most exposed, skin irritation occurs, but is not remarkable....

In the second article,⁵ Ahmed N. M. Naser, M.D., also of the Institute of Industrial Health at the University of Michigan, reviewed the world's literature covering health aspects of exposure to fibrous glass.

These excerpts from the report summarize Dr. Naser's conclusions:

As far as can be ascertained from a search of the American and European literature, available evidence indicates that fibrous glass is a relatively inert material with no fibrogenic or other significant toxic properties."

A study⁶ of human skin reaction to contact with fibrous glass was conducted in 1957 by Eldred R. Heisel, M.D., formerly Chief, Division of Dermatology, and John H. Mitchell, M.D., Clinical Professor and Head of the Division of Allergy, Ohio State University, Columbus, Ohio. This study included both patch and rubbing tests on two groups of human volunteers. One group of 50 white women had never been exposed to fibrous glass. The second group consisted of 92 persons who had been exposed to fibrous glass for periods ranging from less than one year to as long as 15 years.

Excerpts from the report's summary follow:

"... We were unable to induce sensitivity in the first group (i.e., 50 women) and no sensitization reactions were noted in the second group (i.e., 92 persons).

"Reactions were observed when finely cut fibers were applied to the skin. These varied directly with the size (diameter) of the fiber and were interpreted as being due to mechanical irritation...."

SUMMARY

Medical investigations and scientific medical studies conducted by highly qualified specialists in skin and lung diseases have shown fibrous glass to be a biologically inert substance that produces no permanent injury to individuals exposed to it.

The documented evidence substantiating this conclusion includes investigation of the health of hundreds of fibrous glass workers who have been exposed to the material on a regular basis for as long as 25 to 30 years.

Medical publications on fibrous glass include other studies, investigations and literature reviews. Several of these are listed for information 7-13 purposes.

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Glass Dust: A Study of Its Biologic Effects

PAUL GROSS, M.D.; MARIAN L. WESTRICK, Ph.D., and JAMES M. McNERNEY, M.P.H., Pittsburgh

Introduction

Glass has been produced, used, and broken for many centuries, but because this material is not readily reduced to a fine particle size, it is only with the advent of new and mass production techniques that the problem of a possible health hazard from glass dust could have arisen.

Perhaps because every-day experience of civilized man has taught him that freshly broken fragments of glass possess remarkable and often treacherous potentialities for inflicting incisive skin trauma, the public-at-large seems to have acquired the impression that glass may be lethal when ground finely enough so as to be imperceptible when mixed with food. This impression is reinforced from time to time by sporadic reports in newspapers of attempted homicide by the admixture of ground glass with food.

Efforts have recently been made to clarify the potential pulmonary hazard of two types of glass dust. One of these was dust derived from a mixture of filamentous glass and a plastic.¹ In this investigation, no attempt was made to assess the possible role which glass itself may have played, independently of the plastic component, in the production of the results obtained. Neither was a determination made of the relative amount of glass present in the dust to which the animals had been exposed. Furthermore, although the dust was a rapidly settling one and dust counts had been performed, no indication was given as to

the percentage of the dust which may have been sufficiently fine to be respirable. For these reasons, we believe that this published investigation adds little to our knowledge of the hazard of glass dust as such. The other study² was concerned with dust from glass wool. However, because of certain aspects to be dealt with later, some of the conclusions set forth in the latter study do not seem warranted to us.

We undertook the present investigation in order (1) to study the pulmonary response to a glass dust composed of very finely divided particles and to a glass dust composed of larger flake-like particles; (2) to document the effects of massive ingestion of ground glass so that some scientific information may be available on this subject, and (3) to study the potential of a glass dust with larger flake-like glass particles to irritate mucous membranes.

Materials and Methods

The glass dust used in this investigation was prepared from flakes of glass* which were approximately 5 μ thick. The glass contained, in addition to SiO₂, the oxides of Al, Ca, Mg, and B as major constituents. Also present, but in amounts less than 1% and usually more than trace, were the following elements: Na, K, Ti, Fe, and Zr. Fluorine was also present in traces to 0.3%.

A coarse dust was prepared by milling the glass flakes so that the resulting particles passed through a 325 mesh sieve.

In order to obtain a very fine glass dust, the milled flakes were suspended in water and atomized under a pressure of 90 ψ . The jet of suspended glass fragments was made to impinge against a tool-steel baffle at near-sonic velocity. Thereby very small particles were produced. These particles, like those of kaolin and quartz which were produced in a similar manner as control dusts, became admixed with an appreciable amount of iron from the abraded tool-steel baffle. The

* Fiberglass Flake.

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This investigation was supported by a grant from the Owens-Corning Fiberglass Corporation, Toledo, Ohio.

GLASS DUST

dusts were collected by means of electrostatic precipitators.

This investigation was composed of three parts:

1. Eye irritation study
2. Ingestion study
3. Pulmonary study

Eye Irritation Study.—Two experiments were performed, each using six eyes of albino rabbits. In the first, the lids were held open only long enough to dust the ocular conjunctiva thoroughly with the finely milled glass flakes (the largest particles around 400 μ but the vast majority less than 50 μ). In the second experiment, the lids were held open about one minute after the dusting. The eyes were examined 24 hours later with the aid of fluorescein.

Ingestion Study.—Young male white rats from Carworth Farms, approximately 28 days old, were divided into four groups of 10 each after a two-week observation period for acclimatization. These groups were given the following diets:

1. 10% finely milled glass flakes (by weight) incorporated in basic laboratory diet, *ad lib*. The basic laboratory diet consisted of pulverized Purina Laboratory Chow.

2. Pair-fed control; each rat fed only as much basic laboratory diet as its mate from the preceding group consumed on the previous day.

3. 50% finely milled glass flakes (by weight) incorporated in basic laboratory diet, *ad lib*.

4. Control; basic laboratory diet, *ad lib*.

The animals were kept on this regimen for a full year. During this time, weekly records were kept of the body weights and of the general appearance of the animals. Biweekly records were kept of the food consumption in all except the group receiving 50% glass in the diet. Due to much spillage and greater rate of consumption of the diet, the record of food consumption in the latter group was made more frequently.

At the end of the year, all surviving rats were weighed and then killed with ether. Autopsies were performed, and the liver and kidney weights were recorded. Sections were prepared from each of the following organs for microscopic examination: esophagus, forestomach, stomach, duodenum, jejunum, ileum, colon, liver, spleen, kidney, adrenal, testis, lungs, and heart.

Pulmonary Studies.—These studies consisted of one in which dusts were deposited by intratracheal injection and one in which the pulmonary deposition was accomplished by inhalation.

A. Intratracheal Injection: It was desired to test the pulmonary response to a relatively coarse glass dust and also to a very finely divided one. Because the coarse glass dust tended to settle very rapidly in an aqueous suspension it was found necessary to use corn oil (Mazola) as a suspending medium in order to accomplish the injections.

TABLE 1.—Physical Data of Intratracheal Injections

Dust	% Flakes	Particle Size	Amount Injected, Mg.	Suspending Medium, 1 MI.
Glass	45	*	20	corn oil *
Glass	15	1.5 μ ±	20	water
Quartz	15	1.5 μ ±	20	water
Kaolin	15	0.75 μ ±	20	water
	45		1 ml.	corn oil *

* This material passed through a No. 60 U. S. standard sieve. On microscopic examination it was found that, although a small number of particles was in the neighborhood of 400 μ , the vast majority was in the region of 50 μ or less.

* Manta.

* Surface mean diameter (2 μ) $\pm$ 20% determined by optical sizing with oil immersion lens at a magnification of $\times 975$.

For the fine glass dust and the necessary control dusts (kaolin and quartz), water proved to be satisfactory. There were five groups of 15 rats each. Four of these groups were used for the two glass dusts and the two control dusts, whereas the fifth group was injected intratracheally with corn oil as an additional control. The physical data of these injections are given in Table 1.

The injections were administered under light ether anesthesia by means of a blunt-tipped No. 18 gauge spinal needle introduced between the vocal chords. This was accomplished under direct observation by means of a self-retaining, illuminated speculum.*

Three rats were killed from each group at 6 months and the survivors at 12 months. Autopsies were performed on these rats plus all that had died of spontaneous disease. Paraffin sections were cut of the lungs after formalin fixation. These were routinely stained with hematoxylin and eosin. Some were also stained with Van Gieson's stain and by the Gordon and Sweet method for reticular fibers. Selected slides were subjected to incineration in order to study the topographic relationship of the dust.*

B. Inhalation Exposure. In this study, both rats and guinea pigs were used. In addition to exposures to glass dust, animals were exposed to quartz dust and to kaolin dust, respectively. Accordingly, there were three groups of 40 rats each and three groups of 15 guinea pigs each. Each group was housed in a separate inhalation chamber. The dust exposures were for six hours per day, five days per week, for one year.

The method of generating the dust cloud, its dispersal, and control were similar to that previously described.* The experimental conditions in the inhalation chambers and the particle size distribution of the various dusts are listed in Tables 2 and 3.

TABLE 2—*Experimental Conditions of Inhalation Exposure*

	Glass		Quartz		Kaolin	
	Rate	Guinea Pigs	Rate	Guinea Pigs	Rate	Guinea Pigs
Dust concentration, mg./cc. m. ³	18.6	17.1	21.6	24.0	27.0	28.4
Average particle size, μ †						
Optical microscopy, $\times 970$		1.1		1.2		0.7
Electron microscopy, $\times 18,720$		0.08		0.04		0.04
Air flow, cu. m./hr.		4.0		4.0		4.0

* The calculated numbers of particles per cubic foot in the different chambers vary as follows:

from 3.7×10^4 to 1.6×10^5 if based on optical microscope sizing;

from 1.7×10^4 to 8.6×10^5 if based on electron microscope sizing.

† Surface mean diameter (2nd $\sigma/2$ nd σ).

In an attempt to minimize infections, all animals throughout the laboratory were given a saturated solution of sulfathiazole for eight hours once every week as drinking water.

Deaths caused by pneumonias made 13 replacements necessary among the guinea pigs during the first five months of exposure. After the conclusion of the 12-month exposure period, the animals were pastured for another year. After 6 months and again after 12 months of exposure,

Results

1. *Eye Irritation Study*.—The glass particles were very effectively cleared from the ocular conjunctiva by blinking. Careful search for corneal defects failed to reveal any abnormality.

2. *Ingestion Study*.—The significant data, including those of average food and glass

TABLE 3—*Particle Size Distribution of Inhalation Chamber Dusts**

Particle Diameter, μ	Glass		Quartz		Kaolin	
	% by number	% by mass	% by number	% by mass	% by number	% by mass
<0.4	88.2	2.0	64.8	1.2	32.0	3.5
0.4	10.0	3.9	10.4	1.6	33.2	18.1
0.6	6.6	5.3	6.0	3.1	9.6	17.6
0.9	4.2	11.3	8.8	14.8	3.4	21.1
1.3	2.8	22.8	7.6	39.0	1.6	29.8
1.8	2.0	43.2	2.4	39.0	0.2	9.9
2.5	0.2	11.6	0.2	7.3		

* Determined by optical sizing with oil immersion at a magnification of $\times 970$.

several animals from each group were killed for the purpose of sampling. All survivors were killed at the end of the second year. Autopsies were performed on all animals killed as well as on those that had died of intercurrent infections. Sections were prepared and stained as in the intratracheal injection series. Incineration studies were also performed on selected slides in this series.

consumption, mean body weights, and organ weights, are given in Table 4. A graphic representation of the gain in body weight is given in Figure 1.

Throughout the feeding period of one year, there was no appreciable difference

TABLE 4—*Average Food Consumption, Body Weights, and Organ Weights of Male Rat Receiving Various Levels of Glass in Basic Laboratory Diet for One Year*

Level	Survival	Total Diet Consumed, Av/Rat/Day, Gm.	Food Consumed, Av/Rat/Day, Gm.	Glass Consumed, Av/Rat/Day, Gm.	Mean Wt., Gm.		Mean Gain, Gm.		Organ Wt., % of Body Wt.	
					Initial	Final			Liver	Kidney
10% glass	9/10	19.6	17.6	1.55	138	430	292		3.3	0.7
Pair-fed control	8/10	17.2	17.2	—	137	411	274		3.3	0.7
Ad lib control	9/10	19.1	19.1	—	141	480	339		3.3	0.7
30% glass	10/10	34.2	17.1	17.1	128	411	283		3.1	0.7

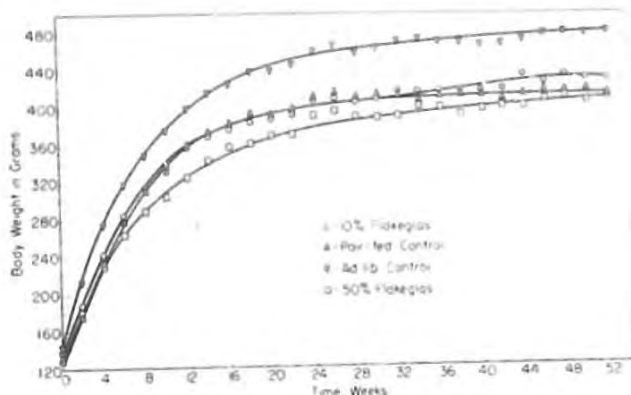


Fig. 1.—Growth curves of male rats fed normal diets and glass dust.

between the control groups and those receiving glass in their diet as judged by general appearance, behavior, growth (Fig. 1) and survival (Table 4). All of the rats on the 50% glass diet survived the experiment. The deaths in the other three groups appeared to have been caused by pneumonia and were associated with weight losses.

The average total diet consumed per rat per day on the 10% glass level (Table 4) approximated the amount eaten by the *ad libitum* controls. The rats on the 50% glass level compensated for the large amount of non-nutritive material in their diet by almost doubling their daily food intake so that the actual amount of food consumed each day approximated the corresponding values for the 10% glass and the pair-fed control groups. The growth curves of these three groups closely approximate each other.

The average weights of the liver and kidneys as listed in Table 4 indicate that there is no significant difference between the values obtained for the experimental groups and the corresponding values for the controls.

Microscopic examination of the various organs from all of these animals revealed no significant abnormality. There was no

evidence of trauma, ulceration, or degeneration of the gastrointestinal mucous membrane.

3. Pulmonary Studies. A. Inhalation of Glass Dust—Guinea Pigs. Grossly, no significant changes were noted. Microscopically, there were widely scattered, relatively small foci of clustered alveoli which contained massed dust cells. Proliferation of alveolar lining cells about the dust cells caused widening of some of the alveolar walls. The dust cells varied in color from tan to brown and gave a positive reaction for iron. The lung tissue between the dust foci was generally normal. No collagen formation attributable to the dust could be demonstrated.

The lymph nodes contained dust cells in the form of small foci and also loosely scattered.

Foci of spontaneous disease were fairly common and had to be differentiated from the experimentally induced lesions. The former included foci of interstitial eosinophilic pneumonia, focal adenomatosis, and a peculiar lipid pneumonia in which signet-ring type cells were a striking feature.

There were also four instances of an interesting disease of connective tissue characterized by focal interstitial pulmo-

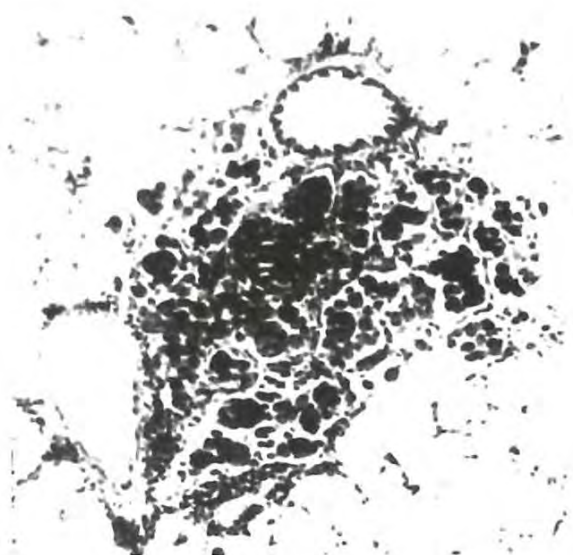


Fig. 2. Tumor. The
300, expanded and printed
as shown in Figure 1.
The tumor is a paratracheal lymph node.
In addition to lymph
vessels, scattered dark, ill-
defined phagocytes are present.
The latter are limited to
identical in the magnifica-
tion. Hematoxylin and
eosin. 195. K. fixed
80% of the original.

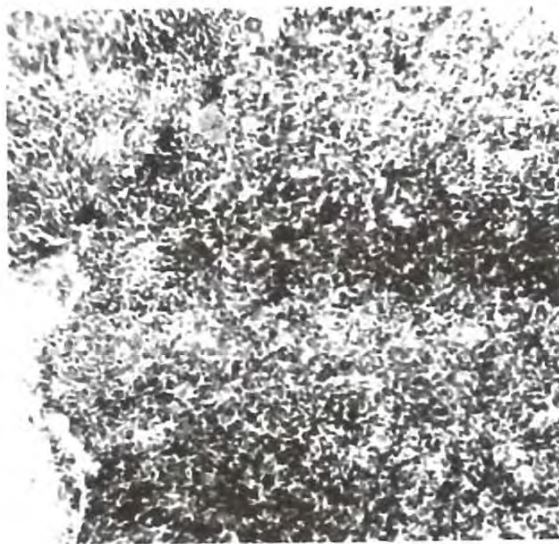


Fig. 3. Tumor. The
300, expanded and printed
as shown in Figure 1.
The tumor is a paratracheal lymph node.
In addition to lymph
vessels, scattered dark, ill-
defined phagocytes are present.
The latter are limited to
identical in the magnifica-
tion. Hematoxylin and
eosin. 195. K. fixed
80% of the original.

nary fibrosis associated with chronic bronchitis and emphysema of cigarette smokers, and is not at all in addition to pulmonary disease. The gross appearance of the lungs of the rats was similar to that of the aorta, pulmonary artery, renal pelvis, the heart, and testes. The rats were diseased, which resembles a disease of human lungs recently described¹⁸ and is the subject of a separate publication.

B. Inhalation of Glass Dust (Rats). No significant microscopic lesion was noted. The microscopic indications of dust exposure were minimal and consisted of widely scattered small foci of clustered alveolar macrophages tan to brown in color. There were also the usual porta- and perivascular collections of lymphocyte-like cells not unusual in the lungs of rats and guinea pigs. Some of these cellular foci contained a few dust cells. The alveolar walls often were thickened in the regions of the dust cells by a proliferation of lining cells. A rare alveolar giant cell was found. The intervening lung tissue was normal except for an occasional focus of spontaneous disease. There was less evidence of dust storage in

the lungs than in the aorta, the wall of the esophagus, and the heart after the inhalation of 12.5 mg of dust/kg body weight.

C. Intratracheal Dust (Rats). The rats that had inhaled 12.5 mg of dust/kg body weight contained more alveolar dust than rats of such exposure in which the alveolar macrophages had been removed by repeated pleurocentesis. In addition, although the rats that the bronch nodes of these rats also contained the usual dust cell foci, such dust cell foci were also present in the lymph nodes of rats that had been killed. They consisted of small collections of tan-colored macrophages and were not associated with the notation of connective tissue.

D. Intratracheal Injection (Guinea Pigs) of Glass Dust. The lungs showed no significant gross changes. Evidence of the injections could be seen microscopically in the form of scattered small accumulations of transparent plate- and spindle-shaped fragments of glass, and sometimes occupying a single alveolus or a small cluster of alveoli. These collections of glass fragments were associated with increased proliferation of alveolar lining cells, and some became surrounded by a

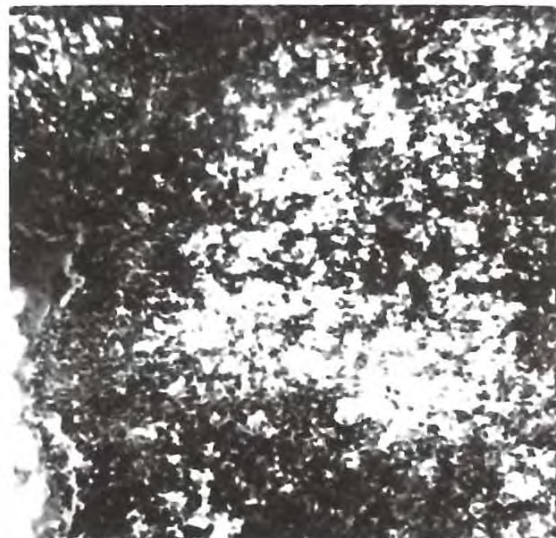


Fig. 4. Glass dust (12.5 mg/kg body weight) in the lungs of a rat. The dust is represented by the small, dark, irregularly shaped particles in the tissue. It is easily recognized from the appearance in Figure 3. (H&E, 100 $\times$, focal field 152, center, right)



Fig. 10. Lymph node (K-3173) showing cellular structures. Large, pale-staining cells with granular cytoplasm are reticuloendothelial cells. Mild cellular cell proliferation is present about the blood vessels. Hematoxylin and eosin; X 100. (Courtesy of the author.)

Fig. 11. Lymph node (K-3173) showing cellular structures. Large, pale-staining cells with granular cytoplasm are reticuloendothelial cells. Mild cellular cell proliferation is present about the blood vessels. Hematoxylin and eosin; X 100. (Courtesy of the author.)



Fig. 7. Same cell as in Figure 6, but with superimposed with pattern showing densely aggregated dust which is practically limited to the foot of large, pale cells. $\times 197$. Reduced 8% on the original.

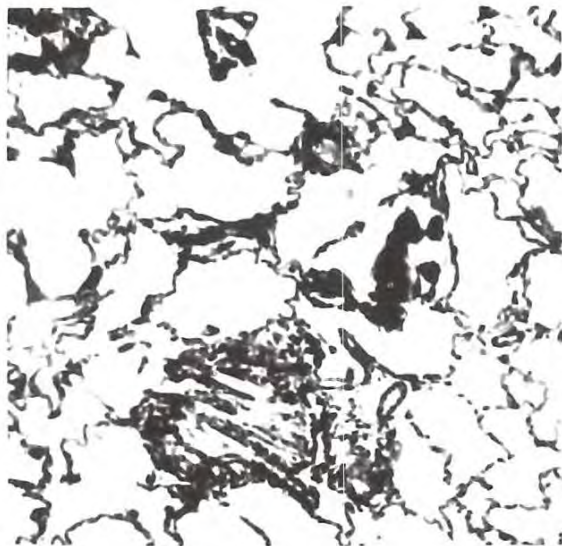
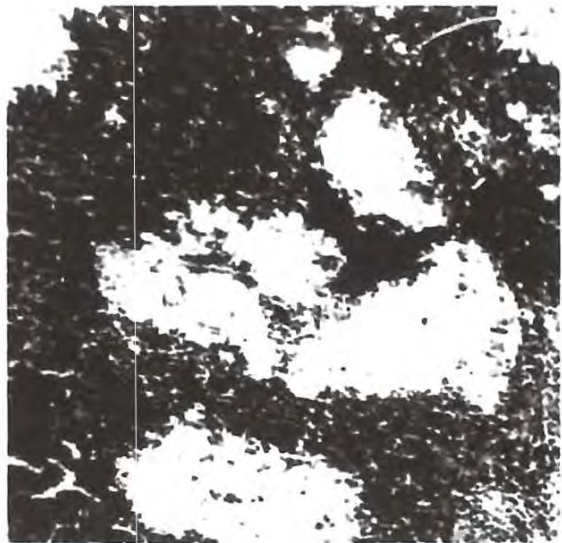
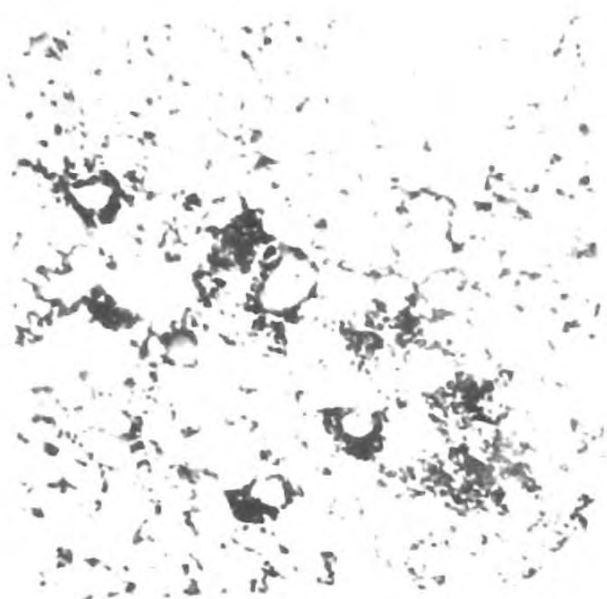


Fig. 8. Rat 4081 excreta 4 intracutaneously with 25 mg. coarse glass dust and half 1 cc. sea water. One of the welch injected, or all deposits of glass dust. The size of the glass plates is indicated by the dots with parallel sides. These dots are associated with proliferation of glycocal cells and an occasional giant cell. The deposit is delimited by glycocal walls. Hematoxylin and eosin. $\times 197$. Reduced 8% on the original.



In addition to scattered, insignificant foci of spontaneous disease in surviving animals, there were 12 deaths from pneumonia. Some pneumonias were of the eosinophilic, interstitial type, others were frankly purulent, while still others had a lipidic component.

One instance of systemic connective tissue disease was found in which there was focal pulmonary fibrosis with basophilia and calcification of elastic tissue.

B. Inhalation—rats. Grossly, the lungs of this group of animals showed irregular consolidations of a fleshy character. The hard, gray-white, nodular, cartilage-like consolidation typical of silicosis was not seen. The lymph nodes were enlarged and firm-to-hard.

The microscopic examination revealed widespread formation of nodules typical of silicosis, except that no collagen was present at 6 months and relatively little at 12 months. After this time, there was increasingly greater collagenization of the silicotic nodules. In addition to the formation of nodules, there was widespread and increasingly severe alveolar stasis of lipidic debris and macrophages. This was associated with widening of alveolar walls, which developed collagen with the passage of time. The number of silicotic nodules and the amount of alveolar debris was usually sharply reduced in animals which had pneumonoma or pulmonary edema.

The lymph nodes showed variable degrees of fibrosis and extensive replacement of lymphoid tissue by masses of reticuloendothelial cells.

C. Intratracheal injection—rats. As in the preceding group, the gray-white cartilage-like, nodular consolidation usually seen in well-developed experimental silicosis was not seen in the lungs of these rats. Instead a more fleshy, irregularly distributed consolidation was found.

Microscopically, numerous silicotic nodules were present which were cellular and contained no collagen at six months. At the end of the year, the nodules in most

animals demonstrated considerable amounts of collagen. However, there were a few rats in which collagenization of the nodules was still absent at this time. There was much alveolar stasis of lipidic debris and macrophages. In the regions of alveolar stasis, there were many cholesterol crystals with associated giant cells. The alveolar walls adjoining plugged air spaces were widened by proliferation of the lining cells, and, in the older animals, focal collagenization of the thickened walls was noted.

The lymph nodes were enlarged and showed varying amounts of fibrosis. Much of the lymphoid tissue was replaced by masses of dust-containing reticuloendothelial cells.

Kaolin. A. Inhalation—guinea pigs. Aside from slight pleural mottling of opaque tan, no significant gross change was noted in the lungs.

Microscopically, there were widely scattered relatively small foci of clustered alveoli plugged by massed dust cells similar to those of glass dust. Here, also, there was widening of adjoining alveolar walls by proliferated alveolar cells. No collagenization was found. The intervening lung tissue was normal except for occasional foci of spontaneous disease.

The lymph nodes contained large masses of dust cells but showed no connective tissue reaction to their presence.

A number of spontaneous deaths occurred. Many of these guinea pigs had a chronic pneumonoma in which lipid-containing cells of signet-ring type were a striking feature. When pulmonary edema was demonstrable in the sections, the dust foci appeared disrupted and reduced in number. There were also three animals with the same type of systemic connective tissue disease as was noted in the other groups. This was associated with focal interstitial pulmonary fibrosis, elastic tissue basophilia, and calcification.

B. Inhalation—rats. Except for slight tan pleural mottling, no significant abnor-

malities were noted grossly in the lungs of this group.

There were numerous relatively small, scattered dust foci composed of packed, large, dust-filled macrophages which plugged alveoli and were delimited by alveolar walls. Delicate collagen fibers could be seen coursing between the cells and were also occasionally noted about the periphery, in the alveolar walls. Some of the dust foci contained a sprinkling of small lymphocyte-like cells. The intervening lung tissue was unaffected by the dust.

In the presence of spontaneous disease, the dust foci were often disrupted, so that loose, clay-filled macrophages were found scattered in the air spaces over large areas and the number of discrete dust foci was reduced.

The lymph nodes contained large masses of dust-filled macrophages, but no collagen formation was demonstrable.

C. Intratracheal injection rats. The gross findings were similar to those of the preceding group. The kaolin dust foci microscopically were generally small and widely scattered. Their structure was identical to that of the preceding group. The intervening lung tissue was unaffected by dust and essentially normal.

The lymph nodes were also very similar to those of the preceding group.

Mazola Control. Grossly, the lungs appeared normal. Microscopically, the lung sections showed only an occasional alveolus containing a group of unattached brown-pigmented macrophages. No collagen formation was noted, and the lymph nodes were normal throughout.

Comment

It is highly probable that the relatively large flat surface presented by the glass flakes caused them to become oriented parallel to the conjunctival surface due to surface attraction of the conjunctival fluid. This parallel orientation diminished the probability of injury from the sharp edges of the flakes.

It would seem that the effect of the glass in the diet was entirely that of a bulky, biologically inert material. The depression in the growth curve of the 10% glass group as compared to that of the *ad libitum* group can be accounted for by the fact that, although an appreciable amount of the dietary intake of the former group was non-nutritious, this dietary intake was approximately the same as that of the *ad libitum* group.

Inasmuch as there were no significant changes in general behavior, organ weight, or the microscopic appearance of the various organs, it is probable that the slight depression in the growth of the experimental groups is due to a less than optimum average daily food intake of adult rats (19-20 gm.). If the rats on 10% glass level had not been satisfied by the bulk of their diet but would have increased their daily intake by 10%, it is probable that there would have been no discernible difference between this group and the *ad libitum* control group.

The lack of irritating qualities to skin or mucous membranes of the flake-like glass dust particles is probably due to two important properties of this material. The first is the insolubility, and hence, biologic inertness, of glass in general. The second is that because of the large, relatively flat surface of these dust flocculi in particular they tend to orient themselves parallel to the surface and to float upon the film of fluid present on mucous membranes. The sharp edges of the particles thereby are given little opportunity to inflict tissue damage.

In view of the insolubility of glass, the absence of toxicity is not surprising, even from such massive dosage as is represented by the animals that ingested an amount of glass equivalent to the weight of their food, every day for one year. The significant difference in the weight curve of the above animals as compared to that of the control group can be explained on the basis that the former could not ingest sufficient food to equal the caloric intake of the controls.

logical investigations. A case in point is the present investigation in which it was found that 4 out of 16 guinea pigs exposed to glass dust suffered from a striking, hitherto undescribed interstitial pulmonary fibrosis with calcification. Since, to our knowledge, no such lesion has previously been reported or seen in animals, it would follow that it was the glass dust that caused the pulmonary fibrosis. We were fortunately saved from falling into this error by the fact that it has been customary in this laboratory to run two additional series of animals as controls whenever an unknown dust is under investigation. These two additional series of animals are exposed to quartz and kaolin dusts, respectively, under conditions as nearly identical as possible to those of the experimental dust. As previously indicated, interstitial pulmonary fibrosis identical to that in the glass-dust group was also found in animals exposed to kaolin as well as to quartz dust. This seems to point not to the glass dust but to some as yet undetermined factor in the technique as a causative factor.

The failure of our quartz dust to produce silicotic nodulations in guinea pigs and the temporary inhibition of silicotic fibrosis in rats had not been anticipated. Investigation disclosed that the quartz dust contained considerable iron, and the latter modified and inhibited the silicotic manifestation.⁸ At the same time, it was found that similar admixture had also occurred with the kaolin and glass dusts. It was this admixed iron which gave the alveolar dust cells in all groups the tan to brown pigmentation.

The question may be raised as to the extent to which admixed iron may have modified the pulmonary response to glass dust. A positive answer cannot be given. However, inasmuch as the inhibitory effect of iron is only of temporary character in the case of quartz dust in the rat, it would seem reasonable to anticipate no more than a temporary inhibitory effect, if any, in the case of glass dust in the rat. The fact that the effects of glass dust in rats which

had been killed at two years were even more trivial than those killed at one year speaks against any inhibitory effect of iron. Furthermore, we have had abundant previous experience with the pulmonary effects of kaolin dust without iron contamination, and we do not see any significant difference in the histopathologic picture produced in the lung by kaolin dust whether contaminated by iron or not. In spite of the anchoring effect of iron, the pulmonary involvement due to quartz was much more extensive and severe than that due to glass.

It has been inferred that glass dust in filamentous form may be biologically more active than glass dust in the form of, say, spherules because of the mechanical irritation of the sharp points.² This is, of course, pure conjecture and is based on a similar conjecture made in regard to the biologic activity of asbestos fibers. Unfortunately, there are at present no data which could serve either to support or to reject this assumption. However, a filamentous dust of glass like material (aluminum silicate) has been shown recently to be biologically inert.⁹ Besides, if the theory of mechanical irritation should apply to silicates like glass and asbestos, it should also apply to the sharp-edged crystals of quartz. Yet, the mechanical theory of the biological activity of silica has been discarded.¹⁰ It is apparent that any difference between filamentous and flake glass of identical composition will tend to disappear if the glass is ground finely enough (as was done during this investigation).

Summary and Conclusions

A study was made of the biologic effects of glass dust upon the conjunctiva, the gastrointestinal tract, and the lungs of experimental animals.

No effect of glass dust and glass flakes was demonstrable upon the conjunctiva.

No toxic or traumatic effect was demonstrable upon the gastrointestinal tract. There was only slight depression of the growth rate when rats ingested a diet con-

taining as much ground glass as food. This depression was due entirely to a lower food intake caused by the bulkiness of the glass diet.

The pulmonary changes in guinea pigs exposed to fine glass dust for one year and those of rats either exposed to the same dust or given injections intratracheally with it were minimal in character and consisted only of small focal alveolar dust cell collections without fibrosis. In the pulmonary lymph nodes there were also focal intracellular glass dust deposits which were likewise not associated with reactive fibrosis. There was no bronchial disease attributable to the dust.

Compared to kaolin, the pulmonary effect of fine glass dust was even more benign, because the stored dust remained alveolar instead of occupying interstitial positions and therefore was more readily mobilized and cleared from the lung.

Coarse glass dust injected into the lungs of rats behaved like inert foreign material and was associated with minimal foreign body reaction without significant collagen formation.

Industrial Hygiene Foundation, Mellon Institute

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Airborne Fibrous Glass Particles

Chest Roentgenograms of Persons With Prolonged Exposure

By JOHN W. WRIGHT, M.D., Cleveland, Ohio

THE question to be explored is whether or not prolonged exposure of workers to an environment containing asbestos fibers, those coated with silicic acid, but devoid of other material, with lungs exposed to existing lung injury associated with recognizable pattern of pneumoconiosis, develops. The answer can be expected to be a good deal of human protection of workers in many of the plants. The products of asbestos are a factor in whether or not the information at this point is particularly concerned with asbestos. The asbestos is the asbestos, and it is of course under the same conditions of exposure, exposure. Available for examination is the chest roentgenogram of all employees, correctly, are the payroll of a large plant at which fibrous glass is produced from glass waste, formed with a binder of silicic acid, formed in a shape, is each final product, and packed for shipment. The plant has been in operation since 1925, and since 1935. Many of the employees are still at work.

In this plant, neither the asbestos nor the asbestos is produced from waste, and is in a continuous process. The fibers are formed either by blowing, or by drawing along thin streams of molten glass, or by spinning the material into off-springs. During or immediately after the formation of the fibers, a binder is applied which contains chiefly a phenol-formaldehyde resin in which an insecticide is sometimes added. Pyrocarbon is added as a lubricant to improve the handling characteristics of the product. The glass wool, with its binder, is then "cured" by heat at temperatures ranging from 140 F. to 340 F., following which it is formed into the desired products, inspected, and packaged. The plant is designated primarily as a glass wool plant, and produces material broadly classified as: 1. residential and commercial building insulation; 2. appliance and equipment insulation; 3. automotive and aircraft and marine products production; 4. pipe insulation; 5. duct and duct insulation; 6. roofing products and insulation; 7. miscellaneous products, including bonded tile and ceiling board; 8. filtration products; 9. decorative and reinforcing mats.

Exposure

There is no free from the foregoing description that mechanical energy is applied to the glass wool, not only in the process of formation but even more so during production of the final product. Activities such as blowing the heat of glass wool, chopping and tearing the edges of the fibrous glass wool, and the acoustic tile chopping and the fiber rope glass wool, are, in the degree of logging fibrous glass wool and packaging and shipping finished products are examples of the kind of mechanical handling which has the potential for raising fine to respirable sized particles of fibrous glass.

A recent study on the effect of retention of the airborne glass wool particles at the finishing, some of the workers involved in the above processes has been carried out and will be reported in detail elsewhere. Dusts and fibers were removed from the air by electrostatic precipitation, filtration through membrane filters, or use of stand and impingers. A high volume sampler equipped with a type S filter was also used in some areas. Without regard to the kinds of material contribu-

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ples, these concentrations varied from 0.93 mg/cu m to 13.3 mg/cu m for an average throughout the plant of 2.24 mg/cu m. With respect to numbers of particles, the lowest average of samples in any area was 0.09 million particles per cubic foot and the highest 0.32 million particles per cubic foot, with an average throughout the plant of 0.22 million particles per cubic foot. Of these total particles counted, fibers made up less than 1%. The median diameter of the fibers was 6 μ , 85% being in the range from 2 μ to 10 μ . Averaging data from samples throughout the plant indicated that 16% of the fibers were under 40 μ and 6% under 20 μ in length. The concentration of both formaldehyde and phenol throughout the plant was less than 0.5 parts per million (ppm).

The atmosphere at the breathing zone during this study cannot be taken as representative of conditions existing in earlier years since extensive exhaust and ventilation facilities are currently in operation. This data is mentioned solely to provide a bench mark indicating that the population to be reported was exposed through the years to higher concentrations than those indicated here.

Population Studied

Since we were interested in effects of long-term exposure, no person was included in the study who had been employed by this company fewer than ten years. Thus, one could assume a sustained cumulative exposure and also the lapse of sufficient time to permit changes to develop which might be recognized in the chest roentgenogram. Employment records and the experience of the plant physician revealed nothing unusual with respect to "labor turnover" in the first few years of employment. There is nothing to suggest that the population employed ten or more years represents a highly selected group in terms of tolerance to any foreign material. Since the manufacturing process begins with raw materials, of which one—free crystalline silica—is known to possess the capacity to produce changes in the thoracic roentgenogram, and since we desired to confine our attention to persons whose exposure was limited essentially to fibrous

glass, none of the personnel involved for more than a year in the manufacturing process at a stage earlier than the actual formation of fibers was included in the study. A few individuals who had worked very briefly in the pre-fiber stage and subsequently moved to the later stages were included. Utilizing these criteria, the chest roentgenograms of every employee, men and women, who had worked in what might be called the factory area for more than ten years, were used in the study.

We had hoped to obtain a control group whose only exposure would have been to such fibrous glass as might have reached the ambient air outside the plant itself. This proved impracticable, since the clerical staff and management personnel had frequent occasion to walk into various parts of the factory, and hence had received some exposure to fibrous glass. Also, some members of the administrative staff had worked for an appreciable period of time in one capacity or another inside the factory operations. Roentgenograms of a population from outside the factory and business office would have introduced bias due to unavoidable film identification of this group by the examiner. It was decided, therefore, not to attempt to obtain controls in the strict sense of zero exposure.

The final study group was therefore composed of all employees currently on the payroll who had had ten or more years of employment in the factory area, and in addition, all male employees on the clerical or management staff with ten years or more of employment. In this way all films examined had identical identification markers and the film examiner had no way of knowing whether films were from factory employees or from the clerical and management staff.

Chest roentgenograms of 1,401 individuals were available for examination. Prior to study of the films, 12 of this original number were dropped from the study. One was too large in stature for technically satisfactory chest roentgenograms. The remaining 11 were excluded because prior to employment by this fibrous glass company, they had had a long occupational exposure to free crystalline silica in the ambient air, most of them having worked for more than ten years in a foundry on occupations

known to be hazardous due to this type of exposure. The final correlations were made utilizing a study group of 1,389 employees.

Grouping the Population

Two procedures were used to arrange employees on a scale of light or minimal through heavy exposure. The first was to group them in terms of whether or not they were hourly wage earners in contrast to being salaried. In general, hourly wage earners worked in the manufacturing part of the plant, while most salaried employees were in the administrative group and worked predominantly in the office building, with a few in supervisory position within the plant. Since some individuals in the study had worked appreciable periods both as salaried personnel and as hourly wage earners, it was necessary to divide the entire group into three categories. Arbitrarily, we designated anyone who had worked 75% or more of his time on salary as in the clerical category. Anyone who worked a like percentage of his time on an hourly wage was placed in the factory category. Those individuals who fit neither of these classifications were considered as "not classified." This separation of categories was accomplished after all chest roentgenograms had been examined and the data derived from them punched on the appropriate card.

To rank the employees in the second way, a committee composed of the plant physician and supervisory personnel, all of whom had had experience within the plant going back almost to the time it began commercial operations, considered the total work history of each person in the study and placed each one into one of four categories based upon the committee's interpretation of the intensity and duration of the total exposure of the particular employee. The committee members utilized not only their broad experience with past conditions in the plant, but also the results of a thorough survey of industrial hygiene completed just before the study was undertaken. Complete work records designating the principal areas and number of years each worker had spent in these areas during his work life in the plant and offices were available and were utilized

whenever called for. While it is difficult to group that judgment enters into such a grouping, there can be little question that the extremes—groups I and IV—have had a markedly different severity of exposure to fibrous glass, whereas those in groups II and III may not be as meaningfully separated as one would like.

Group I includes persons who have been exposed through all or a major portion of their employment and have never had persistent direct exposure to an environment containing a substantial quantity of air borne fibrous glass. A few factory personnel were in this group either because their entire employment had been on the repair of machines in a separate portion of the building, or because their factory work had been around inactive machinery where exposure to fibrous glass was brief and minimal. Group II included employees with at least one year or more, up to five years, exposure to fibrous glass where the level of airborne material would be judged to be considerably more than minimal. Examples of this category were persons whose duties required them to be walking around the factory area throughout each working day; those who may have worked in boxes where cleaning or packing was going on; or those who worked at the loading areas at the termination of manufacturing lines. It also included machinists who worked in all parts of the plant but who frequently did repairs within the factory area and therefore had an appreciable cumulative exposure. Group III included persons comparable in all respects to those in group II but whose exposure was known to have extended over a period of at least five to ten years. It should be recalled that all members of groups II and III had been employed in this plant for a minimum of ten years and the exposures referred to just above were in terms of cumulative exposure, not necessarily continuous in nature. Group IV included all persons who had ten or more years of exposure in such areas as wool packing, fiber reconditioning, pipe insulation, sawing, slitting, and grinding operations, and any other job which by its type or location within the plant was thought to have required an intense exposure to fibrous glass persistently and over a long period of time. All per-

known to be hazardous due to this type of exposure. The final correlations were made utilizing a study group of 1,389 employees—

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To rank the employees in the second way, a committee composed of the plant physician and supervisory personnel, all of whom had had experience within the plant going back almost to the time it began commercial operations, considered the total work history of each person in the study and placed each one into one of four categories based upon the committee's interpretation of the intensity and duration of the total exposure of the particular employee. The committee members utilized not only their broad experience with past conditions in the plant, but also the results of a thorough survey of industrial hygiene completed just before the study was undertaken. Complete work records designating the principal areas and number of years each worker had spent in these areas during his work life in the plant and offices were available and were utilized

in making each designation. While it is obvious that judgment enters into such a grouping, there can be little question that the extremes—groups I and IV—have had a markedly different severity of exposure to fibrous glass, whereas those in groups II and III may not be as meaningfully separated as one would like.

Group I includes persons who have been exposed through all or a major portion of their employment and have never had persistent direct exposure to an environment containing a substantial quantity of airborne fibrous glass. A few factory personnel were in this group either because their entire employment had been on the repair of machines in a separate portion of the building or because their in-factory work had been around inactive machinery where exposure to fibrous glass was brief and minimal. Group II included employees with at least one year or more, up to five years, exposure to fibrous glass where the level of airborne material would be judged to be considerably more than minimal. Examples of this category were persons whose duties required them to be walking around the factory area throughout each working day; those who may have worked in boxes where cleaning or packing was going on; or those who worked at the loading areas at the termination of manufacturing lines. It also included machinists who worked in all parts of the plant but who frequently did repairs within the factory area and therefore had an appreciable cumulative exposure. Group III included persons comparable in all respects to those in group II but whose exposure was known to have extended over a period of at least five to ten years. It should be recalled that all members of groups II and III had been employed in this plant for a minimum of ten years and the exposures referred to just above were in terms of cumulative exposure, not necessarily continuous in nature. Group IV included all persons who had ten or more years of exposure in such areas as wool packing, fiber reconditioning, pipe insulation, sawing, shitting, and grinding operations, and any other job which by its type or location within the plant was thought to have required an intense exposure to fibrous glass persistently and over a long period of time. All per-

sons in this group had had prolonged exposure to fibrous glass in areas where the concentration in the air was considered to have been highest. The arranging of persons into these four groups was done independently, with no possible chance that the committee could know the results of the evaluation and the description of the chest roentgenograms. The results of these groupings were punched on the record cards after all film data had been entered.

Methods

A technically satisfactory single posteroanterior (PA) view of the chest was obtained for each person in this study. Artifacts caused by motion or extremes of over- or underexposure were not acceptable and films were repeated until technically satisfactory. For each person, there was an acceptable film made within six months of the time the entire group was evaluated. For many individuals, previous serial films going back as many as twenty-five years were available for comparison. Films of persons from all categories were deliberately mixed before being sent to the examiner in order to minimize the effect of day to day variations in the examiner's judgment. Since all films bore the same designations and imprints the examiner could not know which category of person any particular film represented. The films were examined by the author in a span of six consecutive weeks.

The soft tissues and bones of the thorax were examined, but described only if a definite abnormality was visible. Evidences of thickening or calcification of the pleura, flattening or obliteration of the costophrenic angle and position of the diaphragm were noted. The cardiac silhouette was noted as to whether its greatest transverse diameter was more or less than 50% of the greatest trans thoracic diameter and also as to whether or not the pulmonary cone was prominent.

Evaluation of the shadows seen with the lung parenchyma posed much greater difficulty since it was desirable that their salient features be described without regard to whether the examiner thought they were normal or abnormal. The most conspicuous shadow complex seen in the normal lung parenchyma is the system of branching linear densities narrowing progressively from the lung root to the periphery, and thought to be caused predominantly by the major trunks of the pulmonary vascular system. For want of a more precise term this pattern

will be referred to as the branching vascular pattern (BVP). Examination of large numbers of well persons of different ages whose lives have been spent outside occupations ordinarily considered to be dirty shows considerable variation in this pattern with respect to prominence or size and density of individual linear shadows, distribution of the shadows, and degree to which they show tiny beads, presumably at branching points, caught in a particular radiological silhouette. Each film in this study was compared to three films used as standards, chosen from a college student population as representative of the variation in size and density of BVP commonly observed in normal healthy persons with no history of exposure in a dirty trade.

Five categories of BVP were thus established. Category I had a BVP thinner and more tenuous than the least of those in the three standard films. Categories II, III, and IV corresponded to one of the three specimen films and category V exhibited a prominence of BVP greater than the category IV standard film.

In the films of normal healthy persons one sees relatively few spherical or irregular-shaped densities, although in almost every roentgenogram of the chest one can see one or more of these isolated nodules. Each film was therefore described in terms of whether excessive nodulations were seen and whether they were diffuse, focal, massive or calcific. In addition if the nodules seemed to be characteristic of an established and recognizable pattern of disease, as for example the healed phase of histoplasmosis, a note to this effect was made. In the chest roentgenogram of normal healthy persons, one also sees sparse short and thin linear densities which do not appear to be components of the BVP. As with the nodular densities, if the number of such small linear densities was thought to be well within the limits expected for normal persons, no mention was made of them. On the other hand, if there was an unusual profusion of linear densities, this was noted and categorized on a comparative scale as to whether they were focal or diffuse. With respect to any other sort of abnormal pattern of densities, a place was made available for description of them on the record. The presence or absence of cavities, whether thin or thick-walled and whether single or multiple, was noted. Among normal individuals there is a great variation in the size, position, and density of the complex of shadows making up what is referred to as the lung root. Each film was categorized in terms of whether or not the lung roots seemed usual or unusual as to size, posi-

tion and density. The presence of long, single linear densities or atypical branching densities either alone or associated with confluent densities such as commonly represent the end stage of lung abscess or tuberculosis was also noted. In those instances where previous films were available, a comparison was made between the earliest and the more recent to indicate whether or not any new densities had appeared, whether any change had occurred in previously noted densities, including the BVP, or whether there had been any change of the prominence of the lung roots or the size and contour of the heart shadow. Finally, the films of each individual were classified as being within the limits of a normal or abnormal shadow pattern.

Results

After completing the observation of this entire set of films, it was apparent to the examiner that there was nothing peculiar or unusual which occurred in the films with sufficient frequency to suggest the presence of a new or heretofore undescribed or an known type of radiologic shadow pattern which could be associated with exposure to fibrous glass. In fact, no unusual shadows were seen that have not been observed in men whose lives have been spent in an environment thought to be free from any potential pulmonary hazard. No films showed any unusual prevalence of short linear densities associated with the normal BVP. In one instance was the pattern associated with asbestosis or berillium granulomatosis seen. In two instances a diffuse nodular pattern was observed; the first was a film showing small, discrete, nodular densities scattered throughout both lungs with very large and dense lung roots. Subsequent examinations and the course of events following the disclosures of this film clearly established a diagnosis of Boeck's sarcoid. In the second case, the current film showed no abnormality but earlier films had shown a very faint nodular density scattered throughout both lungs with moderate enlargement of the lung root, all of which disappeared or returned to normal in subsequent films. The second case also was thought to be Boeck's sarcoid. With these two exceptions, no film showed the pattern associated with silicosis or siderosis.

The frequency with which variations in

the chest roentgenogram pattern were found in each exposure category is shown in Table 1. No example of pleural calcification was observed in this entire series. Rather extensive areas of thickening of the pleura shadow occurred in six instances, three in category I and three in category II, but none occurred in categories III or IV. Combining all instances of pleural density and obliteration of the costophrenic angle, there were 39 thought to have some recognizable alteration of the pleura. Table 1 reveals that the distribution of these is about the same in all categories.

A few examples of large air-containing spaces thought to be bullae were encountered, but with no unusual frequency in any category.

A beaded or nodular appearance of the BVP was seen in 24 persons. Table 1 shows the distribution being greatest in frequency in persons least exposed.

Given an opportunity to examine films of the chest taken a number of years prior to the time that an individual develops the classical picture of silicosis or asbestosis, one can often persuade one's self that a premonitory stage of either of these diseases is represented by an increase in prominence of the branching vascular pattern and, in silicosis, by an increase in size of the shadows making up the lung root. With this in mind, this series was evaluated in terms of the prominence of the BVP and lung roots and also in terms of whether or not any increase was observed in these shadows in persons for whom more than one film was available. Lung root shadows were not categorized by subdivisions, but instead a simple statement was made as to whether or not the shadows impressed the examiner as being unusually large and dense. The BVP designations I through III, as those commonly seen in the chest films of any group of normal individuals whose experience has been devoid of exposure to the inhalation of known potential fibrous materials. Even IV and V are at times seen in a singular group of persons.

In Table 1 the distribution of BVP, IV and V, large lung roots, and any increase in either BVP or lung roots is shown singly and in various combinations in relationship to categories of severity of exposure to fibrous glass. It is apparent that the fre-

glass inhaled by the persons in this study was not coated, the majority was covered by a phenolic resin, cured at 450 F to 550 F. This process converts the resin to a film which is inseparable from the fiber and is insoluble in water, strong and weak acids, weak alkali, salt solutions, and organic solvents. This material is thought to be biologically inert; the findings of this study tend to support this belief.

The petroleum oil lubricant used to condition the fiber is known as "heavy cylinder stock oil." In the process of curing, the more volatile components of this oil are removed, but 2% to 5% of the total mass of the end product is composed of a material similar to petroleum jelly. The biological potential of this material is not known, especially its long-term effects.

Within the duration and exposure limits characterizing this study, there was no recognizable abnormal pattern of shadows in the chest roentgenogram of the persons involved. This suggests strongly that if the materials evoke an inflammatory or fibrogenic response in humans, it is at a level too low to be visible in chest roentgenograms. Some classes of biologically active material require years to develop gross evidence of their fibrogenic influence, but one would expect that twenty-five years would be sufficient to reveal meaningful evidences of inflammation or fibrosis.

With respect to neoplasia, some materials believed to be carcinogenic may have a longer lapse time than that for fibrogenesis. For this reason, as well as those of design of the study, this report cannot be presumed to answer the question of the neoplastic potential in the environment studied. The experi-

ence of a sufficiently large cohort followed over a lifetime is needed for this purpose.

In recent years there has been much concern over the possibility that users of various products are injured thereby. Frequently allegations regarding injury are ascribed to a specific material even though the user may be simultaneously exposed to a number of other materials. Study of population thus exposed cannot incriminate the actual injurious agent. In this study, although the population may have been exposed variously during the hours away from work, one can conclude that the in-plant environment of fibrous glass evoked no response recognizable in the roentgenogram.

Summary

The effect, as revealed in the chest roentgenogram, of ten to twenty-five years of exposure in an environment containing airborne particles of fibrous glass has been studied. No unusual pattern of radiologic densities was observed.

The frequency of various radiologic appearances known to occur in the general population was no higher in those with greatest exposure than in those with the least.

J. S. Bishop, M.D. of the Corning Fiberglass Corporation, and his staff, measured and arranged the roentgenograms and evaluated the work histories for this study.

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Fiber Glass

Chemistry and Technology

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GLASS has been known to man for many centuries. Today, glass has such widespread and commonplace usage that it is accepted as an essential part of our everyday lives. A fact that is little known or appreciated is that glass technology has advanced more in the last 50 years than in the preceding 1000-plus years since its discovery. Scientific research during the 20th century has developed a diversity of new properties and forms of glass.

One of the newer forms is fibrous glass. Its applications have resulted in a rising interest in its properties and in the possibility of biological effects upon those who come in contact with it. The purpose of this article is to dispel some of the mystery that seems to attend the manufacture and handling of fibrous glass.

What Is Glass?

Glass is defined by the American Society for Testing Materials as: "an inorganic product of fusion which has been cooled to a rigid condition without crystallization." It is described as "a supercooled liquid." It is considered neither a true solid nor a true liquid, but it cools to a state somewhere between the two.

Chemically, it is termed amorphous rather than crystalline and it is classed as a silicate. Glass is one of the most stable substances known to man.

Although there are hundreds of glass formulations in use today, the majority of commercial glass compositions are quite similar, varying only in minor quantities of this or that chemical to provide the properties and characteristics suitable to the eventual application of the glass.

Commercial glasses are formed from silicon

dioxide and the oxides of various metals and other minerals. Trace amounts of other chemicals are added as required.

What Is Fiber Glass?

Fiber glass is generally classified as a borosilicate glass because it contains boric oxide. This component provides greater heat resistance, lower thermal expansion, and great resistance to chemical attack.

To those unfamiliar with glass composition and production, the manufacture of fibers of glass might appear to be a comparatively simple task. In reality, there were innumerable problems to be overcome before glass fibers could be produced on a commercial scale.

Fibrous glass was first produced for the commercial market in the early 1930's. Most of the fibers produced at that time were used as a filtering medium and were, when compared to many present-day fibers, coarse and inflexible.

In a general way, glass fibers can be categorized as wool and textile fibers. Textile fibers are usually continuous filaments, often referred to as yarns, and are formed in continuous or mending fibers, whereas the wool fibers are formed in varying lengths.

Another variation is in the fiber diameters. Fiber diameters will range from twelve-one-hundred-thousandths of an inch (.00012 in.) to four-hundred-one-hundred-thousandths of an inch (.00100 in.). By comparison, human hair diameters range from sixty-six-one-hundred-thousandths of an inch (.00066 in.) to more than seven-hundred-one-hundred-thousandths of an inch (.00115 in.).

The diameter of glass fibers is varied to provide the characteristics needed in the eventual application or product to be made. When fibers

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are manufactured for a given application in product, the diameters are controlled within close limits and the range of variation is only a few hundred thousandths of an inch.

Regardless of the ultimate use to be made of the glass fibers, they are almost invariably coated with a number of materials known as binders, lubricants, sizes, and coupling agents. These coatings serve several purposes: (1) they bind the individual fibers together so that the final form is established and maintained; (2) they protect the surface of each fiber; and (3) they may serve as a bonding agent if the fibers

are to be incorporated in concrete or other materials.

Chemicals Used

Raw Materials—Basic Materials

By no means do all of the chemically listed bodies enter any one line of business. The following chemical types of fiber glass:

- E-glass
- A-glass
- C-glass (chemical resistant)
- D-glass
- F-glass

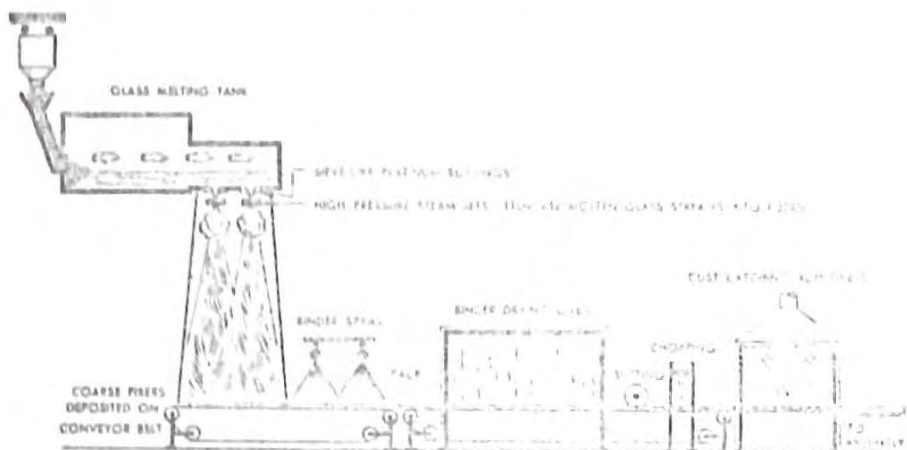


Fig. 1. Schematic representation of air filter process.

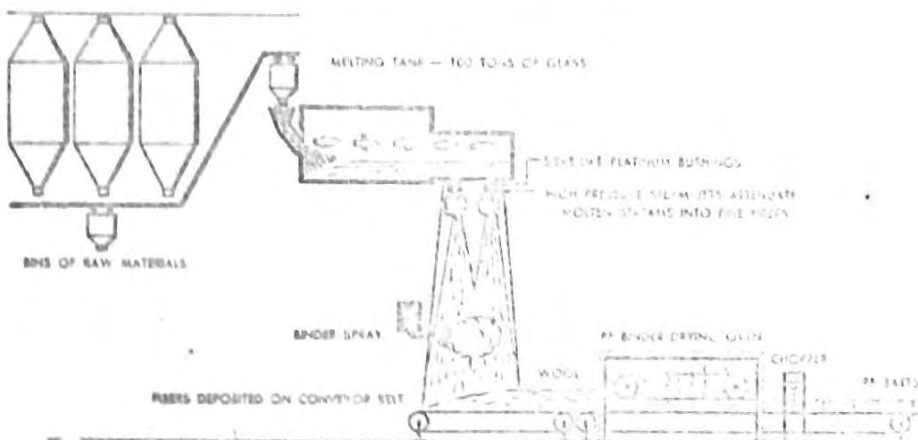


Fig. 2. Schematic representation of wool forming line.

fluorspar
sodium sulfate
boric acid
limestone
rutile (titanium dioxide)
zircon
charcoal
barium compounds
sodium nitrate
cullet

Chemicals Used in Binders

phenol formaldehyde resin
urea formaldehyde resin
melamine formaldehyde
resin
polyvinyl acetate
vinyl resin
urea
silicones
various dyes
ammonium sulfate
starch
carbon dispersion (black
pigment)
epoxy resins
ammonium hydroxide
mineral oil

Manufacturing Process

In the manufacture of glass fibers there are two basic processes: one is for the manufacture of the "wool" products and the other produces the textile yarns. Only the wool production process will be discussed in this article. The wool fibers are used for thermal insulations, filters, sound control, air handling systems, etc.

The Glass Batch The raw materials used

in glass manufacture are received in the batch house where most of the operations are automated. Each constituent of the batch is weighed and mixed into a homogeneous batch. The batch is then conveyed to furnaces where

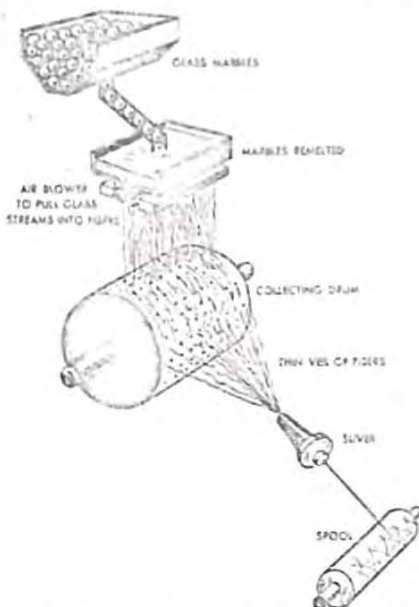


Fig. 4. Schematic representation of staple fiber yarns process.

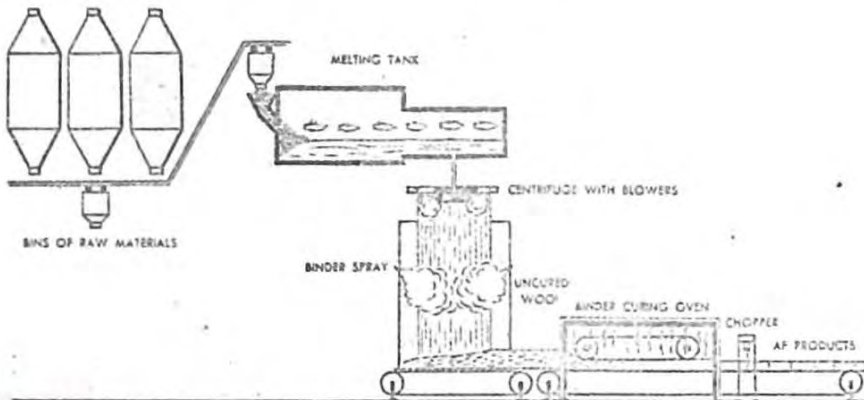


Fig. 3. Schematic representation of AF forming line.

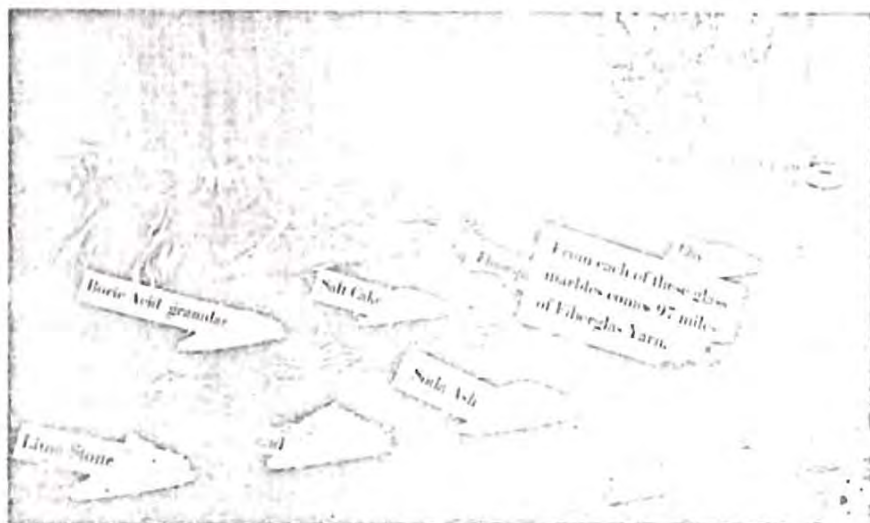


Fig. 5. The chemical constituents of fiber glass textile yarns.

it is automatically fed into the furnace as required.

The Furnace Glass furnaces are composed of two main areas: the crown or upper portion—where the heat is generated, and the melter or lower section—where the batch is melted. Temperatures range from 2400°F. to 2900°F., depending upon the type of operation required.

The molten glass flows from the furnace through a flow chamber to the forehearth.

The Forehearth and Forming Operation The purpose of the forehearth is to provide molten glass for the fiber-forming operation.

There are several methods by which fibers are formed. A general description will provide an understanding of the process.

The molten glass flows into the fiberizer which is made of a heat and corrosion resistant metallic alloy. The fiberizer contains hundreds of outlet orifices through which the molten glass travels in tiny streams of uniform diameter. A series of fiberizing units are required in each forehearth to produce the quantity of glass fibers needed.

Upon leaving the fiberizer, each glass stream is met and transported by steam or air jets which accelerate the rate of flow and further reduce (attenuate) the stream into fibers of desired diameters by the tractive or pulling force

exerted on each glass stream. The speed of glass flow and the fiber diameter will be affected and can be controlled by the temperature of the glass and the steam or air pressure used.

Binder and Lubricant Application As the attenuated fibers move away from the fiberizers, the binders and lubricants are applied by a spraying process.

Wool Fiber Product Forming The glass wool fibers coated with uncured binders and lubricants proceed to a conveyor where they collect in the desired mass and depth as required for the ultimate product being formed. The density of the fiber mass or blanket on the conveyor is controlled by the fiber production rate and the speed of the conveyor.

A straight-line conveyor is used to produce the mat, batt, and pack products because all fabricating operations can be accomplished from the beginning to the end of the conveyor line.

As the glass blanket is conveyed away from the collecting area, it enters a curing oven where the thermosetting binder is cured at temperatures ranging from 400°F. to 500°F. Certain products must be restricted to an established thickness and density and this is accomplished in the curing oven by pressure rolls and plates.

Following the curing operation, the blanket



Fig. 6. One process for fiber glass formation.



Fig. 7. Fiber glass appliance insulation before and after compression packaging.

edges are trimmed and vapor barriers of paper or foil are applied. Some products are slit and cut to specified dimensions while others continue to the end of the conveyor in blanket form where they are rolled into packages.

Typical Products

Fiber glass is now 30 years old. During that time it has been accepted as a part of daily life in thousands of its applications.

Among its valuable properties are: its inertness and chemical stability; its dimensional stability; its resistance to flame, moisture, and other corrosive agents; its exceptional strength-to-weight ratio; its resilience and flexibility; its dielectric or nonconductive properties; and its thermal and acoustical insulating qualities. It is impractical for this article to include any large portion of common applications. The list that follows is merely representative.



Fig. 8. Fiber glass pipe insulation finishing.



Fig. 9. Fiber glass roofing coming off forming line.

Building materials for home, commercial and/or industrial structures including products used inside these buildings

- Thermal and acoustical building insulation
- Acoustical ceilings and ceiling systems
- Furnace filters
- Duct systems and duct insulation
- Decorative wall panels
- Pipe insulation
- Industrial equipment insulation
- Roofing systems and roof insulation
- Cold storage insulation
- Marine insulation

Components of manufactured products

- Thermal and acoustical automotive insulation
- Acoustical automotive headliners
- Automotive safety padding
- Aircraft insulation
- Thermal and acoustical appliance and equipment insulation
- Air filtration media for building equipment, clean rooms and white rooms, and baby incubators and similar products

Liquid filter cartridges for purifying or clarifying fuel, paint, and other products
Vibration isolators for industrial equipment

Textile products

In addition to the foregoing uses of "wool-type" fibrous glass, the textile yarns are used in the following products:

- (1) Decorative and home furnishings:
 - Curtains and draperies
 - Shower curtains
 - Bedspreeds
 - Window shades
 - Tablecloths
 - Mattress ticking
- (2) As reinforcements for plastics in:
 - Vehicle bodies and components
 - Appliance components
 - Sports and recreation equipment
 - Boats and marinas
 - Industrial equipment components exposed to corrosive atmospheres (e.g., pipe, ducts, tanks)
 - Furniture and stadium seating
 - Concrete pouring forms
 - Gasoline and chemical storage tanks
 - Space craft and missile components
 - Military weaponry and body armor
 - Submarine fairwater
 - Deep submersible exploration craft
 - Bathroom tubs, showers, and other components
 - Building panels
 - Polarizing light panels

Biologic Effects of Exposure

Fiber glass, like all common forms of glass, is a chemically inert substance. It possesses no known toxic properties. On a mechanical basis, spicules may abrade the skin leading to superficial and transitory irritation. Among fiber glass

makers, particularly new workers who are most exposed, skin irritation occurs, but is not remarkable. Although free silica is a major primary ingredient of the batch before melting, no free silica occurs in the glass fibers or in the ultimate products. Silicosis is not a fiber glass hazard. The present consensus is that, because of the particle size, fiber glass is not respirable, and thus is unlikely to reach the alveoli of the lungs, but it may reach the tissues of the upper respiratory tract. In animal experiments the introduction of fiber glass particles into the respiratory tract has failed to provoke pneumoconiosis. However, some minor tissue changes have been observed, chiefly reflecting a foreign-body reaction. Fiber glass is not a known carcinogen. Animal experimental work with fiber glass particles introduced into the lungs has failed to provoke lung cancer. No clinical cases of lung cancer have been traced to fiber glass as the source.

A fair number of chemicals in ancillary use in fiber glass production may possess some harmful properties. As example, the number includes partially polymerized resins of phenol formaldehyde, urea formaldehyde and epoxies, and fluorides and trichloroethylene. Such substances involve the fiber glass makers, whose exposures are not contemplated in this presentation, but do not carry over to secondary fabricators, to installers, or to consumers.

All considered, fiber glass, on a relative basis, is one of the least harmful substances known to industry.

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Pulmonary Hazards from Exposure to Glass Fibers

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THE TECHNOLOGY, chemical composition, and other characteristics of fibrous glass have been reviewed by McCord elsewhere in this issue.¹ This present review is designed as a critical evaluation of the pertinent investigations concerning pulmonary hazards from exposure to glass fibers. Neither the raw materials that are used in the formulation of fibrous glass nor the ancillary ones that may be associated with it in various applications will be appraised. Other materials that bear some resemblance in name or application to fibrous glass have been sometimes inadequately differentiated from it. These include mineral wool, Fiberglas Reinforced Plastic and Plexiglass. Thus, while Fiberglass Reinforced Plastic is a structural material composed of fiberglass and a plastic in approximately equal proportions, Plexiglass is not a glass at all, it is a brand name of a certain plastic. The confusion has extended to misnaming a clinical entity claimed to be due to inhalation of glass fibers. This will be discussed in more detail in reviewing the effects on human health.

Experimental Studies on Animals

Gross *et al.*² investigated the biologic effects of glass dust on the conjunctiva, the gastrointestinal tract, and the lungs of experimental animals. The pulmonary studies consisted of one in which dusts were deposited by intratracheal injection in the rat, and one with rats and guinea pigs in which pulmonary deposition was accomplished by inhalation. Because of the method used in its preparation, the glass dust employed contained an appreciable amount of iron. Also, the animals were exposed to a considerably higher concentration of a dust much more finely divided than that to be expected under conditions of industrial exposure.

This higher concentration is presumably optimal for the development of lesions. Inhalation exposures lasted 6 hr. per day, 5 days per week, for 1 year. The pulmonary reaction to the inhalation of fine glass dust was no more severe than the reaction to equally fine kaolin dust, and frequently less so. Histological examination of the lungs revealed a nonfibrogenic reaction which consisted essentially of small collections of alveolar dust cells, with no evidence of interstitial deposition or bronchial or bronchiolar disease. Gross *et al.*, suggested that finely divided glass dust is more benign than is kaolin. This was attributed to the fact that retained glass dust remained alveolar instead of migrating to the interstitial tissue; therefore, it was more readily mobilized and cleared. Coarse glass dust administered into the lungs of rats by intratracheal injection acted as inert foreign material and was associated with minimal foreign-body reaction, with no significant collagen formation. However, the high iron content of the glass dust used in the experiments and the shape of the particles as flakes or plates rather than fibers are two factors that might have influenced the pulmonary response observed. The actual role played by these two factors can only be a matter of speculation.

Schepers and Delahant,³ and Schepers⁴ studied the effect of glass fibers on the lungs of rats and guinea pigs. Glass fibers of three different diameters, ranging from 1 to 8 μ suspended in normal saline, were used for intratracheal injection in the guinea pig. Lung pathology was studied at different time intervals, varying from 4 to 78 weeks. It was found that glass fibers of the largest diameter were relatively inert. No animals died, and the pulmonary lesions were minimal. However, in the animals that were intratracheally injected with glass fibers 3 μ or less in diameter, focal atelectasis was the dominant lesion. Histological examination revealed bronchiolar damage and accumulation of glass

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fibers in the hilar lymph nodes, without evidence of well-defined fibrosis. Using an inhalation chamber, the same investigators^{2,4} also exposed rats and guinea pigs to glass fibers, approximately 6 μ in diameter, at a concentration of 1.4-2.2 million particles per cubic foot of air. The maximal period of exposure was 40 months. It was found that inhalation of glass fibers did not evoke an early acute reaction. However, it produced interstitial and peribronchiolar fibrocellular infiltrates, together with endobronchiolar lesions with a tendency to epithelial hyperplasia. In both the intratracheal injection and inhalation experiments, no control animals were studied and no reference was made to spontaneous incidence in the animal species used of the observed pulmonary pathological changes. The importance of this must be emphasized, especially since the period of exposure occupied a considerable portion of the normal life-span of the animals. Thus, it would be mere conjecture to assess the significance of the findings in these experiments.

Schepers *et al.*⁵ and Schepers^{6,7,8} also investigated the effects of Fiberglas Reinforced Plastic in experimental animals. These reports are cited to make a clear distinction between fibrous glass and Fiberglas Reinforced Plastic. (The full name of the latter material is "Fiberglas-reinforced calcium carbonate filled polymerized polyester resin.") The filler may sometimes be calcium sulfate instead of calcium carbonate, and glass fibers constituted 10 to 30% in the different blends used in those experiments. In addition, it contained minute quantities of catalysts and retardants, the identity of which was not stated. Thus, it is unjustifiable to use the observations made in those investigations to illustrate the biological effects of exposure to fibrous-glass dust. In fact, the authors emphasized that they were concerned with the effects of the pulverized conglomerate of Fiberglas, plastic, and filler, rather than with the effects of individual constituents. Nevertheless, Fiberglas Reinforced Plastic has not been adequately differentiated from fibrous glass in other reports dealing with the effects of the latter material.

The observations of Schepers⁸ on the tissue-damaging effects of flake glass and two varieties of Fiberglas Reinforced Plastics, are but one example to illustrate the fact that similarities in the composition of some materials do not necessarily lead to similarities in their biological effects. While all the three substances were

found to be comparatively inert, Fiberglas Reinforced Plastic with a calcium carbonate filler produced a slightly more local reaction than did the Fiberglas Reinforced Plastic with a calcium sulfate filler. Flake glass had the least effect, and none of these materials resulted in neoplasm formation.

The effect of inhalation of glass fibers in experimental animals also has been investigated at the Saranac Laboratory.^{9,10} Inhalation of glass fibers continued to show negative results over a period of 19 months of daily exposure, and none of the fibers floating in the air could be found in the lungs of exposed animals. Experiments with glass fibers, 3 μ or less in diameter, also failed to reveal evidence of pulmonary retention. It was concluded that glass fibers used for insulating purposes could not be inhaled into the lungs, since they are at least three times as thick as the fibers used in those experiments. It has been suggested that the tendency of glass fibers, unlike asbestos, to form felt-like masses upon any object with which they come in contact, probably prevents their inhalation. However, a slight degree of chronic inflammation, similar to that observed with "harmless" dusts, was observed. This was attributed to accumulation of nonfibrous particulate debris that resulted from the violent heating used to break up the fibrous glass material.

Effects on the Human Respiratory System

Bjore *et al.*¹¹ studied cardiac and pulmonary functions in 6 workers who had been exposed to glasswool and rockwool, and 8 workers who had been exposed to asbestos. The period of exposure in both groups ranged from 7 to 30 years. The study included clinical, radiologic, electrocardiographic, hemodynamic, and spirometric investigations. While the findings in the group exposed to asbestos appreciably deviated from normal, and were in agreement with the finding of other investigators, no detectable functional impairment¹² was observed in the workers exposed to glass wool.

Murphy¹² reported a case of an electrical worker who developed hemorrhagic bronchitis after exposure to dust from fragmented fiber glass insulation on electrical water heaters. This man's disease was called "fiber glass pneumoconiosis." The use of such nomenclature may have not been justifiable. The 50-year-old worker developed cough, weight loss, and hemoptysis following exposure to clouds of fine grey dust which irritated his eyes and skin. Under

Fiberglas Health Hazard Investigation

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St. Louis, Missouri

THE development and use of a new basic material frequently gives rise to a question as to the possible health hazards for workers handling the material. Glass fiber products, now widely used for thermal and electrical insulation, are still of such comparatively recent origin that the question of a possible health hazard is not unnaturally associated with them.

This association, while it is to be expected because of the newness of the products, is due in part to a special circumstance. The word glass is surrounded by connotations which suggest a hard, shatterable, transparent material. Although Fiberglas is glass in a new physical form, and with physical properties which make it unrecognizable as glass by persons who are familiar only with glass in its more conventional forms, the old connotations may cling to the new products.

Purpose of the Investigation

THIS article summarizes an investigation which was carried out by the author during the month of June, 1941, at the request of WILLIAM H. BARTLEY, of the law firm of Bartley and Mayfield, St. Louis. Mr. BARTLEY is attorney for the International Association of Heat and Frost Insulators and Asbestos Workers, Local Union No. 1, of St. Louis, Missouri.

The purpose of the investigation was to determine the existence or non-existence of health hazards in connection with Fiberglas thermal insulating materials which are used or may be used by members of the union. The investigation was conducted by the author as an unbiased, independent consultant, in no way, other than in carrying out the assignment, associated either with the union or with Owens-Corning Fiberglas Corporation, manufacturers of Fiberglas.

Throughout the course of the investigation the author was given complete cooperation by Owens-Corning Fiberglas Corporation, and untrammelled access to all its pertinent records. The author, however, accepted no statements made by the corporation's officials as valid unless supported by his objective examination of the records, and by objective evidence adduced from disinterested sources and his own research.

The investigation covered the following specific phases:

1. Pulmonary disorders, diseases of the upper or lower respiratory system, and gastro-intestinal diseases,
2. Eye, ear, nose and throat infections,
3. Dermatitis.

Fiberglas a Form of Mineral Wool

FIBERGLAS is true glass, but, chemically, it is substantially identical with mineral wool. It can properly be classified as a new, refined form of mineral wool.

[NOTE: DR. SIEBERT, formerly assistant professor of pathology, Washington University School of Medicine, is Pathologist and Director of Laboratories of DePaul Hospital, Lutheran Hospital, St. Louis; St. Joseph's Hospital, Alton, Illinois, and St. Mary's Hospital, Cairo, Illinois. He is a member of the American Board of Pathology, American Association of Pathologists and Bacteriologists, American Association of Experimental Pathology, and the Society of Experimental Biology and Medicine.]

eral wool. The fact that mineral wool has been used for many years without causing any known industrial diseases, while by no means conclusive, in itself suggests that there is nothing in the inherent physical characteristics of Fiberglas that is likely to introduce any serious occupational hazard.

The chemical and physical characteristics of binders used in manufacturing and processing Fiberglas, like the chemical and physical characteristics of the glass fibers, are not of a kind from which serious industrial health problems would theoretically arise. This conclusion is based upon examination of formulas used in the manufacture and processing of Fiberglas products.

1. No free silica is present in the material known as Fiberglas.
2. No carcinogenic hydrocarbons or other hydrocarbons could be present in Fiberglas because of the extremely high temperatures employed in fusing the batch during the manufacturing process.
3. No harmful fumes could be given off, since no substances are present in the Fiberglas which would volatilize within the recommended service ranges.
4. Absent from Fiberglas is any free sodium silicate.

5. Only three binder substances are present in glass wool products used for industrial insulation. These are bakelite, a refined mineral oil, and bentonite.

6. Bentonite is a refractory clay that is employed as an ingredient of candy with the cognizance of the United States Food and Drug Administration. There is no evidence from factory experience that either bakelite or the mineral oil used for binding substances is the cause of a health hazard.

Medical Literature Searched

AN important phase of the investigation was an exhaustive search of world medical literature for references to possible health hazards in the handling of Fiberglas. This search failed to reveal references to any new or unusual occupational hazards in the handling of these products.

In view of the continually expanding market for Fiberglas products since 1932, and in view of the promotion and advertising campaigns directed toward increasing their sale, the scarcity of references to the subject in the literature of science and medicine, and the lack of controversy or interest in the subject on the part of persons usually alert to possible industrial hazards, cannot be attributed to a lack of familiarity with the existence and use of the products. On the contrary, it warrants the conclusion that the handling of the products is attended by no unusual occupational hazard.

Fiberglas Free of Dusting Characteristics

ON the basis of his own careful observation, and dust counts made in Owens-Corning's Newark, Ohio, plant, the author finds that Fiberglas is remarkably free of the dust which characterizes certain products. Because of the length of the fibers and the "pack strength" of Fiberglas, there is negligible dusting of the material during handling. The glass fibers break, rather than shatter, along clean edges at points where they are abraded by sawing, chopping or cutting during production line or fabricating operations.

The following extract is from "Researches of Mellon Institute 1940-41":

"L. U. GARDNER, Director, Saranac Laboratory, has reported on the researches which the Foundation supports at his institution, saying in part: 'We have been studying the effect of inhaled glass wool. Apparently even the finest textile glass set up as an atmospheric dust is not inhaled in appreciable concentrations and does not produce any dangerous effects upon the lung.'"

A further report on Dr. GARDNER's work appears in the Annual Report of the Saranac Laboratory for the Study of Tuberculosis of the Edward L. Trudeau Foundation for the Year 1940. The following statement is made on page 11 of the report:

"The apparent lack of pulmonary damage from exposure to glass wool dust is being demonstrated by an inhalation experiment. For over a year animals have now been exposed to as high a concentration of this material as could be maintained, with no serious effect upon their lungs. The dust has been produced by breaking into short lengths the finest textile glass that is made but none of the fibers that are floating in the air can be found in the lungs of the exposed animals. Their tendency, unlike that of asbestos, to form felt-like masses upon any object with which they come in contact, probably prevents their inhalation. Slight amounts of chronic inflammation similar to those observed with other harmless dusts, have resulted from accumulated particulate debris. These very fine, non-fibrous particles are the by-product of the violent beating used to break up the material."

X-ray Plates of Employees

THE author examined several hundred stereoscopic x-ray plates of the chests of employees of Owens-Corning Fiberglas Corporation. These were selected at random by the author from the files of some 1500 plates taken since the company, in 1939, instituted the policy of making x-ray plates of the chests of all Newark employees. Among the plates selected for special inspection were those of employees working in the batch house where the raw materials for Fiberglas are handled, and the plates of employees with records of silicosis.

Beginning in May, 1939, serial stereoscopic chest films were made of the 15 men who are now working, or who have worked, in the batch house. A repeat examination of all of the men was made in June, 1941. In no case was there any radiographic evidence of pulmonary disease.

Three cases of silicosis were discovered by the company. None of these men had ever been employed in the batch house, but their employment does require the handling of Fiberglas products. It is the policy of the company neither to refuse employment to, nor discharge, any man for whom safe employment can be found. The following covers the occupational histories of these three workers:

1. CASE NO. 1 worked for another company as a molder from 1924 to 1929, and roentgenograms of the chest were made in 1929 because of the presence of tuberculosis in his family. Chest plates were again made in 1936 because of suspicion of silicosis. He was first employed by Owens-Corning Fiberglas Corporation on June 6, 1940.

2. CASE NO. 2 was employed as a bottle blower and machine operator from 1901 to 1930. He was first employed by Owens-Corning Fiberglas Corporation on March 26, 1941. Silicosis was diagnosed following an

x-ray examination of the chest made on May 12, 1941.

3. CASE NO. 3 worked for 12 years as a molder before entering the employ of Owens-Corning Fiberglas Corporation on October 25, 1939. Silicosis was diagnosed following an x-ray examination of the chest on November 1, 1940.

In each of these silicosis cases there is a sufficient causative factor in the man's previous employment history or other circumstances, and the evidence is entirely inadequate to indicate that the condition of any one of these three men should be attributed to any silicosis hazard encountered in handling Fiberglas, even assuming such a hazard to exist.

Physicians Give Fiberglas Clean Bill

EXAMINATION of the records of disability and absenteeism in the office of the plant physician of the Owens-Corning Newark plant reveals that there is no unusual incidence of respiratory infections among Owens-Corning employees. Statements furnished directly to the author by Newark physicians whose practices are limited to eye, ear, nose and throat diseases, and who are in no way affiliated or associated with the company, indicate that these specialists notice no unusual incidence of respiratory disturbances among their private patients employed by the company, as compared with their private patients who are not employees.

The following letter, dated June 17, 1941, written to the author by JOHN E. HENDRICKS, M.D., of Newark, is typical of letters received from Newark eye, ear, nose and throat specialists:

"I am one of five E.N.T. [ear, nose and throat] specialists located in this city. I have practiced my specialty in Newark for three years following a four-year residency in an Eastern hospital. During these three years I have treated my share of Owens-Corning employees. I can safely say that I have noticed no unusual preponderance of head or throat infections among these people as compared to my other patients who are not employed there.

"If I ever make a table showing percentages of different diseases that I have treated, I feel sure that there would be only a normal proportion of Owens-Corning employees in each classification."

On the basis of the evidence, the author finds that workers handling Fiberglas products are subjected to no special occupational hazards that manifest themselves in eye, ear, nose or throat disorders, pulmonary disorders, diseases of the upper or lower respiratory system, or diseases of the gastro-intestinal tract.

Temporary Skin Irritation

NEW employees of Owens-Corning Fiberglas Corporation engaged in handling Fiberglas products occasionally suffer a mechanical irritation of the skin for a few days at the start of the job. Except for extremely rare cases of workers who may be allergic to an ingredient of binders or lubricants used in processing glass fibers, the irritation disappears within a few days. The new workers apparently become immunized against the action of the fibers, and they suffer no further discomfort.

The company recommends that workers wear clothing loose at the neck and wrists, and that they use plenty of warm water and soap when the day's work is done. Soap and water has proved most effective in removing particles of fibers, whether of glass or any other material, from the skin. The company has found that there is seldom, if ever, need of gloves, armlets or other protective clothing. In the absence of gloves or similar protective clothing there is less tendency for fiber particles to be rubbed into the skin.

Two Hundred Employees Examined

ON June 3, 1941, the author made an examination of the wrists, arms, ankles, necks, eyes, shoulders and waists of approximately 200 production employees engaged in handling Fiberglas products in Owens-Corning's Newark plant. These workers were employed on production lines and at fabricating operations, cutting, sawing, shaping, transporting and packaging glass thermal insulating wool, both bonded and unbonded.

The author did not see a single case of eye irritation or conjunctivitis during this inspection. Only one worker who had begun to work in the plant four days before on a fabricating job showed evidence of significant skin irritation. One 22-year-old employee who told the author he had worked in the plant for two and a half years said that, although he had become accustomed to handling Fiberglas and suffered no discomfort while continually on the job, he had noticed a mild skin irritation that lasted a few days after returning to the job following his two-week vacation.

"As long as I am working right along," he told the author, "I stay toughened up to it. But if I haven't been handling it for a while, like when I'm on my vacation, then it takes a few days to get over the itch when I start in again."

Many of the workers who were examined said the temporary irritation was similar to blisters on the hands that are the result of unaccustomed manual work while the hands are getting toughened to a job. "It don't bother me none," was the reply repeatedly heard by the author in response to his question as to whether the handling of Fiberglas products caused irritation.

Biopsy Specimens Taken

AT THE request of the author, DR. H. A. CAMPBELL, the Newark plant physician, removed two biopsy specimens of skin from the arm of the newly-employed worker who showed a moderate degree of skin irritation. Serial microscopic sections of this tissue showed a mild degree of capillary dilatation in the corium of the skin. In addition, there were a few scattered lymphocytes around several of the dilated capillaries. The epidermis was entirely normal. No glass particles were found. There was no evidence of fibrosis, bacterial infection, allergy or hyperkeratosis.

Several of the serial sections revealed a small amount of a brownish pigment in the corium which showed no inflammatory reaction around it. It is assumed that this pigment is bakelite, one of the substances used as a binder for the glass fibers. The fact that there was no foreign body reaction around this indicates that this material is an inert substance, and that it does not produce either a foreign body reaction or a fibrosis. The extremely mild inflammatory reaction in the corium of the skin would result from any slight mechanical irritation.

Report of Louisville Dermatologist

DR. ADOLPH B. LOVEMAN, Louisville dermatologist, reporting on an examination of 10 workers who handled Fiberglas, writes, "I did not find any evidence of a definite contact dermatitis in any of the employees although some complained of a mild itching when handling the product. The dermatitis produced was unquestionably of a very mild and transient nature."

"I did examine one patient who complained a great deal of a skin eruption and who felt certain it was due to the Fiberglas. He presented, however, a toxic eruption,

and upon questioning him I learned it had been present at times prior to working with Fiberglas. Nevertheless, we removed him from this work for a period of two or three weeks, after which I again examined him and he presented the same type of eruption. In this particular case, it is my opinion that his teeth were probably the foci of infection."

The author believes that the following generalization is warranted by the evidence:

Contact with glass wool may cause ephemeral discomfort among workers inexperienced in handling the material. The skin irritation, however, cannot be regarded as a progressive dermatitis, for it is non-toxic and usually disappears in a few days. In extremely rare instances, where a worker may be predisposed to secondary effects of mechanical skin irritation because of idiosyncrasy, allergy, or the presence of foci of infection elsewhere in his body and otherwise dissociated from the effects of mechanical irritation, lesions may persist. But these cases are encountered with such rarity that they do not justify incriminating glass fiber as a causative factor.

No Claims for Workers' Compensation Validated

FIBERGLAS products have been produced in the Newark, Ohio, plant of Owens-Corning Fiberglas Corporation over a period of nine years. The number of employees has ranged from approximately 20 when production was started in 1933 to more than 1800 in 1941. During this nine-year period no claims for workers' compensation, on the basis of an industrial disease arising out of employment in the manufacture and handling of Fiberglas, have been validated in the State of Ohio.

A letter dated June 17, 1941, from Mr. J. A. SHAW, Claims Referee of the Industrial Commission of Ohio, Legal Section, addressed to the author reads, in part, as follows:

"For your information, we recognize diseases of the respiratory tract as compensable occupational diseases, such as silicosis, asbestosis, anthracosis, and siderosis. As a matter of fact, there is a provision under the law to recognize any disease of this type which is caused by the inhalation of any injurious dusts."

"We endeavor to keep a complete statistical record in the Department of Safety and Hygiene of all cases of this type. I have made a thorough check with this department, and to date we have never had a claim filed for a disease of the upper respiratory tract caused by the alleged inhalation of Fiberglas particles."

Health Better Than Average

THE health of Fiberglas workers is regarded as better than average by the Aetna Life Insurance Company, which underwrites group insurance (life, disability, sickness and hospitalization) for employees of Owens-Corning Fiberglas Corporation. In a letter to an executive of the corporation dated June 11, 1941, JOHN A. HILL, Toledo General Agent of the Aetna Life Insurance Company, wrote, in part, as follows:

"The health of your employee has been better than average. This is evidenced by the fact that we now have in effect a 35% reduction in rate (from standard) on both the sickness and hospitalization insurance carried by your employees. This is the result of nearly four full years' exposure, and develops a rate lower than any similar industry that we have insured."

"There is in effect a permanent and total disability feature in connection with your life insurance and as yet there has been only one claim from an employee, and this was due to a non-occupational

accident, far removed from the course of his occupation or from any health hazard in his occupation."

The author regards as particularly significant the following paragraph from Mr. Hill's letter:

"... our Casualty Company carries what is known as products liability at an extremely low rate, and no claim has ever been presented, indicat-

ing, of course, that there is no hazard in the use of Fiberglas or any of its products."

In submitting his best judgment, based on his professional interpretation of all facts assembled in the course of the investigation, the author finds that there is no silicosis, fibrosis or dust hazard connected with working with Fiberglas, and that the manufacture, fabrication and application of Fiberglas products is attended by no unusual occupational hazards.

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The Pulmonary Reaction to High Concentrations of Fibrous Glass Dust

A Preliminary Report

*Paul Gross, MD; Marianne Kaschak; Ethel B. Tolker;
Mary Ann Babyak; and Robert T. P. de Treville, MD, ScD, Pittsburgh*

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The pulmonary response, in both rats and hamsters, to the presence of fibrous glass dust, is characterized by relatively small accumulations of macrophages without significant stromal change. Whether the fibrous glass dust used was coated (either with a phenol-formaldehyde-type resin or with a textile-type binder) or not, there was no demonstrable difference in reaction. This biologic "inertness" of fibrous glass dust persisted in spite of an extremely high total exposure—24 months to dust concentrations which averaged somewhat in excess of 100 mg/cu m.

WITH INCREASED use and production of fibrous materials of various kinds, there is likely to be an increase in the amount of dust derived from incidental comminution of their fibers. This is particularly true for fibrous glass products. A fiber is defined as a particle whose length is at least three times its diameter.

The hazard posed by the inhalation of fibrous glass dust was first investigated by Leroy U. Gardner¹ who concluded that the dust of glass wool was not injurious to lungs because it was not respirable. The problem of how hazardous the inhalation of the dust of one particular fibrous glass product might be was investigated by Schepers et al² who studied a dust composed of fibrous glass and

a plastic. Unfortunately, these investigators failed to ascertain the relative amounts of glass and plastic in the dust to which the animals had been exposed. Nor did they determine how much of the glass in the dust was fibrous and how much nonfibrous; and how much of the fibrous dust was respirable, ie, what percentage of the glass fibers in the dust was less than 3μ in diameter. The lack of this vital information vitiates the conclusions drawn as applied to fibrous glass per se. In another publication, dealing with fibrous glass derived from glass wool, Schepers³ concluded that glass wool dust caused endobronchiolar and peribronchiolar lesions, a "tendency toward bronchial epithelial hyperplasia" and interstitial pneumonitis. The validity of these conclusions may be questioned in view of the fact that such changes as are described and attributed to glass dust occur commonly in stock rats as endemic disease; but no mention is made in the report that the lungs of control rats had been studied.

The pulmonary response to the inhalation of a nonfibrous glass dust was investigated in this laboratory more than ten years ago.⁴ This dust was prepared from gossamer-thin flakes of glass ball-milled and then further comminuted to an average particle size of 1.1μ by optical microscope sizing and 0.06μ by electron microscope sizing. Inhalation (rats and guinea pigs) of this dust (17 and 18 mg/cu m for one year) and its intratracheal injection (25 mg, rats only) resulted in a pulmonary response composed solely of focal macrophage reaction. There was no

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Reprint requests to 5231 Centre Ave, Pittsburgh 15232 (Dr. de Treville).

Summary of Glass Dust Administration in Animals

Type of Dust	No. of Hamsters	No. of Rats	Intratracheal Injection Dose, mg	Concentration, mg/cu m	Inhalation Chamber	
					No. of Rats Killed for Sampling	
					6 mo	12 mo
Uncoated	30	...	...	135	5	5
	...	30	...	135	5	5
	12	...	3 x 3.5	...	...	...
	...	15	3 x 3.5	...	...	...
Coated with phenol-formaldehyde resin	...	30	10 x 3.5	...	...	...
	30	...	...	106	5	5
	...	30	...	106	5	5
	12	...	1 x 3.5	...	...	...
	12	...	2 x 1.75	...	...	...
Coated with starch binder	...	30	3 x 3.5	...	...	...
	...	30	3 x 3.5	...	...	...
	...	30	10 x 3.5	...	...	...
	30	...	...	113	5	5
Controls	...	30	...	113	5	5
	12	...	3 x 3.5	...	...	...
	...	15	3 x 3.5	...	...	...
	...	30	10 x 3.5	...	...	...
Totals	20	20	...	...	...	...
	170	260	...	...	30	30

fibrosis. The glass dust was transported to satellite lymph nodes but there it was also not associated with fibrosis.

Although another vitreous fibrous dust (ceramic aluminum silicate) has been demonstrated to be biologically "inert" when inhaled or injected intratracheally,⁵ the possibility of severe pulmonary damage from the inhalation of dust derived from fibrous glass has recently been given attention because of the delayed occurrence of tumors associated with exposures to still another fibrous dust—*asbestos*.

Against this background relative to the pathogenicity of fibrous dusts and glass dust in particular and the recent reaffirmation of the concept that nonfibrous particulates of asbestos were nonpathogenic,⁶ we are reporting the two-year results of an investigation into the pathogenicity of fibrous glass dust. Two specific questions were to be answered: (1) Does the fibrous shape of glass particles render fibrous glass dust pathogenic? And (2) does a coating on the glass fibers of either a phenol-formaldehyde type resin (used in the construction or insulation industry) or a starch type binder (used for textiles) contribute to its pathogenicity?

In subsequent communications we shall report on the surviving animals that lived beyond the present two-year period, and on the rate at which the fibrous glass was

cleared from the lungs of the animals. We shall also report on the postmortem pulmonary findings in a series of 18 long-term employees of the oldest fibrous glass factory in the world. Some of these men had worked in the production of fibrous glass when the industry was in its infancy up to the present—a period spanning about 30 years.

Materials and Method

In order to ensure deposition of fibrous glass dust upon the alveolar membrane, a specially made fibrous glass averaging 1 μ in diameter was obtained through the cooperation of the three largest producers of fibrous glass. One batch of this glass was coated with a phenol-formaldehyde resin, another batch was coated with a starch binder, and a third batch was left uncoated. The different batches of fibrous glass were ball-milled until the fiber lengths were predominantly around 50 μ or less.

The dust-dispersing apparatus consisted of a glass L-shaped reservoir which had a spiral worm-feed mechanism in the horizontal limb. This was turned by a small 6-rpm motor. The slowly turning worm transported small amounts of dust at a fairly constant rate to the end of the horizontal tube which connected with a 4-inch diameter vertical lucite tube. The lower end of the lucite tube was continuous with the vertical limb of a rounded ensemble of galvanized 4-inch tubing (stovepipe) which, with the vertical lucite tube, formed the letter "d." The



Fig 1.—Left, Dust focus in rat that died after 22½ months of exposure to 135 mg/cu m of uncoated fibrous glass dust. Macrophage containing alveoli are clustered about alveolar duct, not atelectatic, and septal walls are not fibrotic. Some alveolar lining cells are slightly hypertrophied and hyperplastic. Enlarged macrophages contain numerous fine opaque granules. Some alveoli show necrosis of macrophages by loss of cell walls and

disappearance of nuclei (hematoxylin-eosin stain, $\times 150$). Center, Same field after impregnation with silver. Although there is focal increase in argyrophilic fibers of a few alveoli (right and upper left) collagenization is absent and anatomic integrity of air spaces is intact (Gordon and Sweets, $\times 150$). Right, Composite incineration picture of same field shows large amount of glass dust associated with macrophages and cytoplasmic debris ($\times 150$).

short vertical limb opposite the lucite tube was bisected by an inserted squirrel-cage blower which caused a turbulent refluxing of the dust through the blower. A small ¼ inch tube, entering the upper horizontal limb of the sheet metal ensemble, injected air into the system at a rate of 1 liter/min. This injected air carried the finer and disaggregated dust particles upward into the upper portion of the vertical lucite tube. The latter connected with the inhalation chamber by means of a right angle bend. Each of the three inhalation chambers had its own dust-dispersing apparatus.

The inhalation chambers measured $2 \times 4 \times 4\frac{1}{2}$ feet ($0.61 \times 1.22 \times 1.37$ meters) and contained 36 sq feet (1.02 cu m). They were constructed of wood except for a ½-inch lucite front panel. Each chamber held 50 rats and 30 hamsters housed in 20 wire cages suspended from tracks fastened to the top of the chamber. The cages were rotated each day to assure that all animals within the chambers were subjected to comparable exposures. The spent chamber air was pulled at the rate of 1 liter/min

through a 10-inch furnace type filter containing a preweighed filter paper. The increase in weight of the filter paper at the end of the day's run divided by the volume of air flowing through the chamber equalled the average concentration of glass dust during the exposure. The average concentration of dust in the chambers was in the neighborhood of 100 mg/cu m (Table). The exposures were for six hours per day, five days per week, for 24 months. After each six-hour exposure, the animals with their cages were removed from the inhalation chamber and placed in a rack in a large room.

Periodically the glass dust in the inhalation chambers was sampled by means of a millipore filter through which chamber air was pulled at the rate of 1 liter/min for six hours. The glass dust was removed from the millipore membrane and placed on a glass slide. With immersion oil as mounting medium and a phase contrast photomicroscope, 70% to 76% of the dust particles were found to be fibrous and 24% to 30% nonfibrous. The average diameter of the fibers was found to be 0.5μ within a very narrow



Fig 2.—A focus of alveolar fibrosis as encountered in a few rats. This rat died after 22½ months exposure to uncoated glass dust. Presence of cholesterol needle spaces with lipidic macrophages and their debris in air spaces (alveolar stasis) seems to explain fibrosis adequately (hematoxylin-eosin stain, $\times 150$).

range. The average length was in the neighborhood of 10μ , with an approximate range of 5μ to 20μ .

Five rats and five hamsters from each chamber were killed with ether after 6 and 12 months of dust exposure, respectively, for sampling purposes. These and all animals which died during the exposure were subjected to autopsy during which the lungs were distended with 10% formalin under a head of 12 cm of water. The remainder of the animals were pastured after 24 months of dust exposure. They will be allowed to live out their lives in order to obtain data on the tendency of the animals exposed to glass dust to develop pulmonary neoplasms.

An additional 150 rats and 60 hamsters received intratracheal injections of the same dusts as were dispersed in the inhalation chambers. The Table lists the dosage (3.5 mg dust suspended in 1 ml water) and the number of such injections which were carried out with the

animals under light ether anesthesia and with the aid of a laryngoscopic speculum.

After paraffin impregnation of blocks cut from the distended lungs, sections were cut at 6μ . These were stained routinely with hematoxylin and eosin. Pertinent fields from each section were photographed on 35 mm film. The sections were then decolorized and impregnated with silver by the Gordon and Sweets technique. The same fields were then rephotographed. These photographs yielded detailed data on the cell-stroma interrelationship so important for ascertaining the anatomic status of an alveolar wall. After these last photographs were taken, the cover slips of the sections were removed and the sections were incinerated for one hour at 600°C in an electric muffle furnace. After the slides were treated with concentrated hydrochloric acid, washed, and dried, the same fields were photographed for the third time, but on this occasion, under dark-field conditions. The last photographs, when correlated with the previous ones, gave specific information regarding the relation of glass dust deposits to anatomic changes in alveolar walls. Because the index of refraction of the glass dust is very close to that of the embedding medium, the dust is practically invisible except after incineration. The study of incinerated sections was of particular importance to determine if focal alveolar fibrosis was or was not associated with the presence of glass dust deposits.

A series of 20 rats and 20 hamsters from the same shipments as that of the experimental animals served as laboratory controls. They, like the experimental animals, had access to food and water ad libitum. These control animals were housed in wire cages similar to those of the animals exposed to dust, but they were not kept in inhalation chambers. Therefore, unlike the experimental animals whose air supply was limited during the dust exposure, the controls had unlimited supplies of fresh air at all times.

Results

It has not been possible to detect differences in the tissue reaction of the three different types of fibrous glass dusts; whether uncoated or coated with a phenol-formaldehyde type of resin or with a starch binder. For this reason, one description of the results applies equally to those for all three dusts.

Inhalation Series.—Rats.—Grossly, no significant abnormality of the lungs of these

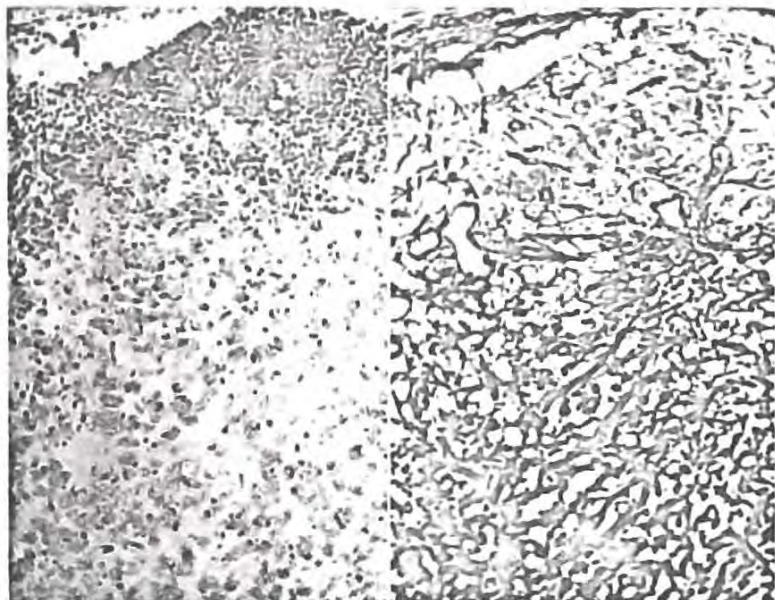


Fig 3.—Left, Portion of greatly enlarged satellite lymph node from rat which inhaled fibrous glass coated with phenol-formaldehyde resin for 21½ months and died during exposure. All but cortical rim of lymphoid tissue has been replaced by massed macrophages, many necrotic. Between macrophages are thick bundles of palestaining collagen (hematoxylin-eosin stain, $\times 150$). Right, Same field after impregnation with silver. Normal reticulin stroma is in subcapsular cortex; elsewhere, delicate, sparsely distributed reticulin fibers have been replaced by dense network of coarse collagen bundles (Gordon and Sweets, $\times 150$).

animals was observed except for pneumonia and the endemic presence of chronic bronchitis and its sequelae.

Microscopically, the outstanding abnormality from the earliest planned observation period (six months) to the end of the dust exposure (two years) was the presence of macrophage-containing alveoli clustered around respiratory bronchioles. These macrophages were filled with glass dust (Fig 1). The number of macrophages per alveolus was not so great as to completely fill the air spaces nor were the latter atelectatic. Occasional extracellular glass fibers were also observed. The character of the dust macule as observed in Fig 1 is typical of the dust reaction in all animals.

The most significant observation regarding these dust accumulations was the fact

that the alveolar septa in the proximity of the glass dust deposits were not thickened. There was focal increased cellularity of alveolar walls and the alveolar surface cells were enlarged; but there was no evidence of collagenization of alveolar septa, or for that matter, of any other component of lung tissue in the vast majority of the animals. (There were a few exceptions which will be described here.) Slight arborization of alveolar argyrophilic stroma was demonstrable in some regions of dust accumulation.

In the lungs of a few rats that had been exposed to high dust concentrations for almost two years, several foci of septal collagenous fibrosis were found. The alveoli here contained macrophages and cellular debris. Among this cellular debris, cholesterol needle spaces were found (Fig 2). Although



Fig 4.—Left, Dust focus in lung of hamster which died after 21½ months exposure to uncoated fibrous glass dust. Alveolar duct running lengthwise (left of center) has most of its evaginating alveoli filled with massed, well preserved macrophages (hematoxylin-eosin stain, $\times 150$). Center,

Same field after silver impregnation shows alveolar architecture undisturbed and no septal thickening with slight focal arborosence of alveolar reticulin. (Gordon and Sweets, $\times 150$). Right, Incineration composite of same field shows large amount of glass dust in macrophages ($\times 150$).

glass dust was also present, it was less abundant than that seen in regions without fibrosis.

Although little evidence of lymphatic transport of glass dust was demonstrable at the six-month period, the amount of glass dust in some of the satellite lymph nodes near the end of the two-year exposure was massive, occupying as much as one half of their moderately enlarged volumes. Yet, with all this lymphatic transport, none of the glass seems to have been arrested in the interstitial tissues, for none was detected there.

At six months, small clusters of large phagocytic cells filled with glass dust but without a significant associated stromal increase were demonstrable in some of the satellite lymph nodes. At 12 months, the accumulations of glass-filled cells were larger; but generally, the dust foci were not associated with an increase in argyrophilic stroma. In a few rats near the end of the second year, many of the phagocytic cells in

the satellite lymph nodes were necrotic and much of the stroma was heavily collagenized (Fig 3).

Hamsters.—Except for the animals that died of pneumonia, the lungs of these hamsters showed no gross lesion. As in rats, alveoli clustered around respiratory bronchioles and alveolar ducts contained dust-filled macrophages. The latter were more numerous and more crowded than in rats. Atelectasis was absent (Fig 4). In the main, the presence of glass dust in the lungs of hamsters caused changes that differed from those seen in the lungs of rats in two other important respects: the presence of ferruginous bodies⁷ which were abundant in hamsters (Fig 5) but absent in rats, and the presence of laminated, calcific alveolar microliths in the lungs of some hamsters killed at the 24-month period. These were likewise not seen in rats. These microliths were found amidst the collections of dust-filled macrophages.

In contrast to the satellite lymph nodes of



Fig 5.—Ferruginous bodies in lung of hamster which died 19½ months after three intratracheal injections of 3.5 mg each of textile binder-coated fibrous glass dust. Note bipolar clubbing and transverse ridges suggestive of early segmentation (hematoxylin and very light eosin stain, $\times 150$).



Fig 6.—Bland pleural thickening in hamster which died after 23½ months exposure to uncoated fibrous glass dust; no evidence of active inflammation within or beneath pleura. Plentiful cells in parenchyma are RBCs due to hyperemia and hemorrhage which developed during agonal period (hematoxylin-eosin stain, $\times 150$).

rats, those of hamsters were not enlarged, even at 24 months. The satellite lymph nodes of hamsters killed at 24 months contained small and larger clusters of large pale phagocytic cells filled with glass dust. The silver-impregnated preparations showed that these phagocytic cell clusters were not associated with an increase in stromal elements.

Intratracheal Injections.—It has previously been reported that fibrous dusts of diverse composition when injected intratracheally into rats are capable of causing the production of polypoid masses of inflammatory tissue in smaller bronchial divisions, and fibrous glass was one of the dusts.⁸ Such lesions were found in the rat lungs four days after the intratracheal injections. However, animals killed after one year or later failed to show any evidence of such bronchiolar lesions, suggesting that these polypoid proliferative lesions were reversible.

The later results of intratracheally injected fibrous glass dusts in rats were similar to those that developed in this species from the inhalation of these dusts, except that the size of the dust foci was smaller and the number of such foci was reduced. No alveolar fibrosis or other significant septal changes were observed.

Since hamsters were not sampled within one week after the intratracheal dust injections, it is not known whether or not these species also reacted to the fibrous dust injections with polypoid inflammation in the air-conducting tubes. No lesion was seen in these structures in the animals sampled at the six-month period. The hamster lungs responded to the intratracheal injections with the development of dust foci very similar to those observed in rats. As in the case of hamsters which inhaled the dusts, ferruginous bodies were abundant in the dust foci

caused by intratracheally injected fibrous glass dust. However, alveolar microliths were not found. There was one additional finding in the lungs of these hamsters which was also present, but to a lesser extent in hamsters and rats that had inhaled the glass dusts. This was a diffuse, bland, acellular, collagenous pleural fibrosis (Fig 6). The incinerated sections showed the presence of glass dust in an occasional macrophage situated in the lumen of one or a few of the subpleural alveoli; no glass dust could be seen in the thickened pleura itself.

Comment

We were confronted with two immediate problems in the design of this study. One was concerned with the diameter of the glass fibers required to allow its particles to reach the alveoli. Some helpful data were obtained from a recent pilot study in which we found that the dust isolated from the lungs of Pittsburghers contained many naked fibers and the vast majority of these fibers (75%) had diameters between 1.5μ and 2.5μ ; whereas less than 6% had diameters greater than 4μ . Inasmuch as the average diameter of the commercially available fibrous glass is about 9μ , we felt that this would be unsatisfactory as a source for respirable dust to which animals were to be exposed. We, therefore, asked for and obtained fibrous glass with an average diameter of 1μ .

The second problem involved the magnitude of the dust concentration to which the animals were to be exposed. Although the threshold limit value (TLV) of a "nuisance" dust has been set at 15 mg/cu m ,⁹ our previous experience with glass dust⁴ and a vitreous fibrous dust⁵ suggested that animals could be exposed to dust concentrations of fibrous glass substantially higher than the TLV of a "nuisance" dust without encountering serious problems such as alveolar proteinosis¹⁰ and endogenous lipid pneumonitis^{11,12} because of alveolar clearance failure.

In a preliminary experiment (unpublished data) during which we exposed rats to fibrous glass dust concentrations that ranged as high as $2,000\text{ mg/cu m}$, we were surprised at the relatively small amounts of dust that could be found in the lung sections. This result led us to consider a glass dust concen-

tration in the inhalation chambers of 100 mg/cu m as probably safe provided that the dust was in the category of "nuisance" dust. This dust concentration proved to be very close to the limit which allowed adequate performance on the part of the alveolar clearance mechanism, as was indicated by the focal collagenous alveolar fibrosis associated with macrophagic debris and cholesterol needle spaces that were found in a few rats. The alveolar fibrosis was very likely a sequel of endogenous lipid pneumonitis^{11,12} and the latter, in turn, appears to be related to faulty clearance of breakdown products of macrophages.

Atelectasis is commonly found associated with the presence of nonfibrogenic dust in air spaces^{12,14} and may be attributed to impaired surfactant secretion by alveolar lining cells. The noteworthy absence of atelectasis in the animals of the present study, in spite of the presence of abundant fibrous glass dust, suggests that the surfactant secretion remains unimpaired. This, in turn, is another indication of the biologic "inertness" of this dust.

The formation of microliths in the lungs of some hamsters surviving 24 months of glass dust inhalation is interpreted as still another indication of alveolar clearance failure caused by the extremely high dust dosage. This clearance failure is definitely dose-dependent inasmuch as microliths were not found in animals exposed to dust for less than 24 months, nor were they formed in the lungs of hamsters receiving intratracheal injections of the same dusts.

The polypoid inflammatory lesions in the air-conducting tubes of rats, that developed after intratracheal injections of fibrous glass, were apparently reversible since they were not to be found in rats sampled later in the year. Furthermore, the lesions were considered to be artifactual since they did not occur in rats which inhaled the same dusts.⁸ The explanation offered was that during the process of injection, the fibers align themselves parallel to the rapidly flowing stream of fluid, the velocity of which increases as it passes from the barrel of the syringe into the narrow bore of the needle. Because of this orientation and the force of the injection, the mass of the fibers tend to impinge point-first upon the mucosa of the branching

air-conducting tubes. Thus, mechanical injury was inflicted upon mucosal surfaces by the technique used to introduce the dust into the lungs.

The incongruity of lymph nodal fibrosis combined with the absence of pulmonary fibrosis secondary to dust deposits in rats may be explained possibly on the basis of two proposals. First, the tissues responding to the fibrous dust in the two different sites are quite different. In the lung, the alveolar membrane of endodermal origin responds; whereas in lymph nodes, mesenchymal tissues of mesodermal origin respond.¹⁵ Second, the accumulations of phagocytic cells appear to be avascular; and the larger the cell masses, the more vulnerable they appear to be to metabolic restrictions. This would explain the necrosis of these cells and the subsequent collagenous fibrosis found in lymph nodes with large deposits. Although the absence of lymph nodal fibrosis in ham-

sters is explainable on the basis that large dust deposits did not occur in this species, there is no explanation why lymphatic transport of glass dust was minimal in hamsters.

The delayed transport of fibrous glass dust to satellite lymph nodes is another feature characteristic of a dust that has little or no pathogenic potential.^{16,18} This is explained on the basis of the concept that dust particles can penetrate into the lung interstitium only as naked particles and that, therefore, their lymphatic transport is contingent upon their attaining an extracellular status. The rapidity with which dust particles are transported to satellite lymph nodes, therefore, reflects their cytotoxicity. Since non-pathogenic particles would not be expected to cause death and disintegration of the cells which have engulfed them, their extracellular status, and hence their penetration of the alveolar membrane and subsequent lymphatic transport, would be delayed.

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DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE
PUBLIC HEALTH SERVICE
Consumer Protection and Environmental Health Service
Environmental Control Administration

REFER TO:

March 14, 1969

Mr. Don Sheldon
c/o Forsythe Bergmann-Vanc: -492
1201 30th Street, N.W.
Canton, Ohio 44703

Dear Mr. Sheldon:

This letter is in response to your telephone request for information as to inhalation hazards from the use of fibrous glass insulation material in duct work.

Fibrous glass and similar materials such as glass wool and rock wool are practically inert materials which of themselves do not have any significant toxic effects. The principal difficulty arising from working with any glass material used in insulation work would be irritation and itching of the skin. Small fibers become imbedded in the skin and cause itching and mechanical irritation of the skin. These areas may serve as focal points of secondary infection. Such infections may be prevented by good hygiene practices, as with any wound or abrasion. If not properly treated, such infections may become quite serious and constitute the greatest danger of working with fibrous glass. The fibrous glass acts only as a mechanical agent which opens a pathway for bacteria to attack the skin.

Studies of the inhalation of dust from fibrous glass materials have not shown them to produce any toxic manifestations or poisonous effects beyond the skin irritant properties which are mechanical in nature. In a rather recent article, "Pulmonary Hazards from Exposures to Glass Fibers," by Ahmed N. M. Nasr (Journal of Occupational Medicine 9:345, July 1967), the author concludes that, "As far as could be ascertained from a search of the American and the European literature, available evidence indicates that fibrous glass is a relatively inert material with no fibrogenic or other significant toxic properties." This article and others indicate in essence that there is no demonstrable pulmonary effect due to long-term exposure to fibrous glass. One other comment in the article by Dr. Nasr is, "The size of glass fibers in common application and their tendency to form felt-like masses upon any object with which they come in contact, probably prevent inhaled glass fibers from penetrating into the pulmonary alveoli."

You also mentioned asbestos. Asbestos is not to be confused with fibrous glass. There is no similarity chemically nor physiologically between fibrous glass and asbestos.

Even studies made with fibrous glass plastic products have not indicated there were any responses other than the category of an "inert dust." Dr. Schepers, in one of his articles, states that the most important fact elicited by this experimental inquiry into the biological action of inhaled Fiberglas plastic dust concerns its negligible toxicity. In other studies by Dr. Schepers with sheet fibrous glass plastic materials, he likewise reports that judging by the responses in the three animal species studied, the dust behaved as a biologically inert substance.

In a later study by Dr. Schepers he explored by intratracheal and intravenous injections, subcutaneous implantation, cutaneous irritation and ocular instillation the theoretical possibility that fibrous glass plastics or other components might gain access to subcutaneous tissues, may irritate the cornea, or may become lodged in some other organ. He concluded that, "None of the substances caused fibrosis except in a case of Fiberglas by subcutaneous implantation. Necrobiosis did not occur. Lesions resolved through phagocytosis of retained particles."

In a study by E. B. Helzel and J. B. Mitchell, "Cutaneous Reaction to Fiberglas," (Industrial Medicine and Surgery 26:647, December 1957), the authors report that they were unable to induce sensitivity by means of patch and rubbing tests. Any reactions observed were interpreted as being largely due to mechanical irritation which was transitory in nature.

We are enclosing a list of references on fibrous glass. You can probably locate many of these in public or university libraries available to you. We hope the information furnished will be helpful and if we can be of further assistance, we shall be glad to do so.

Sincerely yours,

E. Elbridge Merrill, Jr.

E. Elbridge Merrill, Jr.
Industrial Hygienist
Scientific Reference Service
Bureau of Occupational Safety and Health

Enclosure

EXPOSURES OF INSULATION WORKERS TO

AIRBORNE FIBROUS GLASS

REPORT TO HEALTH AND SAFETY COMMITTEE
NATIONAL INSULATION MANUFACTURERS ASSOCIATION

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BERKELEY

MAY 1, 1970

FINAL REPORT

EXPOSURES OF INSULATION WORKERS TO

AIRBORNE FIBROUS GLASS

W. Clark Cooper, M.D., J. LeRoy Balzer and Douglas P. Fowler
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This study was undertaken as part of our investigation of the work environment of insulating workers; and is a continuation of a previously reported study on the exposures of insulation workers to asbestos fibers and other environmental stresses.^{1,2}

The stated objective of this part of our study was: "To define qualitatively and quantitatively the airborne concentration of fibrous glass in the breathing zones of workers engaged in the fabrication, application, finishing and other operations involved in the installation of prefabricated and other fibrous glass insulation materials." To attain this objective: (1) over 100 samples of fibrous glass insulation products were examined; (2) the conditions of use of these materials were evaluated during visits to over 50 construction sites where the materials were being installed by members of the International Association of Heat and Frost Insulators and Asbestos Workers Local 16; (Northern California and Northern Nevada) (3) the actual concentrations of airborne fibrous glass were determined during representative installation operations by taking and analysing samples of the air in the breathing zones of those workers, and in areas surrounding the operations.

The general work environment of insulating workers has been described previously, particularly in relation to asbestos exposure;² as have been many of the chemical and physical properties of fibrous glass.³ The use of fibrous glass insulation has increased markedly in recent years, and it is now probably the most commonly used thermal and acoustical insulation material. As its uses have increased, so have the variety of forms in which it is produced. It has found widest use in the form of "batts" or "blankets" of light-density fibrous glass either with or without a backing of protective and/or water impermeable material such as foil, vinyl, or treated paper. In this form it is used by the insulation workers studied to thermally insulate ventilation ductwork. Another important source of exposure to the insulation worker is the handling of higher density "boards" of fibrous glass (which may be coated with Neoprene or other substances) used for the thermal or acoustical insulation of walls, ceilings and other flat surfaces, particularly in mechanical rooms and ventilation plenums of large buildings. A third primary source of glass fiber exposure is in the installation of fibrous glass pipe insulation, in which half-round sections of compressed fibrous glass are used for thermal insulation. Fibrous glass products are also used by insulation workers for acoustical/thermal insulation of mechanical equipment, and are formed into special shapes by them on job sites.

Because of the varying emphases of local contractors, it is difficult to estimate a "typical" exposure to fibrous glass, but it is probable that

more than half of an average insulator's work experience (in the Northern California and Nevada local union studied) is with fibrous glass insulation of various types. However, usage varies and some insulators' experience may be almost totally with glass. Further, the use of these materials is continuing to increase.

The job classifications used by Balzer¹ for the description of work with asbestos-containing products do not appropriately describe work with fibrous glass products, since one man will ordinarily fabricate, apply, and finish a single piece of fibrous glass material. Therefore, such classifications are not used, except where definite separation of work tasks was observed.

Fibrous Glass Insulation Materials

Fifty-seven fibrous glass insulation products were supplied by manufacturers cooperating in the project. Additional samples of 50 products were collected in the field, some of which were duplicates of those supplied. From each product, a representative sample was taken, including portions of all apparent types of material, and this sample was examined under phase contrast microscopy at 430X using a mounting medium with a refractive index below that of glass. The diameters of 50 individual fibers were estimated by comparison with a Porton eyepiece reticle, and the size distribution of fibers in each product determined. Although it is customary to characterize particle size distributions by geometric mean and geometric standard deviation, this was not done for these data, since the distribution of fiber

diameters did not exactly fit the expected simple log-normal distribution.

Although some of this deviation from log-normality was undoubtedly due to sampling variation, which was then magnified in effect by our relatively small samples, much of the deviation is "real", and was confirmed for several products by sizing 400 to 500 fibers. There was "smoothing" of the size distribution curves, and extension of the range of sizes encountered in larger samples; but the basic conclusions were not changed.

To permit comparison among materials, accumulated data on product fiber diameters are summarized in Table I. The diameter distributions of some typical products are shown in Appendix A, with some comparisons of the results obtained when large and small numbers of fibers were sized.

Exposures of Insulation Workers

The exposures of insulation workers to fibrous glass, like their exposures to other environmental contaminants, cannot be described by time-weighted average exposures because of the nature of the trade. An insulation worker will be exposed, during a working day, to a series of "peak exposures" to the material which he is handling. One man will ordinarily cut, apply, and finish a single piece of glass fiber insulation material. This differs from the situation usually found in a production plant, or in some applications of asbestos-containing products, where a worker may repeat a single operation for most of his working day, so that environmental contamination tends to fluctuate less widely. The

insulation worker moves about frequently, and may work on several floors of a large building in a single working shift, with a consequent variety of exposure to environmental contaminants. Further, the worker who today is installing calcium silicate insulation in an enclosed boiler room, with extremely high concentrations of airborne asbestos fibers, may tomorrow be wrapping ducts with fibrous glass in a relatively open area of the same building, with natural ventilation from open windows serving to remove airborne glass fibers from his vicinity nearly as soon as they are produced. Exposures, then, cannot be accurately characterized as though they were constant, but must be related to the work being performed at the time of the exposure. Short term sampling periods are required in order to assess the environmental exposure.

Concentrations of Glass Fibers in Air

In table II are shown the mean fiber diameters of the fibrous glass materials used, and concentrations and sizes of the airborne glass fibers found during installation of fibrous glass insulation at the construction sites from which air samples were taken.

Sampling techniques used in this study have been described previously.² Briefly, air for breathing zone samples was drawn through 0.8 μ m (mean pore size) Millipore membrane filters by NSA Monitaire pumps at flow rates of 2 to 3 liters per minute. Area samples were taken in the same way, using pumps with flow rates of 25 to 30 liters per minute. The filter holders for the area samples were about 5 to 6 feet above floor level and 10 to 20 feet away from the locations of the installation operations. Sampling periods were 20 to 60 minutes.

Table IV.

		Material		Air Samples: Breathing Zone				Air Samples: Area			
Operation	Site #	mean fiber diameter (μ m)	f/ml	mean fiber diameter (μ m)	* Length %		f/ml	mean fiber diameter (μ m)	* Length %		
					<78 μ m	>78 μ m			<78 μ m	>78 μ m	
Duct Wrapping	1	5.3	1.26	4.7	24	76	0.09	4.0	43	57	
		6.3	0.90	4.0	40	60					
		6.4									
	2	4.0	0.51	3.4	46	54	0.02	5.5	40	60	
			0.79	3.6	36	64					
	3	4.1	1.40	2.6	62	38	0.03	3.7	78	22	
			1.33	2.3	60	40					
	5-IV	7.5	0.80	2.5	83	17					
8	5.8	1.20	6.2	37	63	0.07	3.7	62	38		
	5.5	2.34	5.0	42	58			0.03	4.5	54	46
Wall & Plenum Insulation	4	7.2	0.53	7.4	30	70	<0.01	-	-	-	
							0.09	6.2	50	50	
	5-I	10.2	3.26	8.4	47	53	0.08	7.9	49	51	
	7-I	8.1	4.18	3.5	59	41	0.47	2.8	55	45	
		7.6									
	7-II	7.8	3.08	3.8	60	40	0.34	2.2	50	40	
		7.1									
	Pipe Insulation	5-III	8.5	0.93	3.1	71	29				
6			0.48	4.1	40	60	0.05	2.3	86	14	
		6.7	0.57	3.4	59	50	0.05	2.6	88	12	
		5.0					0.03	2.6	86	14	
		6.0					0.02	3.1	66	34	
		5.6					0.09	3.5	66	34	
							0.09	2.9	65	35	
Fan Housing Insulation		5-II	6.9	1.57	3.5	88	12	0.20	3.4	82	18

* 78 μ m is the equivalent length of the major division of the Porton reticle with our optical system.

number of work sites were visited, those described were selected for sampling as representative of the insulators' work environment. With each description is a figure showing the diameter distribution of the material used, the breathing zone samples and the area samples.

DISCUSSION OF RESULTS

The exposures of insulators to airborne fibrous glass during the application of fibrous glass insulation products ranged from 0.5 to 8 fibers/ml with a median of 1.26 and mean of 1.8 fibers/ml. This is significantly lower than their exposures to other fibrous materials.² The potential exposures of other construction trades to airborne glass fibers as a result of "fallout" from insulating operations were still lower, ranging from below 0.02 fibers/ml to 0.47 fibers/ml with a median of 0.07 fibers/ml and a mean of 0.1 fibers/ml, at a distance of 10-20 feet from the insulator during his application of fibrous glass materials.

The maximum levels were found in confined spaces, where there was little circulation of air, and where clean-up (including sweeping and packing of scraps of fibrous glass residue) had generated high concentrations of airborne dust of all kinds. Although, as stated previously, it is difficult to generalize about the occupational environment of the insulator, it is probable that exposures of this sort are relatively rare. It is most common for clean-up to take place only at the end of a work shift. Because of the bulkiness

of fibrous glass insulation products, cutting is usually done in a relatively open area which affords the space necessary for layout and measurement.

One of the striking findings of the study, as shown in Figure 1, is that the mean fiber diameter of the parent products is not a reliable predictor of the mean diameter of the fibers in the aerosol generated from these products. Although the determinants of "respirability" of airborne fibers are not fully agreed upon, it is probable that respirability is diameter-dependent.⁴ Timbrell's experiments, using the aerosol spectrometer, suggested that fibers with densities of less than 3.5 g/cm^3 and large aspect ratios (length to width ratios) could be considered "respirable" if the fiber diameter was less than 3.5 micrometers. Fibrous glass (with a density of $2.4 - 2.6 \text{ g/cm}^3$) fits his definition and thus glass fibers less than 3.5 microns in diameter will be defined as "respirable." As can be seen in Figure 2, about one half of the airborne glass fibers generated from the handling of fibrous glass insulation materials are below this upper limit. Thus, it would seem that the often encountered statement that the diameter of the majority of glass fibers is such that they will not penetrate to the lower respiratory tract is true of the parent materials, but generally not of the airborne fibers which are of primary concern in occupational health.

Although our observations might appear to be at variance with those of Johnson et al., who recently studied the airborne dust concentrations in glass fiber insulation and textile manufacturing plants, our conclusions are essentially the same as theirs; the concentrations of small diameter airborne fibers are very low.

Figure 1

ACCUMULATED PERCENTAGE DISTRIBUTION OF FIBER DIAMETERS FROM ALL SITES

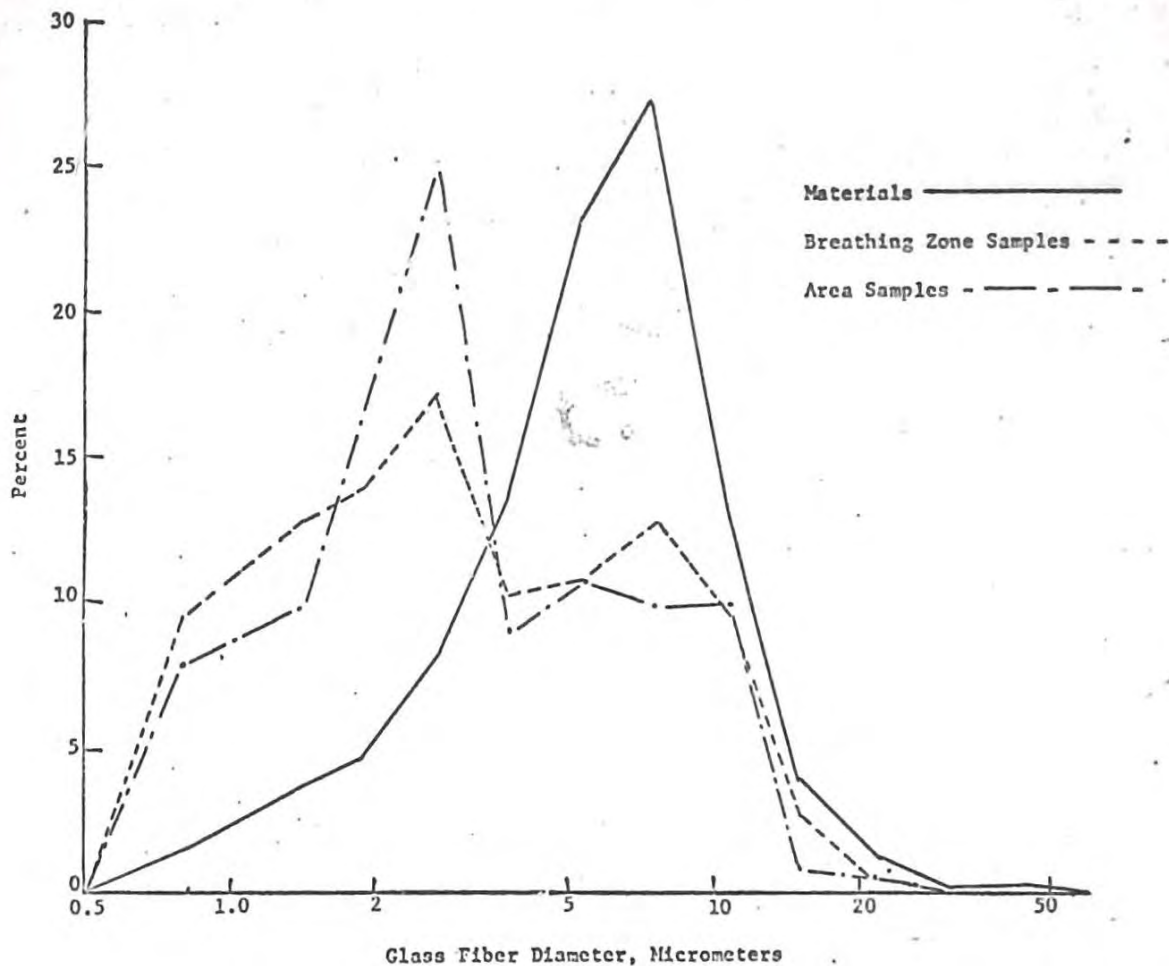
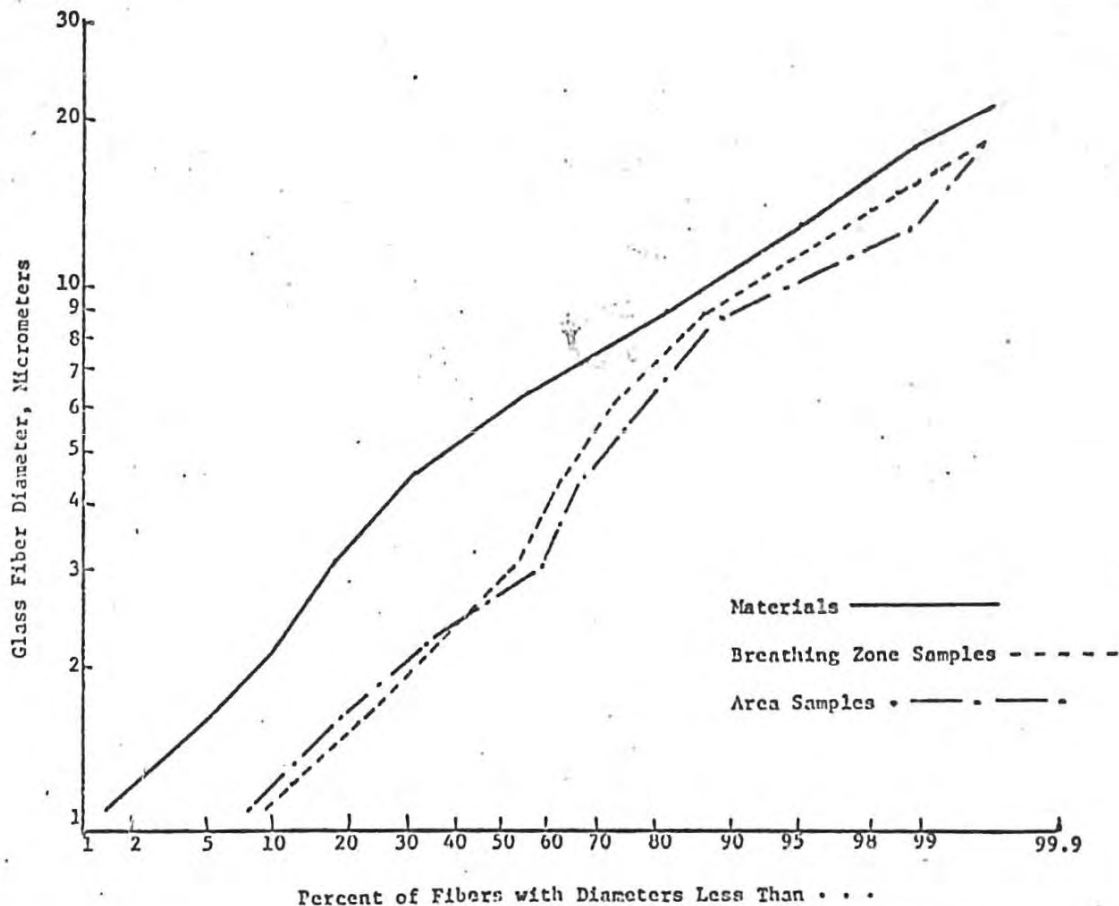


Figure 2

CUMULATIVE DISTRIBUTION OF FIBER DIAMETERS FROM ALL SAMPLES



What do the concentrations of fibrous glass which were found mean in terms of potential hazard to health? The consensus of present opinion is that fibrous glass, although mechanically irritating to the skin and upper respiratory tract, is per se relatively inert and that it does not cause chronic pulmonary disease when inhaled.^{5,7,8,9} Although Milby and Wolf have reported cases indicative of respiratory irritation in individuals who had removed insulation materials or otherwise been exposed to fibrous glass,¹⁰ and Murphy in 1961 reported a case of hemorrhagic bronchiolitis after heavy exposure to dust during removal of glass insulation,¹¹ no documented cases of pneumoconiosis or other chronic pulmonary diseases due to fibrous glass are known to us. Intubation studies in lower animals have shown relatively little biologic activity.^{12,13}

This is the basis upon which the American Conference of Governmental Industrial Hygienists, in 1969, set the tentative threshold limit value for fibrous glass at the same level as that for inert dusts, now 10 mg/m^3 (for fibers under .5 to 7 micrometers in diameter). Although gravimetric determinations of glass are not feasible in the construction trade, the fiber counts that we observed have been calculated to represent amounts of glass far below the TLV. These were the concentrations during actual installation operations and not time-weighted averages. The insulator's time-weighted average would be much lower than the figures given.

Because there are unquestionably respirable fibers in most fibrous glass materials and there are many possible variations in coatings, binders, etc.,

prudence dictates that there be continued surveillance of the exposures of various groups to fibrous glass as is being done by the Public Health Service.⁵ The assumed absence of chronic effects should be checked by appropriate long-term observation of those whose work involves frequent or daily inhalation of glass.

Conclusions

1. Insulation workers, during the actual application of fibrous glass insulation products were exposed to airborne concentrations of glass fibers ranging from 0.5 to 3 fibers/ml, with a median of 1.3 fibers/ml and mean of 1.8 fibers/ml.
2. The computed gravimetric concentrations of airborne glass fibers less than 7 μ m in diameter did not exceed the tentative Threshold Limit Value of 10 mg/m³ during any peak period. The majority of these measurements were less than 1.0 mg/m³.
3. No time-weighted average could be computed which would be representative of a work day exposure. In any case, it would be considerably less than the peak concentrations measured.
4. Insulators work with fibrous glass containing materials from less than 10% to 100% of their time depending on employer and type of construction site.
5. It is doubtful that the concentrations of airborne fibrous glass found in our study are of long-term biological significance.

The technical assistance of Mr. Jack Murchio and microscopy of Mr. Arturo DeLeon are gratefully acknowledged.

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Exposure to Fibers in the Manufacture of Fibrous Glass

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As part of its study of health hazards associated with the manufacture and processing of fibrous materials, the Bureau of Occupational Safety and Health is conducting a cohort analysis of past workers in the fibrous glass industry. Environmental surveys to estimate the degree of exposure of fibrous glass workers to fibers that are respirable indicated that the exposure had been negligible. Present average concentrations of fibers in air are low, as is the total airborne glass concentration. Concentration ranges and averages in glass fiber insulation and glass textile fiber manufacturing are compared with those existing in other mineral industries for potential health implications.

Introduction

THE BUREAU OF OCCUPATIONAL SAFETY AND HEALTH of the U. S. Public Health Service is presently conducting a cohort analysis of past workers in the fibrous glass industry. The personnel records were obtained for the subject workers from five of the oldest plants in the United States that manufacture fibrous glass products. Mortality data will be developed for this group of workers from records of the Bureau of Retirement and Survivors Insurance, Social Security Administration. This records study is being undertaken in conjunction with the U. S. Public Health Service study of mortality rates in the asbestos product manufacturing industry. Since the two populations of industrial workers selected are exposed to two different fibrous materials, age-adjusted death rates by cause can be compared for possible relation to industrial dust exposures.

An environmental survey of the fibrous glass plants to estimate the exposure of work-

ers to airborne fibers of respirable size was conducted when the personnel records were obtained. The sampling techniques and methods of characterizing exposures that had been developed for the asbestos study were used in this study also. This paper summarizes the concentrations of airborne dust and fibers in five fibrous glass factories, four manufacturing insulation, and one making textiles.

Background

Glass has been known to man for many centuries, but it has only been since the 1930's that glass has been produced in the fibrous form which is the basis for many unique and useful applications. Fibrous glass is classified into two groups: (1) wool fibers and (2) textile fibers. Wool fibers are used in the manufacturing of acoustical and thermal insulation, acoustical ceiling material, air filters, and similar products. Textile fibers are used in fabrics, insulating tapes, and reinforcements for plastics and other materials. The fibers differ in that the textile fibers are produced in a continuous filament and are generally smaller in diameter, ranging to

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a low of 4 microns (μ) in diameter. The wool fibers are produced in varying lengths and are usually about 5 to 14 μ in diameter, depending on the application.¹ The fiber diameters are varied to provide the desired characteristic for the particular application but are closely controlled for each application.

Concern about possible health hazards associated with fibrous glass has been increased by the evidence that links asbestos fibers with carcinoma of the lung. This asbestos experi-

ence contrasts with that for fibrous glass, where neither animal experiments nor human experience to date has demonstrated significant effects on the respiratory tract.

Gross *et al.*² investigated the biological effects of glass dust on the lungs of experimental animals. They found that inhalation exposure caused a pulmonary reaction no more severe than would be expected from fine kaolin dust and a nonfibrogenic reaction with no bronchial or bronchiolar disease. Schepers and Delahant,^{3,4} studied the effects of glass fibers of different diameters in the lungs of rats and guinea pigs. They found that the larger-diameter fibers were relatively inert when administered by intratracheal injection, but that injected glass fibers 3 μ or less in diameter provoked bronchiolar damage without well-defined fibrosis. The latter condition was also reported from their inhalation studies with 6- μ -diameter fibers.

Human experience to date indicates that fibrous glass causes only upper respiratory irritation.^{5,6} Cardiac and pulmonary function studies revealed no functional impairment in workers who had been exposed for a number of years. In reported cases of lung disease associated with fibrous glass exposure, the disease has been acute, and infection has played a major role.⁷ The consensus of the toxicological literature seems to be that fibrous glass is a biologically inert material.

Procedure

The environmental portion of the field study of the fibrous glass industry utilized some of the same sampling procedures employed in the study of the asbestos product industries.⁸ The workers' exposures were characterized by a variety of sampling techniques to estimate the dust concentration in terms of the gross dust exposure, respirable dust exposure, gross fiber exposure and respirable fiber exposure. Membrane filter sampling, however, was selected as the principal method to be used in this study. The sampling medium used was Millipore Type AA membrane filter with a pore size of 0.8 μ . A 37-mm plastic filter holder was used for sampling at a flow rate of 1.7 liters per minute. Gross airborne samples were collected

TABLE I
Gross (Glass) Dust Concentrations by
Plant and Operations
(milligrams/cubic meter)

Operations	Plant No.*	Mean	Range
Batch and Marble	1	N.R. ^b	N.R.
	2	N.R.	N.R.
	3	1.34	0.18-5.96
	4	12.29	2.69-21.80
	1-4 ^c	5.82	0.18-21.89
Forming	3	0.12	0.12-
	1	0.20	<0.01-0.94
	2	0.44	0.06-1.70
	3	0.18	<0.01-0.66
	4	0.46	0.04-1.74
Spinning and Twisting	1-4	0.32	<0.01-1.74
	3	0.06	0.04-0.22
Waste Recovery	3	0.11	<0.01-0.40
	5	0.16	<0.01-0.48

(a) Plants 1 through 4 are insulation plants; 5 is a textile plant.

(b) N.R.—no results.

(c) Composite of results for plants 1 through 4.

TABLE II
Respirable (Glass) Dust Concentrations by
Plant and Operations
(milligrams/cubic meter)

Operation	Plant No.*	Mean	Range
Batch and Marble	1	N.R. ^b	N.R.
	2	N.R.	N.R.
	3	0.15	<0.01-0.31
	4	0.55	0.06-1.03
	1-4 ^c	0.35	<0.01-1.03
Forming	3	0.36	0.19-0.52
	1	0.03	<0.01-0.24
	2	0.03	<0.01-0.20
	3	0.07	<0.01-0.45
	4	0.09	<0.01-0.47
Spinning and Twisting	1-4	0.06	<0.01-0.47
	3	0.05	<0.01-0.36
Waste Recovery	3	0.10	<0.01-0.65
	5	0.12	<0.01-0.73

(a) Plants 1 through 4 are insulation plants; 5 is a textile plant.

(b) N.R.—no results.

(c) Composite of results for plants 1 through 4.

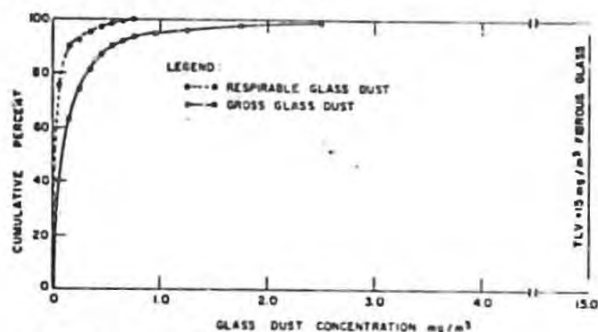


FIGURE 1. Cumulative frequency distribution of glass dust samples by plant.

with the holder open; for the respirable samples, the filter holder was fitted with a 10-mm nylon cyclone described by Lippmann and Harris⁹ as meeting the "respirability" criterion suggested at a 1961 Los Alamos conference sponsored by the U. S. Atomic Energy Commission's Office of Health and Safety. Both personal and general air samples were collected with the 37-mm filters. Personal samples were collected for periods varying from 2 to 7 hours by means of battery-powered diaphragm pumps worn by the workers. Gross airborne and larger-volume "respirable" dust samples were collected with high-volume samplers operating at a rate of 35 cfm. An Aerotec-2 size selector was used prior to the filter for the collection of "respirable" samples.¹⁰

The samples were analyzed by two methods. A portion of each of the Millipore samples was rendered transparent and counted at 430X magnification by the procedure detailed by Edwards and Lynch.¹¹ Fibers, defined as elongated particles with a length-to-width ratio of 3 or greater, were counted in three size ranges: total fibers, fibers longer than 5 μ , and fibers longer than 10 μ . A Porton eyepiece reticle was used to define the counting field and as an aid in the sizing of the fibers. The other portion of the Millipore filters and the high-volume filters were analyzed for total silica by the Talvite method.¹² On the basis of a composition of 50% total silica for fibrous glass, the dust concen-

trations were computed in terms of the amount of glass dust present in the atmosphere.

Results

Means and ranges of concentrations of glass dust are presented in Tables I (gross) and II (respirable) by plant and principal operation. The batch and marble operations occur prior to the forming of the fibers; therefore, an exposure to fibers would not be anticipated. In operations where a fiber exposure would be expected, the airborne concentrations in all five plants were well below the 1969 threshold limit value (TLV) of 15 mg/m³ proposed by the American Conference of Governmental Industrial Hygienists.¹³ Many of the raw materials used in the batch plant have a high percentage of free silica; the hazards from quartz exposure that may exist in some of the plants are generally recognized and will not be discussed in this paper.

Mean gross and respirable dust concentrations are shown in Figure 1. The line at the far right of the graph is the proposed TLV of 15 mg/m³. It can be seen that the concentrations found are well below the proposed TLV.

Table III (gross) and Table IV (respirable) present the means and ranges of fiber concentrations in total fibers, fibers longer than 5 μ , and fibers longer than 10 μ . The

TABLE III
Gross Fiber Concentrations by Plant and Operation
(fibers/cubic centimeter)

Operation	Plant No. ^a	Total fibers		Fibers greater than 5 μ		Fibers greater than 10 μ	
		Mean	Range	Mean	Range	Mean	Range
Batch and Marble	1	—	—	—	—	—	—
	2	1.14	1.14—	0.24	0.24—	0.12	0.12—
	3	0.18	0.18—	0.07	0.07—	0.	0.—
	4	0.20	0.20-0.72	0.05	0.—0.08	0.03	0.—0.05
	1-4 ^b	0.31	0.06-1.14	0.11	0.—0.24	0.03	0.—0.12
Forming	5	16.37	16.37—	0.91	0.91—	0.03	0.05—
	1	0.0	0.0—	0.0	0.0—	0.0	0.0—
	2	0.09	0.04-1.95	0.20	0.—1.01	0.15	0.—0.80
	3	0.30	0.07-0.73	0.11	0.02-0.29	0.08	0.02-0.23
	4	0.33	0.08-0.86	0.17	0.—0.69	0.13	0.—0.67
Spinning, Twisting and Waste Recovery	1-4	0.37	0.04-1.95	0.16	0.—1.01	0.12	0.—0.80
	5	0.20	0.06-0.40	0.04	0.—0.13	0.02	0.—0.07
	5	0.33	0.03-1.26	0.08	0.—0.38	0.03	0.—0.25

(a) Plants 1 through 4 are insulation plants; 5 is a textile plant.

(b) Composite of results for plants 1 through 4.

workers' exposures were evaluated in terms of the airborne fiber concentration to compare the exposure in the fibrous glass manufacturing industry to the exposures found by the same methods in the asbestos processing industry, although there is no evidence that glass fibers produce effects on the lung comparable to those of asbestos dust. One possible reason for this apparent difference would be the absence in the fibrous glass industry of exposure to fibers of a respirable size.

In Figure 2, the fiber concentrations in

fibrous glass plants are compared with those in asbestos textile plants. The bar chart shows that mean concentrations of fibers found in the fibrous glass industry are well below those found in the asbestos textile industry. It should be pointed out that the asbestos textile plants have adopted dust control measures, and there may be little or no disease developing at the dust levels shown. In fact, the average asbestos concentration is less than half the 1969 proposed TLV for asbestos of 12 fibers per cubic centimeter over 5 μ in length.¹² In contrast, the fiber concentra-

TABLE IV
Respirable Fiber Concentrations by Plant and Operation
(fibers/cubic centimeter)

Operation	Plant No. ^a	Total fibers		Fibers greater than 5 μ		Fibers greater than 10 μ	
		Mean	Range	Mean	Range	Mean	Range
Batch and Marble	1	—	—	—	—	—	—
	2	3.64	3.64—	0.97	0.97—	0.54	0.54—
	3	0.66	0.41-1.03	0.16	0.10-0.26	0.08	0.02-0.16
	4	0.30	0.08-0.67	0.10	0.02-0.23	0.04	0.—0.07
	1-4 ^b	1.53	0.08-3.64	0.41	0.02-0.97	0.22	0.—0.54
Forming	5	0.09	0.03—	0.04	0.04—	0.	0.—
	1	—	—	—	—	—	—
	2	0.41	0.04-2.95	0.12	0.—0.56	0.08	0.—0.35
	3	0.15	0.02-0.45	0.04	0.—0.14	0.02	0.—0.09
	4	0.19	0.07-0.31	0.07	0.01-0.19	0.03	0.—0.06
Spinning, Twisting and Waste Recovery	1-4	0.25	0.02-2.95	0.08	0.—0.56	0.04	0.—0.35
	5	0.10	0.—0.19	0.02	0.—0.04	0.01	0.—0.04
	5	0.72	0.03-12.67	0.11	0.—1.97	0.01	0.—0.06

(a) Plants 1 through 4 are insulation plants; 5 is a textile plant.

(b) Composite of results for plants 1 through 4.

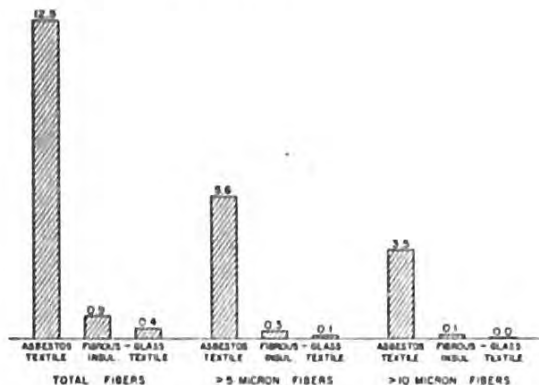


FIGURE 2. Comparison of mean fiber concentrations asbestos textile versus fibrous glass industry (fibers per cubic centimeter).

tions found in the glass fiber industry were one-fortieth of the proposed asbestos TLV. No sample taken in the fibrous glass industry reached the concentration level of the proposed asbestos TLV. Our results confirm the findings of prior investigators that the fibers produced in these fibrous glass plants were too large to result in significant concentrations of respirable fibers.

In the plants surveyed, little or no local exhaust ventilation was being used at most operations. Therefore, it would seem unlikely that airborne fiber and dust concentrations higher than those found in our survey would be encountered unless the present process is changed.

Summary

In this paper the results of environmental studies of four plants manufacturing fibrous glass insulation and one plant manufacturing fibrous glass textile products are presented. The results in terms of airborne concentration of glass fibers and total dust would indicate that the workman's exposure to these materials is negligible. The airborne concentrations found in the fibrous glass industry are contrasted to those found in similar environmental samples taken in a study of the asbestos textile industry. Exposures in

the asbestos textile industry to fibers are of the order of twenty times those in the fibrous glass industry.

Animal experiments and human experience reported in the literature seem to indicate that fibrous glass is a relatively inert material with no fibrogenic or other significant toxic properties.

From the environmental results found in this survey and data from the previous toxicologic and worker studies, there appears to be no reason to class the manufacture of fibrous glass products as a hazardous occupation.

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Poison Control Education

The Education Committee of the American Association of Poison Control Centers has prepared several visual aids that concern themselves with prevention of accidental poisoning. These include:

Slide talk, *The Deceivers*—40 35 mm color slides and suggested commentary covering incidence, hazards, preventive measures and first-aid suggestions. (\$12.50)

25 35 mm captioned color slides. Suitable for use in an automatic projector with its self-contained screen. (\$6.00)

Color filmstrip, *Go Seek and Hide*, with an illustrated script. Designed for elementary school children, it discusses chemical hazards in the home and how accidental poisoning can be prevented. (\$6.50)

Poster Exhibit Set, *In the Blink of an Eye*—Three colorful posters—green, tan and violet—printed on heavy stock. Each poster measures 25" x 35" and is suitable as basic material for eye-catching exhibit. Instructions for making several types of exhibits are included. (\$3.50)

First-Aid for Poisoning Card—A 6" x 9" card of instructions for handling poisoning emergencies. Quantities of 200 or less are free. A reproduction proof suitable for local printing is available free for those who need larger quantities.

Poster, *Syrup of Ipecac*—a reproduction proof suitable for local printing is available free. The poster is 8 1/4" x 11" and shows the picture of a bottle of syrup of ipecac and the legend "Parents, Get Me and Keep Me Handy."

All requests should be made to: American Association of Poison Control Centers, Education Committee, c/o Academy of Medicine of Cleveland, 10525 Carnegie Avenue, Cleveland, Ohio 44106.

June 1970

EROSION OF FIBERS FROM INSTALLED FIBROUS GLASS DUCTS

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ADMINISTRATIVE PURPOSES ONLY.

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ABSTRACT

A field study of installed fibrous glass duct systems which had been in service for some time at six different sites was undertaken as a follow-up of a study of new fibrous glass materials which had been tested in accordance with specifications by the Underwriter's Laboratories, Inc. As was the case with the earlier test, air samples collected at the outlets of the installed duct systems showed no evidence of the erosion of fibers. Dust which had settled on superstructures or which had been removed by wiping walls near the sampling sites generally contained some glass fibers. The presence of glass fibers in these dusts is attributed to dust which had accumulated over several years or more from many possible sources.

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Apprehension has been expressed over the health problem due to the possible erosion of glass fibers from the surface of fibrous glass duct liners in contact with air streams in typical heating and air conditioning ducts and subsequent entrainment of these fibers in the air.^{1,2,3} Concern over the problem has spread to the point where a bill to prohibit installation of asbestos or fiber glass liners in interiors of air duct systems has been introduced in the California Legislature⁴ and some scientists and other interested persons are promising support for, or have supported, similar legislation in other states.^{2,5}

The Underwriter's Laboratories, Inc., in their Standard UL 181 on air ducts,⁶ requires that "material of an air duct shall not break away, flake off, or show evidence of delamination when air is passed through typical sections." Consequently, starting in late 1966 and extending through April, 1967, a laboratory study was conducted in which a typical duct configuration, as specified by Underwriter's Laboratories, Inc.,⁶ was tested to determine the quantities and nature of particulates which could be eroded from a variety of fibrous glass materials. From this study of new duct systems it was concluded that during the test period fibrous duct systems and ducts lined with fibrous glass liners showed no evidence of erosion of fibers.⁷

It was felt that the laboratory study should be complemented by a field study of installed fibrous glass duct systems which had been in service for some time. Therefore, a study was undertaken which, along with the determination of the content of fibrous glass materials present in air issuing from the ducts, included examinations of settled dusts removed from structures and walls in the areas serviced by ventilation duct work. The tests were conducted at six sites selected to determine, if possible, quantitative differences in the potential erosion of glass fibers between installed systems which would be expected to produce the greatest and the least erosion. This paper gives the results of the tests carried out at the six sites during the period of February 18, 1969 to August 21, 1969.

PROCEDURES

Sampling Sites

The six different sites of installed systems are listed in Table 1. The table gives the installation dates, the types of ducts, the test periods, and the locations of the two sampling locations in each system. The downstream location was the air inlet from the duct, and the upstream location was the control, representative of either outside air or air from an inlet which could be expected to have less erosion than the downstream site.

Sampling Methods

The atmospheric dust sampler used to remove particulates from the air stream was similar to that described in U.S. Public Health Service Publication #999 AP-12⁸. The airborne particulates were

collected on 106 mm diameter, 3 micron pore WS Millipore filters during a period of approximately 48 hours at an average rate of about 4 cubic feet per minute.

At each sampling site where an air sampler was set up, a strip of 2" wide 3M vinylchloride adhesive tape was suspended near the opening with the adhesive side exposed to the air stream exiting from the opening. This strip hopefully could be expected to trap fibers present in the air but which were not in the immediate area influenced by the sampler. In addition, dust which had settled on ledges, light fixtures, or other superstructures was collected near each sampling location. Appropriate areas of walls or permanent structures in rooms were also wiped with 4" circles of Whatman #41 filter paper to remove surface deposits of dusts.

Samples to be used for testing for pathogenic bacteria were collected at each sampling station with equipment and the procedures described by the Millipore Corporation in their Technical Bulletin 40?

Analytical Procedures

Total Weight of Dusts

Prior to use, Millipore filters were numbered and weighed after conditioning for 24 hours in the balance room. Each filter was inserted into a separate plastic petri dish. After use, the filters were returned in their plastic dishes with record forms to the laboratory where they were again conditioned in the balance room and weighed to determine the total amount of dust collected during a test.

In the case of settled dust, all or a portion of the dust was weighed and was then analyzed as described below for ash weights and

milligrams of "glass". Weights for dust collected on adhesive tape could not be obtained because of the variable weights of equivalent areas of the unexposed adhesive tape. However, ash weights were obtained by ashing the sample strip and subtracting from the resultant weight the weight of ash of an equal area of unexposed tape. Weights of dust collected on Whatman #41 filter papers were obtained by weighing samples and subtracting the weight of an equivalent number of unexposed filters. The samples were then analyzed as described below.

Ash Weight and Milligrams of "Glass"

A wedge representing 10% of the total filter was cut from each filter with the aid of a special template. The remaining 90% portion was placed in a weighed platinum dish and was ashed in an electric muffle furnace maintained at 500°C. The ashed material remaining in the dish was then treated with hydrofluoric acid. After evaporating to dryness, the dish was again placed in the muffle for a few minutes. Following removal and cooling, the dish was weighed to obtain the loss due to the volatilization of the silica which may have been present in the ash. A chemical analysis was made of glass fibers from a number of sources and it was observed that 50% of total silica was equivalent to 100 percent "glass". Assuming that the loss in weight was due solely to the volatilization of silica present in glass fiber, then twice the loss was taken as the amount of "glass" which could have been present in the dust. A change in weight of 1 milligram or less was considered as indicating the absence of "glass".

Determination of Glass Fibers

The wedge representing 10% of the filter was examined by light field microscopy in order to determine the presence or absence of

glass fibers. A number of 1/4 inch circles were punched from different areas of the wedge and were fastened to a glass slide by means of looseleaf paper reinforcing rings. The slide was then inverted over a beaker containing acetone. On applying gentle heat, the vapors of acetone reacted with the membrane filter to produce a clear transparent film in which the dust particles could easily be seen. Several fields in each circle were examined at magnifications of 200 and 900. Any particle having a cylindrical appearance and length to diameter ratio of 2:1 or greater was considered a fiber. In the case of settled dust and the ash of material obtained on wiping a surface, representative portions were spread over a microscope slide and were examined at a number of magnifications for the presence of glass fibers. Before being ashed, the adhesive tapes were also examined microscopically for the presence or absence of the fibers.

Determination of Pathogenic Bacteria

Upon return to the laboratory the filters were incubated at 36°C for 24 hours. They were then dipped in a solution of crystal violet and the colonies were counted in the usual fashion.

RESULTS

The results of the examination of the material trapped on the filters of the particulate air samplers are given in Table 2. As may be seen, except for a downstream sample in the Suburban Department Store, smaller quantities of dust were usually collected by the downstream sampler than by the upstream sampler. This was most likely due partly to the deposition of some of the particulate matter on the walls of the ducts as the air stream moved through the ducts and

partly to the action of the air cleaners in the systems. The slight increase in the quantity of dust in the downstream air in the case of the samples collected at the Suburban Department Store could have been due to the collection of dust produced as a result of pronounced activity in the busy stock room in which the test was made.

In only one downstream sampler (Suburban Department Store) was the calculated quantity of "glass" greater than was present in the upstream sampler. The source of the silica is unknown but is not believed to have been due to erosion of the ducts since examination of the filter on which the dust was collected indicated the absence of glass fibers. The calculated "glass" values listed in this and other tables should not be considered as indicating the presence of fibrous glass material since any silicious material present--quartz (sand), silicate, etc.,--could also be responsible for the loss observed on treatment with hydrofluoric acid.

Microscopic examination of the adhesive tapes suspended at the duct openings near the air samplers showed that in only one case (Hospital B) were glass fibers present (Table 3). Only a few fibers were seen but their presence could not be verified by chemical means. The few fibers on the adhesive tapes set up at Hospital B may have been due to dust resulting from the general construction work including the installation of glass fiber ducts underway in an adjoining wing. It is also necessary to point out that the room in which the air intake was located had been stripped recently for remodeling and that some concrete cutting had been done in the area.

Examination of settled dusts (Table 4) and wipe samples (Table 5) indicate that glass fibers were generally present in these dusts.

Since these samples represent accumulations of dust generally over several years, it is likely that the fibers seen had their origin during periods in which the fibrous glass ducts were first installed. Settled dusts collected near the downstream samplers were generally lower in silicious material than was the case near the upstream samplers. The loss due to treatment with hydrofluoric acid was much greater than could have been due to the few fibers seen in the dust. The source of silica must have been material other than "glass": for example, sand or sandy soil such as was observed to be present in large amounts in settled dust collected at the Hospital A site in Orlando, Florida.

Tests for pathogenic bacteria in the duct systems indicated that, except for Hospital B, concentrations were within the reported range of 0.25 to 6 colonies per cubic foot of fresh air on the outskirts of a town. The number of colonies in the upstream sample at Hospital B, however, was well below the peak of 65 colonies per cubic foot reported as being present in air sampled in a crowded typists' office!⁶

CONCLUSIONS

The conclusion drawn from these studies is that for the period of testing there was no evidence to indicate erosion of fibers from the ducts. The only definite evidence of the presence of glass fibers was obtained on the analysis of dusts which had settled on structures or which were removed from surfaces by wiping. These dusts may represent accumulations of several years or more from many possible sources.

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LEGENDS

TABLE 1 DESCRIPTION OF SITES AT WHICH EROSION TESTS OF
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CULATES REMOVED FROM AIR STREAMS MOVING IN A
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TERIA IN AIR STREAMS

TABLE 1

DESCRIPTION OF SITES AT WHICH EROSION TESTS OF
FIBROUS GLASS DUCTS WERE MADE

SITE	TEST PERIOD	DATE OF INSTALLATION	TYPES OF DUCT & AIR CLEANER	SAMPLING LOCATION
Suburban Office Building, Market Street, Philadelphia, Pennsylv- ania Upstream Downstream	2/18-21/69	1962	A coated fiber glass duct liner, 3 PCF density Type of air cleaner: Electrostatic Pre- cipitator	Both samples in file room on 5th floor At the air outlet 22 feet upstream from the Downstream sampling site and 140 feet from inlet plenum At the air outlet of the duct system
Hospital A, Orlando, Florida Upstream Downstream	3/24-26/69	1963	Type HD 5 PCF density glass fibers (main) and SD-3 1/4 PCF density branches Type of air cleaner: glass fiber filters	In the maintenance shop between the fresh air inlet and louvers to outside 8 inches below the ceiling outlet in the storage room approximately 50 feet from upstream location

Table 1 (continued)

-2-

SITE	TEST PERIOD	DATE OF INSTALLATION	TYPES OF DUCT & AIR CLEANER	SAMPLING LOCATION
Hospital B, Eustis, Florida Upstream Downstream	3/26-28/69	1953	Type HD 5 PCF density glass fibers (main) and SD-3 1/4 PCF density branches Type of air cleaner: glass fiber filters	On first floor inlet between filters and louvers to outside 8 inches below the ceiling outlet in the classroom about 25 feet from upstream air inlet
Downtown Office Building, North High Street, Columbus, Ohio Upstream Downstream	4/14-16/69		Unlined metal duct (Return duct lined with glass wool insulation. Return duct short circuited during test, used 100% fresh make-air during test) Type of air cleaner: glass fiber filters	Inside of unlined metal duct, 18 inches from the louvers to the outside 6 inches below the ceiling outlet about 50 feet downstream from the air inlet

SITE	TEST PERIOD	DATE OF INSTALLATION	TYPES OF DUCT & AIR CLEANER	SAMPLING LOCATION
Suburban Department Store, West Broad Street, Columbus, Ohio Upstream Downstream	5/5-9/69	1967	A rigid round fiber glass duct - 3 PCF density Type of air cleaner: glass fiber filters	In the outside a plenum between the filter bank and fan #3 In the 1st floor supply room 12 inches below ceiling outlet and about 50 ft from the air inlet fans
Municipal Auditorium, Richmond, Virginia Upstream Downstream	8/19-21/69	1965	Uncoated duct liner This is a duo density product - 1/8 inch skin 4 PCF density backed up with 7/8" thick 1 PCF density Type of air cleaner: glass fiber filters	In the sub-basement 6 inches in front of the outside louvers 4 inches below ceiling outlet on the stage in the basement ballroom

TABLE 2

CONCENTRATIONS AND CHARACTERIZATIONS OF PARTICULATES REMOVED FROM
AIR STREAMS MOVING IN A NUMBER OF VENTILATION SYSTEMS

Site	Test Period	M ³ of Air Sampled	Mg Dust Collected		% Ash in Dust	Mgs Loss Due to Treatment with HF	Mgs Calculated As Glass*	Glass Fibers Identified by Microscopic Examination
			Total	Per M ³				
Suburban Office Building, Market Street, Philadelphia, Pennsylvania	2/18-21/69	Upstream	168	<5.0	<0.03	<1.0	0.0	absent
		Downstream	164	<5.0	<0.03	<1.0	0.0	absent
Hospital A, Orlando, Florida	3/24-26/69	Upstream (outside air)	376	11.3	0.030	<1.0	0.0	present
		Downstream	369	4.3	0.012	<1.0	0.0	absent
Hospital B, Eustis, Florida	3/26-28/69	Upstream (outside air)	390	111.4	0.286	7.4	14.8	absent
		Downstream	390	11.2	0.029	<1.0	0.0	absent
Downtown Office Building, North High Street, Columbus, Ohio	4/14-16/69							

(continued)

Site	Test Period	M ³ of Air Sampled	Mg Dust Collected		% Ash in Dust	Mgs Loss Due to Treatment with HF	Mgs Calculated As Glass*	Glass Identification by Microscopic Examination
			Total	Per M ³				
Downtown Office Building, North High Street Columbus, Ohio (continued) Upstream	4/14- 16/69	386	16.6	0.043	50.0	<1.0	0.0	absent
		380	6.6	0.017	50.0	<.10	0.0.	absent
Suburban Department Store, West Broad Street, Columbus, Ohio	5/5- 9/69	347	11.7	0.034	23.7	<1.0	0.0	absent
		344	32.6	0.095	49.0	4.0	8.0	absent
Municipal Auditorium, Richmond, Virginia	8/19- 21/69	381	21.8	0.057	43.2	3.8	7.6	absent
		379	10.9	0.027	26.4	<1.0	0.0	absent

*Calculation based on assumption that all of the SiO₂ is due to glass fibers and that the fibers contain 50% SiO₂.

TABLE 3

RESULTS OF ANALYSES OF PARTICULATES
COLLECTED ON ADHESIVE TAPES

Location	Test Period	Mgs. of Ash per 24 cm ² of Paper	Mgs. Loss Due to Treatment With HF	HF Loss Calculated as Glass (Mgs.)	Glass Fibers Identified By Microscopic Determination
Suburban Office Building, Market Street, Phila- delphia, Pennsylvania	2/18-21/69		Could not be determined. Control (Gruber's "Sticky Paper [11]") was found to be too high in ash content.		
Upstream		6.8			Absent
Downstream		3.8			Absent
Hospital A, Orlando, Florida	3/24-26/69				
Upstream (outside air)		4.2	<1.0	0.0	Absent
Downstream		1.7	<1.0	0.0	Absent
Hospital B, Eustis, Florida	3/26-28/69				
Upstream (outside air)		3.9	<1.0	0.0	Present
Downstream		4.7	<1.0	0.0	Present

(continued)

Table 3 (continued)

-2-

Location	Test Period	Mgs. of Ash per 24 cm ² of Paper	Mgs. Loss Due to Treatment With HF	HF Loss Calculated as Glass (Mgs.)	Glass Fibers Identified By Microscopic Determination
Downtown Office Building Columbus, Ohio	4/14-16/69				
Upstream		14.8	<1.0	0.0	Absent
Downstream		1.8	<1.0	0.0	Absent
Suburban Department Store West Broad Street Columbus, Ohio	4/14-16/69				
Upstream		<1.0	<1.0	0.0	Absent
Downstream		<1.0	<1.0	0.0	Absent
Municipal Auditorium Richmond, Virginia	4/19-21/69				
Upstream		4.2	<1.0	0.0	Absent
Downstream		1.7	<1.0	0.0	Absent

TABLE 4

CHARACTERIZATION OF PARTICULATES REMOVED FROM EQUIPMENT AND LEDGES
NEAR THE AIRBORNE SAMPLING SITES

Site	Test Period	Mgs. of Dust Analyzed	Mgs. of Ash	Mgs. Loss Due To Treatment With HF	HF Loss Calculated as Glass* (Mgs.)	Glass Fibers Identified By Microscopic Determination
Suburban Office Building Market Street Philadelphia, Pennsylvania	2/18-21/69					
Upstream (from light fixture)		16.2	6.2	1.6	3.2	Present
Downstream (from light fixture)		25.7	9.5	2.0	4.0	Present
Hospital A Orlando, Florida	3/24-26/69					
Upstream (near air in- let duct)		104.5	102.3	83.3	102.3	Present
Downstream (top of duct near air vent)		97.5	77.0	17.9	35.8	Present
Hospital B Eustis, Florida	3/26-28/69	No ledges or superstructures present to provide settled dust				

* Findings not valid. See text.

(continued)

Site	Test Period	Mgs. of Dust Analyzed	Mgs. of Ash	Mgs. Loss Due To Treatment With HF	HF Loss Calculated as Glass* (Mgs.)	Glass Fibers Identified By Microscopic Determination
Downtown Office Building Columbus, Ohio	4/14-16/69					
Upstream (from louvers of the air intake system)		102.7	73.8	15.0	30.0	Absent
Downstream (from light fix- tures below ceiling outlet)		107.1	47.5	11.0	22.0	Present
Suburban Department Store West Broad Street Columbus, Ohio	5/5-9/69					
Upstream (removed from air supply fan #3)		100.0	82.5	24.9	49.8	Present
Downstream (from shelving supports be- neath ceiling outlet)		100.0	27.6	6.9	13.8	Present
Municipal Auditorium Richmond, Virginia	8/19-21/69					
Upstream (from louvers at air intake)		150.0	104.4	41.0	82.0	Absent
Downstream (from wall tile in ballroom)		287.4	141.4	28.7	57.4	Present

TABLE 5

RESULTS OF ANALYSIS OF PARTICULATES WIPED FROM SURFACES
WITH WHATMAN #41 FILTER PAPERS

Location	Test Period	Mgs. of Dust Analyzed	Mgs. of Ash	Mgs. Loss Due To Treatment with HF	HF Loss Calculated as Glass* (Mgs.)	Glass Fibers Identified By Microscopic Determination
Suburban Office Building Market Street Philadelphia, Pennsylvania	2/18-21/69					
Upstream (from top of shelf below ceiling outlet)		7.2	1.6	<1.0	0.0	**
Downstream (from top of shelf below ceiling outlet)		40.6	5.1	<1.0	0.0	**
Hospital A Orlando, Florida	3/24-26/69					
Upstream (top of storage tank near air inlet)		29.6	10.7	1.8	3.6	**
Downstream (from top of locker below ceiling outlet)		719.7	445.1	13.7	27.4	**
Hospital B Eustis, Florida	3/26-28/69	Wipe sample yielded insufficient quantity for analysis				

(continued)

Location	Test Period	Mgs. of Dust Analyzed	Mgs. of Ash	Mgs. Loss Due To Treatment with HF	HF Loss Calculated as Glass* (Mgs.)	Glass Fibers Identified By Microscopic Determination
Downtown Office Building Columbus, Ohio	4/14-16/69					
Upstream (from inside of duct near air inlet)		564.9	412.4	90.1	180.2	**
Downstream (from top of storage cabi- net)		293.3	97.0	21.1	42.2	**
Suburban Department Store West Broad Street Columbus, Ohio	5/5-9/69					
Upstream (from the top of #3 air in- let fan)		27.0	23.2	8.2	16.4	Absent
Downstream (from shelf below ceiling outlet)		127.2	37.0	7.1	14.1	Present
Municipal Auditorium Richmond, Virginia	8/19-21/69					
Upstream (from the louvers in the sub-base- ment)		221.5	139.6	66.1	132.2	Present
Downstream (from the ceiling outlet)		44.5	16.5	5.4	10.8	Absent

*First attempts to examine filters were unrewarding because of interference by paper fibers; later examinations were made on ashed filters.

Findings not valid. See text.

TABLE 6

RESULTS OF TESTS TO DETERMINE PATHOGENIC
BACTERIA IN AIR STREAMS

LOCATION	TEST PERIOD	CUBIC FEET OF AIR SAMPLED	NUMBER OF COLONIES PER FT ³ OF AIR
Suburban Office Building Market Street Philadelphia, Pennsylvania	2/20/69		
Upstream		10	0.5
Downstream		10	0.0
Hospital A Orlando, Florida	3/26/69		
Upstream		11	2.0
Downstream		11	0.3
Hospital B Eustis, Florida	3/28/69		
Upstream		11	26.0
Downstream		11	11.0
Downtown Office Building Columbus, Ohio	5/6/69		
Upstream		11	0.2
Downstream		11	1.0
Suburban Department Store West Broad Street Columbus, Ohio	5/9/69		
Upstream		10	0.0
Downstream		10	0.8
Municipal Auditorium Richmond, Virginia			
Upstream		10	1.2
Downstream		10	1.9

EVALUATION OF THE EFFECT OF AIR VELOCITY
ON FIBROUS GLASS DUCT AND DUCT LINER SYSTEMS



THE KETTERING LABORATORY
in the
Department of Environmental Health
College of Medicine
UNIVERSITY OF CINCINNATI, CINCINNATI, OHIO

E

Material: Sample of particulate matter collected during the Air Erosion Testing on a duct system after 7 years of service at Cape Kennedy*

Submitted: September 11, 1967 by C. D. Smith, Product Testing Laboratories, Building 300, Owens-Corning Fiberglas Corporation, P.O. Box 415, Granville, Ohio

Required: Determination of quantity and nature of material eroded from the duct system

Analysis Number	Sample Number	Test Number + Location	Duration of Test	M ³ of Air Sampled	Mgs Dust Collected		% Ash in Dust	Mg Loss Due to Treatment With HF	Mgs of Glass Found	Glass Fibers Identified by Microscopic Examination
					Total	Per M ³				
7A-5037	O-64	#26 Upstream	136 hrs.	905	16.9	0.019	30.5	0	0	none
7A-5038	O-65	#26 Downstream	136 hrs.	785	7.3	0.009	24.0	0	0	none

*See the attached description sheets

Note: As was the case in earlier tests, the loss due to treatment with HF was insignificant, generally being less than 1 mg., equivalent to 0.001 mgs./M³ of air.

From the Kettering Laboratory in the Department
of Environmental Health, College of Medicine
University of Cincinnati, Cincinnati, Ohio

Reported: 10-30-67

F

KEITTING LABORATORY
COLLEGE OF MEDICINE, UNIVERSITY OF CINCINNATI
EDEN AVENUE, CINCINNATI, OHIO 45219

Material: Samples of particulate matter collected at the grill openings of a duct system at the Plakington House, Milwaukee, Wisconsin

Date of Test: May 6 to May 8, 1968

Submitted: May 10, 1968, by S. R. George, Owens-Corning Technical Center, Granville, Ohio

Required: Determination of quantity and nature of material eroded from the duct system

Analysis Number	Sample Location	Duration of Test	M ³ of Air Sampled	Mg Dust Collected		% Ash in Dust	Mg Loss Due to Treatment with HF	Mg of Glass Found	Glass Fibers Identified by Microscopic Examination
				Total	Per M ³				
8A-2936	Room 709	48.3 hr.	344	8.2	0.024	9.8	0	0	None
8A-2938	Room 845	26 hr.	184	15.6	0.085	41.6	0	0	None

Note: The loss due to treatment with HF was insignificant, generally being less than one mg.

Reported: 5-20-68

EVALUATION OF THE EFFECT OF AIR VELOCITY
ON FIBROUS GLASS DUCT AND DUCT LINER SYSTEMS

By

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Cincinnati, Ohio

September, 1967

Summary

A study of the effect of air velocity on fibrous glass materials in contact with the moving air stream in typical heating and air conditioning ducts was made to determine if fibers were eroded from the fibrous glass materials and entrained in the air stream.

No fibers were found in the air stream exiting from seven separate duct systems constructed from commercially available fibrous glass duct materials exposed to air traveling at 3000 fpm continuously for 136 hours.

No fibers were found in the air stream exiting from two duct systems composed of flexible, wire reinforced, fibrous glass duct materials tested under the same conditions.

In a similar test of 16 different duct lining materials, only one fiber was observed by the microscopic examination of dust removed from the air stream. Since this fiber was the only one observed in over 2000 hours of testing of liner materials, it has little or no significance as a measure of erosion and could have been due to the fabrication of the lined duct at the test site.

It may be concluded that during this test period fibrous duct systems and ducts lined with fibrous glass liner showed no evidence of erosion of fibers.

Purpose

Concern has been expressed over the possible erosion of glass fibers from the surfaces of fibrous glass duct liners or duct materials in contact with the air stream in typical heating and air conditioning ducts and the subsequent entrainment of these fibers in the air stream. Tests were devised to simulate a typical duct configuration incorporating fibrous glass materials and to subject them for an extended period of time to a continuous stream of air moving at a velocity in excess of the velocity for which the materials under test are normally recommended and then to continuously gather a sample of the particles, if any, in the air stream under isokinetic conditions.

Background

Modern air conditioning and heating systems utilizing ducts for the distribution of air are commonly treated for the control of heat and sound. The most common method of obtaining sound absorption in duct systems has been to line the duct with absorbing material. A good sound absorbing material is typically porous so that it can absorb the energy of impinging sound waves. The porous structure presents a number of potential problems of strength and hygiene that must be met before a good sound absorbing material can be considered useful as a duct lining material or a duct material in itself. Other considerations, including fire resistance, are also important. Besides the need for good

acoustic properties, the American Society of Heating, Refrigerating and Air Conditioning Engineers recognizes four requirements for a good sound absorption material for use in ducts:

1. adequate strength to avoid breakage and crumbling;
2. fire resistance and compliance with building code requirements;
3. particles should not fray off at the higher design velocities;
4. freedom from odor when either dry or wet. (1)

Fibrous glass duct materials have been used for sound absorption within ducts since 1946. The blanket or board is cemented or attached mechanically to the inside surface of the duct. The amount of lining required can be calculated on the basis of the sound energy and the frequency that needs to be absorbed.

In 1960 the basic fibrous glass duct lining material was developed into a form in which it could serve as the duct itself rather than as just the liner. The major development involved an increase in the density of the material. In addition, a facing strong enough to allow grooving and folding of the fibrous material and subsequent forming into the air duct was added to the outside surface. This outside surface facing also provides a vapor barrier, giving the fibrous glass duct material all the essential characteristics necessary for the control of heat and noise.

Tests have been developed by the Underwriters' Laboratories (UL) to describe a series of requirements which fibrous glass material should meet in order to be acceptable for

use as a duct material. These requirements are included in Standard UL 181 of the Underwriters' Laboratories. (2)

Besides measuring fire resistance characteristics, the UL tests also evaluate the strength characteristics of the duct materials, the resistance to mildew and mold attack and other aspects that an adequate material should exhibit.

UL 181 also requires that the susceptibility of fibrous glass duct materials to air stream erosion be measured. The tests are made on a duct assembly following a simulated aging process and are conducted at a velocity two and one-half times the rated velocity of the duct system material for four hours. The air is discharged into a collection chamber fitted on one side with a baffle and on the other with a 200 USS standard screen over the outlet. The standard requires that no macroscopic particles of duct material be found in the chamber or on the screen following the four-hour exposure nor can the interior duct surfaces show evidence of cracking, flaking or delamination.

The question of how these materials would perform under a velocity closer to the rated velocity of the materials and for an extended period of time was considered of interest and the National Insulation Manufacturers Association asked the Kettering Laboratory to conduct the investigation in late 1966. The testing program was completed in April, 1967.

Test Procedures

The test was designed to subject to a controlled stream of air either actual sections of fibrous glass duct material formed

into the elements normally found in duct systems or duct sections of the same size and configuration lined with the fibrous glass lining material. The exposure was continued for a reasonable time during which period sampling for particulates entrained in the air stream was continuous. At the conclusion of the exposure period the sampling filter was examined for the presence of fibrous glass particles.

A test velocity of 3000 fpm was selected. Most of the fibrous glass duct materials are not recommended for use with air velocities over 2400 fpm and the 25% higher test condition duplicates satisfactorily the maximum condition that might occur in a properly designed and installed conventional air distribution system. The recommended air velocity for main ducts in conventional air systems varies from 1200 to 1800 fpm with a maximum recommended velocity of 1600 fpm for ducts in other than industrial buildings and only 2200 fpm in industrial buildings. (3)

A period of 136 hours was selected for each test. Outdoor air was used continuously, no attempt being made to control the air temperature or humidity. Tests were conducted in Granville, Ohio, during the months of July, 1966, to March, 1967. There was no evidence that the air temperature or humidity had any effect on results of the tests.

Test Conditions

To provide a standard test situation, the test set-up was designed along the lines of apparatus used by the Underwriters' Laboratories in its evaluation of air ducts according to UL 181.

The set-up, illustrated in Figures 1, 2, 3, and 4 provided:

1. a mechanism for filtering a source of air to eliminate as many foreign particles as possible;
2. a method of measuring the particle content of the filtered air stream to provide a baseline for subsequent measurements after the air passes through the test section;
3. a fan to accelerate the air stream to the desired velocity;
4. a method for eliminating the turbulence caused by the fan to provide nearly uniform air entry to the test section;
5. a test section containing elements typical of a normal duct system; and
6. a method for measuring whether any fiber particles have been entrained in the air stream passing through the test section.

Test Set-up

In the test set-up used, shown in Figures 1, 2, and 3, the air was drawn through a filter box, the filters being protected from the elements by a containing box with a louvered air entrance. Two feet downstream from the filters, the air passed through a flow nozzle which gave a reasonably flat velocity profile. The upstream sampling device which provided the baseline measure of particles in the entering air stream was located just ahead of the transition connecting the flow nozzle and the blower. A damper assembly on the lower intake provided flow rate control.

On the discharge side of the blower, air was passed through a straightening section two feet long, constructed in an egg crate configuration with 2 inch square openings. The straightener was located in a duct 12 inches x 12 inches x 8 feet which ended in a transition section over which the entrance side of the test section was slipped. Test sections either 11 inches x 11 inches square or 12 inches in diameter could be attached to the transition section. At the end of the test duct, another metal duct of the same size was slipped inside the discharge end of the test section to hold the test probe for the downstream sampling.

The test section consisted of a straight run connected to a three piece 90-degree elbow (to provide a typical duct turbulence condition) followed by a second straight run of the test material. After the test assembly was attached to the blower, the ducts were blown out for a period of one hour at the test velocity to clear out any construction fragments. Average velocity was measured by a Hastings Precision Air Meter, using an 11 point traverse for square ducts and a 9 point traverse on round ducts.

Samples were collected through the use of an isokinetic flow nozzle of approximately 1/2 inch diameter located in the center of the air stream. The sampling nozzle attached to a funnel arrangement was connected to a filter holder fitted with a 3 micron pore size, 104 mm diameter, Type WS Millipore^(R) filter. The filter holder was connected to a suction pump to provide the isokinetic velocity condition. Figure 4 shows the sample collection mechanism.

The Materials Tested

Rigid Duct Systems. Fibrous glass duct systems are made with materials that combine a thickness of glass fibers and phenolic binder with either a metal foil or vinyl film vapor barrier on the exterior surface. The test materials vary in density. The smaller diameter round duct and the standard duty flat board shape have a density of 3 pounds per cubic foot (pcf); the larger diameter round duct has a density of 4.5 pcf and heavy duty board has a density of 5 pcf.

Flexible Duct Systems. Flexible fibrous glass duct uses a pliable form of the fibrous glass and phenolic binder surrounding a spiral wire reinforcement on the inner surface. A plastic film vapor barrier covers the outside.

Duct Liner. Fibrous glass duct liner comes in batt or blanket form. It is similar in formation to the duct board material, generally consisting of materials having a density of 1.5 pcf or 3.0 pcf but without the exterior vapor barrier surface. In some cases the inner surface has a thin sprayed film or coating of neoprene to provide a lower coefficient of friction.

Forms

Seven different samples of rigid duct board material, supplied by three manufacturers were tested. The rigid samples are described in Table 1. Two different forms of flexible fibrous glass duct having a nominal thickness of one inch were tested. Table 2 gives their characteristics. In Table 3 are listed the 16 different forms of fibrous glass duct that were tested. These were supplied by four manufacturers.

Fabrication of Test Samples

All fabrication of fibrous glass ducts and lined sheet metal ducts was according to manufacturer recommendations. Fabrication of the test sections was by skilled workers employed by an experienced sheet metal contractor.

Rigid Rectangular Duct. All rigid ducts formed from fibrous glass duct system boards were made by V-grooving the inner face of the board as dictated by the dimension of the final duct, folding along the grooves that formed the first three corners and then stapling the flap that forms the fourth corner. The seam along the flap was made either with mastic and tape or heat-seal tape as recommended by the supplier. Duct sections were each four feet long and the two 8-foot straight runs in the test configuration were made by joining together two sections. Joints were grooved according to instructions, with the male end placed on the upstream side and stapled and taped according to directions. The elbow was formed in three sections, laid out in accordance with recommendations, grooved, folded, and assembled.

In a typical duct section there were four cut edges (along each corner) and a single cut edge around the perimeter at each joint. Normally the folding operation covers the raw edges exposed by the grooving, but the test conditions duplicated the amount of cut edge that good workmanship would normally expose within the duct. Figure 5 shows the typical configuration used and the relative location of the cut edges present in the configurations.

No cutting or grooving is required to form the rigid round duct as it is supplied in 6-foot seamless lengths. With rigid round ducts two joint systems are used. To form joints in ducts made with square ends, a sheet metal sleeve is inserted inside the butted ends and the joint is taped.

To form joints in ducts made with molded ends, the male end is slipped within the female and the joint is taped. The elbow required was made from three segments of the duct held together by small sheet metal clips, then taped.

Flexible Round Duct. There was no cutting required with the flexible duct samples. They are also made with male and female ends and joints are made by slipping one within the other, stapling the overlapping outer face and taping. Three six foot lengths were used to form the test section, two straight and the third for the 90 degree turn.

Duct Liner. Duct liners were cut and affixed according to normal practices. Cutting is usually done with a knife along a straight edge, though folds are used in corners where appropriate, with or without grooving as the thickness requires. Liners were attached with adhesive or mechanical fasteners according to the usual recommendations. The lined ducts tested had one less seam than the rigid rectangular fibrous glass duct configuration as the liner is in eight foot lengths and did not have to be joined to form the straight run sections.

Analytical Procedures

Total Weight of Dust

Prior to use, Millipore filters were numbered and weighed after conditioning for 24 hours in the balance room. Each filter was inserted into a separate plastic box and the lot was shipped to the test site. After use, the filters were returned in their plastic boxes with record forms to the Kettering Laboratory where they were again conditioned in the balance room and weighed to determine the total amount of dust collected during a test.

Ash Weight and Mg. of Glass

A wedge representing 10% of the total filter was cut from each filter with the aid of a special template. The 90% portion of the filter web was then placed in a weighed platinum dish and was ashed in an electric muffle furnace maintained at 500°C. The material remaining represented the ash in the dust. The contents of the dish were then treated with hydrofluoric acid, and after evaporating to dryness the dish was again placed in the muffle furnace for a few minutes. Following removal and cooling, the dish was weighed. The anticipated loss in weight was expected to be due to volatilization of silica, which constituted about 50% of the glass fibers as was determined from analysis of products produced by one plant. In no test was the weight loss greater than the error in weighing (± 0.5 to ± 1 mg.) and, therefore, all findings listed as milligrams of glass and HF loss are reported as zero. This assumption appeared to be justified on the basis of the absence of visible glass fibers and the

failure of dust removed from the downstream air to show ash values greater than those shown by dust removed from the upstream air.

Determination of Glass Fibers

The wedge representing 10% of the filter was examined by light field microscopy in order to determine the presence or absence of glass fibers. A number of representative 1/4-inch circles were punched from different areas of the wedge and were fastened to a glass slide by means of loose-leaf paper reinforcing rings. The slide was then inverted over a beaker containing acetone. The vapors of acetone evolved on applying gentle heat, reacted with the membrane filter to produce a clear transparent film in which the dust particles were plainly visible. Several fields in each punched circle were examined under magnifications of 200 and 900. Any particle having a cylindrical appearance and length to diameter ratio of 2:1 or greater was considered a fiber.

Results

Results of the tests are shown in Table 4. As may be seen, smaller total quantities of dust were generally collected at the downstream sampling point than was the case at the upstream sampling point. This was most likely due to the deposition of some of the particulate matter present in the entrance air on the walls of the ducts as the air stream moved through the duct.

In general, there were no significant differences between the ash contents of the dusts collected at the entrances and those of dusts collected at the exits of the test ducts. Significant increases in the ash content of the dusts collected at the downstream sampling station would have been due to glass fibers eroded from the ducts. That this did not occur is further confirmed by the failure of the chemical treatment to indicate increases in the silicious material in the dusts collected downstream over that in dusts collected upstream.

While no fibrous duct particles were observed by the naked eye in any of the 25 tests, one fibrous glass particle was detected microscopically in dust collected in testing one of the 16 liner materials. This fiber was the only one observed in dusts collected downstream in over 2000 hours of testing of liner material. Its presence cannot be taken as proof of erosion since it could also have resulted from fabrication of the duct at the test site.

Conclusions

It may be concluded that fibrous glass duct systems and ducts lined with fibrous glass liner showed no evidence of erosion of fibers during this test period when exposed for a continuous period of 136 hours to an air stream moving at a velocity of up to 3000 feet per minute, a velocity 25% higher than the recommended operating velocity for the material under test.

References

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Table 1

CHARACTERISTICS OF RIGID DUCT SYSTEMS TESTED

Rectangular Board

<u>Sample</u>	<u>Thickness (inches)</u>	<u>Density (pcf)</u>	<u>Coating</u>
A(#1)	1	3.25	No
B(#2)	1	5.00	No
C(#22)	1	3.50	No
D(#23)	1.5	5.00	No
E(#8)	1	3.00	No

Round Duct

<u>Sample</u>	<u>Size</u>	<u>Coating</u>
F(#24)	12" diameter	No
G(#3)	12" diameter	No

(Numbers in parentheses refer to order in test program.)

Table 2

CHARACTERISTICS OF FLEXIBLE ROUND DUCT SYSTEMS TESTED

<u>Sample</u>	<u>Size</u>	<u>Coating</u>
H(#4)	12" diameter	No
I(#25)	12" diameter	No

Table 3CHARACTERISTICS OF FIBROUS GLASS LINERS TESTED

<u>Sample</u>	<u>Thickness (inches)</u>	<u>Density (pcf)</u>	<u>Coating</u>
J(#6)	0.5	1.5	Yes
K(#5)	1	1.5	Yes
L(#7)	1	3.0	Yes
M(#10)	0.5	1.5	Yes
N(#9)	1	1.5	Yes
O(#11)	1	Dual	Yes
P(#13)	1	1.5	Yes (joint coat)
Q(#14)	1	1.5	Yes
R(#12)	1	3.0	Yes
S(#17)	0.5	1.5	Yes
T(#18)	1	1.5	Yes
U(#20)	0.5	1.5	Yes
V(#19)	1	1.5	Yes (joint coat)
W(#16)	0.5	3.0	Yes
X(#21)	1	3.0	Yes
Y(#15)	1	Dual	No

(Joint coating refers to practice of covering butt joints with a "battered" coating of mastic; liners were tested with and without the joint coating.)

Table 4

RESULTS OBTAINED IN THE EROSION TESTING OF FIBROUS GLASS AIR DUCTS

<u>Sample and Location</u>	<u>M³ of Air Sampled</u>	<u>Mg Dust Collected</u>		<u>% Ash in Dust</u>	<u>Mg Loss Due to Treatment with HF</u>	<u>Mg of Glass Found</u>	<u>Glass Fibers Identified by Microscopic Examination</u>
		<u>Total</u>	<u>Per M³</u>				
<u>Fibrous Glass Duct Systems</u>							
<u>Rigid Rectangular</u>							
A							
Upstream	845	39.0	0.046	10.0	0	0	None
Downstream	880	39.5	0.045	8.9	0	0	None
B							
Upstream	940	22.5	0.024	10.7	0	0	None
Downstream	910	19.4	0.021	10.9	0	0	None
C							
Upstream	900	20.6	0.023	17.0	0	0	None
Downstream	775	16.3	0.021	15.9	0	0	None
D							
Upstream	890	28.8	0.032	18.0	0	0	None
Downstream	765	20.9	0.027	17.7	0	0	None
E							
Upstream	900	38.3	0.043	30.6	0	0	None
Downstream	810	26.6	0.033	29.0	0	0	None

Table 4 continued

-2-

<u>Sample and Location</u>	<u>M³ of Air Sampled</u>	<u>Mg Dust Collected</u>		<u>% Ash in Dust</u>	<u>Mg Loss Due to Treatment with HF</u>	<u>Mg of Glass Found</u>	<u>Glass Fibers Identified by Microscopic Examination</u>
		<u>Total</u>	<u>Per M³</u>				
<u>Fibrous Glass Duct Systems</u>							
<u>Rigid Round</u>							
F							
Upstream	925	19.0	0.021	19.0	0	0	None
Downstream	780	15.9	0.020	23.3	0	0	None
G							
Upstream	945	25.7	0.027	30.7	0	0	None
Downstream	905	22.6	0.025	29.0	0	0	None
<u>Flexible Round</u>							
H							
Upstream	925	23.7	0.025	16.0	0	0	None
Downstream	880	17.9	0.020	16.8	0	0	None
I							
Upstream	900	19.5	0.022	21.5	0	0	None
Downstream	775	15.5	0.020	22.6	0	0	None

Table 4 continued

-3-

<u>Sample and Location</u>	<u>M³ of Air Sampled</u>	<u>Mg Dust Collected</u>		<u>% Ash in Dust</u>	<u>Mg Loss Due to Treatment with HF</u>	<u>Mg of Glass Found</u>	<u>Glass Fibers Identified by Microscopic Examination</u>
		<u>Total</u>	<u>Per M³</u>				
<u>Metallic Duct, Fibrous Glass Lined</u>							
J							
Upstream	960	25.0	0.026	22.0	0	0	None
Downstream	865	18.9	0.022	21.0	0	0	None
K							
Upstream	945	35.3	0.037	18.4	0	0	None
Downstream	865	27.9	0.032	22.2	0	0	None
L							
Upstream	900	37.5	0.042	17.0	0	0	None
Downstream	790	27.0	0.034	14.4	0	0	None
M							
Upstream	920	21.2	0.023	17.9	0	0	None
Downstream	830	17.8	0.021	17.9	0	0	None
N							
Upstream	925	22.7	0.025	20.7	0	0	None
Downstream	790	21.7	0.028	29.5	0	0	None
O							
Upstream	960	19.9	0.021	16.6	0	0	None
Downstream	800	13.8	0.017	13.0	0	0	None
P							
Upstream	960	24.1	0.025	18.6	0	0	None
Downstream	815	17.9	0.022	19.5	0	0	None

Table 4 continued

-4-

Sample and Location	M ³ of Air Sampled	Mg Dust Collected		% Ash in Dust	Mg Loss Due to Treatment with HF	Mg of Glass Found	Glass Fibers Identified by Microscopic Examination
		Total	Per M ³				
<u>Metallic Duct, Fibrous Glass Lined</u>							
Q							
Upstream	935	25.5	0.027	20.0	0	0	None
Downstream	800	18.4	0.023	25.0	0	0	None
R							
Upstream	960	25.1	0.026	13.1	0	0	None
Downstream	790	17.6	0.022	13.4	0	0	None
S							
Upstream	970	17.3	0.018	20.2	0	0	None
Downstream	795	13.0	0.016	19.4	0	0	1 fiber 30μ long + 3μ in diameter
T							
Upstream	925	32.5	0.035	10.8	0	0	None
Downstream	810	14.1	0.017	14.9	0	0	None
U							
Upstream	890	30.1	0.034	7.1	0	0	None
Downstream	780	23.1	0.030	8.3	0	0	None
V							
Upstream	890	20.6	0.023	13.0	0	0	None
Downstream	775	20.0	0.020	7.5	0	0	None

Table 4 continued

<u>Sample and Location</u>	<u>M³ of Air Sampled</u>	<u>Mg Dust Collected</u>		<u>% Ash in Dust</u>	<u>Mg Loss Due to Treatment with HF</u>	<u>Mg of Glass Found</u>	<u>Glass Fibers Identified by Microscopic Examination</u>
		<u>Total</u>	<u>Per M³</u>				
		<u>Metallic Duct, Fibrous Glass Lined</u>					
W							
Upstream	980	17.7	0.018	13.0	0	0	None
Downstream	806	13.6	0.017	8.8	0	0	None
X							
Upstream	830	34.6	0.042	14.2	0	0	None
Downstream	765	28.3	0.037	12.7	0	0	None
Y							
Upstream	935	21.0	0.023	18.1	0	0	None
Downstream	795	16.0	0.020	12.5	0	0	None

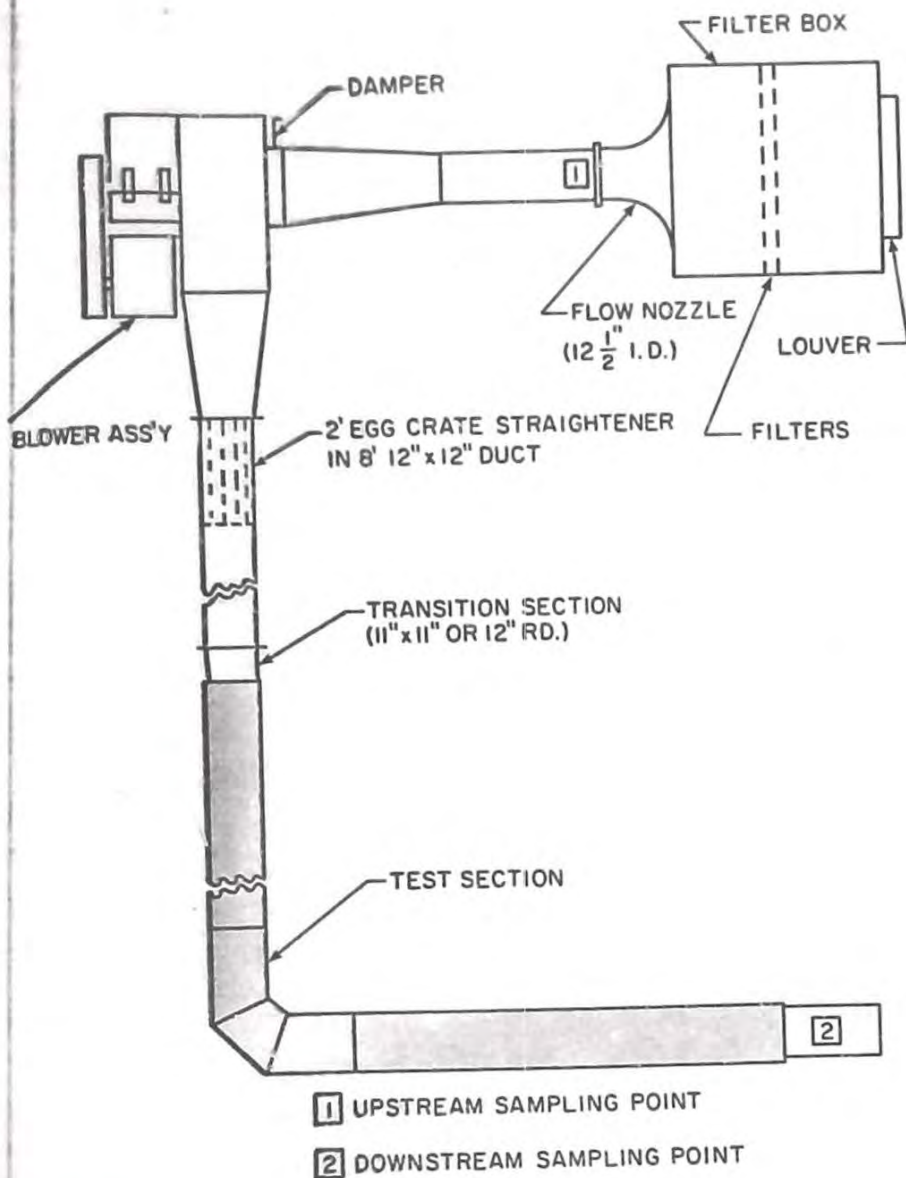


Figure 1. Plan of NIMA Test Set-up Showing Location of Sampling Points

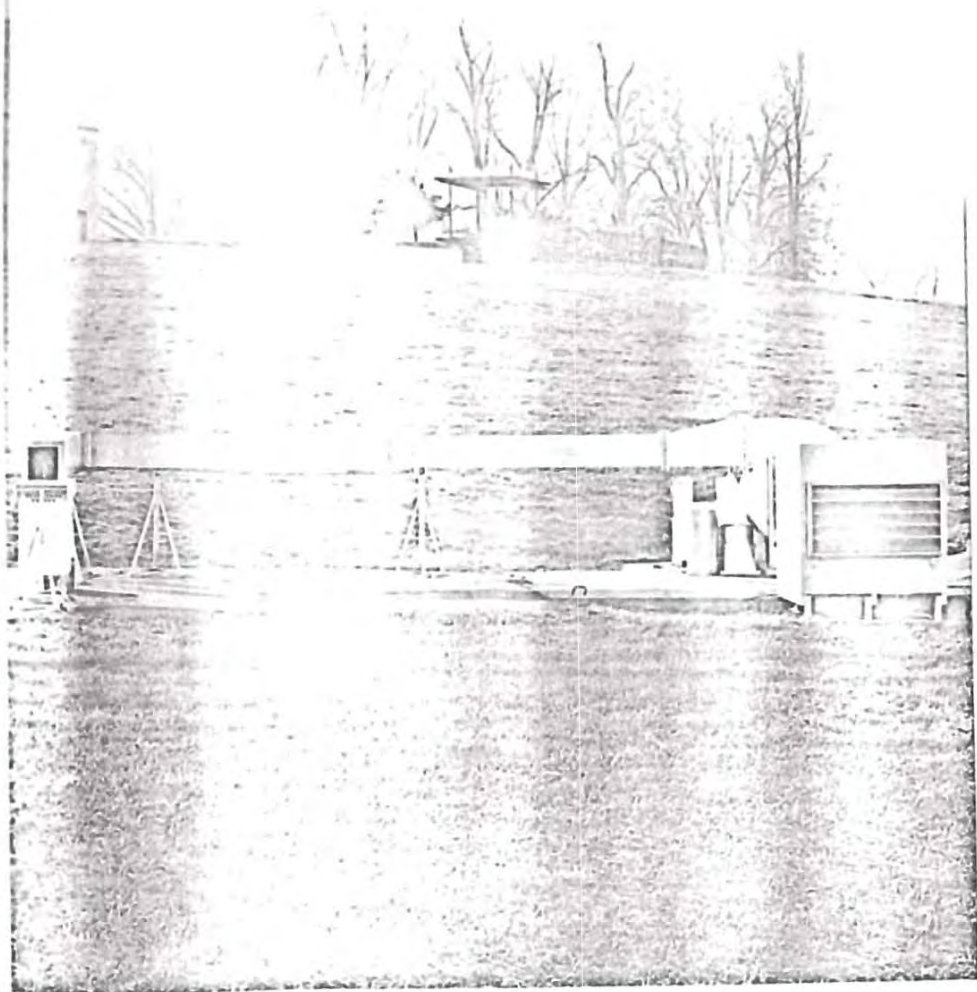


Figure 2. Photograph of Test Set-up in
Operation in Granville, Ohio

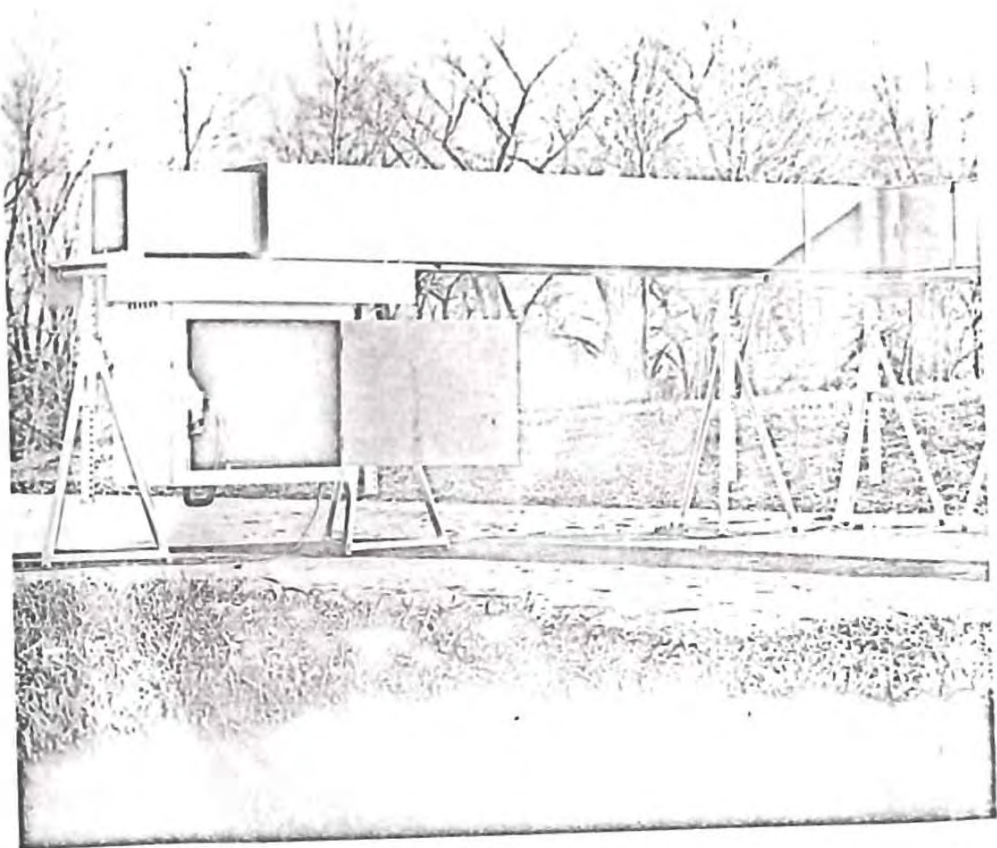


Figure 3. Photograph Showing Downstream Sampling Location and Pump Attached to Iso-kinetic Sampling Nozzle

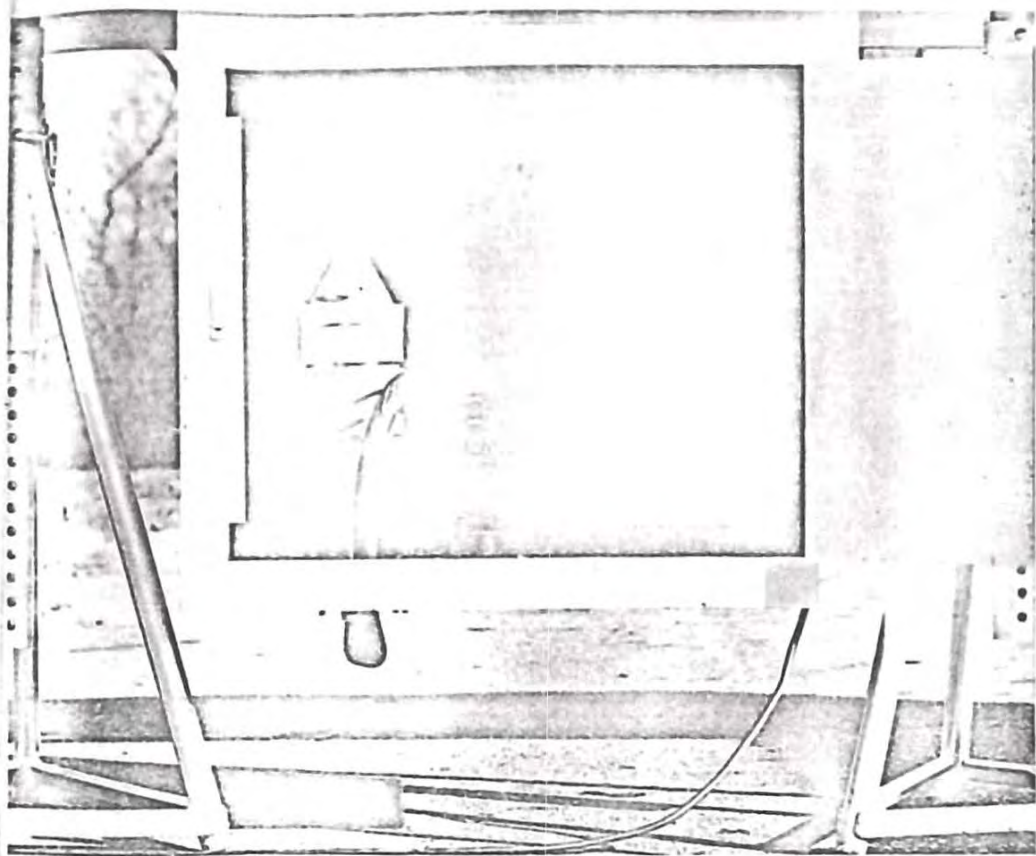


Figure 4. Close-up of Pump and Filter Housing

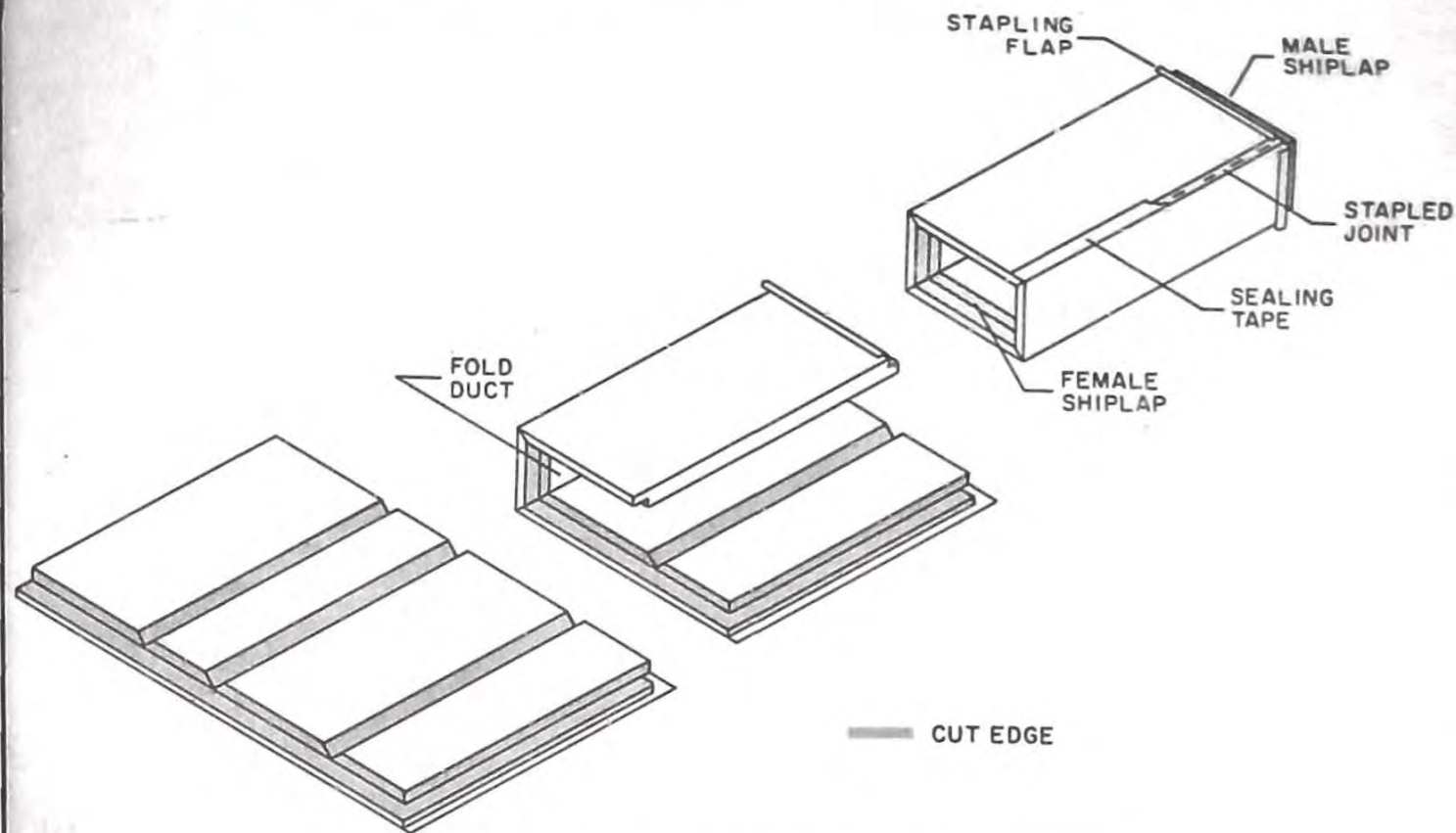


Figure 5. Steps in the Construction of Rigid Rectangular Duct from Fibrous Glass Duct Board

7

FIBROUS GLASS LINED AIR TRANSMISSION SYSTEMS:
AN ASSESSMENT OF THEIR ENVIRONMENTAL EFFECTS

REPORT TO HEALTH AND SAFETY COMMITTEE
NATIONAL INSULATION MANUFACTURERS ASSOCIATION

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June 1, 1970

FIBROUS GLASS LINED AIR TRANSMISSION SYSTEMS:
AN ASSESSMENT OF THEIR ENVIRONMENTAL EFFECTS

W. Clark Cooper, M.D., J. LeRoy Balzer and Douglas P. Fowler

This study was undertaken as part of a continuing project which is aimed at determining the exposures to airborne fibrous materials of certain occupational groups, and of the general public. The stated objective of this part of our study was "...to obtain quantitative estimates of the amounts of fibrous glass introduced into living and working areas from ventilation or heating ducts lined with fibrous glass."

To attain this objective, we sought the most direct method available for making such estimates. Measurement of the concentrations of glass fibers in air before and after passage of that air through fibrous glass lined air transmission systems was selected.

SAMPLING METHOD

All air samples were taken by drawing air through Millipore type AA (0.8 μ m mean pore size) organic membrane filters at flow rates between 0.1 and 2.0 cubic feet per minute. Samples taken from moving air streams were taken "iso-kinetically"; air velocities at the faces of the sample filter holders matched (within $\pm 25\%$) the velocities of the air streams from which samples were taken. This was accomplished by selection of filter holders with suitable size openings for the general range of the velocity of interest and then adjusting pump flow rate to bring the face velocity within tolerance.

ANALYTICAL METHOD

Analysis of samples was by the standard method developed by Lynch et al. of the United States Public Health Service.¹ A randomly selected portion of the exposed filter was mounted and "cleared" by addition of a mounting medium matching the refractive index of the filter medium; 100 randomly selected microscopic fields (area of each field = $(78 \mu)^2$)² were examined under phase contrast microscopy at 430X magnification. The microscopic field was defined by a Porton eyepiece reticle, which was also used to determine fiber size (after calibration against a stage micrometer). From this examination we determined the number of fibers per unit area of filter, the sizes of those fibers, and made a preliminary identification. This preliminary identification was based on the morphology of the fibers seen. The presence or absence of biological cellular structure; the presence or absence of synthetic resin coating; the appearance of the broken ends of the fibers; variation in fiber diameter in individual fibers; and the gross morphology (curved vs. "rod-like"; bundled vs. individual etc.) were all found to be significant identifiers of certain types of fibers. Each sample (and each fiber previously counted within those samples) was further examined using petrographic microscopy, for more complete identification of fibers.

From the combination of microscopic methods six categories of fiber types were developed:

1. Amorphous synthetic mineral (fibrous glass)
2. Crystalline natural mineral (asbestiform minerals)
3. Other mineral fibers (includes amorphous fibers not identifiable as fibrous glass, fibers smaller than the limit of resolution for positive identification but believed to be mineral on the basis of morphology and size, and other mineral fibers such as the naturally occurring platy-serpentine.)
4. Biological fibers (animal and insect parts, plant fibers, yeasts, and molds.)
5. Synthetic organic fibers (Nylon, Rayon, Orlon, et al.)
6. Unidentified fibers.

Characterization of fibers was based primarily on our examination of samples of fibrous materials, with some reliance on descriptive studies from the scientific literature.² Our samples were obtained either from manufacturers of the material in question or from samples of fibrous glass liners at installation sites.

SENSITIVITY AND RELIABILITY OF THE METHOD

It is essential to consider the sensitivity (or lower limit of detection) and reliability (reproducibility and accuracy) of the method, since we must define the limits within which our quantitative descriptions are valid. These will be dependent on inherent limitations of the method, as well as the care and skill with which the method is applied.

The sensitivity of the method of sampling and analysis is primarily dependent on the limits of resolution of the microscopic examination of the filters. Our obvious lower limit of detection is one fiber, in the total area of the filter examined. The smallest fiber which can be seen (and identified as fibrous) in the phase contrast microscope has a diameter of 0.5 μ m. The smallest fiber which will give an identifiable pattern in our petrographic examination has a diameter of about 1.6 μ m. Since it is critical to determine the crystallinity of fibers to differentiate between glass and other mineral fibers, our lower limit of detection is one glass fiber [with a diameter of 1.6 μ m, or greater, and a length (from the definition of "fibrosity"³) of 4.8 μ m,] per 7800 μ m² of filter.

It is more appropriate, however, to convert our "absolute" sensitivity (of one fiber) to a volumetric sensitivity, expressed as fibers per liter. To calculate sensitivity on this basis, we divide our limit of detection (one fiber) by the fraction of the filter taken for analysis (approx. 1/1500), divide the result (approximately 1500) by the sample air volume in liters. As an example, consider a sample taken at a flow rate of one cubic foot of air per minute (28.3 liters per minute) for 16 hours. The total volume sampled is 27, 168 liters, and the sensitivity is $1500/27, 168 = 0.055$ fibers per liter.

It is theoretically possible to increase sensitivity by several methods. Perhaps the most apparent of these is examination of a greater proportion of the filter. This is not a practically valuable method, since it rapidly becomes burdensome in terms of time for microscopy. We estimate that the

time required to accurately count, size, and identify the fibers found in 100 microscopic fields is approximately one hour. If, for the sake of completeness of analysis, we wished to examine the total area of a filter, this would require approximately 28 man-weeks for microscopy alone, without consideration of the time required for analysis of data. We have found that 100 fields is the optimum for our examination, primarily because of the eye-strain involved in this exacting analysis. Further, we feel that it is more productive to examine additional samples of air taken at different times rather than attempting to extrapolate from a few intensively examined samples.

Another apparent method for increasing our volumetric sensitivity would be the taking of larger air samples; this was done for several samples (and is reflected in the lower limits of detection for several of the systems in Table I). In these samples, (some of which were taken for as long as 80 hours) it was noted that there was a definite trend toward "loss" of fibers. This was due to the large quantities of miscellaneous particulate material, which tended to cover and obscure the fibers, making microscopic examination very nearly useless.

Although our sensitivity is an important factor to be considered in data evaluation, perhaps of more importance is the reliability of the method. We do not attempt to make any numerical definition of our accuracy; determination of this would require extensive preparation, dispersion, and sampling of known concentrations of fibrous glass aerosols in the range of concentrations reported here, and the present state of aerosol technology has not

advanced to the point where this is possible. The other major component of reliability is reproducibility the degree with which successive measurements of the same (or purportedly similar) quantities are in agreement.

In our case we wish to know how closely we are able to describe the variation in concentrations estimated from the same air. There are several points at which sampling variation can have an effect on our results. In the progression of events which culminate in the counting of fibers on a prepared filter mounted on a microscope slide, the first point at which variation might be expected is the air sampling. We are unable to give a quantitative description of the reproducibility of our results at this point; sequential samples are not representative of the same body of air, sampling from side-by-side filter holders introduces disturbance into the flows of air into the filters, and (as previously stated) we are unable to generate known aerosols (or uniform aerosols) in which this variation could be measured.

However we can test the reproducibility of rereading the same mounted sample and the variation introduced by taking several different mounts of the same filter. The coefficient of variation for rereading the same mounted sample was 6.0%. Several portions of the same filter were mounted and counted on several occasions; the differences ranged from 0 to over 200%. It is our feeling that this is the most important determinant of sampling variation in our method.

SELECTION OF SAMPLING SITES

One of the most challenging portions of the study was the selection of sites for sampling. The obvious aim was to select air transmission systems which would be representative of all such systems. Early in the project it was realized that this was an impossibility; the variety of types and uses of fibrous glass products in air ducts precluded a complete survey. Further, it was found to be difficult to describe the systems which were seen. There were discrepancies between the extent of fibrous glass lining described in plans and that which was seen in the systems; operating engineers (in charge of the buildings surveyed) frequently were found to have an inaccurate idea of the locations and extent of such liners; and the systems were frequently difficult to inspect, making an on-site evaluation difficult. Nevertheless, we do not feel that our study is invalid because of these factors. We selected, from 30 building systems which were surveyed (either by a review of the plans, or physical inspection), 13 systems from which air samples were taken. In these 13 systems, we were certain (from physical inspection) of the presence of fibrous glass in at least one portion of the systems; samples were taken of the air after it had passed through these portions. [The assumption was made that any effect of fibrous glass lining on the quality of the air coming through the system would be noted after passage of the air through the section of the system which was known to be lined].

RESULTS

Data from the 13 systems are summarized in Table I. Because of constraints on available sampling sites, it was not possible to obtain samples from equivalent sites at each of the systems. Because it was most convenient to take air sampling in three buildings on the University of California's Berkeley Campus, several hundred samples were taken in these, listed as Systems 1, 2 and 3. Systems 4 through 13, where the number of samples was less, are grouped together. Individual sample results for these sites are reported in the Appendix.

Although there were wide variations in concentrations of glass fibers in outside air, within ducts, and within buildings, air within ducts showed consistently lower concentrations than outside air. Figures 1 and 2 illustrate the relationships observed.

Although there were far too few fibers in the accumulated air samples from any one system to draw a statistically valid conclusion regarding the size distribution of glass fibers, the general ranges were 1.6 micrometers to 35.3 micrometers in diameter. An estimated geometric mean was 4.5 micrometers.

DISCUSSION

In the 13 systems studied, there did not appear to be any measurable increase in air-borne fibrous glass due to the passage of the air stream over fibrous glass duct linings. If there was erosion, it was offset by

deposition and entrapment of fibers. In nearly all instances, air within ducts had fewer glass fibers than either outside air or air immediately after passage through inlet filters. The decrease was both in concentrations of air-borne glass fibers and in the proportion of total fibers identifiable as glass. Over 95% of matched individual air samples (taken the same day in the same building) showed such a relationship.

Statistical comparison of the sizes of the fibers which went into and came out of duct systems showed differences for "other fibers" as well as glass fibers. The concentrations and characteristics of the total fiber "populations" in air passing through the systems studied were changed, but the causes of these changes could not be determined with the limited data available.

To place the concentrations of fibers which were observed in proper perspective, we would like to emphasize that the numbers were quite small. In spite of the relatively large volumes of air sampled in the three systems which were intensively studied, glass fibers were relatively rare. The highest concentrations were found in ambient air. In ambient air samples in the San Francisco Bay Area (which included but were not limited to the results reported in Table I), we have found glass fiber concentrations to range from <0.05 to 10 f/L , with a mean of approximately 0.2 f/L . The estimated weight of the "average" glass fiber which makes up

deposition and entrapment of fibers. In nearly all instances, air within ducts had fewer glass fibers than either outside air or air immediately after passage through inlet filters. The decrease was both in concentrations of air-borne glass fibers and in the proportion of total fibers identifiable as glass. Over 95% of matched individual air samples (taken the same day in the same building) showed such a relationship.

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sweeping generalizations. Conclusions based on the 13 systems studied should not be extended to other systems less well designed, poorly maintained, or constructed from materials not intended for use in air moving systems. We do feel that the systems we studied are widely representative and that the periods of sampling and numbers of samples (over 600) were adequate to indicate that properly designed, constructed and maintained air transmission systems lined with fibrous glass do not add glass fibers to the air stream.

SUMMARY

1. In 13 air transmission systems lined with fibrous glass, there was a decrease in concentrations of glass fibers in air after the air had passed through the systems, both in number of glass fibers and in the relative proportion of total fibers which were identifiable as glass.

2. Data did not preclude the occurrence of erosion, but any which occurred apparently was more than offset by deposition and entrapment of fibers in filters and elsewhere within the systems.

3. Glass fibers within buildings correlated more closely with fiber concentrations in ambient air than with air exiting from the air supply systems lined with fibrous glass.

4. The concentrations of total fibers and glass fibers in ambient air, in air exiting air supply systems, and in building areas were extremely low and do not appear biologically significant.

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- 2) McCrone, W. C., Draftz, R. G. , and Delly, J. G.: The Particle Atlas; Ann Arbor Science Publishers, Inc., Ann Arbor, Mich., 1967.
- 3) Timbrell, V.: The Inhalation of Fibrous Dusts, Annals of the N. Y. Academy of Sciences, 132:255-273, Dec. 31, 1965.
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Table I

GLASS AND OTHER FIBER CONCENTRATIONS IN
THIRTEEN AIR TRANSMISSION SYSTEMS
(FIBERS PER LITER)

Outside Air						Filtered Outside Air					Air Within Ducts					Building Space Air				
Fibers per Liter	Average Concen- trations		Range of Concen- trations			Average Concen- trations		Range of Concen- trations			Average Concen- trations		Range of Concen- trations			Average Concen- trations		Range of Concen- trations		
System	Glass	Other	Glass	Other	% ^a	Glass	Other	Glass	Other	% ^a	Glass	Other	Glass	Other	% ^a	Glass	Other	Glass	Other	% ^a
1	0.26	1.51	<0.1- 0.56	0.5- 2.6	15	0.05	0.24	<0.04- 0.45	<0.04- 1.2	16	0.03	0.15	<0.02- 0.2	<0.02- 0.63	15	0.17	1.35	<0.05- 0.55	0.1- 2.6	10
2	0.27	2.42	<0.05- 1.23	0.2- 9.3	12						0.05		<0.05- 0.60		8					
3	*	*	*	*		0.11	0.22	<0.01- 0.92	<0.01- 0.93	34	0.02	0.14	<0.01- 0.19	<0.01- 0.56	12					
4-13	4.47	3.33	0.4- 8.8		57						1.22	1.43	<0.1- 7.0	<0.1- 10.0	46	3.6	8.16	<0.1- 9.0	<0.1- 20.0	31

^a % of all fibers which were glass. Taken from accumulated fiber data; may not match concentration data.

* Outside air concentrations probably similar to those in Systems 1 and 2.

FIGURE 1

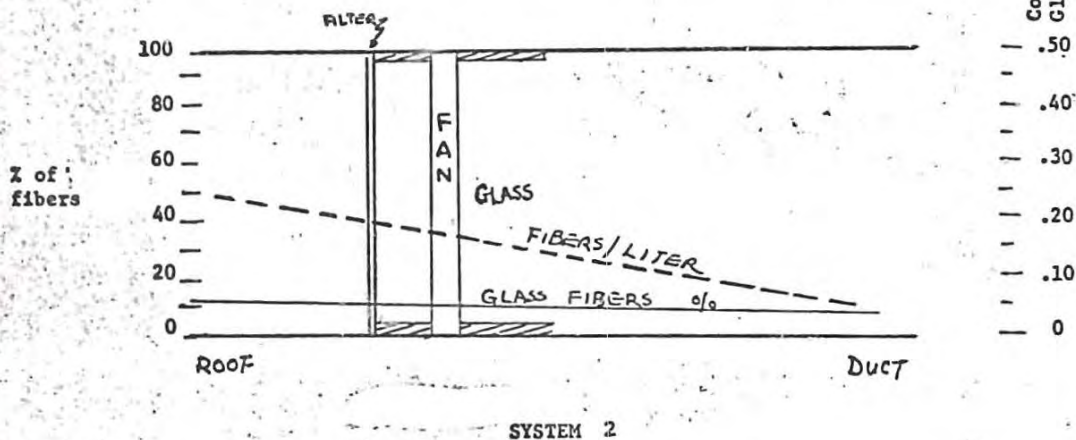
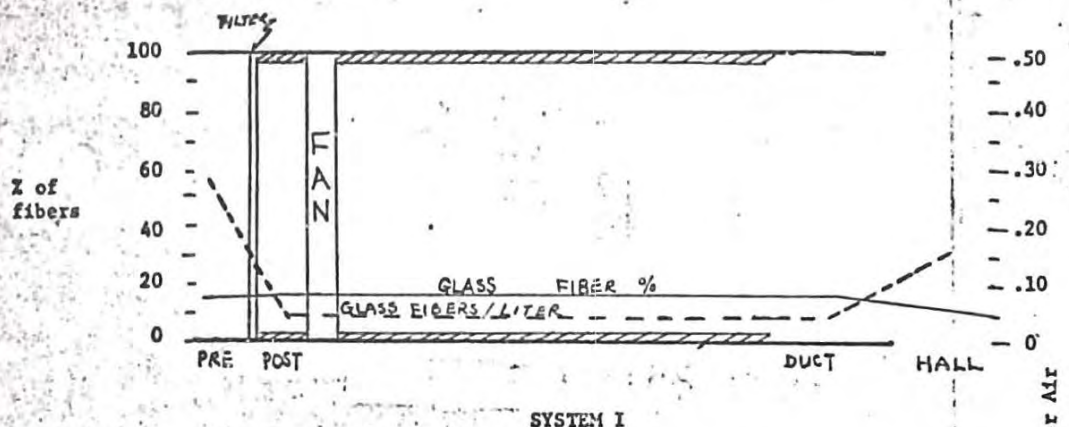
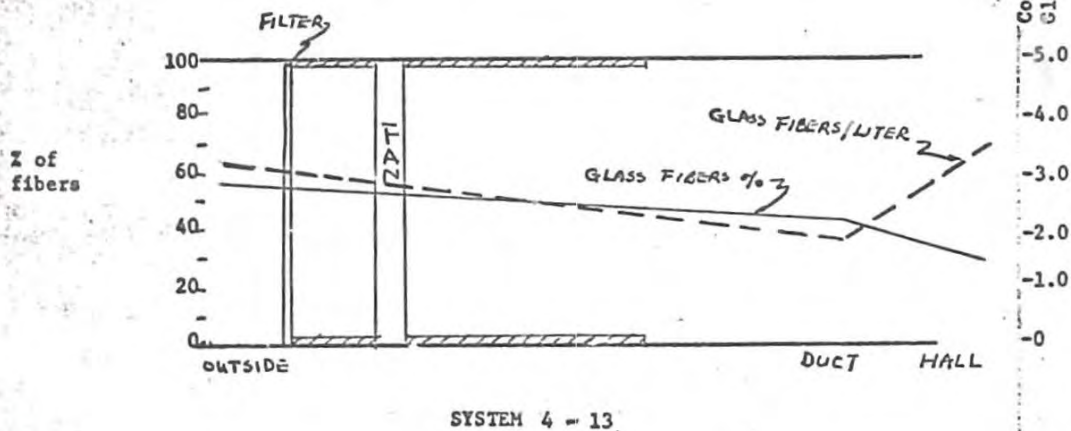
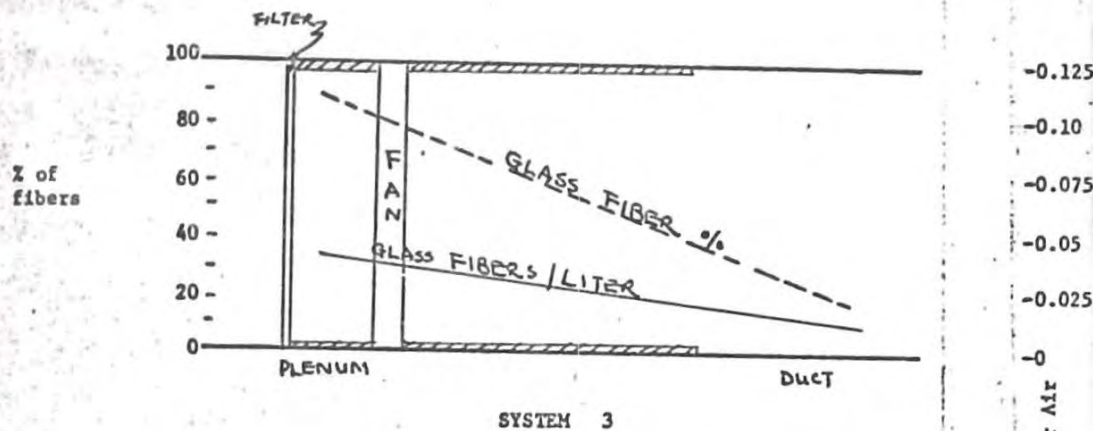


FIGURE 2



APPENDIX
DESCRIPTIONS OF SYSTEMS

SEN. C. L. LOM 1982

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APPENDIX

A-1

System No. 1. This is a large science building (about 7 years old) containing classrooms, laboratories, offices, and shops. The total volume of the air supply system is about 300,000 cubic feet per minute. Air is taken in through a ground level inlet, filtered through a fibrous filter, heated or cooled by passage through water-containing coils, and discharged into a large main air shaft for delivery to the floors of the building. This air shaft is lined with 2" thick coated fibrous glass insulation of 5 lbs. per cubic foot density, and air is taken from it to the occupied spaces within the building through unlined sheet metal ducts. Exhaust of air from the building is accomplished by the chemical fume hoods in the laboratories, and exhaust outlets are on the roof of the building (about 9 floors above the inlet). There is no recirculation of air.

Maximum air velocity in the system (in areas lined with fibrous glass) is about 2500 fpm at the fan outlet immediately prior to discharge into the main air shaft. Velocities decrease with height of the shaft, as portions of the air are taken for each of the floors in turn, until an effective zero velocity is reached at the top of the shaft at roof level.

In order to define the concentrations of fibers within this system, air samples were taken over a two-month period at four sites within the system. Samples were taken at the inlet of the system before filtration of the air (designated: "Pre"), immediately after the filter, before entry of the air into the fan and passage through the duct system (designated: "Post"),

Within an outlet duct discharging into the 8th floor hallway (designated: "Duct"), and in the 8th floor hallway about 50 feet away from the outlet of the duct above (designated: "Hall").

System No. 2. This system serves the ground and first floors of a moderate sized building, containing classrooms, laboratories, and offices. The building is about 10 years old. Although the main ducts both upstream and downstream of the fans contain fibrous glass lining, this extends (linearly) only about 30-40 feet; and the bulk of the duct work is unlined sheet metal, externally insulated with fibrous glass. Total capacity of the system is 25,000 cfm, and maximum velocity in the ducts containing fibrous glass is 1500 fpm. The system draws air from a plenum on a roof surface at the 3rd floor level and discharges with an exhaust fan through stacks at the 6th floor roof level. There is no recirculation of air.

The fibrous glass lining is a low density 1-inch blanket, with obvious tears and patches removed, particularly in the damper area where it has apparently been used as a walkway and work surface without protection.

Because of the design of the system, it was not practicable to sample the air after filtration but before passage through the ducts. In order to obtain a reasonable comparison, simultaneous samples were taken at the roof level entry to the system and within a duct on the first floor.

System No. 3. This system serves part of another moderate sized building on campus, again with classrooms, offices and laboratories. This

building was built about 5 years ago. The volumetric rate in this system is about 50,000 cfm, and the maximum velocity over the fibrous glass lining is about 2000 feet per minute. The duct work in this system is more extensively lined with fibrous glass than in either of the first two systems. The liner is 6 lb. density, 1-inch thick, and is used in all main supply ducts up to the final delivery ducts leading to the occupied spaces which are served. For all spaces, then, only the last "run" of duct is unlined, varying from 4 to 50 feet in length. Exhaust of air is through a separate exhaust system, discharging at roof level, while air is supplied to the system through a ground level entry. There is no recirculation of air.

Air samples were taken at two points within the system: immediately after the filter before entry into the fan and duct works ("Plenum" samples), and within an outlet duct on the 4th floor, supplied by the same fan ("duct" samples). There are about 160 feet of lined duct between these two points.

System No. 4. New Construction. The opportunity to determine the amount of erosion from a new system was presented by the completion of a new building on the campus. This system is of relatively low velocity and is lined throughout with fibrous glass. There is no recirculation of air in this system. Samples were taken of the general air inside and outside the building during construction and of the air within the ducts during the first "blow out" of the system as well as of the air during initial stages of balancing the system.

The observed concentrations of glass fibers within the ducts during the initial "blow out" were 0.4 f/L, 0.5 f/L, and 0.6 f/L. In the air of the rooms served by the above ducts, the concentrations were 1.0, 0.7, and 1.6 fibers per liter.

The outside air, which was drawn through the system unfiltered at this time, contained 0.8 glass fibers/liter.

A month later, samples were taken when the system was again activated. At this time, outside air contained 1.5 glass fibers/liter; air after passage through the duct work contained 0.2 glass fibers/liter; and room air contained 0.3 glass fibers/liter.

Samples were taken overnight at this time, after the ventilation system had been shut off, and outside air was found to contain 0.2 glass fibers/liter while air inside the building also contained about 0.2 glass fibers/liter.

System No. 5. This 7 year old system serves a drafting room in a highschool arts and crafts building. The system consists of a furnace/forced air "conditioning" system and serves only the one room. Air is taken in at ground level inlet, heated (depending on need), and dispersed through a fibrous glass-lined duct system with two branches; one about six feet long and the other about 28 feet long. The lining is 1 1/2 lb. per cubic foot faced liner. Exhaust from the room is by diffusion through doors and windows and through a single exhaust vent exiting at roof level of the single story building. There is no exhaust fan. Velocity is about 300 feet per minute in the supply ducts. At the time samples were taken,

the system had been shut down for about two weeks. The system was reactivated for our visit and had been operating for about one-half hour before sampling was begun.

Two samples of the outside air at the entry to the system showed concentrations of 5.9 and 8.8 glass fibers/liter while a sample of outside air taken on the other side of the building contained 6.8 glass fibers/liter. The air after passage through the six foot duct contained 0.6 glass fiber/liter; after the 28 foot duct the concentration was 0.8 glass fibers/liter. The air within the room served by the system contained 5.2 glass fibers/liter.

System No. 6. This system serves about one-fourth of a grade school building and was installed in 1967. Filtration of the air is by standard disposable "furnace filters". Air enters the system at roof level and is distributed through a combination of heavy duty fibrous glass duct system and flexible fibrous glass ducts. Representative outside air samples could not be obtained at this site. Samples were taken within the building at three sites: within the diffuser openings of two separate "runs" of the fibrous glass duct system and of the general room air in the room served by the two ducts. Removal of air from the building is by forced exhaust to a roof-top vent separated from the entry to the system by about 50 feet and an intervening gable roof. The level of glass fibers at the first duct outlet, downstream of about 55 feet of duct system and six feet of flexible fibrous glass duct, was 0.3 fibers/liter. At the second duct system, downstream of about 20 feet of heavy duty duct and again about six feet of flexible duct, the concentration was 0.4 fibers/liter. Room air concentration was 9.0 fibers/liter.

System No. 7. This is a newly constructed (1969) junior college business administration building. The system recycles air, the respective percentages of outside and recirculated air depending on the inside/outside temperature differential, with control of the percentage controlled automatically by thermostat. At the time of the visit, the percentage of recirculated air was about 35%. The return air passes through a false ceiling plenum which is lined with sprayed fireproofing. Although it was not possible to obtain a sample of this material, it was the impression of the building manager that the material contained asbestos. Air was filtered by a standard medium efficiency filter, both the outside air entering the system for the first time and the portion of the return air which was to be recirculated. The portion of the air exhausted from the rooms of the building which was to be "dumped" was discharged at roof level of the two story building. Maximum velocity was about 950 fpm. Air samples were taken in the filtered air stream entering the system (concentration: 2.5 glass fibers/liter), within a duct outlet downstream of about 60 feet of duct lined with a "multi-density" duct liner (concentration: 2.6 glass fibers/liter), in the air stream entering the return air system from the room (less than about 1 fiber/liter), and within the room served by the supply duct sampled, where the concentration was 3.8 glass fibers/liter.

System No. 8. This is a large system serving a college library building. The system has been extensively remodeled and altered since original installation about 6 years ago. Building plans were incomplete, and details of the extent of duct lining were sketchy, but it appeared

likely that most supply ducts were nearly completely lined with fibrous glass of varying densities. Maximum air velocity over lined portions of the duct work was judged as probably below 1500 feet per minute. The system makes use of recirculated air, and the percentage at the time of the visit was about 80% recirculated and 20% outside air. Entry of outside air to the system is through a ground level inlet, and exhaust of the portion of the return air which is not recirculated is through a roof level (fifth floor) discharge. Single samples showed concentration within the stream of unfiltered outside air was 2.4 glass fibers/liter; within the plenum, where outside and recirculated air are mixed after filtration through a "medium-efficiency filter", the concentration was 0 (less than 0.4 glass fibers/liter); at a duct outlet on the third floor of the building downstream of about 80 feet of lined duct, the concentration was 1.5 glass fibers/liter; at the outlet of a duct serving a ground floor room, the concentration was 7.0; and the air within the ground floor room contained 0 (less than 0.3 glass fibers/liter). This last duct was about 150 feet long and appeared to be completely lined with fibrous glass, although of a different type than that called for by the material specifications for the building.

System No. 9. This again was a large system, serving a college arts building which had been constructed in 1964. Air was also recirculated in this system with the percentage recirculated at the time of the visit about 85%. Maximum velocity of the air stream in the lined portions of the duct

system was judged to be not greater than 2000 fpm. The fibrous glass product used in this system was medium density faced liner. Exhaust from the system was through a roof-top discharge, about six stories above the ground level entry. Single samples at the entry in the unfiltered outside air stream indicated a concentration of 0.4 glass fibers/liter; of the filtered, mixed, return and outside air was 1.3 glass fibers/liter. Within two supply ducts on the first and fourth floors of the building, downstream of, respectively, 40 feet and 150 feet of lined duct, the glass fibers/liter were respectively 0.3 and 0 (less than 0.1).

System No. 10. This 5 year old system was somewhat different from those previously discussed since it was a high-pressure, high-velocity system. Hot and cold air were supplied separately to general areas of the building, such as a single large room or a group of small offices, through high-velocity unlined ducts, and mixed for delivery through lined ducts in small fibrous glass lined plenums, with the mix controlled by area thermostats. The plenums served an additional purpose as "sound traps" for suppression of fan noise. The system recirculated nearly all air with only about 10% outside air added. All outside and return air was filtered after mixture through a high efficiency, double filter system. The maximum velocity of air traversing the fibrous glass liner, after passage through the high-velocity portion of the system was about 500 fpm, and it was reported by the building engineer that the system had no fibrous glass in the air stream except downstream of the mixing plenums/sound traps. Samples were taken of the mixed/filtered air stream and within a supply duct 28 feet

downstream of a mixing plenum, with air velocity of about 300 fpm through the length of the duct to the single outlet. The concentration of glass fibers in the mixed inlet air after filtration was 2.1 fibers/liter; the air within the tested outlet duct contained 0.1 glass fibers/liter.

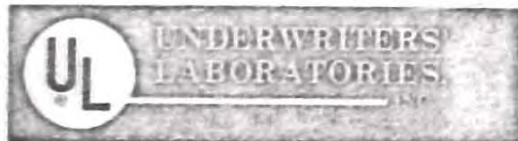
System No. 11. This system, also about 5 years old, served a junior college classroom and office building and was a "medium-high velocity" system, with air passing through fibrous glass lined ducts at an average velocity of about 1500 fpm for most of the distance from fans to the rooms served. As in the previous system, cold and warm air were separated for most of their travel and mixed in plenums before delivery to general areas of the building. Entry of air was through roof-level grilles at about the third floor level, while discharge was at the fourth floor level, about 100 horizontal feet from the entry. The fibrous glass liner in all visible portions of the duct work was medium density. Air was recirculated here also, with a mix of about 25% outside air to 75% recirculated air. Samples of air were taken at the entry plenum in the stream of unfiltered outside air where the concentration was 0.8 fibers/liter; downstream of the filter through which passed the return air before mixing with the outside air for filtration and delivery to the building, there was a concentration of 0.3 fibers/liter. The concentration within a supply duct downstream of about 200 feet of lined duct was 0 (less than 0.5 glass fibers/liter).

System No. 12. This system, installed in 1966, served a cafeteria

within an office building. Air velocity was low throughout the system and the maximum length of fibrous glass lined duct was about 25 feet. The liner in the system was light density and all air used was exhausted. Valid samples of the outside and room air were not obtained because power supplies to the samplers were turned off during the course of the sampling time. The concentration of glass fibers at the outlet of the supply duct was 0.9 glass fibers/liter, a level which is similar to concentrations of glass fibers in the outside air obtained at nearby sites during subsequent sampling.

System No. 13. This system serves the 11th through the 19th floors of a large office building. Air is not recirculated in this system but is discharged after passage through the building. The exact extent of fibrous glass liner in this system could not be determined even after extensive plan review with the assistant building superintendent and physical inspection of the system with him. He had been in charge of the building inspection for the tenant firm during construction of the building. Maximum velocity of air through the system, at points lined with fibrous glass, was about 2200 feet per minute. The liner specified in the plans was a medium density faced liner. Samples of air were taken within three outlet ducts, downstream of about 150-200 feet of (presumably) lined duct. The concentrations of glass fibers/liter of air were 0.8, 1.5, and 1.3 fibers/liter. It was not possible to obtain samples of outside air at the entry of this system.

22

STANDARD $\frac{1}{2}$ SAFETYAIR DUCTS
UL 181-1970

90 Following the above test, the inner and outer surfaces of the sample shall not be ruptured, broken, torn, ripped, collapsed, or separated; joints and seams shall remain intact and not be fractured; and there shall be no evidence of other damage to the sample which would cause it to be unusable.

Erosion

91 Material for ducts and connectors shall not break away, flake off, or show evidence of delamination or continued erosion when air is passed through typical sections at a velocity of two and one half times the manufacturer's rated velocity, but not less than 2500 feet per minute.

92 One or more of the samples of duct sections previously subjected to the impact test (paragraphs 90-95) and one or more samples of connectors selected in accordance with paragraph 91 and previously untested are to be subjected to the test for erosion. The sample or samples selected are to be arranged in an L-shaped assembly. When sections of duct are to be tested, the leg of the assembly connected to the fan outlet is to be the section which had been subjected to the impact test and the down leg is to be of similar untested material having a length of not less than six times the major inside dimension of the duct section. A connector for this test is to be 12 feet long. For rigid ducts, the 90-degree bend is to be made with an elbow as normally employed for installations. Flexible duct and connectors are to be bent at a radius equivalent to the inside diameter of the duct or connector to form the 90-degree bend. The entire assembly is to have similar cross sections. See Figure 5.

EROSION TEST

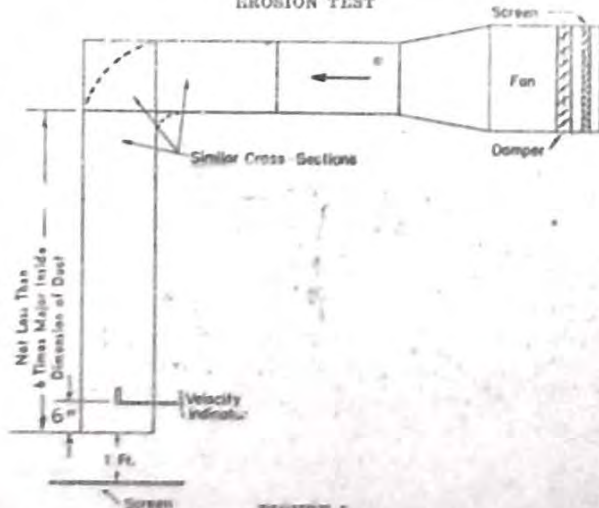


FIGURE 5

- 90 Connection to the outlet of the fan is to be by means of a transformation piece of any suitable material to provide for uniform air entry to the test sample. The fan is to have sufficient capacity to provide a maximum velocity of two and one-half times the maximum velocity recommended by the manufacturer, but not less than 2500 feet per minute of air at room temperature.
- 100 The air velocity for purposes of the test is to be measured by a pitot tube, or direct-reading velocimeter, positioned in the center of the outlet end of the sample. Room air handled by the fan and ranging in temperature between 60 and 100° F (15.6 and 37.8° C) may be employed.
- 101 The inlet of the air fan used for the test is to be covered with a double layer of cheesecloth (14 to 15 square yards per pound and known to the trade as "count of 32 by 25").
- 102 A damper, if employed for regulating velocity in the duct system, is to be located between the screen and the fan or between the fan and the inlet to the test sample.
- 103 A collecting screen consisting of a double layer of cheesecloth, as specified in paragraph 101, stretched taut on a frame sized to provide for an area not less than five times the inside cross-sectional area of the test sample, is to be located 1 foot from the outlet end of the test sample.
- 104 Samples are not to have been exposed to relative humidities greater than 70 percent during the 24-hour period prior to the test.
- 105 Air is to be passed through the sample at approximately test velocity with the collecting screen removed for a period of at least 1 hour but not more than 24 hours. The collection screen is then to be placed in position.
- 106 The test is then to proceed at test velocity and continued for a period of 4 hours. The collecting screen is to be examined for macroscopic particles at the end of each hour during the test period by taping the screen with the adhesive side of transparent tape, or tapes, in order to remove and record any eroded particles. At the end of the test period, there is to be no evidence of continued erosion, and interior surfaces of the sample are not to show evidence of cracking, flaking, peeling, or delamination. For the purpose of this requirement, continued erosion is considered to be either a constant or increasing rate of erosion.

STATEMENT ON PROPOSED REGULATION R-71-16

ASBESTOS AND SPRAY INSULATION

STATE OF ILLINOIS

POLLUTION CONTROL BOARD

PRESENTED BY

Clifford L. Sheckler, Chairman

National Insulation Manufacturers Association
Health and Safety Committee

ON BEHALF OF THE

National Insulation Manufacturers Association
7 Kirby Plaza
Mt. Kisco, New York 10549

October 19, 1971

Gentlemen, I am Clifford L. Sheckler. I represent the National Insulation Manufacturers Association. I serve as Chairman of the National Insulation Manufacturers Association Health and Safety Committee.

With regard to the proposed Pollution Control Board Regulation R-71-16, Asbestos and Spray Insulation, we respectfully call your attention to Part III, Section 302, paragraph (g) which reads as follows:

"Any plenum or other structure coated with or containing fibrous insulation and used in the circulation of air in a building shall be thoroughly cleaned of all debris and waste insulation. All applied fiber-containing insulation within a plenum or duct shall be coated with a sealant which precludes exposure of the fiber-containing material to the circulating air."

We urge you to consider revising Part III, Section 302, paragraph (g) by inserting the word "sprayed" in both the first and second sentences as follows:

"Any plenum or other structure coated with or containing (sprayed) fibrous insulation and used in the circulation of air in a building shall be thoroughly cleaned of all debris and waste insulation. All (sprayed) applied fiber-containing insulation within a plenum or duct shall be coated with a sealant which precludes exposure of the fiber-containing material to the circulating air."

We are requesting this revision to clarify the fundamental purpose of the proposed Part III, Section 302, paragraph (g) to specifically relate to "sprayed" fibrous insulation.

We believe this clarification is required because fibrous glass insulation is applied in air transmission systems. This material is applied by hand in board or blanket form and is not sprayed. Unlike sprayed insulation, fibrous glass duct insulation materials are preformed and bonded.

Fibrous glass duct insulation materials meet the requirements of nationally recognized standard-making bodies and are approved for use by key governmental agencies.

Extensive industrial hygiene engineering research studies have been carried out by highly qualified university affiliated industrial hygiene engineers and physicians. Studies both in the laboratory and in actual installations demonstrate no significant erosion from the standpoint of either health or comfort.

The subject of safety and health has been utilized extensively by a certain few individuals and groups for the purpose of eliminating fibrous glass thermal and acoustical insulation from the inside of ducts. This strategy and campaign has been pursued unsuccessfully. The preponderance of medical and engineering scientific research to date shows fibrous glass insulation to be a biologically inert, safe, effective material.

Published authoritative medical scientific research studies over the past 30 years show fibrous glass to be a biologically inert substance which presents no health risk greater than any other innocuous dust.

The American Conference of Governmental Industrial Hygienists has scrutinized the scientific research studies concerning the pulmonary effects of fibrous glass and has classified this material as an inert or nuisance-type dust. The Federal Occupational Safety and Health Act Standards of 1971 have confirmed this classification.

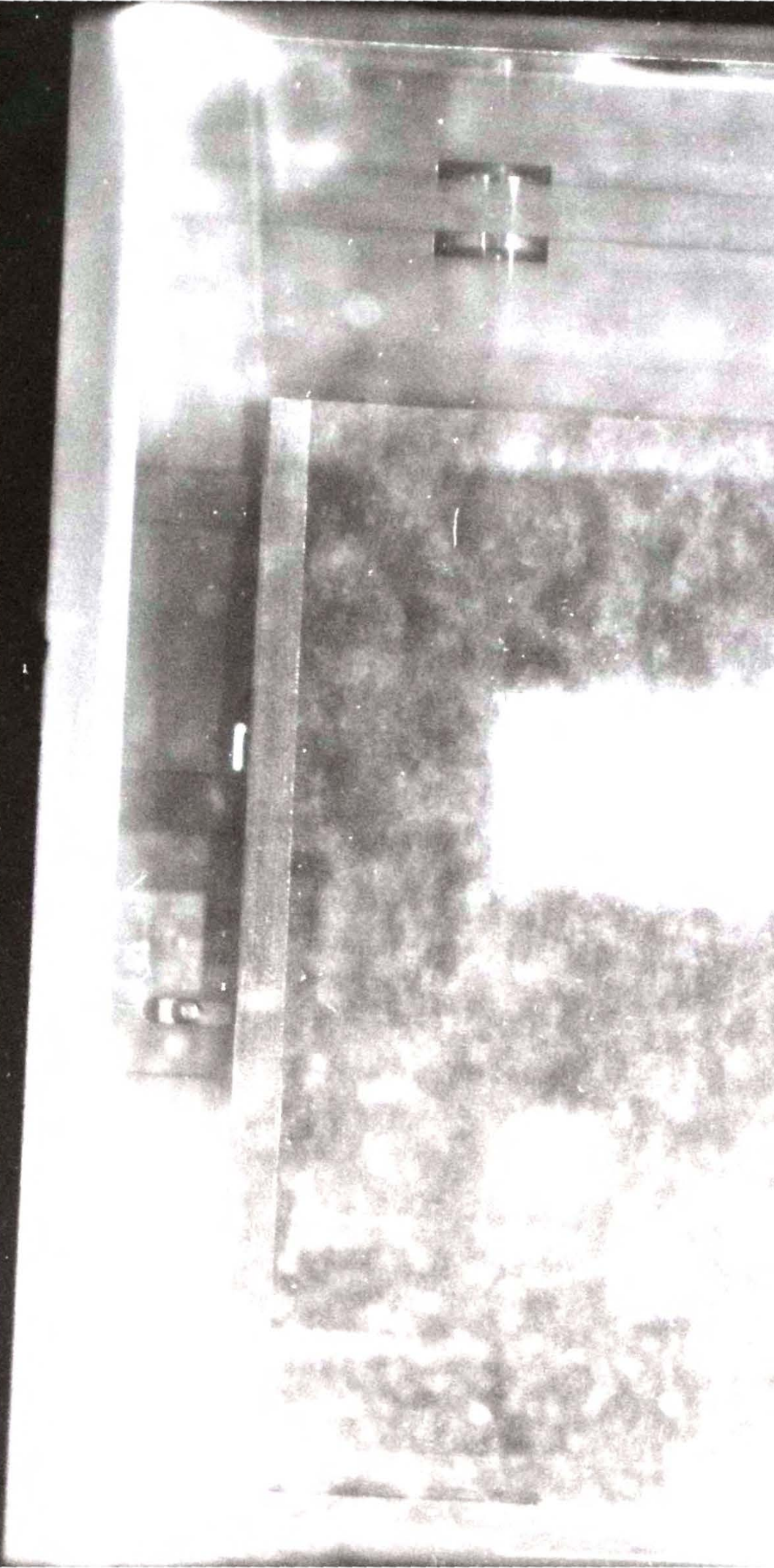
All substantiating evidence concerning the health aspects of fibrous glass are listed in our attached National Insulation Manufacturers Association booklet, "Health Aspects of Fibrous Glass". One copy of the supporting medical and engineering scientific data is enclosed for committee review.

We wish to thank you for the opportunity of appearing before your Committee.

Clifford L. Sheckler, Chairman
National Insulation Manufacturers Association

R71-16

EXHIBIT 25



572
+ 25

SUBMISSION OF

CATERPILLAR TRACTOR CO.

to

State of Illinois

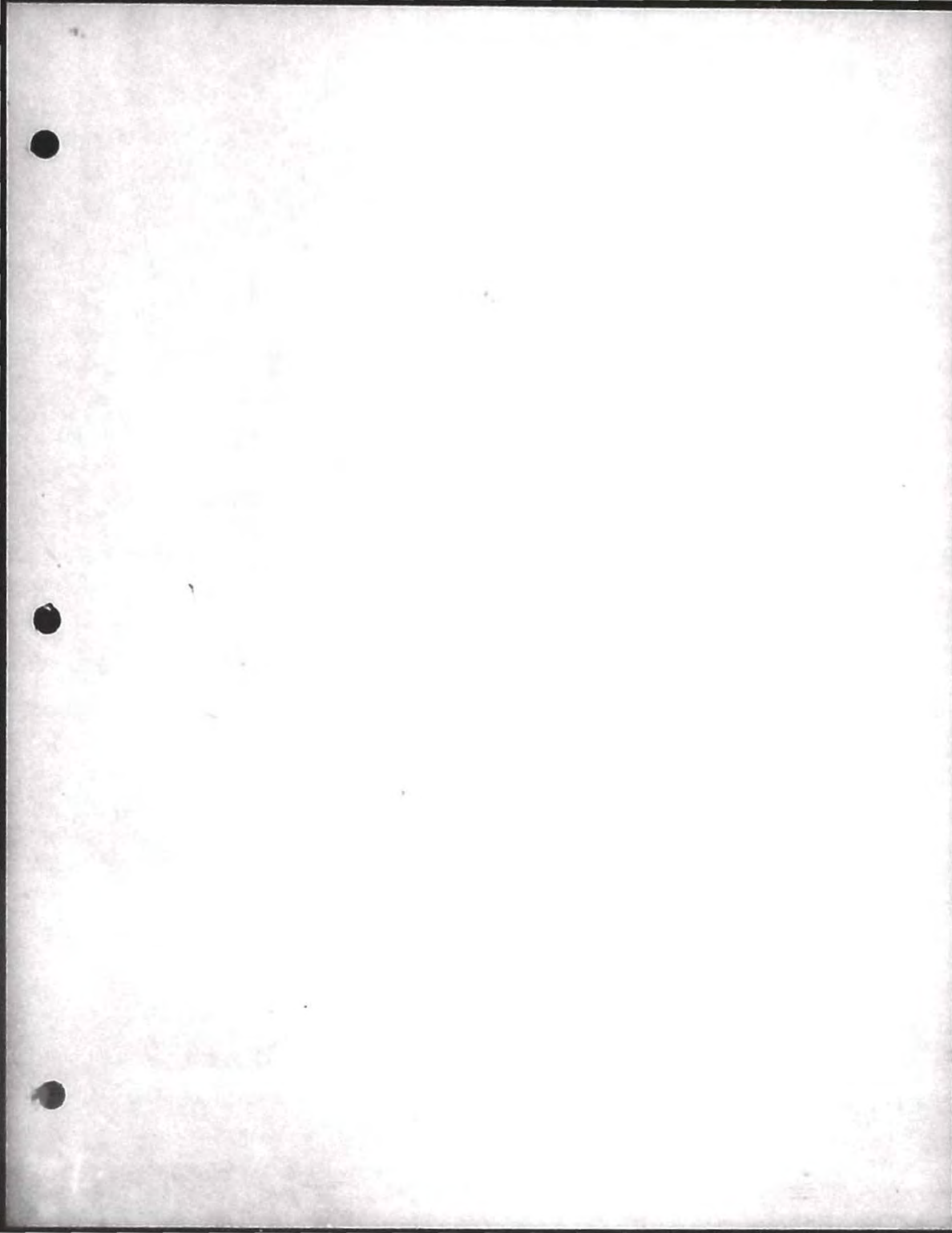
Pollution Control Board

at

Regional Hearing on Asbestos and Spray Insulation

Waukegan, Illinois

October 19, 1971



PREFACE

It is the sincere desire of Caterpillar Tractor Co. to cooperate with and assist the State of Illinois Pollution Control Board in the preparation of the proposed Asbestos and Spray Insulation Regulations R71-16 in accordance with the Environmental Protection Act of the State of Illinois.

The purpose of this booklet prepared by Caterpillar Tractor Co. is to supplement the statement and document the information presented by the Company at the Regional Hearing on Asbestos and Spray Insulation Regulations at Waukegan, Illinois on October 19, 1971

Included in this booklet are: (1) Caterpillar's concerns regarding the possible prohibition of asbestos brake linings; and (2) Caterpillar's recommendations relative to the proposed Regulations which would ban asbestos brake linings by 1975. Also included in Appendix B is a copy of Caterpillar Tractor Co.'s oral testimony presented at the Waukegan Hearings, October 19, 1971.

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I. INTRODUCTION

A. Purpose of Booklet

The purpose of this booklet is to supplement and support the oral statement made by Caterpillar Tractor Co. at the Regional Hearing on Asbestos and Spray Insulation Regulations at Waukegan, Illinois on October 19, 1971. Recommendations and suggestions contained herein are intended to apply only to products of a type and kind built by Caterpillar Tractor Co. The information presented is brief; the intent is to provide the Company's position relative to the proposed ban on the use of asbestos brake linings.

B. Caterpillar Product Line

Caterpillar currently produces 52 models of track or wheeled earthmoving equipment (in addition to 53 models of Towmotor material handling equipment), all of which have braking systems which utilize asbestos friction material. The following listing gives the number of models in each specific product line (Appendix A pictorially illustrates the Caterpillar product line):

- 6 - models of track-type tractors
- 6 - models of wheel-type tractors and compactors
- 5 - models of track-type loaders
- 7 - models of wheel loaders
- 5 - models of pipelayers
- 13 - models of wheel tractor-scrapers
- 6 - models of motor graders
- 2 - models of off-highway trucks
- 2 - models of off-highway tractors

52 - Total Models

II. CATERPILLAR CONCERNS

A. Proposed Regulation R71-16, "Asbestos and Spray Insulation"

The specific portion of the proposed Regulation that concerns Caterpillar Tractor Co. is Part VII - Asbestos Containing Products, Section 702, which states:

"The use of asbestos in the brake lining of vehicles manufactured after January 1, 1975, and sold for use in Illinois is prohibited."

B. Definition of a Vehicle

The lack of a definition for the word "vehicle" in the proposed regulation is of concern to Caterpillar Tractor Co. The Illinois Vehicle Code (Section 1-217) defines a vehicle as "every device, in, upon or by which any person or property may be transported or drawn upon a highway...". Although this definition could provide some clarification to the proposed regulation (if included), it is obviously quite general. Earthmoving equipment does not normally operate on public highways and is not designed for such use, although certain equipment such as the 613 Elevating Scraper is small and light enough to be driven on the highway in its movement from one job site to another; motor graders may also be used to repair and maintain existing roads.

Regulatory statutes typically have differentiated between motor vehicles and earthmoving equipment. It is, therefore, the Company's suggestion that this distinction should be recognized

and dealt with by including the following definition of vehicle in Section 103 of the proposed Regulations:

Vehicle - Any self-propelled device designed primarily for transporting persons or property on a street or highway.

A specific definition of this type would properly exclude from the proposed Regulation earthmoving and construction equipment -- not designed primarily for use on public highways.

C. Reasons for Caterpillar Concern

Unaware of the full intent of the proposed Regulation and uncertain as to whether or not Caterpillar product is included in the proposed Regulation, the Company felt it mandatory to present its case assuming the intent was to include earthmoving equipment.

1. Prohibition of Asbestos Brake Linings

It is the Company's concern that all "use of asbestos in brake lining...is prohibited." Asbestos brake linings are used in all Caterpillar product because of their demonstrated safety and durability characteristics (which will be discussed in more detail later). At this time, Caterpillar is unaware of a safe substitute for asbestos brake linings (our experience with sintered metal brake material has been unsatisfactory).

The brakes on some of Caterpillar's equipment are totally enclosed. The brakes on many models are immersed and rotating in cooling oil. In these cases, asbestos wear products

cannot get into the atmosphere except possibly when the brake compartment covers are removed for service. Other models have brakes with shields which are not air tight and thus brake lining wear products can escape to the atmosphere. The characteristics of these wear products are described in Section III of this booklet.

Some Caterpillar models use caliper disc brakes, which are not enclosed, and the brake pads, containing asbestos, can emit their wear products directly into the air.

2. Some Types of Disc Brakes Are Not a Solution

In the explanation of Proposed Regulations (R71-16), the statement is made: "It is contemplated that the ban will necessitate design changes, probably to disc brakes..."

As previously mentioned, Caterpillar Tractor Co. currently utilizes caliper disc brakes on some of its models; however, asbestos friction material pads are still utilized in this type of brake because of the safe, sure stop they provide.

It must be pointed out that caliper disc brakes are not an alternative solution to banning the use of asbestos brake linings. In fact, with the open nature of caliper disc brakes, it is easy for brake-pad wear products to be emitted.

III. PROBLEM IDENTIFICATION

A. Types of Caterpillar Product Brakes

Asbestos-type friction material is used for brake lining throughout the entire line of Caterpillar Tractor Co.'s earthmoving equipment. A few of the models are shown in Figure No. 1: A is a wheel-type tractor, B is a motor grader, C is a wheel-type

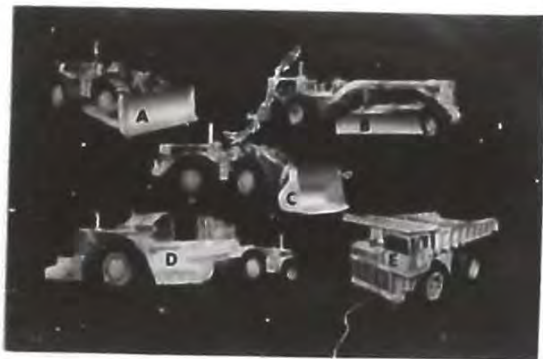


FIGURE NO. 1
EXAMPLES OF CATERPILLAR EARTHMOVING EQUIPMENT

loader, D is a wheel tractor-scraper, and E is an off-highway truck. Asbestos friction material is not used in Caterpillar's brakes from happenstance but is chosen for its performance, reliability and safety. Some of the products use dry friction surface brakes and some are bathed in cooling oil. For example, the off-highway truck (E) has totally enclosed oil cooled multi-disc brakes while the wheel tractor-scraper (D) has shoe brakes. Each product (e.g., truck, wheel tractor-scraper, etc.) requires a specific brake type and friction material depending on its primary application.

As early as 1937 Caterpillar was conducting research work to identify operating characteristics of all known types of brake lining material. It is a necessity that Caterpillar provide its customers with the best performing and safest brakes possible. So far, a material superior to asbestos-type that fulfills all the requirements needed for the heavy-duty brakes required on earthmoving equipment has not been found.

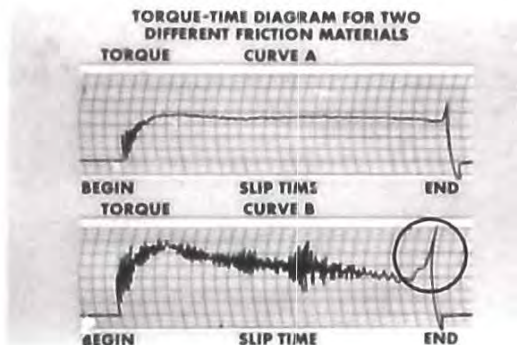


FIGURE NO. 2
TORQUE CURVES OF FRICTION MATERIALS

Figure No. 2 is a strip chart recording of the torque-time relationship for two different friction materials. These two traces illustrate the frictional behaviour throughout a brake application. In curve A, note the smooth deceleration or braking as the wheel speed is reduced to zero, from beginning to end of stop. This is one of the characteristics we expect from asbestos-type material. On the other hand, curve B of a non-asbestos friction material shows undesirable speed change conditions. The trace exhibits considerable frictional vibration. This results

in noise and undue stress in the whole brake system. The abrupt change to a static level at the end of braking (as circled in Figure No. 2 on the right of curve B) causes instability and an erratic, unsafe brake operation.



FIGURE NO. 3
TRACK-TYPE LOADER WITH RAISED BUCKET

If a track-type loader (see Figure No. 3) stops abruptly, rather than smoothly, while backing up with a raised bucket full of rocks, some of the rocks leave the bucket and continue in the rearward direction and end up in the operator's lap. This safety hazard also applies to other earthmoving machines which can spill a load due to an abrupt stop.



FIGURE NO. 4
TRACK-TYPE TRACTOR
ENCLOSED BRAKE COMPARTMENT

The enclosed brake compartment in Figure No. 4, shown with covers removed, has band brakes (noted by arrows), which are used either dry or bathed in oil. In either case, due to the enclosure, brake wear products cannot be emitted into the ambient air during normal operation. When servicing these compartments, they are exposed to the ambient air and the products found (Figures No. 11 and 12 and discussion) are powder rather than fibrous in nature and submicron in size.

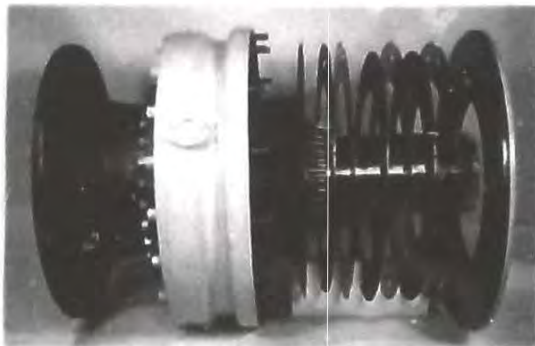


FIGURE NO. 5
OFF-HIGHWAY TRUCK
ENCLOSED, OIL COOLED, MULTIDISC BRAKE

Figure No. 5 is an example of multidisc brakes which are totally enclosed and bathed in cooling oil. The only time brake wear products from this type brake are exposed to the ambient air is during servicing. This brake was originally designed to use sintered metal friction material. Poor performance, reliability, and safety requirements necessitated a change to asbestos-type material.



FIGURE NO. 6
FRICTION PLATE AND MATING PLATE
FROM BRAKE IN FIGURE NO. 5

Figure No. 6 shows the asbestos containing friction plate used in the previously mentioned multidisc off-highway truck brake design. This design of disc brake system cannot emit wear products into the air during operation due to its oil environment and the fact that it is contained in an enclosed compartment.

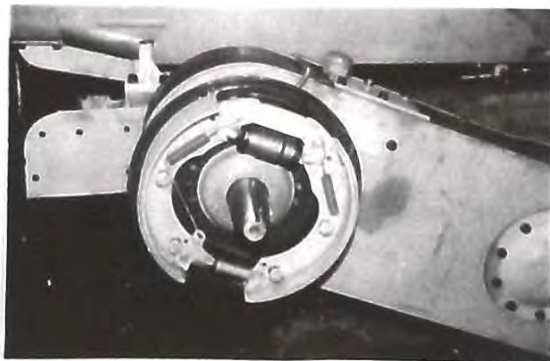


FIGURE NO. 7
MOTOR GRADER SHOE BRAKE (DRUM REMOVED)

Figure No. 7 shows a shoe brake located in the wheels of rubber-tired earthmoving equipment. Motor grader shoe brakes of this type normally are shrouded to protect the operating mechanism. The shrouding may allow brake wear products to be emitted into the ambient air since it does not provide an airtight compartment.

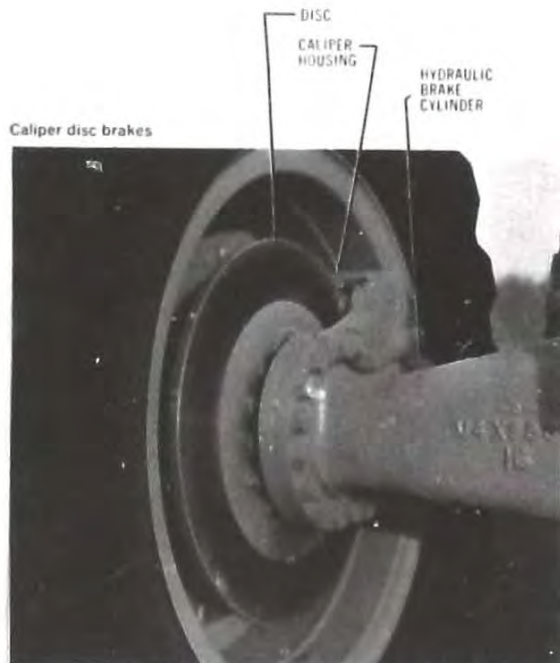


FIGURE NO. 8
WHEEL TYPE LOADER CALIPER DISC BRAKE

Conventional caliper disc brakes are also used on some Caterpillar product and are shown in Figure No. 8. The pads in the brake caliper contain asbestos. With this type of brake system any brake wear products are emitted directly into the ambient air.

The heavy-duty performance of Caterpillar machines and their braking requirements are considerably more stringent than automotive applications and need to be explained. It is normal for track-type or wheel loaders to go forward and reverse as frequently as 180 times per hour, all day long, and often three shifts a day. This means as many as 360 brake applications per hour. Bulldozing operations also require severe usage of brakes since track-type tractors are steered by the brakes.

Caterpillar 666 Wheel Tractor-Scrapers weigh 250,000 pounds when loaded to capacity. Typical applications will find these machines going downhill when fully loaded. The brakes on a 250,000 pound scraper, stopping from 45 miles per hour on the level, convert enough energy into heat to melt 41 pounds of iron. (A typical passenger car stopping from this same speed would develop enough heat to melt 2/3 of a pound of iron.) Of course, downhill operation increases the energy dissipation further. Off-highway trucks weigh slightly less and must perform in similar situations.

Pipelayer brakes must hold as much as 200,000 pounds of pipe suspended over a trench, while men are working beneath the pipe. Track-type tractors often operate in conditions where very steep slopes must be negotiated (in forward and reverse). Since these machines are steered by the brakes, the safest possible linings must be used to prevent brake failure which could result in a machine attaining runaway speeds on these steep slopes.

It is Caterpillar's candid opinion that the prohibition of asbestos from brake linings would place a severe penalty on the performance and safety of earthmoving equipment.

B. Brake Wear Products

In the December 1968, Air Pollution Control Association Journal, Jeremiah Lynch presented the results of his study entitled "Brake Lining Decomposition Products." In an initial study, dust samples taken from automobile brake drums were examined. Electron micrographs did not reveal the presence of any free fiber. Additional study, on a laboratory basis, was conducted. Decomposition products of various brake linings were sampled under simulated operating conditions. In all but a very few cases the linings tested showed less than 1% free asbestos fiber in the decomposition product. This is a significant finding since the original linings averaged 50% asbestos by weight. In the isolated cases where significant free asbestos fiber was found, the test conditions were such that brake failure would have occurred if the linings had been subjected to like conditions in a vehicle.

Lynch states that in all but the most extreme driving conditions, only a very small fraction of the 30% - 50% asbestos present in brake lining escapes into the ambient atmosphere as a free fiber. Wear apparently occurs not by abrasion but in minute areas of intense heat at points of contact between the drum and lining. At these temperatures, about 900°F., decomposition products do not generally contain free fiber, but are a different mineral resulting from the thermal breakdown of the asbestos.

Asbestos Fiber Analysis



FIGURE NO. 9
Asbestos fiber from new brake lining
5000x

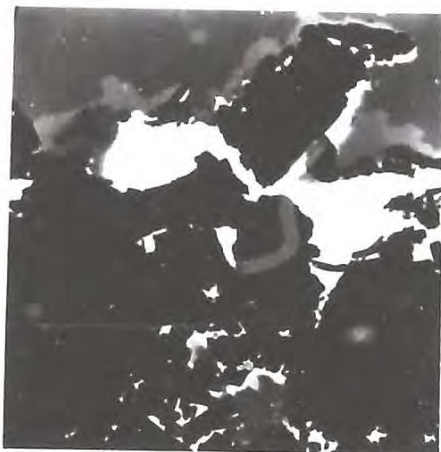


FIGURE NO. 10
New brake lining material after
heating to 1200°F. for 30 minutes.
5000x

All fibers in Figures No. 10, 11 and 12 are below 7 micron in length.

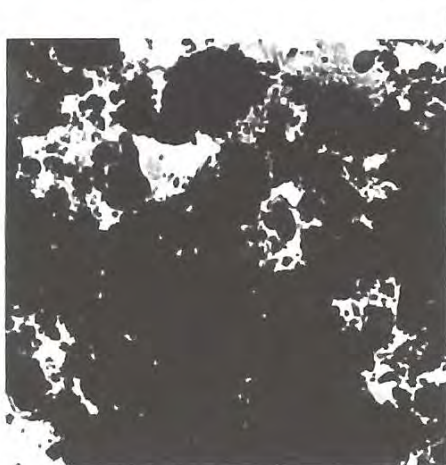


FIGURE NO. 11
Debris from dry brake compartment
5000x



FIGURE NO. 12
Debris from dry brake compartment
5000x

35 Microns - Thickness of a human hair at 5000x

Recently, tests were made by Caterpillar's Research Department on the wear debris found in an enclosed, tractor, dry brake compartment which corroborates Lynch's findings. The four photographs on the opposite page were made with an electron microscope at 5000 x magnification. Note the 5 micron (two ten thousandths of an inch or 0.0002") scale in the center of page No. 14. Section 601 in the proposed Regulation states that "only fibers of 5 microns or greater in length...shall be counted" in the emissions from a factory into ambient air. The scrapings from an unused asbestos brake lining shown in Figure No. 9 obviously have fibers longer than 5 microns in length. Heating these scrapings (which provides a laboratory simulation of continued brake applications) from an unused brake lining destroys the fibrous nature. This is shown in Figure No. 10. Mechanically rubbing the deteriorated fibers in a brake would further powder this material. Figures No. 11 and No. 12 show the actual debris found in dry brake compartments. Any fibers visible are submicron in size. With the intense heat resulting from brake application, it is expected that a breakdown to sub-micron debris would also occur in enclosed, oil cooled brake compartments.

If the standards in Section 601 of the proposed Regulation were applied to brake emissions into the air, the brake wear products of heavy-duty earthmoving equipment would be under 5 microns in size and therefore be within threshold limits.

It should be pointed out that there are well over 100 different natural minerals having a degree of fibrous structure. Some investigators consider respirable fibers to be ubiquitous (everywhere at the same time) and feel that much more definitive information must be obtained on their identification, source, and biologic response.

A good example of this is talcum powder which is found in almost every home and is a measurable source of asbestos fiber. Tremolite, another form of asbestos, occurs naturally in most talc used in this country and is thus found in talcum powder. Industrial talc may contain as much as 30% tremolite. Since more than 750,000 tons of talc are used in this country annually, it must be considered to be a prime source of respirable asbestos fibrils. (The finest single fiber shown in Figure No. 9 can be called a fibril.)

C. Asbestos Usage - Caterpillar vs. U.S. Consumption

Finally, it should be pointed out that of the more than 800,000 tons of asbestos used in this country each year, only about 3% of this goes into the manufacture of friction materials such as brake linings. Caterpillar's dry brake asbestos material usage amounts to 0.006% of the total asbestos usage in the U.S.A. and less than 0.2% of the total friction material usage of asbestos in the U.S.A. It is felt that the amount of free asbestos fiber that can potentially become airborne, as a result of breakdown of brake linings of earthmoving equipment, is quite low and unlikely to be significant from a health standpoint.

IV. OTHER CONCERNS

A. An Illinois Standard Would Dictate Worldwide Caterpillar Requirement

The establishment of a standard which prohibits the use of a material in vehicles which are manufactured and sold for use in Illinois has the effect of setting national or even international standards. It is impossible to know the ultimate destination of Caterpillar machines as they are assembled and to provide for selective assembly or alternate brake systems for those to be sold in Illinois. In order to provide competitive leadership on a worldwide basis, Caterpillar has established the policy to use interchangeable parts on all models so that specifications can be followed for purchase and manufacture at any plant in the world

If there were a material that would do the same job as asbestos in brakes, it is highly improbable that it could be a direct substitute. Other mechanical parts would have to be modified to adapt this new material. The results would be an "Illinois only" machine with parts only available locally, not worldwide. The customer could immediately react to this and buy a standard machine in a neighboring state and move it into Illinois as a used machine.

Caterpillar Tractor Co. has long been a leading and vitally important company in Illinois. As the state's largest private employer with over 41,000 employees and 6 manufacturing facilities, it has become the largest dollar exporter of product in the United

States. Over 52% of the \$2.1 billion annual sales reached in 1970 were outside the United States. Illinois would not be the leading state in exports to other countries without Caterpillar.

Imposition of a ban on the use of asbestos in brakes would impose a severe restriction upon Caterpillar Tractor Co.

B. Effect of Proposed Regulations on Illinois Caterpillar Dealers

The requirement for a specific design of earthmoving equipment for Illinois could result in a hardship for Caterpillar dealers and Illinois earthmoving contractors, and a subsequent loss of sales tax to the State. Special brake assemblies to meet the Illinois requirement would, of necessity, result in higher first cost of product. This higher first cost would result in higher bid prices for road building and earthmoving contracts. Thus, with a fixed State budget for road improvement, etc., less work would be done, and fewer machines would be sold.

V. SUMMARY AND RECOMMENDATIONS

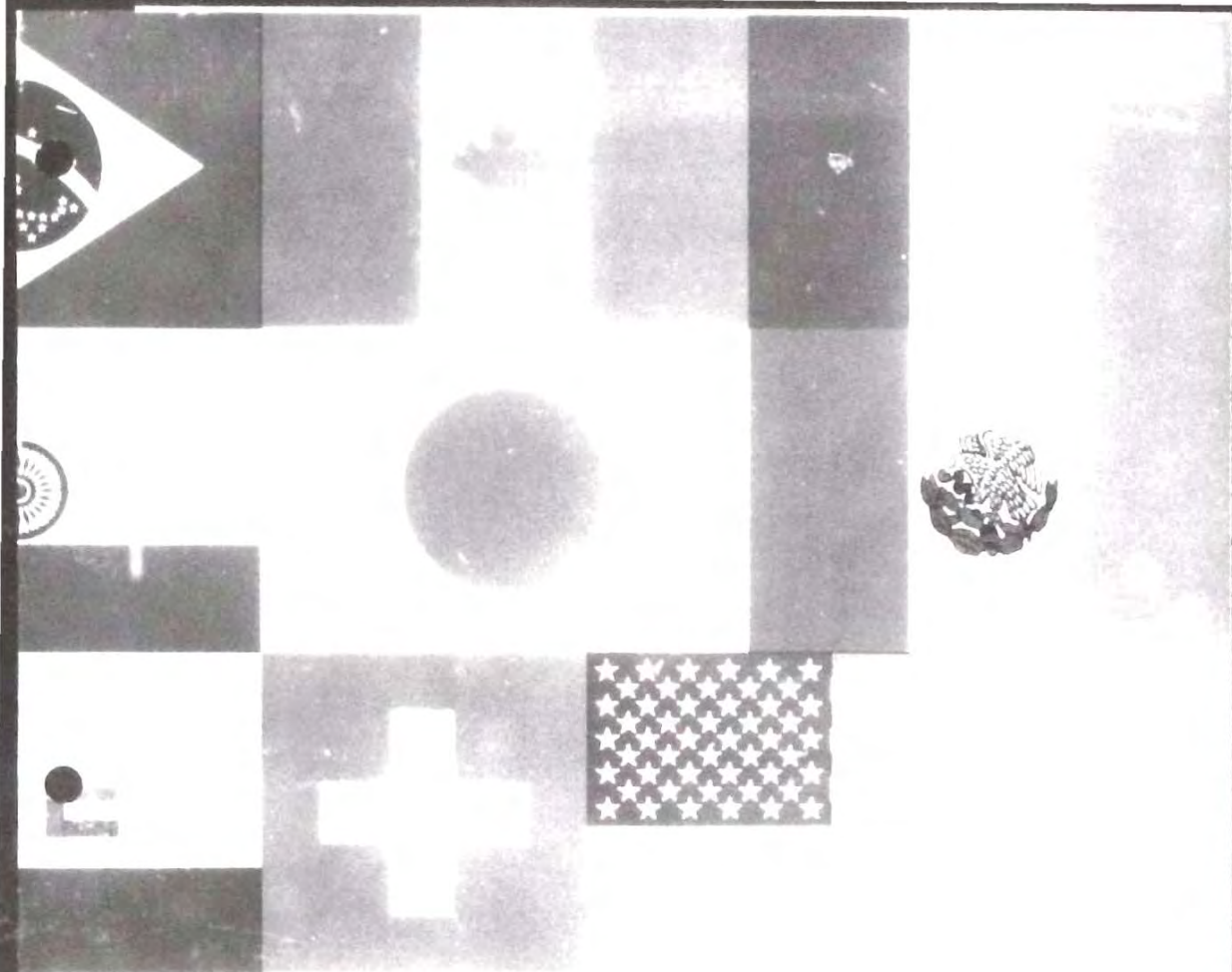
Caterpillar Tractor Co. sincerely believes that the information presented in this booklet establishes reasonable doubt about the real contribution of earthmoving equipment to the asbestos environmental problem. It has been pointed out that each Caterpillar product uses a distinctive brake system design which is influenced by its working environment, job application spectrum, performance criteria and safety requirements. However, all Caterpillar product brake systems use asbestos friction material since it has evolved over the years as the most satisfactory material, all aspects (particularly safety) considered. Caterpillar Tractor Co. tests and total Company usage of asbestos both indicate that asbestos friction material used in our product does not significantly contribute to the level of asbestos fibers in ambient air -- at least not to the point where its use should be prohibited!

Based on the aforementioned points and the significant interstate and international commerce aspects, Caterpillar Tractor Co. questions whether the intent of the Asbestos and Spray Insulation Regulations is actually accomplished by the proposed ban of asbestos brake linings. If research work and data on ambient concentration levels of asbestos fibers which are detrimental to society establish a need for asbestos brake control and/or regulation, Caterpillar recommends that Section 702, Part VII of R71-16 (Asbestos and Spray Insulation Regulations) be modified to allow the following exceptions:

1. That asbestos brake linings (friction material) may be used in a brake system when the friction material operates in either a dry or oil-wetted environment in a totally enclosed brake compartment which prevents the emission of brake wear products into the ambient air.
2. That asbestos brake linings (friction material) may be used in a brake system, when the friction material operates in a non-enclosed brake compartment, until conclusive medical evidence proves that the emitted brake wear products contain harmful amounts of asbestos fibers.

If all evidence indicates the need for asbestos brake controls and/or regulations, Caterpillar would further recommend that reasonable threshold limits of asbestos fiber be established by qualified personnel (based on scientific fact and foreseeable technological advances). Test procedures which measure the concentration of asbestos fibers must also be developed which are understandable, repeatable, and realistic. Place the burden on the manufacturer to design his product to meet reasonable, attainable standards -- don't just prohibit the use of asbestos for brakes.

As mentioned at the beginning of this booklet, Caterpillar Tractor Co. agrees in principle with the objectives of the Environmental Protection Act. The Company stands ready and willing to assist the Board in the establishment of asbestos regulations which serve the intent of the legislation.



PRODUCT LINE





WORLD-WIDE

Under the flags of almost every nation in the world, people recognize the big yellow machines that do the heavy work. On roads and dams, in fields and forests, at building sites and airports, on docks, in mines and quarries, around factories and warehouses. Behind these familiar machines are Caterpillar people and facilities, our subsidiaries, and the world-wide dealer network.

We are mechanics, secretaries, officers, sales consultants, parts suppliers, service specialists, financial experts, assembly workers, researchers, engineers, independent dealers and salesmen... all working multinationally to build, sell and service those machines that mean progress wherever you see them.

And we make many different ones... because progress takes many directions. This booklet covers the breadth and depth of the Caterpillar product line: the scrapers, graders, loaders, wheel dozers, compactors, crawler tractors, pipelayers, trucks and engines that men use to change the face of the earth... for the better.

Flags represent countries in which Caterpillar has factories, parts depots, offices and affiliates. Arranged alphabetically, they are:

ARGENTINA	CHINA	THAILAND
AUSTRALIA	FRANCE	PANAMA
BELGIUM	GERMANY	SINGAPORE
BRAZIL	INDIA	SOUTH AFRICA
CANADA	JAPAN	SWITZERLAND
FRANCE	MEXICO	UNITED STATES

CATERPILLAR PRODUCT LINE

WHEEL TRACTOR-SCRAPERS/

High speed haulers for building roads, dams, levees, airports. Push loaded and self-loading models—load to capacity in less than a minute. Cushion Hitch models smooth the ride on rough haul roads.

STANDARD SCRAPERS/

Push loaded by tractors, they haul and spread earth at low cost, are the basic machines of most earthmoving spreads. Available in two and three axle versions to match all hauling jobs.



650B
550 Flywheel HP
40/54 cu yd struck/heaped
(31/41 m³)



651B
550 FWHP
32/44 cu yd
(25/34 m³)



650B
530 FWHP
32/44 cu yd
(25/34 m³)



641B
550 FWHP
28/38 cu yd
(21/29 m³)



631C
415 FWHP
21/30 cu yd
(16/23 m³)



621
300 FWHP
14/20 cu yd
(11/15 m³)

TANDEM POWERED/Push loaded earthmovers with additional engine on rear for all-wheel-drive, can climb grades up to 40% fully loaded and work on soft or muddy ground.



660B
950 Flywheel HP
40/54 cu yd struck/heaped
(31/41 m³)



657B
950 FWHP
32/44 cu yd
(25/34 m³)



637
640 FWHP
21/30 cu yd
(16/23 m³)



627
450 FWHP
14/20 cu yd
(11/15 m³)



ELEVATING SCRAPERS/Used to load material into the bowl with chain elevators, chop they load. Each unit is also an excellent finisher.



633C
415 Flywheel HP
12/24 cu yd struck/heaped



J621
615 Flywheel HP
24/48 cu yd struck/heaped

PUSH-PULL/Self-loading pairs hook up separate and haul independently. All-wheel traction and power.



657B Push-Pull
950 Flywheel HP
32/44 cu yd struck/heaped
(25/34 m³)



637 Push-Pull
640 FWHP
21/30 cu yd
(16/23 m³)



627 Push-Pull
450 FWHP
14/20 cu yd
(11/15 m³)

ENGINES/ Diesel and Natural Gas engines—power for industrial, marine, electric power, petroleum and other uses.

Diesel Fuel

INDUSTRIAL RATING

1 term rated HP

2500 RPM
3000 RPM

ELECTRIC SETS

Prime KW 60Hz

50Hz



D399-TA



D398-TA



D379-TA



D353-TA



D349-TA



D348-TA



D346-TA



D343-TA

D342-TA

1300

970

650

430

370

250

180

130

1500

1100

750

500

400

280

200

150

800

600

400

300

200

150

100

75

660

500

330

250

180

140

100

75

550

425

325

225

175

135

100

MOTOR GRADERS / Build and maintain roads, finish grade building sites, highways and airports, construct levees and farm terraces. Operators quickly position blade to ditch, slope or cast. Work tools: blade options, automatic blade controls, blade bowls, ripper-scarifiers.



WHEEL LOADERS / Highly versatile material handlers that load trucks and rail cars, stockpile materials, feed hoppers, sort and carry logs and lumber. All have articulated frame steering for short turns, quick maneuvering. Work tools: general and special buckets, log and lumber forks, ripper-scarifiers.



613
150 FWHP
11 cu yd (8.4 m³)

No. 16
225 Flywheel HP
48,900 lb: basic operating weight
(22,200 kg)

992
550 Flywheel HP
10 cu yd bucket
(7.65 m³)

950
130 FWHP
2 1/2 to 4 cu yd
(1.72 to 3.06 m³)

No. 14E
150 FWHP
30,200 lb
(13,800 kg)

988
325 FWHP
5 1/2 to 6 1/2 cu yd
(4.20 to 4.95 m³)

930
100 FWHP
1 1/4 to 2 cu yd
(1.34 to 1.53 m³)

No. 140
150 FWHP
28,300 lb
(12,900 kg)

No. 120
125 FWHP
24,300 lb
(11,000 kg)

980B
260 FWHP
4 to 5 cu yd
(3.06 to 3.82 m³)

920
60 FWHP
1 1/2 to 1 1/4 cu yd
(1.15 to 1.34 m³)

No. 12F
125 FWHP
27,700 lb
(12,500 kg)

No. 112F
100 FWHP
21,600 lb
(9,800 kg)

966C
170 FWHP
3 to 5 cu yd
(2.30 to 3.82 m³)

and aftercooled T—Turbocharged. NA—Naturally aspirated.

								Natural Gas			
	D336-7A	D334-7A	D333-T	D330-T	3160-NA	3150-NA	3145-NA		G399-7A	G398-7A	G379-7A
HP	350	280	250	165	210	165	160	INDUSTRIAL RATING Continuous HP*	930	700	325
Weight	2200	2200	2200	2200	2800	3000	3200	@ RPM	1400	1200	1200
Capacity	1901 1301	175 150	135 110	90 75	75 50	60 50	50 40	ELECTRIC SETS KW*** 60Hz 50Hz	650 565	500 420	325 280
				125	150						

*High compression ratio with 80° F. (27° C.)

COMPACTORS/Fast modern tamping and sheepfoot compaction on highways, dams, airports. Highly maneuverable with articulated frame steering

WHEEL DOZERS/High speed dozing on roadways and dams, clean up around mining shovels. Articulated steering allows sharp turns for close quarter work.



35 Compactor
400 Flywheel HP
76,900 lb. equipped
operating weight
(35,800 kg)



834 Dozer
400 Flywheel HP
88,800 lb. equipped
operating weight
(40,300 kg)



825B Compactor
300 FWHP
64,800 lb.
(29,400 kg)



824B Dozer
300 FWHP
72,300 lb.
(32,900 kg)



815 Compactor
170 FWHP
38,200 lb.
(17,400 kg)



814 Dozer
170 FWHP
40,700 lb.
(18,500 kg)

TRACK-TYPE TRACTORS/High tractive power for heavy bulldozing, for shattering rock that once required blasting, and for push loading standard scrapers. Log skidders and farm tractors of exceptional power and stability. Work tools: 29 bulldozers, eight rippers, three tool bars.



3x3 D9G
770 FWHP
193,500 lb.
(87,800 kg)

DD9G

770 Flywheel HP
177,400 lb.
(80,500 kg)



D9G
385 FWHP
87,300 lb.
(39,600 kg)



D8H
270 FWHP
63,000 lb.
(28,600 kg)



D7F
180 FWHP
40,000 lb.
(18,100 kg)



D6C
125 FWHP
29,000 lb.
(13,100 kg)



D5
93 FWHP
23,400 lb.
(10,600 kg)



D4D
65 FWHP
16,900 lb.
(7,700 kg)



D6C SA
125 DBHP
26,100 lb.
(11,800 kg)



D5 SA
90 DBHP
20,400 lb.
(9,300 kg)



D4D SA
68 DBHP
15,100 lb.
(6,800 kg)



D5 LGP
93 FWHP
29,000 lb.
(13,100 kg)



D4D LGP
65 FWHP
20,500 lb.
(9,300 kg)

			
Q353-TA	Q343-TA	Q342-TA	Q333-TA
350 1200	350 1800	295 1200	220 1800
250 800	250 210	200 175	150 125

Truck Diesel Engines



OFF-HIGHWAY
HP
@ RPM

HEAVY DUTY
HP @ RPM

MID-RANGE


D349-TA
1130 to 1200
2000


***1693-TA**
375 @ 2000


1180-NA


D348-TA
850 to 990
2000


***1693-T**
325 @ 2100


1150-NA


D346-TA
565 to 600
2000


1674-TA
270 @ 2200


1145-NA


1673-T
250 @ 2200


1140-NA


1140-NA

TRACTOR DRAWN SCRAPERS

Load, haul and dump or short-haul jobs. Often the only practical earth moving method in wet climates. Also built for levees, dams, farm ponds and terraces. Easy loading with low bowl design.



463F
21-28 cu yd struck/heaped
(16.2/14 m³)



463E
19-26 cu yd
(13.6/20 m³)



435E
12/17 cu yd
(9.2/13 m³)



435F
14-18 cu yd
(10.7/13.8 m³)



No. 60
7.9 to 8.3-10.5 cu yd
(5.4/7 or 6.3/8 m³)

TRACK-TYPE LOADERS

Heavy excavators for digging basements, developing building sites, loading from banks. Steel slag cleanout, log loading, other tough jobs. Automatic bucket controls, advanced pedal steering. Work tools: general and special buckets, log and lumber forks, ripper-scarifiers.



983
275 Flywheel HP
4½ to 5 cu yd buckets
(3.44 to 3.82 m³)



977K
120 FWHP
2½ to 3 cu yd
(1.91 to 2.29 m³)



951B
85 FWHP
1½ to 1¾ cu yd
(1.15 to 1.34 m³)



955K
115 FWHP
1¾ to 2 cu yd
(1.34 to 1.53 m³)



941
70 FWHP
1¼ cu yd
(1.0 m³)

PIPELAYERS

Handle pipe for oil and gas pipeline builders, and for water and sewage contractors. Booms, from 15 to 28 feet (4.6 to 8.5 m). Hydraulically controlled counterweights for stability. Flywheel horsepower from 93 to 365.



594
200,000 lb lift capacity
(90,700 kg)



583H
140,000 lb
(63,500 kg)



571
60,000 lb
(27,200 kg)



572
90,000 lb
(40,800 kg)



561C
40,000 lb
(18,100 kg)

OFF-HIGHWAY TRUCKS

High-speed rear-dump haulers for ore, mine overburden, quarry rock, tough highway excavation. Speeds to 43 mph (69 km/h). Oil-pneumatic suspension smooths the ride for fast loading. Cat oil-cooled disc brakes are non-fading.



773 Power Shift
600 Flywheel HP
50-ton capacity
(45.4 t)



769B Power Shift
415 FWHP
35-ton capacity
(32 t)

COM

ing and
dams a
ulated f

WHE

ing on
mining
sharp to



35 C
100 Flyw
78 500 lb
operating
(35 800 lb)



825B
300 Flyw
64 600 lb
(29 400 lb)



815 C
170 Flyw
38 200 lb
(17 400 lb)



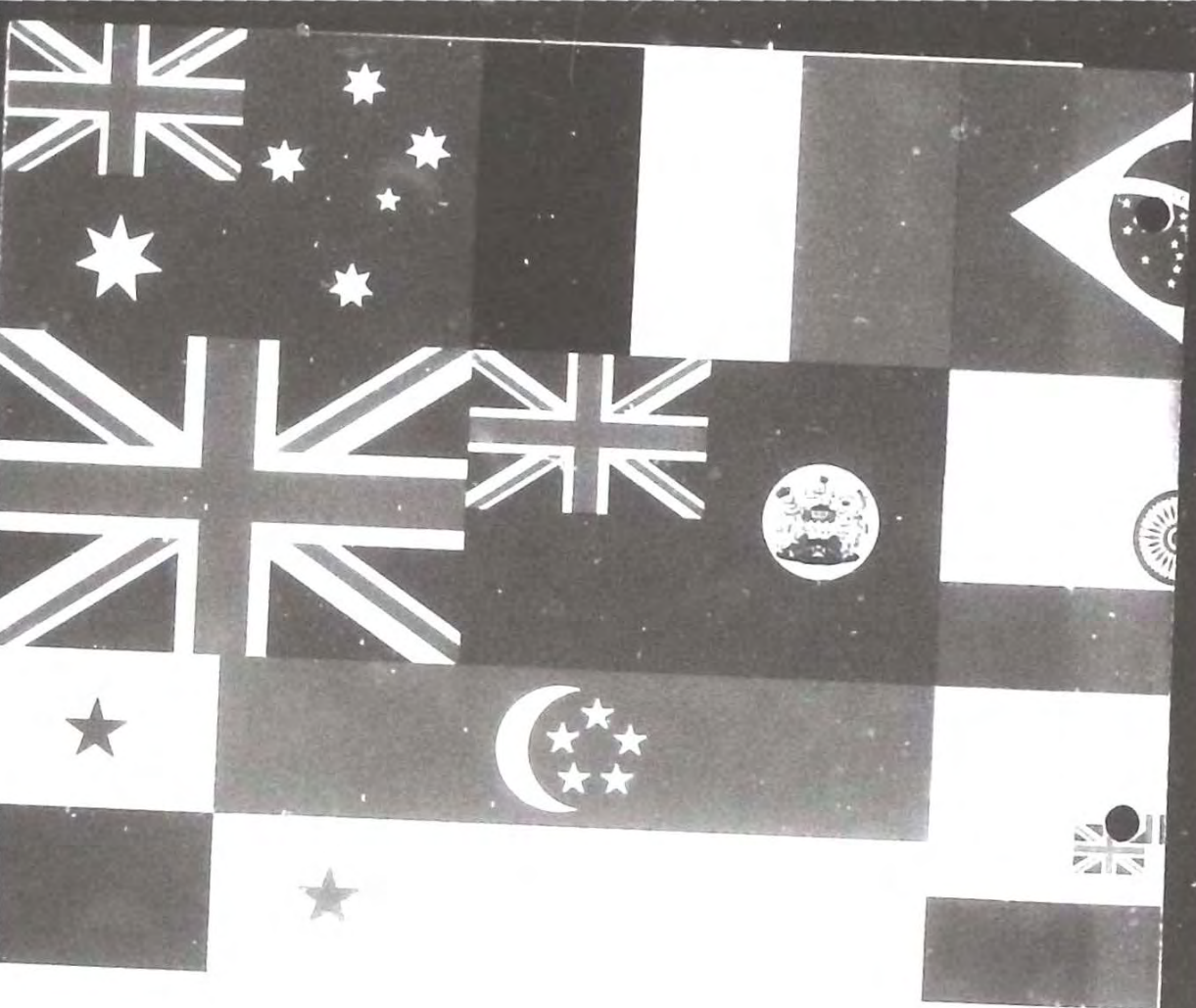
Q353-TA

3

1200

250

200



ORAL TESTIMONY

by

CATERPILLAR TRACTOR CO.

to

State of Illinois

Pollution Control Board

at

Regional Hearing on Asbestos and Spray Insulation

Waukegan, Illinois

October 19, 1971

Mr. Chairman, members of the panel, my name is Fred Engelking. I joined Caterpillar in Peoria in 1937 after receiving a degree in Chemical Engineering from Purdue University. I have been working on friction materials for over 20 years. Presently, I direct the Research and Development programs for nonmetallic materials, including friction material, in the Research Department. I have been a member of the American Society for Testing Materials subcommittee on Friction Materials since 1957.

Caterpillar is pleased to have the opportunity to appear before the Board today. We have six manufacturing facilities in the state, and are the largest private employer in the State of Illinois, with over 41,000 employees. We are vitally interested in the efforts of the Illinois Pollution Control Board to guard the health and environment.

I am here today to present to you information that I hope will assist you in efforts to regulate the use of asbestos and spray insulation in the State of Illinois. Copies of our complete submission are in your hands, and it is our sincere hope that each of you will find time to read the entire booklet. My oral remarks are included in Appendix B.

The Caterpillar product line includes primarily earthmoving equipment. All of this equipment utilizes asbestos brake linings and it is for this reason that we are concerned with Section 702 of Part VII of the proposed Regulation.

Appendix A of the booklet provides a listing of the Caterpillar product line which includes 52 models of earthmoving equipment and 53 models of material handling equipment.

The lack of a definition for the word "vehicle" in the proposed Regulation is also of concern. Typically, regulatory statutes have differentiated between motor vehicles and earthmoving equipment. There are suggestions for the definition of "vehicle" in the written testimony.

There are reasons for our concern:

Asbestos is used in all types of brakes in all Caterpillar machines because it has outstanding safety and durability characteristics. In spite of many years of research, there are no known safe substitutes for asbestos brake linings for our type of equipment. I would like to acquaint you with a few types of machines other than the familiar track-type tractor.

SLIDE 1

Starting in the upper left hand corner and going clockwise, there is a wheel tractor, motor grader, off-highway truck and wheel tractor-scraper. A wheel tractor loader is in the center.

Unlike most vehicles you may know about, we have 5 different types of brakes. They are dry shoe brakes, dry caliper disc brakes, dry band brakes, oil cooled band brakes, and oil cooled disc brakes.

SLIDE 2

Conventional shoe brakes with dry linings are used in wheel tractor-scrappers, motor graders and most wheel loaders. These brakes are always shrouded to minimize the entry of debris from the outside. Brake lining wear products can be emitted into the environment from this type brake. Smaller sizes of this type are used on passenger cars.

SLIDE 3

The second type is called a caliper disc brake. The asbestos-containing pads are forced to grip the disc to produce the braking power. This is the most exposed type of brake and, obviously, wear products can easily enter the environment. Many U.S.-made passenger cars are now using this type brake on the front wheels only. We use caliper disc brakes on all wheels of some loaders.

SLIDE 4

The next three types of brakes are totally enclosed and may be less familiar to you. Wear products cannot be emitted into the environment due to their containment. Track-type tractors and track-type loaders use band brakes. In this photo, the covers have been removed from the compartments so that the brakes are visible. Our larger machines use band brakes bathed in cooling oil such as is shown. The smaller machines use a dry-type band brakes without oil.

SLIDE 5

The fifth type is the totally enclosed oil-cooled disc brake used in our off-highway trucks. These have many discs which are forced to rub against mating steel discs and provide braking power. Due to large rubbing surface area and the oil media, there is very little wear in this type brake. There are no emissions into the environment.

SLIDE 6

This is a typical friction disc with its mating disc as is used in oil-cooled, multiple disc brakes shown in the previous slide.

Break Wear products:

It is my understanding that the technical paper by Mr. Jeremiah Lynch of the U.S. Public Health Service on "Brake Lining Decomposition Products" has been entered into the record in earlier testimony before this Board at Granite City, Illinois.

We have done similar, but less extensive work, on brake decomposition

SLIDE 7

products. This slide shows scrapings from an unused brake as photographed with an electron microscope at high magnification. So you will have a better feel for this high magnification, the thickness of human hair at this same magnification would cover a screen twice as large as we have in this room.

I have included a 5 micron scale on this and the three succeeding slides because Section 601 in the proposed Regulation states that "only fibers of 5 microns or greater in length...shall be counted" in the emissions from a factory into ambient air. We believe that if it is safe for a factory to emit fibers less than 5 microns in length, it should also be safe for a brake to emit fibers less than 5 microns in length into the ambient air.

Additional research by qualified medical personnel may even show that asbestos fibers much larger than 5 microns are safe in the ambient air. Since these are fibers scraped from unused brake material, they are larger than 5 microns.

SLIDE 8

Some asbestos fibers from new friction material were heated in an oven to remove the water of crystallization inherent in asbestos. This destroys the fibrous nature, and mechanically rubbing the deteriorated fibers further reduces the fibers to powder. This is a simulation of the formation of brake wear products. You will note no fibers are longer than 5 microns.

SLIDE 9

A sample of brake wear products was taken from a dry brake compartment. There are a few fibers showing and they are much shorter than 5 microns. Dust or clumps of dust occupy most of the picture.

SLIDE 10

This last slide on brake wear products from a dry brake compartment also shows very few fibers and none of significant size. There are many dust particles.

We believe that our limited study substantiates the findings of Mr. Lynch of the U.S. Public Health Service, that few fibers are emitted as wear products from brakes.

Smoothness of brake operation is vital to safe operation of Caterpillar equipment. You may have experienced a malfunctioning passenger car brake

SLIDE 11

which grabbed and threw you forward at the end of a stop. This pair of curves shows what is happening when you brake a vehicle. In curve A, asbestos material

is stopping the vehicle. The brake torque or stopping power builds up at the beginning, stays smooth and constant, and finally increases slightly at the end of the stop. The sintered metal friction material produces rough braking in curve B. There is a lot of vibration and noise followed by a drastic increase in torque at the end. The sudden increase in stopping power causes material to spill out of machines.

SLIDE 12

If this loader is backing up with a raised bucket full of dirt or rock, and the machine stops abruptly, due to grabbing brakes, the material will spill out of the bucket and end up in the operator's lap. Smooth braking action is absolutely necessary for safe operation of our machines.

Safety:

When a brake is designed for a Caterpillar machine, there are three vital factors: safety, durability, and smoothness of stopping. The last two factors enhance the safety factor. The brake must be able to stop the machine in a reasonable distance, time-after-time. Asbestos brake materials have proven performance and durability.

I would like to summarize and make some recommendations. Caterpillar Tractor Co. questions whether the intent of the Asbestos and Spray Insulation Regulations is actually accomplished by the proposed ban of asbestos brake linings. If research work and data on ambient concentration levels of asbestos fibers which are detrimental to society establish a need for asbestos brake control and/or regulation, Caterpillar recommends that Section 702, Part VII of R71-16 (Asbestos and Spray Insulation Regulations) be modified to allow the following exceptions:

1. That asbestos brake linings (friction material) may be used in a brake system when the friction material operates in either a dry or oil-wetted environment in a totally enclosed brake compartment which prevents the emission of brake wear products into the ambient air.
2. That asbestos brake linings (friction material) may be used in a brake system, when the friction material operates in a nonenclosed brake compartment, until conclusive medical evidence proves that the emitted brake wear products contain harmful amounts of asbestos fibers.

If all evidence indicates the need for asbestos brake controls and/or regulations, Caterpillar would further recommend that reasonable threshold limits of asbestos fiber be established by qualified personnel (based on scientific fact and foreseeable technological advances). Test procedures which measure the concentration of asbestos fibers must also be developed which are understandable, repeatable, and realistic. Place the burden on the manufacturer to design his product to meet reasonable, attainable standards -- don't just prohibit the use of asbestos for brakes.

As mentioned at the beginning of this booklet, Caterpillar Tractor Co. agrees in principle with the objectives of the Environmental Protection Act. The Company stands ready and willing to assist the Board in the establishment of asbestos regulations which serve the intent of the legislation.

LIST OF SLIDES

<u>SLIDE NO.</u>	<u>REFERENCE FIGURE NO.</u>
1	1
2	7
3	8
4	4
5	5
6	6
7	9
8	10
9	11
10	12
11	2
12	3

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— R71-16

EXHIBIT 27

—

Bd Ex. #27

RH-16

The Method Used by the U. S. Public Health Service
for Enumeration of Asbestos Dust on Membrane Filters

George H. Edwards and Jeremiah R. Lynch*

Introduction

In the early epidemiologic studies of the asbestos industry by Fulton (1935) and Dreesen (1938), the impinger was settled upon as the standard method for appraising dustiness. Since few fibers were seen it was necessary to count both grains and fibers to obtain a statistically useful count despite the assumption that asbestosis was caused by fibers. This inconsistency notwithstanding, dust control based on impinger results has greatly reduced the incidence of asbestosis where it has been conscientiously applied in the asbestos textile industry. However, a number of investigators (Lynch and Smith, 1955; Bell, 1955) have suggested other health hazards associated with asbestos, and a more relevant and sensitive method is required for estimation of the hazard, especially in industries using only pure asbestos in their product.

A further weakness in the impinger method is that it is not amenable to long personal samples. It is believed that personal samples best approximate the worker's actual exposure, and Koach (1966) has recommended a sample duration of ten work shifts for mineral dusts.

New methods were therefore needed, and among those studied were fiber counts at 430X phase contrast on membrane filters that had been made transparent. This paper deals with the development and standardization of this method.

The principal variable which needed to be considered was the mounting procedure in relation to maximum transparency and the effect of time on the dimensional stability of the mount. Methods using immersion oil, air-plane glue or acetone were found to be either too cumbersome or resulted in an imperfectly cleared or mottled filter, particularly as viewed with phase contrast optics. In methods which involved the application of liquid over the dust deposit the movement of the meniscus across the deposit sometimes caused redistribution of the dust. The use of a 50-50

*U. S. Department of Health, Education, and Welfare
Public Health Service - NCHH
Occupational Health Program
1014 Broadway, Cincinnati, Ohio 45202

mixture of diethyl oxalate-dimethyl phthalate was attempted and found to result in satisfactory clearance. However, since this medium dissolved the filter, the migration of particles resulted in changing counts with time and needed to be examined to evaluate the method.

Sampling Procedure

Samples for count are collected on Millipore[®] type AA filters by personal samplers operated by battery powered pumps worn by the employees, and as simultaneous samples collected in conjunction with impingers. The filters are contained in plastic filter holders and are supported on thick pads which also aid in controlling the distribution of air through the filter. Several methods of admitting air to the filter have been attempted. With the face cap removed and the filter completely exposed, the air may enter the filter evenly, but large high velocity particles and foreign objects may also hit the filter and possibly cause damage. As an alternative, the face cap may be left in place and the small plug removed. The passage of air through this 4 mm opening at a flow rate of 2 liters per minute produces a velocity of 265 cm/sec, which yields an impaction parameter of 0.001 for 1 micron (μ) unit density spheres. Although this impaction parameter would predict a very low collection efficiency of 1 μ particles by impaction, some larger particles would tend to be impacted on the center of the filter. This was found not to affect count distribution, which is dominated by small particles; however, it does result in obscuring a small portion of the filter, particularly if many large particles are present, as in friction product plants. The third alternative was to drill six additional 4 mm holes in some face caps and to fit these perforated caps to the filter holder during sampling. This reduced the velocity to 38 cm/sec thus eliminating the impaction problem while still protecting the filter from damage.

Mounting the Sample

The mounting medium used in this method is prepared by dissolving 0.1 gm of membrane filter material per ml of a 1:1 solution of dimethyl phthalate and diethyl oxalate. The exact proportions of the 3 components are not critical, but the medium must have as high a viscosity as possible without being difficult to handle. The index of refraction of the medium thus prepared is $n_D = 1.47$.

To prepare a sample for microscopic examination, a drop of the mounting medium is placed on a freshly cleaned standard (25 mm x 75 mm) microscope slide, using a dropper or applicator. The volume of the drop is approximately 0.05 ml. A wedge-shaped piece about 1 cm by 2 cm is excised from the filter using a scalpel and forceps, and placed dust side up on the drop of mounting solution (Figure 1). A #1-1/2 coverslip carefully cleaned with lens tissue is placed over the filter wedge. Slight pressure on the coverslip achieves contact between it and the mounting medium.

Clearing of the filter with this method is slow, requiring about 15 minutes. The sample may be examined as soon as the mount is transparent.

The transition of the dust-supporting substrate from translucent, solid membrane filter cellulose ester material to a transparent, optically homogeneous gel is very gentle, and at no time does a meniscus sweep across the dust deposit. Rather, the substrate slowly softens beneath the dust, and there is no opportunity for washing away or redistribution of the dust.

The optical homogeneity of the resulting mount is nearly perfect, with only a slight background granularity under phase contrast, which disappears within 1 or 2 days.

Stability

Observers noted a reduction with time in apparent concentration for samples mounted with only dimethyl phthalate and diethyl oxalate. By observation, this loss was due to the migration of the dust particles outward from the center through the gelatinous material of the fixed mount, with a decrease in areal concentration. To determine the magnitude of this loss, a group of samples was mounted using 1:1 dimethyl phthalate and diethyl oxalate only, and counted at one day to three-day intervals for periods ranging up to 76 days. Counts were displayed as fractions in percent of the original, or first day counts (Figure 2). At the end of one week, samples mounted by this method yielded only 75% of their original values. By 30 days after mounting, the counts using this method had declined to 40% of their first day value, and continued at this level with no further decline until counting was terminated 76 days after mounting.

A group of samples mounted in the method described in the section "Mounting the Sample" using high-viscosity mounting media were counted at one-day intervals for eleven days. No loss of areal concentration was apparent over this short period and a second group was similarly mounted and counted for a period of forty-six days. The composite of these two series is shown in Figure 2. No loss of concentration was detectable during the first 30 days, but the count declined to 85% of its original value by the 46th day, when the series was terminated.

A 6 mm square of membrane filter with a vacuum-deposited film of carbon was mounted in the manner described to provide a macroscopic observation of the distortion of the filter areas. After a few days, the square began to disintegrate at the edges, but the major portion showed no discontinuity during the initial 30 day period. By the end of 62 days, most of the square had disintegrated and the fragments migrated over an area 16 mm by 17 mm.

Evaluation

The filter samples mounted in the manner previously described are evaluated in terms of grain and fiber concentration, and fiber size. Binocular research microscopes equipped with fixed mechanical stages have been used. Phase contrast optics fitted to these instruments include an Abbe condenser with rotating phase turret and a 4 mm "high-dry" achromatic objective used for all membrane filter determinations. In all cases 10X Huygens eyepieces, one of which contained a Porton reticle at the level of the field-limiting diaphragm, were used. The left half of the Porton reticle field served to define the counting area or field.

A ribbon filament illuminator was critically adjusted to provide Kohler illumination through a clear blue filter.

Twenty field located at random on the sample were counted and total grains, total fibers, fibers greater than 5 μ , and fibers greater than 10 μ were recorded. Any particle having an aspect ratio of 3 or greater was considered a fiber.

Recording of Data

Since it was recognized early in the study of the asbestos products industry that the large number of samples to be collected would present a burdensome data processing problem, the data were collected on forms designed for transcription to punch cards (Figure 3). Computer programs were then written to calculate dust concentrations and to produce statistical summaries consisting of mean, standard deviation, median, etc., for any group of samples by plant, operation, job, type, etc. These programs, written in Automath (Fortran II) are applicable to any problem using the sample and count forms as source documents. Additional analyses possible are correlation and regression of simultaneous samples and Poisson index of dispersion of field counts for various counters.

Summary

A method for sampling and counting asbestos dust using membrane filters has been developed and evaluated. The mounting medium developed clears the filter gently so as to not redistribute the dust deposit, and produces a mount with adequate dimensional stability for a period of thirty days. This method has been used in developing environmental data in connection with the Public Health Service study of the asbestos products industry, and is recommended for research studies in other dusty trades although not for application to Threshold Limit Values. A system of data handling involving electronic computer processing, which is also applicable to other studies, was discussed.

References

- Doll, R. Mortality from Lung Cancer in Asbestos Workers. *British Journal of Industrial Medicine*, 12: 81-85 (1955).
- Dreesen, W. C., J. M. Dalla Valle, T. I. Edwards, J. M. Miller, and R. R. Seyers. A Study of Asbestosis in the Asbestos Textile Industry. Public Health Bulletin No. 241, U. S. Government Printing Office, Washington, D. C. (1938).
- Fulton, W. B., A. Dooley, J. L. Matthews, and R. L. Houtz. Asbestosis. Pennsylvania Department of Labor and Industry, Special Bulletin 42 (1935).
- Lynch, K. M. and W. A. Smith. Carcinoma of Lung in Asbestos-Silicosis. *American Journal of Cancer*, 24: 56-64 (1935).
- Roach, S. A. A More Rational Basis for Air Sampling Programmes. *Am. Ind. Hyg. Assn. J.*, 27, 1 (Jan-Feb. 1966).

NOTE:

Millipore[®] filters are named in this paper since the method was developed using this brand of filters. However, this does not imply indorsement by the United States Department of Health, Education, and Welfare or the Public Health Service. Similar techniques may be developed for other filter brands.

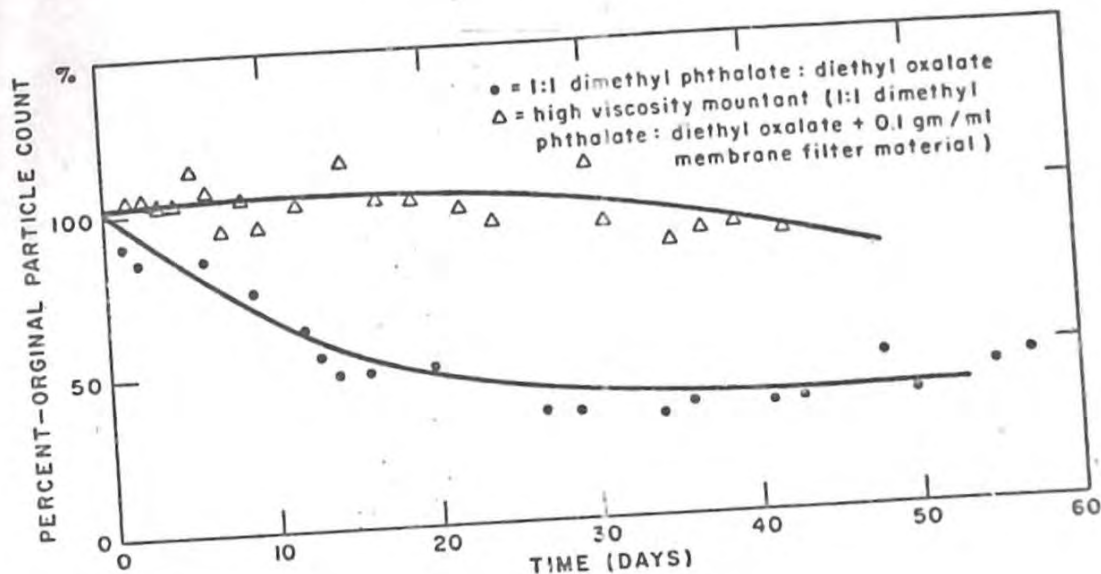


Figure 2. Decrease in percentage of original particle count with time for the original 1:1 dimethyl phthalate: diethyl oxalate mixture and for the high viscosity mixture containing 0.1 gm/ml of membrane filter material.

FCS 7/64

3 1 0 1							
MO. DAY YEAR				PLANT NO.		SAMPLE NO.	
[][][][]				[][]		[][]	
TYPE		NATURE		COUNT		COUNT POWER M.NO.	
[M][N]		[B][G][P][S]		[C][R]		[][][][]	
ILLUMINATION		GRAINS FIBERS > 5 > 10		NO. CTS			
[L][F][P][C]		[][][][][][]		[][][][]			
25		26		27		35 36 37	
BLANK		BLANK		BLANK			

GRAINS		FIBERS		
		TOTAL	> 5	> 10
1	2	3	4	5
39		39		39
43		42		42
46		44		44
49		46		46
52		48		48
		50		50
		52		52
		54		54
		56		56
		58		58
		60		60
		62		62
		64		64
		66		66
		68		68
		70		70
		72		72
		74		74
		76		76
		78		78

BLANK NO. _____

Σ _____

AVG. _____

Figure 3. Count tabulation form used for the evaluation of dust samples taken on membrane filters. M = Membrane filter, U = Elutriated membrane filter, B = Breathing zone sample, G = General a sample, P = Personal sample, S = Sequential sample, C = Count (1st), R = Recount. "COUNT" provides for the technician's initials, "OBJ POWER" indicates the magnification, "M.NO" provides for the microscope number for calibration purposes, and LF and PC are light-field and phase contrast.

— R71-16

EXHIBIT 28

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OWENS-CORNING FIBERGLAS CORPORATION

FIBERGLAS TOWER, TOLEDO, OHIO 43601 (419) 259-3000

S. H. THOMAS
Director of Environmental Control

December 13, 1971

Samuel T. Lawton Jr.
Illinois Pollution Control Board
189 West Madison Street, Suite 900
Chicago, Illinois 60602

Dear Mr. Lawton:

We have the following comments with regard to the proposed final draft of Asbestos Regulations R 71-16.

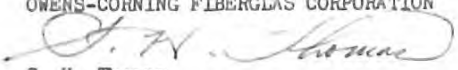
1. Part V, #502. We believe that the use of the word "enclose" in this statement is too restrictive in this sense. We strongly recommend that the word "control" be substituted.

One possible interpretation of the present statement would be that enclosures have to be built around processing equipment. This would make it almost impossible to operate.

2. Part V, #504. We believe the present statement provides opportunity for harassment and burdensome costs. We recommend that the statement be changed to require annual sampling on asbestos emission sources. When major modifications in equipment and processes are made and when sampling results demonstrate the need, more frequent sampling may be required.
3. Part V, #501. The June 30, 1972 time for control of asbestos emissions, i.e. no visible emissions and no emissions of concentrations of asbestos to exceed 2 fibers/cubic centimeter, is unreasonable. We recommend that in order to properly engineer, procure, construct, startup, and evaluate an emission control system that will meet regulatory standards while controlling costs each project should be considered on its own merits.

Sincerely,

OWENS-CORNING FIBERGLAS CORPORATION


S. H. Thomas
Director of Environmental Control

VJK/ds

cc: G. E. Devitt - 11
Dr. J. Konzen - 11
W. L. Kreutz - 26
K. Preston - Bloomington
J. Young - 12

— R71-16 —

EXHIBIT 30

30
United States Gypsum Company

101 South Wacker Drive, Chicago 60606

December 15, 1971

RECEIVED

DEC 15 1971

Illinois Pollution Control Board
189 West Madison Street
Chicago, Illinois 60602

POLLUTION CONTROL BOARD

Attention: Mr. Samuel T. Lawton, Jr.

Re: Proposed Asbestos and Spray Insulation and
Fireproofing Rules and Regulations of
November 30, 1971.

Dear Mr. Lawton:

United States Gypsum Company manufactures numerous products used throughout the country in the economical building of homes and business structures. Some of these products involve the use of asbestos, including, for example, the joint compound used in wallboard construction. The Company maintains a plant for the production of joint compound in Chicago, Illinois. Sales of the product exceed \$4,000,000 for 1971. The plant machinery is worth approximately \$650,000.

Part V, Section 501(a) of the above rules and regulations requires that by June 30, 1972, no visible emission of particulate matter discharge from the manufacture of our joint treatment product, and that no asbestos fiber concentrations, exceeding two (2) fibers per cubic centimeter of air, emit from such manufacture. While some ambiguity exists, we assume that this requirement obtains both inside and outside the manufacturing plant.

A review of this matter leads us to conclude that the manufacturing process now being used cannot be modified to conform to the new requirements by June 30, 1972, if ever. We do not know that all the air within the plant can ever be filtered sufficiently with the existing equipment, or that dust collector equipment is now, or will be in the foreseeable future, available to reduce exterior plant emissions to the required levels. While our research department has been working for some time to discover satisfactory substitutes for asbestos in this

.../...

Illinois Pollution Control Board
Chicago, Illinois 60602

- Page 2 -
December 15, 1971

Re: Proposed Asbestos and Spray Insulation and
Fireproofing Rules and Regulations of
November 30, 1971.

product, none has been discovered yet.

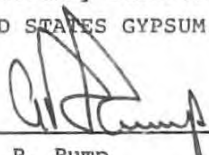
In these circumstances, we are faced with stopping production at the plant or using unsatisfactory substitutes for asbestos. We believe that this will result in losing the plant's market for the product, which will be supplied by sources outside the State of Illinois.

Accordingly, we suggest either that enforcement of Part V be held in suspension until such time as effectuation of the stringent requirements becomes technically and economically feasible or that the manufacturing emission standards be revised to be no more stringent than the standards in the "Emergency Standard for Exposure to Asbestos Dust", Title 29, CFR-Part 1910 Occupational Safety and Health Standards. Effective December 7, 1971, 36 Federal Register 8754.

We suggest that the regulations might also contain some provision for a gradual phase-in of the new program. In view of the sudden shift from no control to severe control, and in view of the substantial amounts of existing equipment which may otherwise be rendered useless, a time table might be established to permit transition from existing Federal standards to the proposed Illinois standards.

Please feel free to contact us if you have any questions with regard to this matter. We will be pleased to provide you with any additional information which you might require.

Respectfully submitted,
UNITED STATES GYPSUM COMPANY

By: 

A. R. Rump
Vice President - Operations

ARR/is

QUALITY PARK
LEARNESCOIR
VERTICAL FILE POCKET
9 1/2" x 14 1/2" x 5 1/4" Exp.
152C-G

Transcripts

R 71-16

RECEIVED

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POLLUTION CONTROL BOARD

BEFORE THE
ILLINOIS POLLUTION CONTROL BOARD

In the Matter of:

ASBESTOS REGULATIONS, CONTROL
OF SPRAY INSULATION.

No. R71-16

Waukegan Public Library
Waukegan, Illinois
October 19, 1971

LEON M. GOLDING & ASSOCIATES
CERTIFIED COURT REPORTERS
 178 WEST MADISON STREET * SUITE 110
 CHICAGO, ILLINOIS 60603
CENTRAL 6-4949

I N D E X

<u>WITNESSES</u>	<u>NARRATIVE</u>	<u>CROSS</u>
Gaddam M. Reddy	196	
Clifford L. Sheckler	204	
Fred Engelking	212	225
Howard E. Ayre	229	242
James Middleton	264	

ANG:hc

BEFORE THE
ILLINOIS POLLUTION CONTROL BOARD

In the Matter of:

Asbestos regulations, control of
spray insulation.

} No. R 71-16

Held on Tuesday, October 19, 1971, commencing
at the hour of 10:00 o'clock a.m., in the Waukegan
Public Library, Waukegan, Illinois, Mr. Samuel
T. Lawton, Jr., Hearing Officer, presiding.

BEFORE:

Mr. Samuel T. Lawton, Jr., Hearing Officer.

ALSO PRESENT:

Mr. Timothy Harker, Administrative
Assistant, Pollution Control Board.

Members of the Press
Members of the Public

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HEARING OFFICER LAWTON: May we get underway, please.

This is the third hearing on the proposed asbestos regulations that have been adopted for purposes of hearing only by the Pollution Control Board of the State of Illinois.

We have had one hearing in Granite City, which was very meagerly attended. We had a very good hearing in Chicago last week and, I am pleased to see how many people have come to this particular hearing.

Now, we have a list of witnesses that have previously indicated their desire to testify.

At this time I will read those that have signified a desire to testify, and then I will ask anyone here who is not on that list who wishes to testify to please make out a little card and give it to Mr. Harker on my right and we will put you in the line-up.

If at the moment you have not made up your mind whether you want to testify that is all right, too. The only thing is that by giving us your name now, you are at least assured of your position on the schedule.

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We have indicated so far a representative of the Illinois Chamber of Commerce. Is he or they present? No.

From the Environmental Protection Agency, Mr. Reddy is here.

MR. REDDY: Yes.

HEARING OFFICER LAWTON: From the National Insulation Manufacturers' Association we have gentlemen here, and Caterpillar Tractor, all right.

Now, I will ask that those who wish to testify who have not been itemized in that list, if they would please do so and give us a slip, if that is your desire.

As I said, if you do not choose to do it, at the time the hearing is completed so far as the people I have listed are concerned, anybody that wants to speak will certainly be able to do so.

The proceedings are being transcribed. Those of you who wish to have copies of the transcript, if you will make arrangements with Mr. Goldstine on my left, you will be able to obtain a copy at your cost, I might add.

The hearing today may or may not be the last one, depending upon what develops today and

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what the Board chooses to do with regard to the proposed regulations.

If the regulations are proposed for adoption and in essentially the same form that they have been previously published, then it is possible that there will not be a further hearing.

If, in fact, the Board feels that further hearings are necessary or, if we propose for adoption regulations that are substantially different from those that are presently under consideration, then it is quite possible the Board will have a further hearing.

In any event, irrespective of whether the Board does or does not have a further hearing, the record of this proceeding will remain open for three weeks, during which time anyone may submit written documentation to the Board which will be incorporated in the record and considered at such time as the regulations are discussed.

Now, at the first hearing in Granite City, I introduced into the record eleven exhibits, which I will summarize very briefly, so you will know what the record consists of; and, then at the Chicago hearing we introduced eleven more. At the Granite

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City hearing we introduced the Newsletter as Exhibit 1, which was the publication of the proposed regulations.

We introduced the formal newspaper notice certificates as No. 2.

No. 3 was the Litton Asbestos report. No. 4 was an article by Dr. Selikoff in the March, 1969 issue of the magazine Environment.

No. 5 was a publication by Dr. Thomson, "Asbestos as an urban air contaminant," in the Archives of Pathology, May, 1966.

Exhibit No. 6 is a publication entitled, "The inter-relationship of selected asbestos exposure indices," in the American Industrial Hygiene Association Journal of September, 1970.

No. 7 was an article by Dr. Nicholson entitled, "Asbestos air pollution in New York City," presented at the International Air Pollution Conference in Washington in December of 1970.

No. 8 was an article on, "Brake lining decomposition products," by Lynch in the Journal of Air Pollution Control Association, December, 1968.

No. 9 was an article by Dr. Cralley,

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"Identification and control of asbestos exposures," in the February, 1971 issue of American Industrial Hygiene Association Journal.

No. 10 was a report of the Philadelphia Department of Public Health, August 31, 1970.

No. 11 was a composite exhibit of testimony by various people before the Chicago City Council Environmental Control Committee at the time when they were considering asbestos regulations.

Those are all identified and admitted into the record at the Granite City hearings.

At Chicago, Exhibit No. 12 was the municipal code of Chicago relating to asbestos control.

No. 13 was a National Insulation Manufacturers' Association publication.

No. 14 was a publication in the American Industrial Hygiene Association by Dr. Cralley and others on, "Fibrous and mineral content of cosmetic talcum products."

No. 15 was a publication on cigarette smoking and asbestos in the American Review of Respiratory Disease, Volume 104, 1971.

No. 16 was an article entitled, "Asbestos fibers in beverages and drinking water," in the

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Nature Magazine of June, 1971.

No. 17 was an article from Science Magazine, Volume No. 173 entitled, "Talc-treated rice and Japanese stomach cancer."

No. 18 was a publication from the Environmental Equipment Users' Association entitled, "Recommendations for handling asbestos."

No. 19 was a publication in the National Academy of Science, 1971, entitled, "Airborne asbestos."

No. 20 was a document prepared by Dr. Harwood, who is here today, and which was published by the Institute for Environmental Quality on the request of the Pollution Board dealing with asbestos and asbestos products.

No. 21 was the report or testimony of Dr. Nicholson at the Chicago meeting.

No. 22 was the statement of Dr. Roemer at the Chicago Meeting, and No. 23 was material furnished by Mr. Punsak on behalf of John-Mansville and others, which was incorporated as a composite exhibit.

Those are the exhibits that are presently in the record.

At the Chicago meeting, Drs. Harwood,

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Nicholson and Roemer and Mr. Punsak were the principal witnesses.

There were one or two others who spoke from the floor and certain questions were asked.

Now, are there any others, beyond the ones whose names I have previously read, or whose organization I indicated wished to testify -- and if you would let me have your name and organization?

Apparently not.

We will hear first from a representative of the Illinois Chamber of Commerce, if there is one present. That organization has indicated a desire to testify at this hearing.

Is there anyone here from that organization? If they show up later, they will not be foreclosed from testifying.

We will move on, then, to the witness from the Environmental Protection Agency. I will ask that all those present who wish to testify will please rise and be sworn at this time.

(Witnesses sworn.)

You can use the podium, if you like. If you would like to put it in some other position, that is all right.

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Is that all right for you?

MR. REDDY: That is fine with me.

HEARING OFFICER LAWTON: I think that way the audience and the Board and the reporter, who is the most important man here, can hear.

GADDAM N. REDDY,

being first duly sworn, testified in the narrative form as follows:

HEARING OFFICER LAWTON: Will you tell us at this time a little bit about who you are and your background and your present association and a general outline of what it is you plan to comment on, please.

THE WITNESS: My name is Gaddam N. Reddy, G-a-d-d-a-m, last name is spelled R-e-d-d-y.

I am employed as an Environmental Protection Engineer with the Division of Air Pollution Control, State of Illinois, Environmental Protection Agency.

I have a Bachelor's degree in chemical engineering from Osmania University and a Master's degree from Southern Illinois University.

I have been requested today by Dr. John J. Roberts, Manager of the Division of Air Pollution Control, to appear today and present the following

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comments regarding the proposed Asbestos and Spray Insulation Regulation.

The Division of Air Pollution Control of our agency concurs with the Pollution Control Board in the need for regulations governing the emission of asbestos and spray insulation material.

The division permit program will be able to handle the requirements implicit in Rule 401(b) of Part IV and Rule 601(a) of Part VI.

Computer process data of the state-wide emission inventory program will be completed in mid-November.

The four digit asbestos code will enable the agency to form a list of potential asbestos emitters which can be the basis of an asbestos inventory of manufacturing facilities, which will be done early next year.

It is requested from the Board that the first paragraph of Part II be changed to read as follows:

"No commercial activity, not otherwise hereinafter prohibited, involving the potential discharge of asbestos fiber or asbestos-containing materials into the

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environment from the construction, alteration, repair or demolition of a structure or from the processing or manufacturing of asbestos-containing products shall be conducted unless the person or entity in charge of such activity complies with the following regulations:"

This would then require all operations that have a potential of emitting asbestos or asbestos-containing materials into the open atmosphere to comply with the regulations rather than only those that do emit asbestos into the open atmosphere.

We are also requesting the deletion of the requirement for an agency operating permit for such activities as construction, alteration, repair, or demolition. We do not at present have a clear estimate of the number of situations of this kind, but this aspect of the proposed standards seems unattainable with the present manpower level of the agency.

We are asking to encourage local commerce to set up a permit program and/or surveillance of such activities would be appropriate. These activities, nevertheless, should be required to

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comply with Rules 201(a, b and c) of the proposed regulation.

If Rule 302(a) is intended to stop all activity on all floor areas not related to the spraying of asbestos when the spraying is in progress, we feel that this rule is unreasonable. The rule should be reworded such that it would relate to the particular area in which the spraying is being conducted.

We are proposing a change in Rule 401(b) to read as follows, start of quotation marks:

"The mechanical exhaustion of dust from such enclosure to the ambient air is prohibited unless such exhaust system is equipped with a properly-sized fabric filter for dust collection or any equivalent device as approved by the agency."

The Environmental Protection Agency requests the Board to add the following rules to Part VI of the proposed rules and regulations.

Rule 601(b).

"No factory, plant or enterprise which engages in the processing or manufacturing of any asbestos-containing product shall enclose all asbestos-handling

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facilities so that exhaust air can be ducted through necessary Air Pollution Control equipment and the gases emitted into the open atmosphere sampled."

Change Rule 601(b) and 601(c) to 601(e) and 601(d) respectively.

Add Rule 601(c) as follows:

"Any person operating factory, plant or enterprise which engages in the processing or manufacturing of any asbestos-containing product shall, at reasonable intervals, prescribed by the agency, sample the exhaust from such factory, plant or enterprise. The ambient air at the boundary of the plant or premises shall also be sampled at reasonable intervals or as required by the agency."

The agency, at present, does not have either sufficient equipment or personnel to conduct tests at each and every potential manufacturing source of asbestos.

In keeping with the philosophy that a license to pollute carries with it the responsibility for protection of the public, it is requested that

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sampling requirements be made the responsibility of the person operating such sources.

The agency, when issuing an operating permit, will indicate the frequency at which such tests are to be made.

The agency concurs with the remainder of the rules and regulations proposed by the Pollution Control Board.

Thank you, and I will try to answer any questions you gentlemen have.

HEARING OFFICER SULLIVAN: Thank you, Mr. Reddy.

I wonder if you could have the agency actually prepare the text of the particular portions of the regulations you are suggesting be modified in the form that you are proposing.

THE WITNESS: That's right. We were planning on bringing certain copies here, but we just could not make it.

I will send it to the Pollution Control Board.

HEARING OFFICER LAWTON: If you can get it in in the next week or two, it would be very helpful in the specific areas that you commented on and, of course, any others.

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Are there any questions that anyone cares to put to Mr. Reddy?

If not, thank you very much for coming --

MR. FOUT: I have one question.

Is he proposing that the ban to the spraying of asbestos onto steel posts -- are you suggesting that they enclose the area to spray it on, is that it?

THE WITNESS: The way the regulation reads right now, it says that all floors on a particular building shall be -- material from all floors shall be removed and that all floors shall be vacuumed, and that it implies that there shall be no activity on any of the floors.

We are suggesting that instead of doing this for all floors, just to limit it to the particular area where the spraying is being conducted.

MR. FOUT: In other words, controlling the spraying part that is getting away; is that what you are proposing?

THE WITNESS: Right.

We are proposing that if there is any particular corner or one particular room where the

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spraying is conducted, that only that room be subjected to the regulation, not the whole building or the total number of floors in the building.

MR. HARKER: Did you propose that we should not have a permit system? I did not catch that part of your testimony.

THE WITNESS: No. We are proposing that activities such as construction and demolition, et cetera, should be handled by a local agency and all the rest of the permits shall be handled by the Agency.

MR. HARKER: Thank you.

HEARING OFFICER LAWTON: Thank you very much.

If you would get to us your specific proposals, we would be very grateful.

(Witness excused.)

Is this a copy of a prepared statement, Sir?

MR. SHECKLER: Yes.

HEARING OFFICER LAWTON: Do you have any more copies?

MR. SHECKLER: I have two more.

HEARING OFFICER LAWTON: You do have two more. All right.

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I do not want to take the one you are going to use.

CLIFFORD L. SHECKLER,
being first duly sworn, testified in the narrative form as follows:

THE WITNESS: Mr. Chairman, Gentlemen, I am Clifford L. Sheckler. I represent the National Insulation Manufacturers' Association. I serve as Chairman of the National Insulation Manufacturers' Association Health and Safety Committee.

With respect to the proposed Pollution Control Board Regulation R-71-16, Asbestos and Spray Insulation, we respectfully call your attention to Part III, Section 302, Paragraph (g) which reads as follows:

"Any plenum of other structure coated with or containing fibrous insulation and used in the circulation of air in a building shall be thoroughly cleaned of all debris and waste insulation. All applied fiber-containing insulation within a plenum or duct shall be coated with a sealant which precludes exposure of the fiber-containing material to the circulating air."

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We urge you to consider revising Part III, Section 302, Paragraph (g) by inserting the word "sprayed" in both the first and second sentences as follows:

"Any plenum or other structure coated with or containing (sprayed) fibrous insulation and used in the circulation of air in a building shall be thoroughly cleaned of all debris and waste insulation. All (sprayed) applied fiber-containing insulation within a plenum or duct shall be coated with a sealant which precludes exposure of the fiber-containing material to the circulating air."

We are requesting this revision to clarify the fundamental purpose of the proposed Part III, Section 302, Paragraph (g) to specifically relate to "sprayed" fibrous insulation.

We believe this clarification is required because fibrous glass insulation is applied in air transmission systems. This material is applied by hand in board or blanket form and is not sprayed. Unlike sprayed insulation, fibrous glass duct insulation materials are preformed and bonded.

Fibrous glass duct insulation materials

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meet the requirements of nationally recognized standard-making bodies and are approved by use by key governmental agencies.

Extensive industrial hygiene engineering research studies have been carried out by highly qualified university affiliated industrial hygiene engineers and physicians. Studies both in the laboratory and in actual installations demonstrate no significant erosion from the standpoint of either health or comfort.

The subject of safety and health has been utilized extensively by a certain few individuals and groups for the purpose of eliminating fibrous glass thermal and acoustical insulation from the inside of ducts. This strategy and campaign has been pursued unsuccessfully. The preponderance of medical and engineering scientific research to date shows fibrous glass insulation to be a biologically inert, safe, effective material.

Published authoritative medical scientific research studies over the past 30 years show fibrous glass to be a biologically inert substance which presents no health risk greater than any other innocuous dust.

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The American Conference of Governmental Industrial Hygienists has scrutinized the scientific research studies concerning the pulmonary effects of fibrous glass and has classified this material as an inert or nuisance-type dust. The Federal Occupational Safety and Health Act Standards of 1971 have confirmed this classification.

All substantiating evidence concerning the health aspects of fibrous glass are listed in our attached National Insulation Manufacturers' Association booklet, "Health Aspects of Fibrous Glass". One copy of the supporting medical and engineering scientific data is enclosed for committee review.

We wish to thank you for the opportunity of appearing before your committee.

I have here, Gentlemen, copies of some of the available scientific information data.

"Threshold limit values of airborne contaminants and intended changes," adopted by ACGIH for 1970.

Industrial Hygiene Foundation of America, Inc., "A report on health aspects of fibrous glass, airborne fibrous glass particles," by George W. Wright.

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"Glass dust: A study of its biological effects," by Paul Gross.

"Fiberglas. Chemistry and technology," by Karey P. McCord.

"Fiberglas, health hazard investigation," by Walter J. Siebert."

"The pulmonary reaction to high concentrations of fibrous glass," by Paul Gross, Mary Ann Caschak, Ethel B. Tolker, Mary Ann Babcak and Robert Treville, from the Department of Public Health, Education and Welfare, Consumer Protection Environmental Health Service.

A copy of the work and paper, "Exposures of insulation workers to airborne fiberglas," by Clark Cooper, J. Leroy Balzer, University of California, Berkeley.

"Exposure to fibers in the manufacture of fibrous glass," by the United States Public Health Service, Douglas L. Johnsen, Jerome Healey, Howard E. Ayer and Jeremiah R. Lynch.

"Erosion of fibers from installed fibrous glass ducts," by Dr. Jacob Cholak, University of Cincinnati, Kittering Laboratory.

"Fibrous Glass lined air transmission systems, an assessment of their environmental effects,"

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by W. Clark Cooper and J. Leroy Balzer and Douglas P. Fowler.

"Standards for safety, air ducts, Underwriters Laboratory."

"Evaluation of the effect of air velocity on fibrous glass duct and duct liner systems," by the Kittering Laboratory, University of Cincinnati.

And I respectfully submit these for your review.

There are, incidentally, additional papers available which we will be very happy to make available to you, if you so choose additional information.

I would like to respectfully call your attention to the Glenn Booklet, Page No. 13.

I am not going to elaborate on it, but there is a major labor-union jurisdictional dispute which is definitely pertinent to the health facts here, and I think it would be important for you to read it. This dispute is between two major construction labor unions.

HEARING OFFICER LAWTON: Thank you.

We will receive your comments and these supporting documents as Exhibit No. 24.

Thank you very much.

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Does anybody have any questions of Mr. Sheckler?

If not, thank you very much, and I believe you mentioned a further publication that would be available?

THE WITNESS: If you so choose, or if there is any additional help we might be in this matter, please call on us. Our address is on the statement.

HEARING OFFICER LAWTON: Thank you.

THE WITNESS: Thank you.

HEARING OFFICER LAWTON: Thank you, Sir.

(Witness excused.)

Do we have a representative of Caterpillar?

MR. ENGELKING: Yes.

HEARING OFFICER LAWTON: We will hear from you now.

MR. ENGELKING: One of the problems we have, and we are now set up, we did want to show some slides with our testimony, and we have a screen behind you, and he is all set up for us to start.

I think it might be perhaps better if you could move over.

HEARING OFFICER LAWTON: I will come down in

front.

Do you have a written commentary also?

MR. ENGELKING: Yes. We have six copies here.

HEARING OFFICER LAWTON: All right.

Let's give one to the reporter.

Do you care to make any comments before the slides, Sir?

MR. ENGELKING: Yes. I will make a few comments before the slides, and I think we had better get this down, and I will discuss the slides with you.

MR. REDDY: Can the EPA have a copy of the report?

MR. ENGELKING: You will have to write to us for it, if you would.

It just happens that I brought so many copies here, and I am not quite sure how many they would like for you to have.

How many copies was it that you wanted?

HEARING OFFICER LAWTON: Of this material?

MR. ENGELKING: Yes.

HEARING OFFICER LAWTON: You have given us about six?

MR. ENGELKING: Six.

HEARING OFFICER LAWTON: Six.

If you can get us four more, I think four

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or five more.

MR. ENGELKING: All right.

HEARING OFFICER LAWTON: And send one direct to the EPA. I think it would be helpful.

MR. ENGELKING: All right. We will get you one then later on here.

HEARING OFFICER LAWTON: Fine.

FRED ENGELKING,

being first duly sworn, testified in the narrative form as follows:

HEARING OFFICER LAWTON: For the record, Sir, will you tell us who you are and who you represent?

MR. ENGELKING: Yes, Sir.

Mr. Chairman, members of the panel, my name is Fred Engelking. I am with Caterpillar Tractor Company. I have been there since 1937. I have received a degree in chemical engineering from Purdue University.

I have been working on friction materials for over twenty years, and presently I direct the research and development programs for non-metallic materials, including friction material, in the Research Department.

I have been a member of the American Society

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for Testing Materials Subcommittee on Friction Materials since 1957.

Caterpillar is pleased to have the opportunity to appear before the Board today. We have six manufacturing facilities in the state, and are the largest private employer in the State of Illinois, with over 41,000 employees.

We are vitally interested in the efforts of the Illinois Pollution Control Board to guard the health and environment.

I am here today to present to you information that I hope will assist you in efforts to regulate the use of asbestos and spray insulation in the State of Illinois.

Copies of our complete submission are in your hands, and it is our sincere hope that each of you will find time to read the entire booklet.

My oral remarks are included in Appendix B.

The Caterpillar product line includes primarily earth-moving equipment. All of this equipment utilizes asbestos brake linings and it is for this reason that we are concerned with Section 702 of Part VII of the proposed regulation.

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Appendix A of the booklet provides a listing of the Caterpillar product line which includes 52 models of earth-moving equipment and 53 models of material-handling equipment.

The lack of a definition for the word "vehicle" in the proposed regulation is also of concern.

Typically, regulatory statutes have differentiated between motor vehicles and earth-moving equipment.

There are suggestions for the definition of "vehicle" in the written testimony.

There are reasons for our concern. Asbestos is used in all types of brakes in all Caterpillar machines because it has outstanding safety and durability characteristics.

In spite of many years of research, there are no known safe substitutes for asbestos brake linings for our type of equipment.

I would like to acquaint you with a few types of machines other than the familiar track-type tractor.

Referring to Slide 1, we have a wheel tractor, it has a bulldozer blade on the front of it.

he28

You have probably seen these on small county roads, and working on shoulders of paved roads. This is a motor grader. This is a highway truck here, and these are in weight up to 50 tons carrying capacity.

This is a wheel tractor loader in the center, it, too, can carry as much as 50 or 60 tons with a gross vehicle weight of a quarter million pounds.

In the center we have a shovel, sometimes known as a wheel-tractor loader.

Unlike most vehicles you know about, we have five different types of brakes. They are dry shoe brakes, dry caliper disc brakes, dry band brakes, oil cooled band brakes, and oil cooled disc brakes.

In this slide, I will show you a conventional shoe brake. It is very similar to what you have on most wheels of your passenger cars. It has the asbestos friction material on each shoe and there is a hydraulic piston which actuates and shoves the shoes apart against the drum. In all cases we are using asbestos material for the brake lining in this type of brake. This is a dry shoe brake.

This is a caliper disc brake, and it is very similar to the caliper disc brakes used on the

he29

front wheels of some of the latest model passenger cars, except, of course, it is larger in size.

This consists of a disc which is rotating and attached to your wheel. The caliper here and inside the caliper are two friction pads which grip the disc as it rotates and provides the stopping capabilities.

The next three types of brakes are totally enclosed brakes and, as such, they are probably unfamiliar to you.

We have removed the cover from this brake here, the compartment, and you must remember that in all cases anywhere products from this type of brake cannot get out into the environment. One type of enclosed band brake is wet and has oil flowing inside the brake drums, which not only would hold and keep any asbestos wear particles and prevent them from getting into the environment, but it does a cooling job for us.

Other machines in the tractor line use the same type of band brake operating on a drum here, but in dry compartments. And these two are totally enclosed so that wear products cannot get out into the environment.

he30

The fifth type is a totally enclosed oil-cooled disc brake which we use on our off-highway trucks. In this type of brake, the friction material is on every other plate and it rubs against these mating plates, when a piston compresses the whole stack together. This, too, has oil flowing through it which can take away any wear products and contain them and prevent them from getting into the environment.

This is a typical friction material disc such as is used in a disc oil-cooled brake in the previous slide. This is from a slightly different model.

The brake wear products are the next subject I would like to discuss, and, as you have mentioned, there has been entered into the testimony Mr. Lynch's paper on brake lining decomposition products.

We have done some similar but less extensive work on brake decomposition products.

If you will turn to the next slide, please. This slide shows scrapings from an unused brake lining material as photographed with an electron microscope at high magnification, and the asbestos

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fibers are quite apparent. We have put a scale on the side there, a five micron scale. And the reason we did this is that in your Paragraph 601, you mention that any fibers shorter than five microns would not be counted as an emission.

And we want to show you that what fibers we find in wear products are, indeed, shorter than five microns and, if that is the proper regulation for manufacturing plants emitting fibers into the air, we feel that it should be a proper one for brakes also.

However, we would suggest that five microns may not be the proper dividing line. This could be ten, maybe fifteen, twenty microns in size, depending upon whatever qualified medical developments turn up as the proper dividing line in size that could create some hazard.

The next slide we took some of the asbestos material and heated it in an oven to try to deteriorate the fibers, and our first efforts at doing this did not work out too well, and we found out that in mounting our heated fibers which we thought would have decayed, but essentially looked like fibers, that you had to crush them, really.

hc32

So the next time we heated some more of the same unused fibers, crushed them by rolling a glass rod over some of this powder, and it powdered it further. And we still ended up with a few fibers here.

Essentially they are less than five microns, and what we are trying to simulate here was the wear condition in a brake. In other words, we generate a lot of heat in a brake, and the brake material rubbing against the drum or the disc further deteriorates the fibers into dust, rather than an actual fiber.

I should have mentioned that all of these black particles are either dust particles, conglomerates of dust particles or fiber. The white happens to be a background.

The next one, please.

We then sampled a brake compartment of a 933 machine, that is a Caterpillar number. And we examined it at the same 5,000 X magnification on the electron microscope, and these black particles again are debris, conglomerates of small dust particles, with a few fibers showing here.

There is one here, and it looks like one

hc33

here and perhaps one here. But essentially it is non-fibrous. The material that we find in the compartment was non-fibrous.

I forgot to mention earlier that at this magnification, it is quite terrific, and the thickness of a human hair is 35 microns, which would be completely off the screen. That is just the thickness of a human hair.

The next slide, please. This last one was another sample out of a brake compartment in a vehicle. Again, we see quite a conglomeration of dirt particles here, dust particles, which could either be from the outside or decomposed fiber into particles.

We see a few fibers left inside. They are kind of hard to find.

The next slide, please.

These two curves show you what it is like when a vehicle stops, and we thought we would like to explain it to you.

When you are driving a passenger car or any one of our vehicles, the situation is somewhat similar. You put your foot on the brake right at this point and, as you press down to stop the vehicle,

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the torque or stopping power increases, and you try to hold your foot on the brake, and you are slowly decelerating until you finally stop at the end.

We also have a little pip, as we call it, here, a slight increase in stopping power. And that is what a first-quality brake looks like, and that Curve A happens to be for asbestos-containing brake material.

We also have Curve B down here, which was run on a similar test, and this was not asbestos material, this was sintered friction material. Again we start down here and apply the brakes, and you will notice the stopping torque or stopping capability building up, and we have a large amount of vibration here. And vibration is noise, and noise is pollution also.

So this is one thing we do not like to have is squeak or squawk in the brakes.

You will notice also that whereas the curve up here is rather level, indicating a very uniform stopping rate, this one has a decaying stopping rate. In other words, the braking capability is getting less as you get near the end of the stop. The worst part, though, is this entire circled area here, and

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this really bothers us. That is, this sudden increase in stopping rate at the very end. In your car, this would throw a passenger against the windshield, which would be kind of a pain in the head but not particularly dangerous.

But in our vehicles, this can be quite dangerous, as I will explain in the next slide.

This is a common operation with our type of machine, which we take a loader which drives his shovel into the bank here, raises it up in the air, and he is going to back up and dump it in a truck, which is out of the picture. When you back up a vehicle like this and abruptly stop due to harsh grabbing friction material, the tractor will certainly stop, the bucket stops, but the material in it doesn't stop and it cascades over the back end down in this man's lap. And this is a very unsafe situation, and one that we are very watchful of on all of our vehicles. The worst ones, if we would change friction materials -- the worst ones would be a loader-type machine. But many of our vehicles are carrying material in the air, and we do not want the material to spill out, due to abrupt brake usage.

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When a brake is designed for a Caterpillar machine, there are three vehicle factors that we have shown: Safety, durability and smoothness of stopping. Now, the last two factors enhance the safety factor. The brake must be able to stop the machine in a reasonable distance, time after time. And asbestos brake materials have proven performance and duration in this regard.

I would like to summarize now and make some recommendations.

Caterpillar Tractor Company questions whether the intent of the asbestos and spray insulation regulations is actually accomplished by the proposed ban of asbestos brake linings.

If research work and data on ambient concentration levels of asbestos fibers which are detrimental to society establish a need for asbestos brake control and/or regulation, Caterpillar recommends that Section 702, Part VII of R71-16 (Asbestos and Spray Insulation Regulations) be modified to allow the following exceptions:

1. That asbestos brake linings (friction material) may be used in a brake system when the friction material operates in either a dry or oil-

hc37

wetted environment in a totally enclosed brake compartment which prevents the emission of brake wear products into the ambient air.

2. That asbestos brake linings (friction material) may be used in a brake system, when the friction material operates in a non-enclosed brake compartment, until conclusive medical evidence proves that the emitted brake wear products contain harmful amounts of asbestos fibers.

If all evidence indicates the need for asbestos brake controls and/or regulations, Caterpillar would further recommend that reasonable threshold limits of asbestos fiber be established by qualified personnel, based on scientific fact and foreseeable technological advances.

The test procedures which measure the concentration of asbestos fibers must also be developed which are understandable, repeatable, and realistic.

Place the burden on the manufacturer to design his product to meet reasonable, attainable standards -- don't just prohibit the use of asbestos for brakes.

As mentioned at the beginning, Caterpillar

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Tractor Company agrees in principle with the objectives of the Environmental Protection Act.

The company stands ready and willing to assist the Board in the establishment of asbestos regulations which serve the intent of the legislation.

Thank you, Mr. Chairman.

HEARING OFFICER LAWTON: Thank you, Sir.

We will receive your composite exhibit as No. 25.

Do you have any questions, Mr. Harker?

MR. HARKER: Yes.

CROSS EXAMINATION

By Mr. Harker:

Q You talked about rather than banning, setting reasonable attainable standards.

Since a numerical emission standard is obviously impossible when you are dealing with brake lining, what kind of standards do you have in mind, Sir?

A We don't know.

We think there should be more work done in this area to satisfy this. This should certainly be work done by medical people as well as engineers

he39

working with them, or scientists working with them to see what the standards should be.

We don't know. We think further work should be done.

Q I take it you are suggesting a standard which would go to the quality of the product used as well as the type of encasement which might be around it?

A We see no reason at all for any regulation for a totally-enclosed brake compartment, because it cannot get out into the environment, and we feel that at the present time the essential thing -- essentially there is enough data and facts already entered into your record here that question the effect of the asbestos fibers, where they really are a problem, when you have got a dry and exposed department.

Q I wasn't going to that. I was going to the kind of standard we might come up with, assuming it were a problem, other than numerical standards and other than a flat ban.

A I don't know. This is something which I think requires a lot more work and effort.

Q On the basis of your study, is the brake

hc40

lining decomposition likely to be -- that is, decomposition of the fiber into some sort of a particle, likely to be greater with heavy equipment than it is with ordinary transportation vehicles?

A I don't think it would be too much different myself. It is strictly a function of temperature, and I think in any case you are going to get up to a temperature where the so-called water crystallization is removed from the asbestos fiber, the fiber is deteriorated, and can then become a dusty particle instead.

Q Did you have any percentage which you could give us of the decomposition product which remained fiber in your study?

A As near as we could see, scanning with the electron microscope in our field, we rarely sell more than two fibers, and these were what we would call typical fields that I was showing in the picture, and what these are quantitatively I have no way of knowing.

MR. HARKER: Thank you very much, Sir.

HEARING OFFICER LAWTON: Thank you very much.

You will get us some additional copies, I take it? I think the demand has already been

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shown for these brochures.

THE WITNESS: I had names on the ones for the Board, but we can get others.

HEARING OFFICER LAWTON: Get us about half a dozen more, if you will.

(Witness excused.)

HEARING OFFICER LAWTON: Has Mr. Ayre arrived?

All right, Sir. We will be happy to hear from you. If you would care -- if you have anything to say.

I wonder if you would be sworn. I do not believe you were.

MR. AYRE: No, I haven't been here before.

(Witness sworn.)

hc42

HOWARD E. AYRE,

being first duly sworn, testified in the narrative form as follows:

THE WITNESS: My presentation will not be quite as polished as that of Mr. Sheckler, or the previous speaker.

I was asked to give my opinion on whatever doubt we have on the measurement of asbestos fibers in emissions from factories.

HEARING OFFICER LAWTON: I wonder if you would identify yourself, say with whom you are associated and a little on your professional background.

THE WITNESS: All right.

My name is Howard Ayre. I am with the National Institute for Occupational Safety and Health of the U. S. Public Health Service.

I have a degree in chemical engineering from the University of Minnesota in 1948, and in industrial hygiene from Harvard University in 1955.

I have been with the National Institute for Occupational Health and its predecessor organization since 1950. My present position, I am Assistant Director of the Division of Field Studies on Clinical Investigations of the Institute.

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As part of my experience in industrial hygiene, I have done dust evaluations. During my entire career in industrial hygiene, I was for several years Chairman of the Committee on Evaluation of Environmental Factors of the American Conference of the Governmental Industrial Hygienists.

Presently, I am on the Joint American Industrial Hygiene Association and ACGIH Committee on Aerosol Hazard Evaluation. I am presently Chairman of the American Conference of Governmental Industrial Hygienists.

My responsibilities do not have anything to do with the emissions from factories into the air. I guess our last air pollution investigation was in Denora, which was before I came with the organization.

However, the measurement methods which have been discussed I understand are the same ones that we have used in factories and that, in fact, are used in British factories as well.

So I thought I would just try and talk a little bit about the sampling method, the method of counting the fibers, the kind of variations we get between counts on the same filter and the

he44

resulting variations in calculated fiber concentrations, the difference, and then the kind of differences we might get if different people examined the same samples.

I am sure someone must have described by now the sampling method, the method as we use it. We use a small plastic filter holder about so big around, air is drawn through a millipore membrane filter in this holder.

We measure the flow rate going through the filter by a flow meter. We time the length of the sample, so we have a sample volume, usually in liters.

Then we multiply this by a thousand to give a volume in cubic centimeters.

Having taken this sample, we place a cap on the plastic holder and send it back to the laboratory.

For sampling exposure of workers, we fasten a little battery-operated sampling pump right on the belt of the worker, place this sample holder on his lapel and take a sample at one and a-half or two liters per minute for, say, an hour.

Using the same holder, you could, in a

hc45

fixed situation, such as emission sampling, you could sample at five or ten or fifteen liters per minute and thus get a very much larger sample volume in the same length of time.

This sampling system is relatively inexpensive. These plastic filter holders containing the filter cost about 60 cents each, and a suitable pump with a flow meter would be about \$100. And the sample time is usually done by something like a wristwatch.

Once these samples have been collected and mailed or carried back to the laboratory, this small plastic filter holder is opened, and this membrane filter is removed. A small slice is cut out and placed on a microscope slide. We put a drop of a chemical on the slide to make the filter transparent, cover it with a small piece of thin glass, and leave it for an hour or two to clear completely.

Actually at this point the microscope slide with the sample on it can be left for days or weeks without the count changing appreciably.

The sample is then examined, using an optical microscope with phase-contrast illumination.

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The objective on the microscope, that is, the lower part of it, has a 40 to 44 times magnification, and the eye piece, the upper part, is a 10 to 20 times, giving a total magnification of about 400 to 450 times.

In the eye piece, we have a ruled reticle which outlines a measured area on the microscope slide.

This reticle we use also has a series of graduated circles, each with double the area of the next smaller circle, so that we can using these graduated circles measure the length of objects on the microscope slide.

With a 43 times objective and a 10 X eye piece, such as we use, one of the middle-sized circles will correspond to about five microns on the filter.

Once having mounted this filter and placed it on the slide, and focused on the area, the technician, while looking from the slide, will select a position over the filter, then look through and focus exactly and record the number of fibers in this area defined in this reticle; mark it down, one, two, three, none, whatever the number of fibers

hc47

is in this field.

In our technique, we count everything we call a fiber, and this is really everything that is more than three times as long as it is wide. Those fibers longer than five microns and those longer than ten microns.

Once he has recorded these numbers, he again looks away from the microscope and moves his filter, and then repeats the process twenty times.

I make quite a point of this looking away, because we have to be rather careful not to try and select the kind of a field we think we should be looking at, but, rather, to look at the field in a random manner.

Each of the fields as we see it is only about one one-hundred thousandth of the total area of the filter.

Having counted 20 fields, we take the average field count and multiply it by the ratio of the total filter area to the area of the field counted. Divide by the sample volume in cubic centimeters.

Then we have our concentration in fibers per CC.

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The fibers can be expected to be distributed randomly over the filter, which means that some fields will have more than the average number, some less. And these variations at times can be fairly high.

They will vary according to a statistical function called the Poisson distribution. So how close the counter will come to the average number per field will depend on the number of total number of fibers he counts.

If we wanted to be, say 90 per cent sure that we were within certain limits, in a 90 per cent confidence interval; if, for example, in 20 fields the technician counts 25 fibers, or one per field, the actual average number might be as high as 31 or as small as 12.

If in these 20 fields, he counted 50 fibers, they might have been as high as 66 and as few as 37.

If he counted 100 fibers in 20 fields, he might have had as many as 116 or as few as 84.

And if he had 200 fibers, there might be as many as 225 and as few as 175.

You can see that as he counts more fibers,

he49

the percentage of variation gets smaller. However, he is looking at dust particles in this field, and, as his number gets larger, we don't increase the accuracy indefinitely, because he will soon come to the point where the fibers are crowding one another. Where his count becomes inaccurate, the fibers are overlapping. He cannot decide whether he should be counting one fiber or two, and this kind of thing. So we do not want to increase this indefinitely.

With 200 fibers, plus or minus 12 per cent, for 90 per cent confidence, we would certainly consider an adequate level for our purposes. And if we increased it very much above that, we would start getting these other problems I mentioned.

Well, if we then translate this kind of a random variation into the fiber concentrations we would measure in the air, we might say that through this little plastic filter holder, a reasonable sample volume would be 300 liters. That would be five liters per minute for one hour or 10 liters per minute for 30 minutes.

So, in this 300-liter sample at two filters per CC, we would have an average of about $4\frac{1}{2}$ per field, if my arithmetic is right, or 90 fibers per

he50

20 fields, and the 90 per cent confidence limits for this would be about plus or minus 20 per cent.

If we had the same sample volume and an actual concentration of two-teaths of a fiber per CC, this would only give us nine fibers per 20 fields, and so the limits, the upper limits, would be 17, and the lower limit would be four.

So here you see we have a factor of about two, in our confidence.

Now, you can increase the concentrations of the fiber on the filter by just increasing the length of the sampling time. Thus, a five-hour sample at ten liters per minute would get you back to our 300 liters per minute, plus or minus 20 per cent.

However, if in this air we had other air contaminants, soot or other kinds of dust, lengthening our sampling time, making a very large sample, a 3,000 liter sample, would tend to obscure the fibers and they might, in fact, become completely obscured, so that we could not see the fibers at all. And this same comment would apply for lower concentrations, if the air were very dirty and there were many other contaminants in this air we were

he51

looking at.

The random variation of fibers on the filter, of course, isn't the whole story. Because we have people looking through the microscope and different people use the microscope somewhat differently. Our ability to see very fine spots or very fine lines will vary. Some of us will see more fibers than another. And if we are using something like the longer than five microns, we would also have a small difference there in judging which was the five micron and which was the four and the six.

I did not have any data immediately available which directly compared fiber counts by different people. We have, as a matter of fact, done comparative fiber counts with other organizations. Mostly the factories producing asbestos products, where they do their own measurements. And we have after some trial and error reached what we have considered acceptable agreement.

This kind of experiment has given me the impression that the same situation applies in an older kind of dust counting with another instrument called the midget impinger. Some years ago I did an analysis of this for our organization, to see

hc52

whether the method of dust counting should be changed for health evaluations.

I found a number of comparisons between different groups counting the same samples or samples taken at the same time. The difference between two results on the same sample, or samples taken at the same time, can come from a number of sources. The microscope, the individual uses may be slightly different.

As I mentioned, our optical abilities are slightly different.

Of course, we have the statistical variations, and then there are some things that I just don't understand.

The best that any of these experiments managed was a coefficient of variation of 15 per cent, and I might -- I will stop on that one for a minute.

This is the standard deviation divided by the average. If we have plus or minus two standard deviations, we include 95 per cent of our results. So that plus or minus 30 per cent, including 95 per cent of our results, was about the best any two groups managed on identical samples.

However, in some cases, with experienced,

hc53

conscientious dust counters, counting presumably identical samples, we had coefficients of variation of 50 per cent, which was more or less our maximum range, but still more typical than the 15 per cent.

So again, to include 95 per cent of our samples with this kind of a coefficient of variation, we say that about one time in 20, one count might be as much as double or as little as half that of the prepared count.

This statistical variation that I talked about previously really only accounted for a small part of this difference, and it was these other differences that seemed to account for most of it.

Now, this sounds like a very inexact method, and perhaps it is.

However, a more interesting fact, I think, was that if we took these same two people that had these occasional wide variations and averaged five or ten or twenty of their samples, we would find that they came up with substantially the same dust concentration and would be within, say, 10 or 25 per cent of one another.

So, to summarize this, I guess I would say that the asbestos concentrations can be readily

he54

evaluated by a fairly simple sampling technique followed by microscopic counts by trained people.

The results of any one sample are only an estimate of the actual concentration at the time of sampling.

To determine our occupational concentrations, and I presume to determine emissions as well, a number of samples should be taken.

If only one sample is used, then one must be prepared to accept the possibility that the actual concentration is as little as one-half the reported concentration or as much as double the reported concentration.

I have the feeling that these same things would apply equally well for emission data as for occupational data.

That is about all I prepared, if you have some questions.

HEARING OFFICER LAWTON: Thank you very much. I know you came here on very close notice. We are grateful for your being here today.

Do you have any questions?

MR. HARKER: Yes, I have some.

HEARING OFFICER LAWTON: Mr. Harker.

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CROSS EXAMINATION

By Mr. Harker:

Q Dr. Ayre, could you give us an idea of the expense involved in training a person for sampling on the one hand and then perhaps training a person for counting on the other?

A All right.

As I said, the sampling technique is very simple. You have a pump with some kind of a flow meter, usually a roto-meter. You look at the ball and you set it at 5 or 10, whatever, draw the sample through the filter and time it, and record all these things.

You could train somebody to do this in something less than an hour.

The equipment, as I said, is probably less than a hundred dollars altogether.

When we come to the microscopic counting, it is a somewhat different story. The phase contrast accessories are somewhat expensive. I would guess, without having looked at a catalog recently, that it would be in the neighborhood of a thousand dollars to get a microscope which would be suitable for this purpose.

he56

They go considerably higher if you wanted to get a very versatile research microscope, and it would cost somewhat less for a typical medical microscope without the phase contrast.

The training of the technician is quite important. Phase contrast is a technique which is very reproducible. You almost have to set the thing up right in order to make it work. However, it is a by-the-numbers procedure to get it set up right.

I would say that someone experienced with a microscope could be trained to use the phase contrast system in a day. If you were starting out with someone who had never seen a microscope, I think we would a week or so, and we would obviously want somebody with reasonable intelligence to do this.

Does that answer your question?

Q Yes.

Are commercial facilities generally available which could be used, let us say, by a local manufacturer to take the samples and send the sample over there to have it counted; and, if so, what would be the general cost per sample?

A Such facilities are available. I am sorry

he57

that I don't know what they would charge.

MR. WILLIAMS: I talked to a test laboratory in Northbrook, and a sample ranged from three to \$20 per sample, depending upon the amount and type of sample, and the analysis that is needed.

HEARING OFFICER LAWTON: Will you identify yourself for the record, Sir?

MR. WILLIAMS: Pardon?

HEARING OFFICER LAWTON: Could you identify yourself?

MR. WILLIAMS: I am Nathan Williams, Illinois Department of Labor, Industrial Hygiene Unit, supervisor.

HEARING OFFICER LAWTON: Thank you.

Please continue.

By Mr. Barker:

Q Why did the ACGIH and the British use a numerical standard for industrial hygiene as opposed to a standard of, perhaps, no visible emissions within the plant?

A I don't think you can work with a mineral in the open and have no visible dust.

About the only cases I know of where people do things like this are with extremely toxic

hc58

materials like plutonium, where it is an extremely small concentration. Further, this has been an evolving situation, when people became concerned about these problems in the United States in the early 1900's, problems with silicosis, pneumoconiosis, and asbestosis. People were exposed to extremely high dust concentrations. These were concentrations of particles per cubic foot or thousands per cubic centimeter.

In the case of quartz, you would have typically 80 per cent of your workers with silicosis after 15 to 20 years, and they were dying of silicotuberculosis.

It was obvious that the dust had to be cut down tremendously to make it safe, and a series of studies by the Public Health Service, by the Metropolitan Life Insurance Company and others, resulted in some numerical dust concentration limits below which it was felt that illness would not occur.

The Public Health Service did a study on asbestos dust in 1937. Unfortunately, the group of workers studied included none who had worked more than 16 years, because that was just what they had

hc59

to look at. And there weren't -- the group without noticeable effects was rather small. So that looking back on it, we can see that the tentative number which was 5 million parts per cubic foot recommended at the time was not safe.

We feel that at a lower value we will have safety, that we will not have workers developing asbestosis, and I personally feel that we won't have excesses of lung cancer either.

The only way we can see to evaluate this -- and, as you probably know, we have some 400 substances for which the ACGIH has threshold limit values, all of which are numerical, except for a few carcinogenic materials, we see no difference between asbestos or quartz or other dusts, and we feel that a numerical limit is the way to go and what we should do is set the limit low enough so that no one is going to be injured.

Q In terms of the great number of invisible fibers involved in something like asbestos, if you are trying to keep a level as low as perhaps as feasible, is it better to deal with the standard that is numerical or simply to say no visible emissions?

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That is what we are driving at, trying to answer.

A Again, I have a problem with this no visible emissions.

If I see something coming off a process, I cannot tell by looking at it whether it is steam, which, of course, is non-toxic, or whether it is, in fact, a fairly low concentration of very fine dust.

The kind of concentrations we are talking about are not really visible anyhow. At 5 or 10 fibers per CC, the air in a factory will clear. I mean, you can look through the air and you won't have any difficulty in distinguishing things 500 feet away. The dust will not obscure your vision.

In the case of asbestos, where we are usually also using other materials. For example, in an asbestos plant, they are also using cotton and rayon and so on. You may see lint particles floating down, but the ones you can see are, by definition, not the ones that you can get into your lungs.

Q One other question about sampling then. Would it be cheaper and could we feasibly

he61

require, permit the sampling to be done with an automatic dust counter and take some sort of a correction factor into account to get an average count and use that method as opposed to the membrane filter method?

A You can use automatic dust counters, and they have a number of advantages.

One of the disadvantages is that they cannot tell asbestos from anything else. So if we are talking about a factory here which is using asbestos as one of its materials, we cannot tell by this automatic dust counter whether we are seeing asbestos particles or seeing some other kind of particles or, as a matter of fact, sometimes seeing smoke or water droplets.

Q I have a question on sampling.

In your experience in industrial hygiene, have you encountered any heavy manufacturing sources which exhaust directly to the atmosphere either through windows or exhaust fans as opposed to some sort of a stack?

A I have seen all kinds of things exhausted by every means, I guess.

Are you talking ~~now~~ about major asbestos

hc62

manufacturers or what?

Q I am not interested in names, but if we have an emission standard, to enforce that standard we have to deal with the stack primarily, to take samples of what is coming out of the stack.

If things are going out the window or out an exhaust fan, I would imagine that measuring an emission standard would be kind of difficult.

For that particular reason, I am curious about the frequency with which exhaustion might be done by means other than a stack.

A Well, I am going to get into my personal feelings now.

I don't really feel that when you are dealing with a material like asbestos, which we recognize as toxic, or quartz, or materials of that nature, that one should try using things like window fans. Because the protection for the worker is very poor.

In order to afford protection to a worker when you have a material you are trying to keep at a very low concentration, and even our workroom standards are quite low, it takes a well-designed ventilation system with a blower, which is something

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other than a window exhaust fan.

The system has to be maintained.

And my personal opinion is that it should always go through a dust collector to prevent large quantities of this material from going out into the community.

Q My point was, to my knowledge that kind of dust collection system and ventilation system is not a legal requirement. And, if it were not a requirement, it may not be practiced.

A Yes.

The point that I am trying to get across is that in my work, we are primarily interested in protecting the worker, and I believe Dr. Nicholson was here, and I am sure he told you the story of the health problems that his insulation workers have had, have been very serious health problems, a lot of asbestos, a lot of lung cancer.

We are looking at workers in factories, we don't quite know what our lung cancer rate is. But we can see a lot of health effects on these people who have worked for 20, 30 and 40 years, eight hours a day in this material.

And I would like to see these people

he54

protected, because their exposure is so much greater than anything that can occur in the community.

And we cannot protect them by using things like window fans and open windows and so on. We have to be collecting material at the source, where it is used, and putting it out perhaps not through one distribution -- it can go through any one of several dust collections. It would not have to go through a central stack.

Q Just one other question, if I may.

We have a proposed provision which would require the cleaning of employees' clothing before they are permitted to leave the work site.

This would be to protect people again, the general population, as opposed to industrial hygiene.

Did you ever do any measurement of the kind of concentrations that might gather on workers' clothes?

A I guess the simplest answer is no.

MR. HARKER: Okay. I have no more.

HEARING OFFICER LAWTON: Dr. Harwood.

DR. HARWOOD: With regard to the sampling, you suggested that an area of plus or minus 50 per cent could be induced on one sample. So if we were

hc65

trying to measure two fibers per CC, this could be either one or three fibers per CC, in fact.

Now, I wondered if you had anything to show how the area would be reduced if we, say, took five samples or two or ten?

THE WITNESS: Yes.

We have such information, but I don't have it very handy.

DR. HARWOOD: I see.

THE WITNESS: I wouldn't have it with me.

This is something that we looked at in connection with trying to get a reasonable way of enforcing the legislative standards in coal mines, and for this we used an average of 10.

I am sorry, I just can't recall what we felt our variation was for an average of 10, but it would have been something on the order of 10 or 15 per cent.

DR. HARWOOD: 10 or 15 per cent possibly -- probably. Okay.

Just one other thing. With regard to the particles that are emitted, I just wanted to talk about the ratio of the particles above and below five micron.

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If we were measuring five micron particles, it is conceivable that a process will emit only fibers less than five micron and will, hence, never be a measurement of fibers from that particular process.

MR. WELLS: Can you conceive of a process in which the emission will only contain fibers less than five microns?

THE WITNESS: Well, the previous speaker showed us what happens when you severely degrade asbestos fibers in little, tiny spots, which is the way I understand the brakes wear, and particles just a few microns at a time get up to very high temperatures and degrade.

And the pictures he showed, which I believe are probably representative of we might find, too, showed that there are not very many fibers of any sort and, under those conditions the fibers are pretty short.

Now, he referred to the paper by Lynch where he worked with one of the asbestos brake manufacturers, and using one of their testing machines induced a very high surface temperature at which the brakes in essence are failing. The brake lining was breaking down, the coefficient of

ha67

friction was going down, the brakes would not really be using it. At this point the resins which bind the fibers were breaking down and whole fibers were being released, long fibers. This would be more like what we would see in a manufacturing process.

To get back to your specific question, where you are handling raw asbestos, mixing it, grinding it, so forth, I can't conceive of a process where you would get nothing but very fine fibers which we wouldn't see by our method.

MR. HARKER: Thank you very much.

HEARING OFFICER LAWTON: Mr. Reddy.

MR. REDDY: I have a few questions, but before that I would like to mention as an answer to Mr. Harker's last question, that the proposed addition to Part 6, the Environmental Protection Agency would require that all plants be enclosed, and that there be no emissions from windows or from fans in a factory or plant.

Now, would you know what length of tube you would need to use before the filter, in case you are using a tube for the filter; or, are you using a filter like you mentioned, that you put it right on the shoulder of a worker?

hc68

Now, is that the filter holder that you put on the shoulder of a worker or is it just a tube that you put in and have a filter at the back?

THE WITNESS: We put the actual filter holder there. Your question is how long a tube could you use in front of the filter?

MR. REDDY: Right.

Or could you use a tube at all or even a pipe?

THE WITNESS: I couldn't really give any figures on how much loss you would have on the walls of a long piece of tubing.

In many cases of dust sampling, they do use some tubing ahead of a filter for stack sampling they commonly do it, as you know. You have to use kind of a small probe sometimes to get up to some of the high velocities. If asbestos were really emitting from a particular place, we would get into some problems here, because these fibers -- these fibrous materials would tend to build up over a small opening and bridge over and so on, you see.

MR. REDDY: That is what I was really driving at.

One of the requirements when we sample

he69

particulate matter, we do it through the stack.

Could we possibly use this membrane filter or this filter that you have described and take the sample out of the stack, which would be representative of what is going through the stack into the atmosphere?

THE WITNESS: I think you could.

If this were a general stack which included the outlet of the furnaces, and all this kind of thing, you would get into this problem that I mentioned before. First of all, this is an organic material which isn't suitable for high temperatures, so you could not use it from a high temperature stack anyhow, and if this were a general stack with all kinds of soot and dust and things, from other processes, these would obscure the material and you would not be able to see the fairly low concentrations of fibers which you are looking for.

MR. REDDY: Would you have any idea of what the pressure drop because of the millipore membrane could be, say a normal-sized millipore membrane?

THE WITNESS: It is dependent on the flow rate through the filter.

MR. REDDY: You mentioned flow rate between 2 and

10. I was talking in that range of 2 and 10 liters per minute.

THE WITNESS: If I recall, it would be something on the order of 2 or 3 inches of mercury, perhaps, and something like 15 liters per minute or 20 liters per minute.

And it would decrease from there down to a few inches of water, the way we use it, at 2 liters a minute.

MR. REDDY: Well, now, again, say for example -- you did say that it was possible to sample a stack iso-kinetically for asbestos?

THE WITNESS: I think you could, yes. I don't see why not.

MR. REDDY: As I see the problem that would arise in this case would be one of the problems that you mentioned just awhile ago, the fibers would tend to collect on the tip of the probe and then to block the probe itself, if the fibers are long enough.

Now, other than that, since the material is of a fibrous nature, to maintain iso-kinetic sampling, and say that we get a representative sample out of the stream, looks quite difficult to

hc71

me now.

The reason I say this is when you have laminar flow inside a stack, it is quite easy to maintain iso-kinetic sampling, but when you have a lot of eddies, the particles then to follow the eddies and it becomes a little bit more difficult to sample the particulate itself.

Now, if there are any fibers in there, wouldn't it be more difficult to get a representative sample?

THE WITNESS: I believe, Sir, that you know a great deal more about stack sampling than I do. And I think you could probably answer your own question better than I can.

Frankly, I have been in industrial hygiene my whole career, and we have not done much in the way of stack sampling.

In fact, I have never taken one myself.

MR. REDDY: These are some of the questions that I have not answered myself, and that I have been trying to find an answer for. I thought maybe you could answer them.

That is the reason that I was wondering in this millipore membrane method, if it could be

hc72

used for stack sampling at all.

I think the second problem is that which would arise in maintaining iso-kinetic sampling itself.

For that you have got to measure the pressure drop across the filter, the flow rate inside the stack, the velocity inside the stack, then try to maintain iso-kinetic sampling at the probe depth.

These are a few of the problems that I would request the Board to look into before they adopt this particular method, you know, for stack sampling or for asbestos.

HEARING OFFICER LAWTON: Are there any other questions?

Yes, Sir.

MR. SHECKLER: Did I understand you to say that only the asbestos dust that is not visible is the part that will bother you?

Did I understand you to say that or did I misunderstand you, Sir?

THE WITNESS: Only the stuff that isn't visible to the naked eye. However, if you see a big cloud of dust, there is also a lot of invisible dust in

hc73

there.

MR. SHECKLER: All the fibers are harmful to you if you breathe it, right?

THE WITNESS: Right.

MR. SHECKLER: Asbestos fiber, whether -- regardless of whether it is five microns or over or under?

THE WITNESS: I wouldn't bother with that. But if you are breathing one kind, you are breathing another.

MR. SHECKLER: I think the question is what is respirable, what is not. I think that is what the question amounts to. Not all fibers are respirable. Is that what you mean, Sir? Not all the fibers you see are respirable?

THE WITNESS: I will go back to the way I started before. The dust you see in the air is too large to make it through your system and get down into your lungs. However, if you see a cloud of visible dust, with that visible dust there is a lot of this very fine dust, fine dust particles that will get down into your lungs, if it is asbestos dust which we are talking about.

Now, the precise length in diameter as

he74

far as the fibers which eventually in sufficient concentrations will cause disease nobody really knows whether it is five microns, whether it is only the fibers longer than five microns, or whether the fibers shorter than five microns contribute to it just as much. No one can say for sure.

For measurement of the fiber concentration, it is convenient for us to use longer than five micron fiber count. It helps us among other things to eliminate other dust which is not asbestos.

MR. SHECKLER: That is my only question.

HEARING OFFICER LAWTON: Yes, Sir.

MR. ENGELKING: When you are examining this under the microscope, I believe you said that you added some kind of a fluid to it and put a cover glass on it.

Is this enough to make the fibers lie down flat or do they still stick up like this, so that you really cannot see how long the fiber is when it is sticking up like this, but you can when you are looking at it this way?

That is one of the problems with an optical microscope, and I wondered whether you had that problem or not.

hc75

THE WITNESS: The fibers appeared to us to be lying down.

We have used the optical microscope at various magnifications and have also done electron microscopy. The fibers, at least, appear to be lying down on this filter. The filter collects pretty much on the surface. They seem to be lying down there, maybe curled up. It is a curly kind of fiber.

I don't think we consider this one of our major problems.

MR. ENGELKING: I did not know an estimating length where it was, but if you are looking at it like this, you can get it pretty good. If it is tilted just a little, then you get a peculiar length when you are looking at it. And when you are looking at it like this, you are not very good at all.

THE WITNESS: They appear to be lying down.

HEARING OFFICER LAWTON: Dr. Ayre, the Wall Street Journal of October 15th had a statement that the Federal Environmental Protection Agency was preparing regulations prohibiting the spraying of asbestos.

hc76

Do you know anything about that? First, if it is anything that you are aware of?

THE WITNESS: They are prohibiting what, Sir?

HEARING OFFICER LAWTON: The spraying of asbestos, much as our proposed regulations.

THE WITNESS: No. I don't really know what they are doing in EPA.

HEARING OFFICER LAWTON: All right.

Well, thank you very much. We are most grateful for your appearing here on such short notice.

(Witness excused.)

MR. REDDY: Mr. Chairman, I have got here proposed limits. Would you want to look at that?

HEARING OFFICER LAWTON: Yes. Could we have this for the record?

MR. REDDY: I am afraid not.

HEARING OFFICER LAWTON: Well, let us have it and we will make copies and return it to you. Will that be all right?

This is the October 4, 1971 issue of publication, "Air and Water News," published by McGraw Hill. We will put that in the record as Exhibit 26.

We will take about a two-minute break here.

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(A short recess was had.)

HEARING OFFICER LAWTON: Mr. Middleton. All right, Sir.

JAMES MIDDLETON,

being first duly sworn, testified in the narrative form as follows:

THE WITNESS: Mr. Chairman, and members of the panel, I am James Middleton, Financial Secretary and Business Representative for the International Chemical Workers Union.

Our members work in the Johns-Manville plant here in Waukegan, approximately 1,300 hourly employees.

I started to work in this plant in 1955. Not too much knowledge was known about asbestos dust or silica dust. The employees didn't have much knowledge about it on the floor, I don't think -- certainly I did not.

From there I went into the service, and I was in a preventive medicine program in the Air Force, which did not deal with asbestos dust. I did get involved with preventive measures.

I returned back to Johns-Manville in 1959 and went back to work in the plant. From there I

hc78

started noticing areas of asbestos dust and silica dust, and it was certainly something new to me. And this is more or less how I got involved in the program now that we are working with the plant at this location on cleaning up the area inside the plant.

I think many employees and myself certainly hear a lot of news about emission controls and outside polluters, but very seldom do we get anybody involved with somebody that is right there on the job, who makes these products, and is exposed to them eight hours a day, five and sometimes six days a week.

That is how I mostly got involved with the asbestos program, and in the spraying.

I think it was March of this year we had one employee that passed away from an abdominal malignancy. He had worked in the plant over 25 years, and he had an injury to the lower part of his body, the stomach area. He was admitted into the hospital and the cause of death was from asbestosis, an abdominal mesothelioma.

I interviewed his wife May 19th.

Then we had another employee that passed

hc79

away March 19th, 1971. The other employee was March 4, 1971.

That more or less triggered off an investigation on the product that Johns-Manville was making in the plant. From there we went into research on this.

I went to the Mt. Sinai School of Medicine, Dr. Selikoff has been working with this asbestos dust problem very diligently. It is kind of sad that medical people can't put a timetable on curing some of these problems that we have. We are going to live with them until they find something to stop a lot of these problems with asbestos.

I do have a lot of notes here. I won't go through them of what I know about asbestos, how it affects the people, the respiratory system, the lungs, areas of shortness of breath, loss of weight and et cetera, on down to -- I have seen them in the hospital. I have been with them days before they passed away. We have had numerous more deaths from disease going up into the lungs and other areas.

That gets us into a program where we went to the company as a union, because our people are involved there, and we asked them for an Environmental

he80

Control Committee, which was something new I think to them, it was something new to the union. As of today we have got that committee in that plant. The union has three people and the company has three that are working on it, cleaning up the dust areas.

When I say dust areas, I mean when a man is unloading the fiber that is shipped into this country, there is going to be a certain amount of asbestos dust that is coming from those bags.

When the man is making the pipe or making the product or the roofing or whatever he is making in our plant, he is going to be exposed to some of this dust, especially when he is machining.

From that standpoint, I will go into that area of the plant, where they make the water pipe, air duct pipe, and from the Environmental Control standpoint, we had a tour the other day, and from the Union's standpoint and the Company's standpoint we mutually went through the department.

They showed us the areas where they are improving on dust collecting systems in the plant, and this is a self-contained dust system.

Don't get me wrong. The problem isn't solved in that department. But the company has told

he81

the union from the standpoint that money is no limit in this Environmental Control program. And we are going to hold them to their word. Because asbestos dust in a product is going to get into our lungs, if it is not controlled some way.

They have a respirator which is approved by the Bureau of Mines. It is not the best, and I don't agree with it. I hope that the company can come into a better respirator. I have seen better ones, I think.

I am not medically inclined to probably argue that point, but I have seen the asbestos sprayer respirator, I think it is a breather type that we have in our plant.

Another area of the plant that we have not toured yet, the company has shown progress down there. Now, how far we can go with controlling asbestos dust in our plant will be something that we are going to have to take and wait and see the attitude, what this representative of our employees in the plant says we are going to do and work with the company on this.

There are other areas that we have agreed that they will move on with bringing in new dust

hc82

collectors, in the controlling of some of the dust.

The reason I didn't speak, Mr. Chairman, on this hearing, I thought it would be better to come and listen, hear and work together, then it would be better. But I did hear something that was brought up awhile ago that I am concerned about, and that is people working on the job in work clothes, and then leaving that plant and going home. I think it is a big factor with the employees in the plant.

I can't help but feel when a man is working around asbestos dust, if he is not required or furnished clothing that he will leave in the plant with the asbestos dust and fibers, because they cling to your clothes. First of all, it gets in the car, second of all he may stop by the drug store, the grocery store, and then he gets home and he probably may sit down on the couch and sit there, and maybe he has got a two-year-old child that comes along and pulls on the couch, bites on the couch, and gets asbestos fibers.

To me I think it would be a real point, real important matter with asbestos.

The emission control into the air from the

he83

plant down there, certainly they are going at it. I think it is going to be a major thing to correct some of it, but as I said awhile ago, they have self-contained control.

A lot of the Johns-Manville officials are here, I don't want to give them a lot of credit for their programs, even though I appreciate what has been done. But the dust problem on the clothing I think is real important, and that will be something that we are going to have to work out.

The Federal Government, the Occupational Safety and Health Act, is certainly going to be a major thing, and Johns-Manville wants to cooperate on this, because it is their life line product, and so far we have got this cooperation, even though it started off slow with a few stalemates. I think we are working it out together.

But, there is no doubt about it. Asbestos does -- is a killer. If it gets into your lungs, there is no cure as of yet.

A university has been working on a drug, they have been using guinea pigs, I guess you would call them, but it will not work on human beings, so it is going to be a long, hard trip for both the

he84

union and the company to get the area in the plant. But they are improving and collecting their dust, and to me that is the day I would like to see that we can get down, where a man can pull off the respirator in our plant and work without it.

Thank you.

HEARING OFFICER LAWTON: Thank you very much.

Have you had an opportunity to review the specific regulations that we are proposing?

THE WITNESS: Yes, Sir.

HEARING OFFICER LAWTON: Do you have any thoughts on anything that might be an improvement?

If you have not now, and you do get a chance to look them over, and if you do have any suggestions we would certainly appreciate getting them from you.

THE WITNESS: Thank you.

HEARING OFFICER LAWTON: We are very grateful for your appearing.

Are there any questions of Mr. Middleton?

FROM THE FLOOR: Yes, Sir.

As Corporate Manager of Accident Prevention and Health Administration for Johns-Manville Company, I would like to thank Mr. Middleton and the union

hc85 for their total and complete cooperation in this matter.

It is a very serious matter, I agree, and together I am sure, as Mr. Middleton has so well stated, we can lick the situation.

Thank you.

HEARING OFFICER LAWTON: Thank you very much.

Any other questions?

Thank you, Sir.

(Witnesses excused.)

This would appear to be all of the witnesses who have previously indicated an intention of speaking.

Mr. Willins, I don't know if you care to say anything, but would you just indicate your presence here and, if you do have anything we would be happy to hear from you.

MR. WILLINS: I think most of the things that I would have to say have been said at this meeting, or the one in Chicago the previous week, and I think somebody here has the copy of the record of what the State of Illinois did for construction workers in the asbestos industry.

HEARING OFFICER LAWTON: Would you mind stating your full name and your title?

he86

MR. WILLIAMS: My name is Nathan Williams, Supervisor of Industrial Hygiene Unit of the Illinois Department of Labor.

HEARING OFFICER LAWTON: Again, if any suggestions or comments occur to you or members of your department, we would certainly appreciate getting them with regard to the specific regulations that are proposed.

Is there anybody who wishes to be heard in this matter? Is there anybody who wishes to make any comment?

If not, I would declare this hearing closed -- yes, Sir.

MR. FOUT: I would like to make one comment.

HEARING OFFICER LAWTON: Would you state your name, please?

MR. FOUT: Ben Fout, President of Local 1107, Johns-Manville.

We have got a committee down there, and I think we have a good plant manager in our plant now, and with the cooperation of the three people that we have picked for our committee and the union, I will agree, in the future we will be striving toward an asbestos-free dust area throughout the

he87

plant, and I would like to go on record as stating that I am opposed to anybody limiting the insulation being sprayed onto steel beams and buildings, as a fire-prevention matter. I think there should be some way to control it.

But I would like to go on record as saying that I am opposed to prohibiting asbestos from being used as insulation, because I think it can be well illustrated that it is a good insulation, properly handled, to keep the building from collapsing and the steel from twisting and, et cetera.

I think the McCormick building illustrated part of this. I think other buildings have done the same thing. If they had handled it properly, I think it is one of the best products there is for insulation, to help save lives in a fire.

I would go on record as stating that. That is all I have to say.

HEARING OFFICER LAWTON: I think our concern is not so much with its use but the manner in which it is applied. But your comment certainly will be noted.

Thank you very much.

As I indicated at the opening of the hearing,

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where I made a very unusual mistake, I forgot to identify myself.

I am Samuel T. Lawton, Jr., a member of the Pollution Control Board.

I mentioned that now so that if there is anything further to be submitted by those who testified or by anyone else who wishes to get something to the Board, into the record, if you would be good enough to address it to me, to my attention at 189 West Madison Street, Chicago, 60602, Pollution Control Board, we will keep the record open for three weeks.

We will make a determination as to whether we will have further hearings in this matter. Although, I am inclined to think that unless the Board comes up with proposals that are a major departure from what we have already had under consideration, or unless there is something new that comes to our attention, the likelihood is that we will not have further hearings.

However, when the regulations are tentatively adopted, they will be published in the Newsletter and generally where there is even a minor departure from what is the basis for the original hearing, additional time is given for

hc89

further comments.

So you will get a chance to see what the Board is considering and have a further chance to comment on the regulations that are proposed.

In this respect, it would be highly desirable for those of you who have a continuing interest in this matter to make sure that you are on the Newsletter mailing list, and for those of you who are not and want to be, address a request to the Clerk of the Board, 189 West Madison Street, Pollution Control Board, and ask to be put on the mailing list.

Anything that the Board does with regard to the subject or any other subjects will be included in future issues of a Newsletter.

On behalf of the Board, I want to thank everybody for being here. I think the combined hearings, certainly the Chicago and Waukegan hearings, have been of great value and extremely helpful to the Board in arriving at its decision.

I thank you all very much and I will declare the hearing closed.

HEARING CONCLUDED.

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STATE OF ILLINOIS }
COUNTY OF COOK } SS:

ARNOLD N. GOLDSTINE hereby certifies that he is the Certified Shorthand Reporter who reported in shorthand the proceedings had in the above-entitled matter, and that the foregoing is a true and correct transcript of said proceedings.



Certified Shorthand Reporter.

RECEIVED
JAN 13 1971

BEFORE THE
ILLINOIS POLLUTION CONTROL BOARD

POLLUTION CONTROL BOARD

In the Matter of:

ASBESTOS REGULATIONS, CONTROL
OF SPRAY INSULATION

No. R71-16

Granite City Courthouse
Granite City, Illinois
October 6, 1971

Handwritten signature

LEON M. GOLDING & ASSOCIATES
CERTIFIED COURT REPORTERS
175 WEST MADISON STREET * SUITE 1810
CHICAGO, ILLINOIS 60602
CENTRAL 6-4949

JAN 21 1982

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BEFORE THE
ILLINOIS POLLUTION CONTROL BOARD

In the matter of:)
)
ASBESTOS REGULATIONS, CONTROL) No. R-71-16
OF SPRAY INSULATION)

Held on Wednesday, October 6, 1971,
commencing at the hour of 9:30 o'clock a.m.,
Granite City Courthouse, Granite City, Illinois,
Mr. Samuel T. Lawton, Jr., Hearing Officer, presiding.

BEFORE:

Mr. Samuel T. Lawton, Jr., Hearing Officer

Also present:

Mr. Matthew M. Swetonic

Members of the Press

Members of the Public

LEON M. GOLDING & ASSOCIATES, By:
Holly Eskandani, Certified Shorthand Reporter.

HEARING OFFICER LAWTON: This is the first of three hearings to be conducted by the Pollution Control Board of the State of Illinois with regard to proposed asbestos and spray insulation regulations.

The first meeting is today in Granite City. We have a meeting scheduled for Friday, October 15th, in Chicago, to be held at the Field Museum, starting at 10:00 o'clock, and we have a meeting scheduled for Tuesday, October 19th, to be held in Waukegan at the Public Library, which will also start at 10:00 o'clock.

Depending on what type of response we get at the two remaining meetings, we may hold additional meetings, either in Chicago or downstate.

We will also keep the record open following the last meeting, whenever that happens to be, for at least a two-week period, to receive any further documentation or input that anyone cares to submit.

My name is Samuel T. Lawton, Jr. I am a member of the Illinois Pollution Control Board, and have been designated by the Chairman to act as Hearing Officer for this particular regulatory proceeding.

The regulations as proposed are contained and have been published pursuant to the statute and our Board rules in Newsletter number 24, dated June 14th, 1971. There are copies on counsel's table for those of you who may wish them, and if you care to get one now before we start, please do so. You will find it in the back portion.

There is a good deal of the Newsletter besides that, and the explanation and the proposed regulations themselves are found in the remaining eight or nine pages at the end. I won't read the explanation in its entirety, but I will endeavor to summarize what it contains, and likewise what the proposed regulations relate to.

The initial portion of the explanation deals with the scientific and medical implications of asbestos use and the possible or alleged dangers that result from its use or its ingestion. The balance of the explanation sets forth the structure of the proposed regulations, and you will note from the table of contents which is included in the basic document itself, the structure of the regulations, containing seven major divisions, the first being the introduction, the authority

for the enactment, the policy of the State, and a definition section.

Part two is a general requirements section, dealing with the need for obtaining permits under certain circumstances, the removal of asbestos from clothing, and the removal of waste.

The third section deals with the prohibition against asbestos spraying and the limitations and safeguards that are inherent in what allowance there would be for non-asbestos spray insulation.

The fourth section deals with the construction, alteration and repair of structures, and the need for safeguards to be imposed under those circumstances.

Part five deals with demolition, and part six deals with manufacturing, which uses asbestos in the course of its manufacturing.

Part seven, a provision that hopefully will generate a considerable amount of interest, and that is relating to a prohibition of brake linings using asbestos in automobiles.

Now, as in all regulatory hearings, the principle objective is for the Board to receive

HEARING OFFICER LAWTON: Exhibit two will be the certificate of publication in the Granite City Press Record, announcing the series of hearings.

There also is a certificate of publication, which will be received from the Chicago Tribune, but I don't have it in my possession yet, and that we can bring in as Exhibit 2B at the time it is received.

(The document above-referred to was thereupon marked Board Exhibit No. 2, for identification.)

HEARING OFFICER LAWTON: I have a preliminary air pollution survey of asbestos published by the Lytton Systems, Inc., under contract with the United States Department of Health, Education and Welfare, and I will put that in the record as Exhibit three.

(The document above-referred to was thereupon marked Board Exhibit 3, for identification.)

HEARING OFFICER LAWTON: I have a report by I. J. Selikoff, entitled, "Asbestos." The source of this publication doesn't appear on the document I have, but I will correct that for the record at such time as we have a further hearing,

and an article entitled, "Asbestos as an Urban Air Contaminant," by A.G. Compson, in the May, 1966 issue of -- I believe it is called, "Pathology, 081." Here again, I will complete the record on the precise source.

(The documents above-referred to were thereupon marked Board Exhibits 4 and 5, for identification.)

HEARING OFFICER LAWTON: There is an article by Jeremiah Lynch, Howard Eyre and Douglas Johnson, entitled, "Inter-relationship of Continued Asbestos Exposure Indices" from the American Hygiene Association Journal, September -- October 1970 issue. That will be marked Exhibit six.

(The document above-referred to was thereupon marked Board Exhibit 6, for identification.)

HEARING OFFICER LAWTON: I have a paper presented by William J. Nicholson on "Asbestos Air Pollution in New York City," presented at the Second International Air Pollution Conference in Washington, D.C., September, 1970. That will be Exhibit seven.

(The document above-referred to was thereupon marked Board Exhibit 7, for identification.)

HEARING OFFICER LAWTON: Exhibit eight will be an article by Jeremiah Lynch, entitled, "Brake Line Decomposition Products," reprinted from the Air Pollution Control Association Journal, Volume 18, Number 12, December, 1968.

(The document above-referred to was thereupon marked Board Exhibit 8, for identification.)

HEARING OFFICER LAWTON: Another article from the American Industrial Hygiene Association Journal, February, 1971, by Louis Craley, "Identification and Control of Asbestos Exposures." I will mark that as Exhibit nine.

(The document above-referred to was thereupon marked Board Exhibit 9, for identification.)

HEARING OFFICER LAWTON: "A Report of The Philadelphia Department of Public Health ad hoc Committee on Environmental and Occupational Hazards of Spray Asbestos Fibers," August 31, 1970, that will be Exhibit ten.

(The document above-referred to was thereupon marked Board Exhibit 10, for identification.)

HEARING OFFICER LAWTON: I have a transcript of hearings conducted by the City of Chicago in consideration of its asbestos regulations, which are dated March 3rd and March 23rd and May 11th,

which I will mark as Exhibits 11A, B, C and D, respectively. 11A is the testimony before the City Council Committee of Irving J. Selikoff. 11B is the testimony of Louis Craley, Chief, Division of Epidemiology and Asbestos Services, Bureau of Occupational Safety and Health, Environmental Control Institute. 11C is the testimony of George W. Wright, Head of the Department of Medical Research at St. Luke's Hospital, affiliated with Western Reserve University School of Medicine. 11D is the testimony of Mr. J. Munroe McNulty, President of McNulty Brothers Company.

(The documents above-referred to were thereupon marked Board Exhibits 11A, B, C and D for identification.)

HEARING OFFICER LAWTON: That represents all of the material that I am proposing to introduce at this particular meeting.

We will now hear from anyone who wishes to comment on these particular regulations. I will ask you that you identify yourself, indicate on whose behalf you are speaking, and be sworn by the court reporter.

Is there anybody who wishes to testify

at this meeting?

(No response.)

HEARING OFFICER LAWTON: Apparently not. Is there anybody who wishes to indicate his presence without testifying? State your name and on whose behalf you are here.

MR. SWETONIC: Matthew M. Swetonic, Asbestos Information Association, New York City.

HEARING OFFICER LAWTON: Either you or someone on your behalf, or several people on your behalf will be testifying at the Chicago hearing?

MR. SWETONIC: That is correct.

HEARING OFFICER LAWTON: And, you have made arrangements with the Board for the time?

MR. SWETONIC: We have, yes.

HEARING OFFICER LAWTON: Is there anyone else who cares to enter their name on the record?

(No response.)

HEARING OFFICER LAWTON: Is there anybody who has any questions that relate to this particular program? If not, I will declare this portion of the hearing closed. Those of you who are here, if you have anything that you want to get into

the record or anything that you want to comment on at future meetings, you are certainly not foreclosed from doing so by the fact that you didn't testify today. We will be happy to see you. I anticipate a little more business at the Chicago and Waukegan sessions, but we did want to give anybody in the Metro-East area an opportunity to express themselves on this subject.

I have a strong feeling, as has happened in other cases, that once we promulgate our regulations and they are published for final adoption, we will hear from people in this area who will be concerned that we weren't given the privilege of testifying, but that is inherent in the administrative process, so we won't be upset.

Those of you who are here and have not testified, if there is anything you want to get to the Board, please send it to my attention, Samuel T. Lawton, Jr., Pollution Control Board, 189 West Madison, Chicago, 60602. If you feel the desire to testify at any future meeting, please contact my administrative assistant, Timothy Harker, who is keeping the agenda for future meetings.

I am going to remain here and keep the

record open on the possible supposition that people may have anticipated that this was to be a 10:00 o'clock hearing. But I will remain here a little while, so we can recess till about five minutes after ten. Thank you all for coming.

MR. SWETONIC: May I ask a question?

HEARING OFFICER LAWTON: Do you want to be on the record?

MR. SWETONIC: It doesn't really matter.

HEARING OFFICER LAWTON: We might as well.

MR. SWETONIC: The question is very simply, I notice the size of your Chicago transcripts. I have a set back at my office that are about this thick (indicating.) I wonder if those are selected transcripts.

HEARING OFFICER LAWTON: Yes.

MR. SWETONIC: Is it possible to know whose testimony is contained there?

HEARING OFFICER LAWTON: Certainly. It may be that we will want to bring in more than this portion of it, but you certainly are free to look at them.

MR. SWETONIC: Okay, thank you.

HEARING OFFICER LAWTON: All right, we will

recess for about 15 minutes.

(A short recess was taken.)

HEARING OFFICER LAWTON: Are either of you gentlemen interested in testifying?

(No response.)

HEARING OFFICER LAWTON: Would anyone else care to put their name on the record or indicate their interests?

(No response.)

HEARING OFFICER LAWTON: Very well, the meeting is adjourned at 10:15 o'clock.

(Whereupon, an adjournment was taken until 10:00 A.M. on October 15, 1971, in Chicago, Illinois.)

STATE OF ILLINOIS)
) SS:
COUNTY OF COOK)

HOLLY ESKANDANI hereby certifies that she is the Certified Shorthand Reporter who reported in shorthand the proceedings had in the above-entitled matter, and that the foregoing is a true and correct transcript of said proceedings.

Holly Eskandani
Certified Shorthand Reporter.

ORIGINAL

RECEIVED

NOV 3 1971

BEFORE THE
ILLINOIS POLLUTION CONTROL BOARD

POLLUTION CONTROL BOARD

In the Matter of:

ASBESTOS REGULATIONS, CONTROL
OF SPRAY INSULATION

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)
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R71-16

Field Museum
Chicago, Illinois
October 15, 1971

LEON M. GOLDING & ASSOCIATES
CERTIFIED COURT REPORTERS
179 WEST MADISON STREET * SUITE 15
CHICAGO, ILLINOIS 60603
CENTRAL 6-4949

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BEFORE THE
ILLINOIS POLLUTION CONTROL BOARD

In the matter of:)
ASBESTOS REGULATIONS, CONTROL) No. R 71-16
OF SPRAY INSULATION)

Held on Friday, October 15, A.D., 1971.
commencing at the hour of 10:00 o'clock a.m.,
in the Field Museum, Chicago, Illinois,
Mr. Samuel T. Lawton, Jr., Hearing Officer.

BEFORE :

Mr. Samuel T. Lawton, Jr., Hearing Officer.

Also Present:

Mr. Jacob D. Dumelle, Board Member
Mr. Timothy Harker, Administrative Assistant
Mr. Walter Romanek, Administrative Assistant

Members of the Press
Members of the Public.

HEARING OFFICER LAWTON: I am going to start out the meeting in our traditional way, and ask that there be no smoking in this room. Not only is it in violation of what the Field Museum requested, but it would be contrary to the objective of trying to achieve a good hearing, so I am sure you will understand.

This is the second hearing of the Pollution Control Board in the matter of asbestos regulations, as proposed. My name is Samuel T. Lawton, Jr.. I am a member of the Board, and I have been designated as Hearing Officer for this particular matter.

On my right is Timothy Harker, Administrative Assistant, and Walter Romanek, Administrative Assistant, in the first row.

We had a preliminary hearing in Granite City last week, which was not particularly well attended, but some exhibits were introduced at that time, which I will review quickly. I have copies here of the Newsletter No. 24, which includes the proposed regulations, and the explanatory material, so I am going to adjourn the meeting for just a moment to accomplish two things, one is to make these available to anybody who wishes to pick them

up, so you can look at them without the need for our reviewing them in detail prior to the hearing.

Secondly, I am going to indicate the witnesses that have already been scheduled, and I will ask those of you who wish to testify to submit a small card -- if you would write your name, the organization if any, that you represent, and the approximate length of time that you feel you will need to testify, it would be very helpful to us, and we could have full schedule in that way.

So I will adjourn the meeting for five minutes for those of you who care to pick up copies of this newsletter may do so, and those of you who wish to testify, please get the cards in to me right away.

(A short recess was taken.)

HEARING OFFICER LAWTON: Please let us know if you want a copy and don't have it. We will make it available.

I have only received two cards. The fact that I haven't gotten any more names would not foreclose anybody from testifying, if you so wish, or asking questions and making comments. I have asked for this only in order to try to program the hearing

as well as we are able to.

The sequence of speakers, assuming they are all here, subject to what I have just said, and those of you who have not indicated your desire to testify or speak, may do so, notwithstanding the fact that I don't have anything from you.

The order will be Dr. Harwood, who will present a study made at the request of the Institute for Environmental Quality, which has been submitted to the Board; Dr. William Nicholson of Mount Sinai School of Medicine; Harold Romer, Assistant Commissioner, Engineering Research and Development, of the New York City Department of Air Resources; a representative of the Asbestos Information Council, whose name at the moment I don't have; Fred Pundsack, of the Asbestos Information; and J. Dolenky, who has asked to speak on a related matter.

Now, we have a further hearing scheduled for October 19 at the Waukegan Public Library, at 10:00 o'clock. On that date, the following organizations have indicated their desire to present testimony: The Illinois Chamber of Commerce, the Illinois Environmental Protection Agency, Caterpillar, Inc., and the National Insulation Manufacturers'

which I will mark as Exhibits 11A, B, C and D, respectively. 11A is the testimony before the City Council Committee of Irving J. Selikoff. 11B is the testimony of Louis Craley, Chief, Division of Epidemiology and Asbestos Services, Bureau of Occupational Safety and Health, Environmental Control Institute. 11C is the testimony of George W. Wright, Head of the Department of Medical Research at St. Luke's Hospital, affiliated with Western Reserve University School of Medicine. 11D is the testimony of Mr. J. Munroe McMulty, President of McMulty Brothers Company.

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HEARING OFFICER LAWTON: Either you or someone on your behalf, or several people on your behalf will be testifying at the Chicago hearing?

MR. SWETONIC: That is correct.

HEARING OFFICER LAWTON: And, you have made arrangements with the Board for the time?

MR. SWETONIC: We have, yes.

HEARING OFFICER LAWTON: Is there anyone else who cares to enter their name on the record?

(No response.)

HEARING OFFICER LAWTON: Is there anybody who has any questions that relate to this particular program? If not, I will declare this portion of the hearing closed. Those of you who are here, if you have anything that you want to get into

the record or anything that you want to comment on at future meetings, you are certainly not foreclosed from doing so by the fact that you didn't testify today. We will be happy to see you. I anticipate a little more business at the Chicago and Waukegan sessions, but we did want to give anybody in the Metro-East area an opportunity to express themselves on this subject.

I have a strong feeling, as has happened in other cases, that once we promulgate our regulations and they are published for final adoption, we will hear from people in this area who will be concerned that we weren't given the privilege of testifying, but that is inherent in the administrative process, so we won't be upset.

Those of you who are here and have not testified, if there is anything you want to get to the Board, please send it to my attention, Samuel T. Lawton, Jr., Pollution Control Board, 189 West Madison, Chicago, 60602. If you feel the desire to testify at any future meeting, please contact my administrative assistant, Timothy Harker, who is keeping the agenda for future meetings.

I am going to remain here and keep the

record open on the possible supposition that people may have anticipated that this was to be a 10:00 o'clock hearing. But I will remain here a little while, so we can recess till about five minutes after ten. Thank you all for coming.

MR. SWETONIC: May I ask a question?

HEARING OFFICER LAWTON: Do you want to be on the record?

MR. SWETONIC: It doesn't really matter.

HEARING OFFICER LAWTON: We might as well.

MR. SWETONIC: The question is very simply, I notice the size of your Chicago transcripts. I have a set back at my office that are about this thick (indicating.) I wonder if those are selected transcripts.

HEARING OFFICER LAWTON: Yes.

MR. SWETONIC: Is it possible to know whose testimony is contained there?

HEARING OFFICER LAWTON: Certainly. It may be that we will want to bring in more than this portion of it, but you certainly are free to look at them.

MR. SWETONIC: Okay, thank you.

HEARING OFFICER LAWTON: All right, we will

recess for about 15 minutes.

(A short recess was taken.)

HEARING OFFICER LAWTON: Are either of you gentlemen interested in testifying?

(No response.)

HEARING OFFICER LAWTON: Would anyone else care to put their name on the record or indicate their interests?

(No response.)

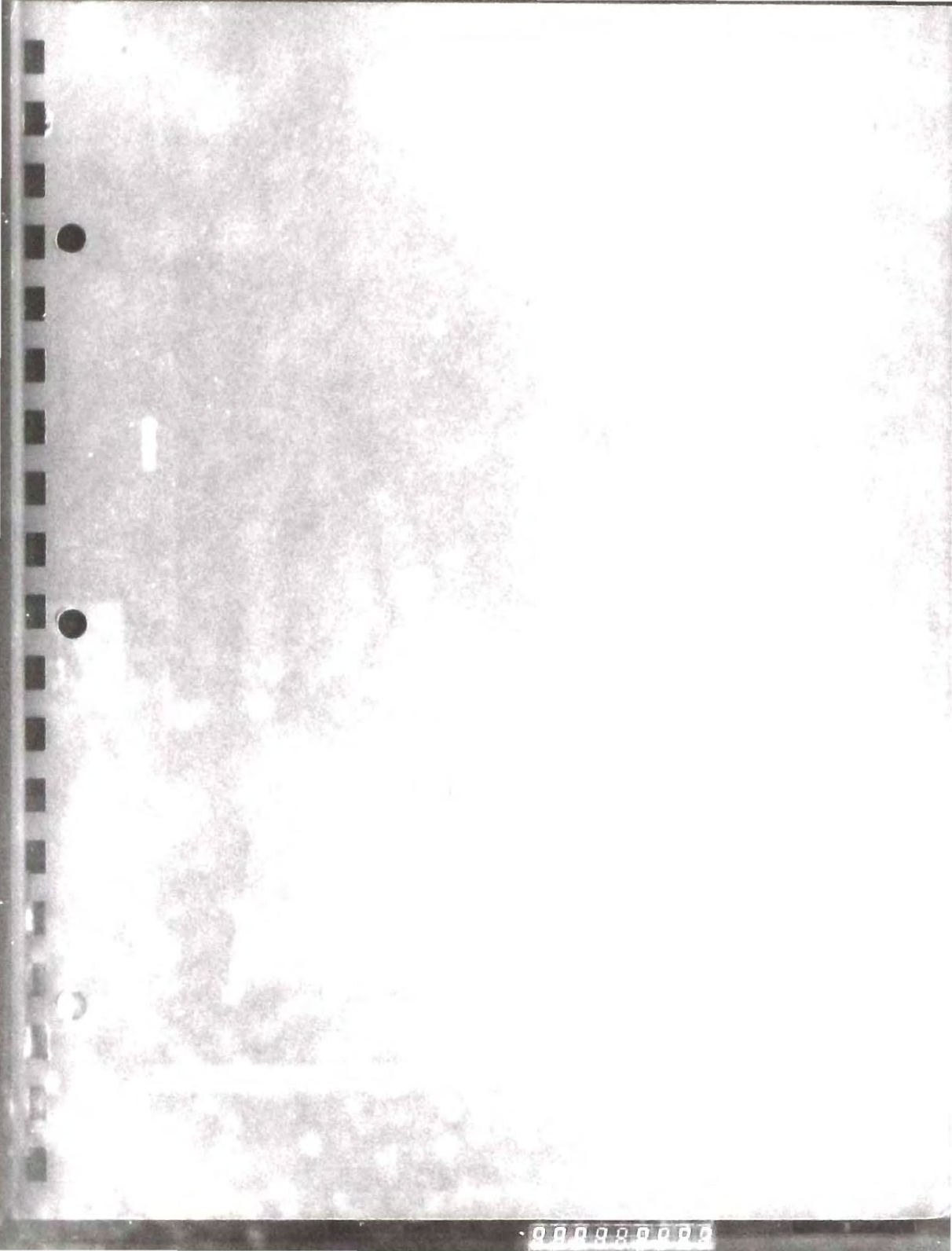
HEARING OFFICER LAWTON: Very well, the meeting is adjourned at 10:15 o'clock.

(Whereupon, an adjournment was taken until 10:00 A.M. on October 15, 1971, in Chicago, Illinois.)

STATE OF ILLINOIS)
) SS:
COUNTY OF COOK)

HOLLY ESKANDANI hereby certifies that she is the Certified Shorthand Reporter who reported in shorthand the proceedings had in the above-entitled matter, and that the foregoing is a true and correct transcript of said proceedings.

Holly Eskandani
Certified Shorthand Reporter.



RECEIVED

NOV 3 1971

BEFORE THE
ILLINOIS POLLUTION CONTROL BOARD

POLLUTION CONTROL BOARD

In the Matter of:

ASBESTOS REGULATIONS, CONTROL
OF SPRAY INSULATION

)
)
) R71-16
)

Field Museum
Chicago, Illinois
October 15, 1971

LEON M. GOLDING & ASSOCIATES
CERTIFIED COURT REPORTERS

172 WEST MADISON STREET - SUITE 10
CHICAGO, ILLINOIS 60603

CENTRAL 6-4949

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HE/gt

BEFORE THE
ILLINOIS POLLUTION CONTROL BOARD

In the matter of:

ASBESTOS REGULATIONS, CONTROL
OF SPRAY INSULATION

No. R 71-16

Held on Friday, October 15, A.D., 1971,
commencing at the hour of 10:00 o'clock a.m.,
in the Field Museum, Chicago, Illinois,
Mr. Samuel T. Lawton, Jr., Hearing Officer.

BEFORE:

Mr. Samuel T. Lawton, Jr., Hearing Officer.

Also Present:

Mr. Jacob D. Dumelle, Board Member
Mr. Timothy Harker, Administrative Assistant
Mr. Walter Romanek, Administrative Assistant

Members of the Press
Members of the Public.

HEARING OFFICER LAWTON: I am going to start out the meeting in our traditional way, and ask that there be no smoking in this room. Not only is it in violation of what the Field Museum requested, but it would be contrary to the objective of trying to achieve a good hearing, so I am sure you will understand.

This is the second hearing of the Pollution Control Board in the matter of asbestos regulations, as proposed. My name is Samuel T. Lawton, Jr.. I am a member of the Board, and I have been designated as Hearing Officer for this particular matter.

On my right is Timothy Harker, Administrative Assistant, and Walter Romanek, Administrative Assistant, in the first row.

We had a preliminary hearing in Granite City last week, which was not particularly well attended, but some exhibits were introduced at that time, which I will review quickly. I have copies here of the Newsletter No. 24, which includes the proposed regulations, and the explanatory material, so I am going to adjourn the meeting for just a moment to accomplish two things, one is to make these available to anybody who wishes to pick them

up, so you can look at them without the need for our reviewing them in detail prior to the hearing.

Secondly, I am going to indicate the witnesses that have already been scheduled, and I will ask those of you who wish to testify to submit a small card -- if you would write your name, the organization if any, that you represent, and the approximate length of time that you feel you will need to testify, it would be very helpful to us, and we could have full schedule in that way.

So I will adjourn the meeting for five minutes for those of you who care to pick up copies of this newsletter may do so, and those of you who wish to testify, please get the cards in to me right away.

(A short recess was taken.)

HEARING OFFICER LAWTON: Please let us know if you want a copy and don't have it. We will make it available.

I have only received two cards. The fact that I haven't gotten any more names would not foreclose anybody from testifying, if you so wish, or asking questions and making comments. I have asked for this only in order to try to program the hearing

as well as we are able to.

The sequence of speakers, assuming they are all here, subject to what I have just said, and those of you who have not indicated your desire to testify or speak, may do so, notwithstanding the fact that I don't have anything from you.

The order will be Dr. Harwood, who will present a study made at the request of the Institute for Environmental Quality, which has been submitted to the Board; Dr. William Nicholson of Mount Sinai School of Medicine; Harold Romer, Assistant Commissioner, Engineering Research and Development, of the New York City Department of Air Resources; a representative of the Asbestos Information Council, whose name at the moment I don't have; Fred Pundsack, of the Asbestos Information; and J. Dolenky, who has asked to speak on a related matter.

Now, we have a further hearing scheduled for October 19 at the Waukegan Public Library, at 10:00 o'clock. On that date, the following organizations have indicated their desire to present testimony: The Illinois Chamber of Commerce, the Illinois Environmental Protection Agency, Caterpillar, Inc., and the National Insulation Manufacturers'

Association. There undoubtedly will be others.

At the meeting in Granite City, I put into the record 11 exhibits, most of them documentation, and some preliminary legal matters. I will review those very briefly, without going into the content.

The first one was the Newsletter No. 24, which contained the regulations, and an explanation. The second was the legal notice in the Granite City Newspaper, and we will also include the notice in the Chicago Tribune, when that is received.

The third is the Lytton Report on Asbestos.

The fourth is an article by Dr. Selikoff, in the magazine, "Environment," March, 1969.

Number five was an article by Dr. Compson, "Asbestos as an Urban Air Contaminant."

Number six, "The Interrelation of Selected Asbestos Exposure Industries," by Lynch, Eyre, and Johnson in the American Industrial Hygiene Association Journal.

Number seven, "Asbestos Air Pollution in New York City," by Nicholson.

Number eight, "Brake Lining Decomposition Products," by Lynch in the Journal of the Air Pollution

No. 14, "Fibrous and Mineral Content of Cosmetic Talcum," an article in the American Industrial Hygiene Association Journal.

No. 15, "Cigarette Smoking, Asbestos and Pulmonary Fibrosis," which is in the American Review of Respiratory Diseases, Volume 104, 1971.

No. 16, "Asbestos Fibers in Beverages and Drinking Water," in the Nature Magazine of July 30, 1971.

No. 17, "Talc-Related Rise in Japanese Stomach Cancer," in the magazine, Science, Volume 173, pages 140-142.

No. 18, "Recommendations for Handling Asbestos," in the Engineering Equipment Users' Association Handbook.

No. 19, "Air-borne Asbestos," a report prepared by the Committee of Biological Effects of Atmospheric Pollutants of the Division of National Sciences, National Academy of Engineering, 1971.

These will all be marked and received into the record.

(The documents above referred to were thereupon marked Board Exhibits 12 through 19, for identification, and were received in evidence.)

HEARING OFFICER LAWTON: Now, at this time, I will ask that all persons who wish to testify or who think they might want to testify, please stand and be sworn.

(Witnesses sworn.)

HEARING OFFICER LAWTON: Thank you very much.

We will now hear from Dr. Harwood.

COLIN HARWOOD,

called as a witness by the Board herein, having been first duly sworn, was examined and testified in narrative form as follows:

THE WITNESS: My name is Dr. Colin Harwood. I am a research scientist in the Fine Particle Research of the Illinois Institute of Technology Research Institute.

My interest in asbestos stems from June, 1970, when I was given the task of producing for the EPA, a document reviewing the asbestos industry, and which formed the basis of the proposed federal regulations on asbestos.

Recently, I have been employed by the Illinois Institute to provide the Board with up-to-date information on the feasibility of the proposed

regulations concerning the use of asbestos.

Much of the information generated is contained in a report given to the Institute and entered as evidence at these hearings.

There are a limited number of copies which I believe will be available to those requiring them.

First of all, a note on the general strategy of the report. The medical evidence on the adverse health effects of asbestos is by no means firmly established. In particular, disagreement is apparent in the areas of the exposure concentration levels which are adverse to health, the particle size and whether it is fibers or fibrils which are most dangerous to health, the type of asbestos most harmful, and the mechanism of the instigation of disease.

However, this apart, there is universal agreement that exposure to asbestos can and does have adverse effects on sufficient numbers of people, such that precautions should be taken to minimize the exposure.

In particular, this should apply where its effect -- where it affects the general public, who

not have the choice of the asbestos factory worker, in accepting that employment. Thus, the philosophy adopted is that although there is no cause for general alarm, asbestos emissions should be curbed to a degree that is readily obtainable and using good modern technology.

Further, a sense of awareness and not fear should be developed in persons handling asbestos products, such that they handle the product with due caution and regard for themselves, their fellow workers, and the general public.

In deciding on an emission level, the strategy of accepting a possible obtainable level has been used. Conversations with Dr. Steve Holmes of the British Asbestos Research Council have revealed that a 0.2 per cc emission is obtainable, and is routine, and they will not allow air to be recirculated in factories unless this level is reached.

Johns-Manville have stated that 1 fiber per cc is acceptable for a sufficient bag house filtering system operating over a time period. Therefore, it would seem that a value of two fibers per cc is eminently reasonable. The only monitoring system that is not complicated to operate and does not

require elaborate apparatus is the membrane filter technique. Very briefly, the procedure is to collect a sample on a membrane filter and make a fiber count of the particles or fibers collected, using a light microscope and magnification of X 430.

As an alternative, the use of best technology as a control technique, without a check other than no visible emissions, is not acceptable, since it gives no measure of the efficiency, no target to aim at, and allows emissions greatly in excess of those here suggested.

The quoting of efficiencies of control equipment on a mass basis is not acceptable, and I have dealt with this in greater length in the report which is available on page 47. The reason for this is that an asbestos fiber has an extremely low mass. In fact, it takes a thousand fibers to equal a mass of one nanogram, which is 10^{-9} grams, a very small weight indeed.

Thus, an efficiency -- a quoted efficiency of 99.999, although very good at first sight, loses its glamor when one considers that 10^{-7} fibers would be emitted for every gram of dust reaching the filter.

With regard to water pollution, which is

dealt with at length on page 40 and 50 in the report, I will make a brief note at this time. Waste water from both manufacturing processes and wet scrubber units designed to limit emissions could give rise to water pollution problems if the waste is allowed to enter streams, sewers, and lakes indiscriminately.

Now, it is known that asbestos is a naturally-occurring product, and hence will occur in water as a natural event. However, the evidence is not clear as to the danger of inducing stomach cancers by water-borne fibers.

Hence, it would seem a sensible precaution wherever possible, to recycle waste water and not dump it into streams, sewers and lakes, et cetera. The efficiency of filtration plants is high, although a recent paper, quoted on page 40 of the report, has shown that a considerable amount of asbestos fibers still continue to pass through the system.

Until the medical evidence is more definite on the dangers of drinking water containing asbestos, it would seem a wise precaution again not to add asbestos to water flows unnecessarily.

The subject of brake linings is dealt with

at fair length in the supplement to the report, Supplement A. The fact is that virtually every vehicle on the roads today carries several pounds of asbestos contained in the brake linings. The action of stopping the vehicle produces asbestos emissions. The emissions are so dispersed over the country that the concentration at any one point is not great, and is certainly well below the occupational exposure levels.

For example, brakes could well be contributing something like 300 fibers per cubic meter, whereas, the occupational exposure level is generally, in a well-conducted plant, of the order of 3,000,000 fibers per cubic meter. So the level is very much less than the accepted occupational exposure levels.

However, it has been shown that the emissions probably do constitute a considerable source of asbestos background levels. The fact is that very high percentages of people are known to have asbestos body in their lungs, and these are people with no occupational exposure to asbestos.

The source of all the fibers in the background is not known. However, in keeping with the policy of limiting emissions to that which is

attainable, using best modern technology, it is suggested that strong efforts should be made towards either using an alternative friction material, or sealing the brake units. It is hard to believe that modern technology could not achieve this result by 1975.

Turning to spray asbestos, building sites are essentially messy places. Since it is impractical to expect workers to constantly sweep up every bit of dust they create, the spraying of asbestos fibers is a process which, by its nature, is bound to increase the number of air-borne particles. The rules laid down by several cities and by the industry itself, should largely eliminate the problem.

However, it would seem that the rules are rarely rigidly adhered to, and indeed, the problem of placing a dust-type piece of plastic against the skeleton girders of a high-rise building are pretty formidable.

The break-down of the rules is particularly apparent when high winds blow. Just to add to that, the City of Chicago has banned the spraying of asbestos-containing materials after December. An evening in a city where this climate exists -- by

climate I mean the awareness that asbestos is to be banned, and if the ban is ever to be lifted, they have to prove that their measures of containing asbestos spray are effective -- one has to go down to any building site where this operation is taking place to observe, for example, plastic sheets which do not seal the area in which the spraying is taking place, and without naming any one site in particular, one would only have to go to several building sites to observe this as a fact.

In conclusion, I would like to stress that the regulations adopted should reflect what is attainable using the best modern technology. To this end, the effort and research of the more progressive asbestos companies is to be commended. It is not possible to wait around until the health effects are precisely defined, since this may take many years.

Research in this area has been going on many years already, and still the foundations are not clearly laid down. There is a great deal of controversy over what constitutes a health hazard. To this end, it is suggested that the Illinois proposals are in keeping with a policy of minimizing emissions, while retaining the many benefits of

that valuable material. Thank you.

HEARING OFFICER LAWTON: Thank you, Dr. Harwood.

Dr. Harwood is a very unusual witness. Normally, the expert witnesses which we have will devote the first 80 percent of their testimony to their own qualifications, and then the balance on what we hope to get. I don't believe there is anything in the record at this point on Dr. Harwood's professional background, and I would be grateful if you would give us a brief summary of what you are doing, with whom, and what you have done in the past from a professional point of view.

THE WITNESS: I think I have dealt with that -- yes. As I stated, I work for the IIT Research Institute, in the Fine Particle Section, which has got a peculiar expertise in the area of fine particles.

Now, it deals with fine particles stemming from powdered material of visible particle size down to the sizes which are not visible to the naked eye, such as the problem we are talking about here, which is the asbestos emissions.

The particles of the respirable range are not in fact visible to the naked eye, particles of five micron size, for example, you never see with the

naked eye. However, the section deals with particles suspended in the air from all these considerations, right down from the unseeable range, the sub-micron range, through to the powdered stage, where the particles are very -- the presence is very obvious.

We do research in air pollution -- half the work is done in air pollution, in which we are talking about the air-borne particles or particles in aerosol form, and the other particle work is with particles which are seeable. We do cover a very wide range of fine particles in this research.

My own training was that I did a Ph.D. in fine particle technology, and which again embraces the whole field.

HEARING OFFICER LAWTON: Thank you, Dr. Harwood.

Mr. Harker has a few questions.

EXAMINATION

By Mr. Harker:

Q Dr. Harwood, would you comment on the provision of the regulations which would require the particular employer who handles asbestos to provide facilities for the removal of fibers from workers' clothing?

A Yes, I have dealt with this, and I didn't

provide booths in which the workers can blow off the material adhering to their clothing.

Now, the British and -- not because I am British, but because I feel it is a better system -- would prefer that the unit suck the material from his clothing, rather than blow it, but any step in this direction would be a positive step, in my opinion.

HEARING OFFICER LAWTON: I was anxious to have Dr. Harwood's testimony in the record before any discussion of the proposed regulations. I think all of you have copies, and I will not go into any substantial detail, but those of you who don't have copies, please let us know and we will see that you are furnished with them.

Also, the report that Dr. Harwood has made on behalf of the Institute for Environmental Quality, and which has been submitted to the Board, is available. If you would care to have a copy, please write to the Clerk of the Board, and make that request at 189 West Madison, Chicago, 60602.

Essentially, the regulations are divided into seven basic sections. The first section states the authority for the enactment of such regulations

and the policy of the State. The second section deals with general requirements that relate to all commercial construction, repair, alteration, demolition or manufacturing using asbestos.

Certain regulatory provisions on waste disposal and scrap piling are in section two. Section three deals with the spraying of asbestos-containing insulation and fire-proofing with certain prohibitions and certain safeguards that relate to the fiber emissions of non-asbestos spraying.

Section four deals with the requirements that relate to construction, and the need for enclosures that will be erected where asbestos is used. Number five deals with demolition, where asbestos fibers are likely to be emitted in the course of that type of work.

Number six sets emission standards for manufacture of asbestos products, and seven contains a prohibition on brake linings using asbestos after January 1st, 1975. It should be emphasized that the Board has approved these regulations for purpose of hearing only, that they do not constitute the law of the state. The purpose of the hearings is to hear from those who feel strongly in regard to any

particular aspect, either for or against, and hopefully as a consequence of this hearing and others, the Board will issue regulations dealing with this very important subject.

Is there anything further you would care to add, Dr. Harwood?

THE WITNESS: No.

HEARING OFFICER LAWTON: Fine. Thank you very much.

We will now hear from Dr. William Nicholson, Mount Sinai School of Medicine.

Mr. Dumelle, a member of the Pollution Control Board, has just arrived. He is on my extreme right.

WILLIAM J. NICHOLSON,
called as a witness by the Board herein, having been first duly sworn, was examined and testified in narrative form as follows:

THE WITNESS: Thank you very much for this opportunity to appear before you and discuss the asbestos regulations that you have proposed.

For purposes of identification, my name is William J. Nicholson, and I am Assistant Professor

of Community Medicine at the Mount Sinai School of Medicine of the City University of New York.

I have served as coordinator of several projects undertaken by the Environmental Sciences Laboratory of Mount Sinai to evaluate the effects of asbestos exposure. These include an industrial hygiene research program designed to identify and control asbestos hazards in the insulation industry, the development of analytical techniques for the quantitation of asbestos in ambient air, and the study of asbestos air pollution in New York City and other cities in the United States.

Additionally, I am engaged in several epidemiological studies of disease associated with occupation or environmental exposures to various substances. Two these studies involve the effects of asbestos on defined populations.

The justification for controlling asbestos as you have proposed in the regulations, is a very valid one, and a very strong one. The history of asbestos diseases -- I would like to spend a little time discussing the effect of asbestos diseases, to set the picture for the control measures you have proposed. That history goes back to about the

and indeed, it was later verified that there was a relationship between the two. For example, in 1947, the Chief Inspector of Factories in Britain found lung cancer in 13 per cent of deaths in which asbestosis was present.

To bring us up to date, 70 years after the first knowledge of asbestosis disease, I would like to give you some data collected by Dr. Irving Selikoff of Mount Sinai Environmental Sciences Laboratory.

He has followed, since January 1, 1943, the 632 members of the New York and New Jersey Locals of the Asbestos Workers' Union, the International Association of Heat and Frost Insulators and Asbestos Workers, the name applied to the union -- the national union of asbestos workers.

These men apply the insulation to the pipes and the boilers of most buildings. They do not apply spray insulation, as we have heard discussed, but they do apply all thermal insulation in most major cities. The material that they used typically contains up to perhaps 15 percent asbestos, and very occasionally more. More often, it will contain much less or even no asbestos whatsoever. In fact,

and indeed, it was later verified that there was a relationship between the two. For example, in 1947, the Chief Inspector of Factories in Britain found lung cancer in 13 per cent of deaths in which asbestosis was present.

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These men apply the insulation to the pipes and the boilers of most buildings. They do not apply spray insulation, as we have heard discussed, but they do apply all thermal insulation in most major cities. The material that they used typically contains up to perhaps 15 percent asbestos, and very occasionally more. More often, it will contain much less or even no asbestos whatsoever. In fact,

half of their workday is with materials that contain no asbestos. So their exposures are at least today considerably less than the massive factory exposures that were occurring at the turn of the century.

Dr. Selikoff's study was that of the mortality experience of these workers' normal ages in 1943, and one could calculate the number of expected deaths that would occur in subsequent years, calculating those deaths on the basis of mortality statistics of the United States, or, in this case, of the New York City area.

These calculated expected deaths are then compared with the experience of that group of workers specifically, and in this case, it is each set specifically so that at a given age, that individual's expected experience was compared with what in fact took place.

From 1943 through 1962, from all causes, 203 deaths were expected of these individuals, individuals who in 1943 had had 20 years of experience in the onset of exposure to asbestos. Rather than 203 deaths, there were 255. More than 50 men died who should not have. If we look at a table which is furnished in a copy of my testimony, you can see

the causes of these "excessive deaths."

45 died of cancer of the lung, trachea and pleura, where only six or seven were expected; 12 died of asbestosis, although none should have died, because asbestosis is not a disease that is found in the general population; three times as many died of cancer of the G.I. tract as were expected.

As the survey was conducted through June, 1971, the data for that are given -- I won't repeat them, but I will summarize it in this way, that at this time, 20 percent of the deaths of that group of people have been from lung cancer. Ten percent, or approximately ten percent have been from major mesothelioma. This is a disease, a cancer of the lining of the lung, or of the abdomen. It is not the lung cancer which is in the interior of the lung itself. It is so rare in the population, that one individual in 10,000 will have been found to have died of it, but when mesothelioma is found, one usually finds an association with asbestos.

Roughly ten percent of these individuals that died of cancer of the G.I. tract, again three times as much as expected, and nearly ten percent died of asbestosis. In total, over 50 percent died

of either some form of cancer or asbestosis. It is a catastrophe, because from this you can say that nearly 40 percent of their deaths are associated with their occupational exposure to asbestos.

I would like to dwell a bit long on mesothelioma and lung cancer, because there are two aspects here that are important. The first is the long lapse of time between the onset of exposure and the incidence of disease. In both lung cancer and mesothelioma, this lapse may be 35 years. In fact, that is roughly the average period of those deaths. The average time between onset of work exposure to asbestos and the incidence of these diseases in these asbestos workers indicates that whatever asbestos does, it takes a long time for it to do it to you.

A second aspect is a strong synergism found between asbestosis or asbestos exposure and cigarette smoking and the incidence of lung cancer. By personal interview in 1962, the smoking habits of 370 of the 632 men were established, and now, using the data on sex, age, and smoking, specific death rates, that is, the death rate where smoking is also established in the general population, it

was found that if you smoke and work with asbestos, the risk of dying of lung cancer is 90 times that if you neither smoke nor work with asbestos. A smoking individual on the average might have something between 10 and 20 times the risk of lung cancer as a non-smoker.

In the 87 members that did not smoke, only one has died of lung cancer. From this, one can say that -- although the statistics are very poor, that it is not that much worse than smoking -- it might perhaps be a factor of ten greater, it might be less, but we know it is not 90 times greater if you do not smoke.

The point here is that combined, the effects of asbestos and smoking is much, much worse than the sum of either one separately. The two factors, acting together in synergism, produce a health effect far greater than each individually.

These data relate to occupational exposures. Were that to be the only issue involved in asbestos disease, the concern for the regulations here today would not be appropriate. Unfortunately, that is not the case. In recent years, it has been found that people only indirectly occupationally exposed

to asbestos have diseases -- that is, workers who work in the vicinity of its application. I am speaking here of pipe fitters and welders, electricians, carpenters who may work on a construction site or in a ship yard, and these individuals now have found -- in these individuals, there have been found cases of mesothelioma in far greater numbers than expected.

In particular, in Holland, 22 cases were found during a six-year period in workers of the de Scheide Ship Yard. These cases were all workers other than insulation workers, the welders, carpenters, and painters. In Scotland, 80 cases of mesothelioma were reported from the years 1950 through 1967. An investigation of the occupational background of the individuals found that they were in other than asbestos work, generally. Maybe one or two were asbestos workers. One of the disconcerting facts of the Scottish data is the rapid rise of the occurrence of mesothelioma -- 51 cases were seen in the years 1964 to 1967, approximately 30 years after the extensive ship building was undertaken during World War II, and the total of the others, the 290 others were found in the previous 14 years, so that

there was a rapid rise of mesothelioma, and that rise can be associated with the rapid increase of ship building undertaken prior to World War II in Great Britain.

Additionally, the spectrum of asbestos-associated diseases extends even to environmental exposures. In 1960, Wagner, Sieggs, and Marchand described 47 cases of mesothelioma found during a four-year period in a northern Cape province of South Africa, an area with extensive oracidolite asbestos mines. Neither were seen in the neighboring Transvaal Province or in 10,000 autopsy cases of workers who had died from exposure to silica.

Of these 47 cases, approximately half were found to have had either industrial or mining exposures to asbestos. Virtually all of the remaining cases were in individuals who simply worked or lived in the vicinity of asbestos mines or mills.

Similar data -- additional data came from London. Newhouse and Thompson reviewed all mesothelioma cases in the London Hospital. They found 76 cases -- of the 76 cases, 31 had occupational exposure to asbestos; of the remainder, 11 had lived before within one half mile of an asbestos

factory. A similar distribution of cases was found by Lieben and Pistawka in Pennsylvania, that is, simply living within half a mile of an asbestos factory under the conditions of that asbestos factory's operation, clearly 30 or 40 or perhaps 50 years ago, gave rise to a disease that is virtually unique to asbestos.

There is one final insidious form of indirect exposure to asbestos. In the last two studies mentioned, 9 of the London mesotheliomas were in family members of asbestos workers, as were 3 of the 46 Pennsylvania cases. This relates directly to the issue that was previously just discussed about the need for workmen's clothing being decontaminated prior to leaving the work place.

Let me now turn to some specific items in the regulations which now relate to the need for controlling exposure, such that experiences as those I have documented will not be appearing in the future.

Firstly, the general requirements of Section 2 are excellent, particularly with regard to the responsibility -- that is, having an individual with direct responsibility for work places and for education, because we found that much of the problem

is that -- at least in marginal operators or uses of asbestos, real knowledge of the health effects are not present, and their laxity and their ignorance -- largely gives rise to procedures that are totally undesirable.

It would go a long way, I believe, to aiding in having better enforcement of procedures, which would be appropriate.

Section 2, the banning of asbestos and spray insulation is entirely appropriate. That entire section I completely concur with, as written. The experience that we have had in New York City is that we have found spray insulation using asbestos to be a major contributor of ambient air asbestos problems.

In New York City, for the past ten years, it was not unusual to see snowfalls of asbestos-containing material anyplace in mid-town or downtown Manhattan, where high-rise buildings were under construction.

In some cases, fire-proofing was done with no controls whatsoever -- that is, they did not even have tarpaulins to contain the material to the job site.

To further verify that such sources were

significant sources of this, we undertook sampling with the Department of Ambient Air Resources, to measure the ambient air levels around such sites up to a quarter of a mile away. Levels exceeding one tenth of a microgram per cubic meter were observed, which exceeds by one hundred times the background levels in suburban areas.

In some cases, this went up to four-tenths per microgram. While one-tenth microgram may seem to be small, it could represent over 10,000,000 minute fibrils of asbestos. These can be broken down into a size approximately two to four hundred Angstroms in diameter, and perhaps a tenth of a micron in length. They are indeed minute, and their number per gram is enormous.

As I mentioned earlier, we don't really know these conditions that existed in the factories that gave rise to the diseases that were seen in the previous studies mentioned. We don't know the air levels that existed 30 or 40 years ago in those circumstances of environmental asbestos exposure associated with diseases. We can't say these levels that were measured in New York City are certain to produce a certain amount of disease. We don't know

but the procedure has been common only in the last ten years. Any disease experience is perhaps 20 or 30 years away. So that the control measures are based on prudence. If they were not implemented, if we were to allow this procedure to continue until another 20 or 30 years elapsed, until dead bodies began to appear, it would be a tragedy if, indeed, that was a significant level.

It is a risk that should not even be taken because to do so would be in essence to be playing asbestos roulette with the lives of millions of people. I concur in what has been done in New York City, where it is banned, and in other cities, Chicago, Boston, Philadelphia, where similar action has been taken.

Moreover, there is an additional continuing risk from this use of asbestos. Often, the undersides of the floors and the area between the ceiling of the room used as a return air plenum, that is, the air conditioning system, involves the used air of the room passing directly in contact with the insulation fiber to a cooling unit, and with the filtration inadequate to remove the fibers and recirculated throughout the building.

In other words, the air stream for part of its time, is in direct contact with asbestos-containing fibers, some of which can erode into that air stream to provide a continuing contamination to the air of major office buildings.

Additionally, the residue -- the asbestos on these buildings provides a legacy of risk in future times, when these buildings are demolished. I really do not know at this time that adequate measures exist, or they have yet to be defined, I believe, that can assure that the demolition of buildings containing extensive amounts of asbestos can be accomplished without a large dissemination into the ambient air.

Moreover, because of the uncertain knowledge of health effects of other mineral fibers which are applied, the precautions in Section 2 which apply to other than asbestos fibers are entirely appropriate. While the term "mineral wool" is a generic term applied to many materials -- they differ considerably, depending upon the source of the material -- while this material has been around for a number of years, that disease might be seen 30, 40 years, perhaps, there is yet to be a defined

study on the population that would give an assurance that indeed this material is safe. Additionally, the possibility of synergism between mineral fibers that are respirable and enter the lung and other environmental contaminants, is not known, and the possibility that disease could arise in the future is possible.

Hence, prudence and control measures which are described, are entirely appropriate.

On other items in Section 4, you might wish to add a qualifying statement that when there is potential release of asbestos to the air, a special enclosure shall be used. I am referring to the section where all uses in construction would require an enclosure.

There are uses in asbestos in construction that give no release of fibers, the installation of vinyl floor tile, for one example. In fact, one can in the insulation industry, even apply asbestos at some times without dissemination of fibers, asbestos cement being one such example, that can now be mixed in plastic bags, where the water is added into the bag prior to the opening of the bag, and the emptying of the cement, which is a wet cement, and is applied

wet -- while in the wet condition, it would not release any fibers into the air.

I cannot emphasize too strongly the need for control during demolition. Those listed here may not be adequate, and close surveillance should be maintained to verify their adequacy. No asbestos should be transported down a building by chutes to trucks. It is hard to imagine that it could be sufficiently wet that there would be no dissemination of asbestos fibers as it descends from the chute into the truck. It may have to be carted down in closed bags.

What is really needed in the area of demolition is a considerable amount of engineering talent looking at the problem of how to contain the asbestos from those buildings that have now been built, when they are demolished.

In reference to Section 6A, I would like to spend a little time on the problem of emission standards and the measurement of asbestos. Any measurement of asbestos, either in occupational circumstances or particularly in ambient air circumstances, is fraught with many difficulties. In ambient air samples, for example, we have found it

extremely difficult to identify asbestos fibers in a map of material that one finds in a background in city air. There are many other fibers which have some properties similar to asbestos, and their appearance in an optical microscope is such that you could not state with certainty whether it was or was not an asbestos fiber.

For example, there are many birefringent fibers with a similar appearance -- they are roughly the same size, short, straight sticks. If you have a very long asbestos fiber, you can see it breaking open and curling across the microscope; it is easy. But most of these fibers -- I am speaking of fibers longer than 5 microns, which are visible to a microscope, but they are still sufficiently small that they cannot be identified ambiguously in an ambient air sample, and I would be somewhat -- I would urge caution in adopting a standard for ambient air such as the one listed here of 0.05 fibers per cubic centimeter of air or longer than 5 microns per cubic centimeter of air.

Moreover, there is another problem associated with measurements using optical microscopy that even in circumstances where all the fibers

can be assured to be asbestos fibers -- you are looking only at the tip of the iceberg. When we have measured occupational -- air concentrations following occupational uses of chrysotile asbestos, the asbestos fiber most commonly used, one finds that the fraction of fibers longer than 5 microns to the total number, there can be perhaps four-tenths of one percent, or a fraction around one percent. Were that a constant fraction, this would be a valid procedure, however, we have found that not to be the case, and that fraction can vary at least ten times. We have found some circumstances when one fiber in 250 will be longer than 5 microns; other circumstances where perhaps one fiber in 20 will be longer than 5 microns.

Hence, an exposure level based on fibers longer than 5 microns is one that is not directly related to the total quantity of asbestos that is in the air. If one is using this to measure the emissions from a bag house, for example, the selective filtration that one attains in a bag house could give a distorted picture of what is really there, that is, the emissions from a filtering system, one would have -- and I now am making a

guess, because we haven't measured directly air samples following filtration by fabric filters, but my guess is that they would contain a much greater proportion of fibers under 5 microns than one would find in the normal air prior to the filtration system.

As the Committee is aware, the National Environmental Protection Agency is considering standards based on procedural requirements for factories, that is, filtration must be used in any air containing asbestos in the factory, and various control measures are suggested. The standard applied is that of no visible emissions. I think this can be an effective standard. If one has access to a factory on a no-notice basis, that you can walk into a work place where asbestos is used, so that there isn't this standard two weeks of cleaning up before an inspector gets there, and one finds no visible emissions at any place in that factory, there is no asbestos lying near the roof near the bag house, or an exhaust from the room, there is none on the window ledges that might be open, if that obtains, there are going to be minimal emissions into the surrounding community, and they would be less than

those proposed here. You just can't have emissions without having some of it lying on surfaces near the source of emissions.

Additionally, the sampling only of the stack emissions from a bag house neglects those emissions that would take place through windows, through doors, through perhaps the room air ventilation system of a factory that had not undergone any filtration. I would suggest that one look strongly at the idea of having rigid controls based upon the absence of any -- finding an asbestos fiber outside the factory or work-site enclosure.

If one wishes additional specific standards, perhaps one could invoke a standard based upon a mass of asbestos per cubic meter of air, where the amount of asbestos fiber is measured using the electron microscope. In these measures, all asbestos is measured and counted with the electron microscope. However, this is extremely costly. Each sample might require \$200 or \$300 for analysis. If the factory wished to, or the work place wished to contest the judgment of an inspector on the no visible emission standard, they could ask for an electro-microscopic analysis, and the factory could undertake to pay for it, although it would be under control of your Board.

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This is one alternative I would propose to this particular standards.

I would like to make one comment with respect to the quantity of asbestos that would be emitted from a bag house. If you have a bag house of 99.9 percent efficiency, a very high level of asbestos in an area might be 0.1 of a milligram, perhaps, if it were the dust collected from a -- from saw or cutting operations that could exceed perhaps a milligram per cubic meter, but factory air typically is always less than a tenth of a milligram per cubic meter, at least today. If that is filtered through a 99.9 percent efficient bag house, one would then have coming out of that bag house, if these figures are correct, less than a hundred nanograms per cubic meter, which is not much more than that we have found in many ambient air samples. They would be diluted into the ambient air, and it would not make that great a contribution to ambient air. Particularly in bag houses, many are very efficient, and this example I have given is one that is probably very conservative. Bag houses typically are more efficient than that, even as they cake up with fiber. The amount will decrease because of the additional material through which it must pass. I have appended to my statement levels

that we have measured in approximately 48 cities in the United States, giving the range, the number of cities within a given range of fibers, and we have analyzed figures 187 composite samples. Of these samples only 11 exceeded ten nanograms per cubic meter.

These data might be used as a criteria, or information from this could be used to adopt a criteria for ambient air standards.

Finally, one comment on the proposed ban of asbestos in brake linings after 1975, the problem here is that we have a dearth of information. Our laboratory -- we have some preliminary data that indicates that air levels near places of extensive braking may be perhaps two, three, four times higher than areas one block away. The areas where the sights were, where measurements were made, were at the stop light following a toll, or a road after a bridge -- that is, cars would pass over, in this case, the 59th Street Bridge in New York City, and the first stop light measurements were made, and similar ones were made in the toll booth at the Long Island Expressway, and they were higher than those made of the same general air levels, and they

indicate two things, one, that asbestos does come from brakes. We have this very preliminary information that can't be used in a definitive way other than to say that emissions occur, and that the problem is not as great as, say, spray asbestos, because there we found much, much higher levels, but the full extent of the problem is not really known. We don't really have adequate data at this time on the contribution of brake linings to ambient air asbestos levels, so perhaps one might qualify this Section 7-B with words such as "unless evidence of safety is demonstrated, the use of asbestos in brakes shall be banned."

The point here is that the onus should be placed upon, in this case, industry or the government to demonstrate that the use of a material is safe, and if it is safe, then it should be allowed, but the safety should be demonstrated, rather than the converse. That is, we should not have to wait many years until a hazard has been demonstrated. That has been proven so disastrous in the past, and I think here is a circumstance where such measurements and such analysis can be obtained and action taken on their basis. Thank you.

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HEARING OFFICER LAWTON: Thank you, Doctor.
There may be questions. Mr. Dumelle?

MR. DUMELLE: Yes.

EXAMINATION

By Mr. Dunelle:

Q I want to thank you for a very complete statement, and the research behind it is extremely important to us, so that we have some data such as this on which to make a decision. Your statements remind me of a great many parallels in other environmental problems, the mercury problem, the PCV, and so forth, and I just hope 35 years from now we are not going to find that PCV is causing something that you seem to indicate.

I wonder first if you could tell me whether the mesothelioma is a disease that is clearly identifiable by most physicians, since you are using it as a marker disease.

A No, in this sense it is not clearly identifiable in many deaths by most physicians. Often, an individual will obviously have a cancer in the chest that can be seen by X-rays, they may take biopsies and discover that the cells are

cancerous. A skilled pathologist, however, distinguishes on the basis of the cells themselves, as to whether the cancer is a mesothelioma or whether it is a protogenic cancer, that is, all the data I gave you relate and all the data I gave you relating to the incidence of mesothelioma, biopsy, autopsy, cells that were looked at by one individual in each of those separate studies, whether it was in the London, South African or Mt. Sinai studies, the studies had material all looked at by individuals who could distinguish. For example, at Mt. Sinai there are pathologists very skilled at this. It is not necessarily a general ability amongst standard physicians, general practitioners.

Q So perhaps the mesothelioma is occurring at a greater rate in the general population than we know, because of the analysis problem.

A It could be at a somewhat increased rate, although studies have been made in which autopsy material of individuals likely to have had misdiagnosis, that is, the misdiagnosis has been found to at least in some of these studies, particularly that of asbestos workers, to lie within certain

categories, that is, often on the death certificate you will find "generalized carcinomatosis." You will find certain cancers of the gastrointestinal tract, where you have a carcinoma in the abdominal cavity.

In a study of death certificates in which these possibilities would exist, material has been looked at, and there have been additional cases found. The incidence in the general population is ill-defined. Some of these studies I refer you to, one of which was done by Dr. Selikoff, does indicate that it is still a very rare disease in people who have never had exposure to asbestos -- whether it is one in ten or one in a hundred thousand, no one is going to give you an argument about, but it is certainly much, much more infrequent than one in ten, as we have been finding here.

HEARING OFFICER LAWTON: Do you have anything further, Mr. Dumelle?

MR. DUMELLE: Yes.

Q In your Table No. 1, where --

HEARING OFFICER LAWTON: By the way, there are some additional copies of this presentation for those of you who want them. If you want to take a

minute now --

FROM THE FLOOR: Is it possible to ask a question from the audience?

HEARING OFFICER LAWTON: Wait until Mr. Dumelle is finished.

By Mr. Dumelle:

Q In Table 1, where you talk -- where you show the initial comparison of both the cause of death and the greater increase for asbestos workers, I wonder if you would know in the construction of this -- these data, is there a correction, and would it be significant, for the possible accidental hazard for these types of workers, as compared to the general population -- there are falls and things of that sort that perhaps the population is not exposed to, I am not sure.

A These workers would perhaps have a very slightly increased rate of death. I don't know what the accidental rate of death is in this group of workers. We have looked at another group of workers in which the dramatic factor was the accidental death rate. These happened to be the roofers. There, the death rate was, if I remember right, approximately five percent in that group of

workers. It is much, much less than that in this group of workers, because it isn't that noteworthy. The accidental deaths were not -- correction, for accidental deaths they were not made, they were simply listed as all other causes of death, and if the accidental death rate was significant, it would even make these statistics worse, that is, they would demonstrate a greater problem, but it would have been a small correction.

Q One other question. You talk about the possible hazards from the duct systems in the high-rise buildings in the asbestos spray, and you state on page 7 that until recently, New York City did nothing at all to analyze the air in such buildings. Are you now doing that sort of a study?

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A We hope to begin that. The problem was that legally, New York City or its representatives which would have in this case been us, had not been allowed into the building, into the privately-owned buildings. We have just very little information on the amount of such room air contamination. It is a big study, and we hope to begin it soon, but we do not, as with brake linings, have adequate information on the air levels in those circumstances.

MR. DUMELLE: That is all.

HEARING OFFICER LAWTON: Mr. Harker?

EXAMINATION

By Mr. Harker:

Q Dr. Nicholson, I just wanted to clarify the point that Mr. Dumelle raised.

You are saying that the difficulty in diagnosing mesothelioma as a cause of death would possibly result in understating the amount of mesothelioma, but not -- would not be a cause for error which in those cases had been diagnosed as mesothelioma?

A The cases that are described are correct. There is nowhere -- there is minimal error in the diagnosis of their cases that I have reported. What is poorly known in the incidence rate in the general population, because one cannot rely upon a death certificate analysis. The cause of death, as listed in a death certificate, where the general practitioner or even the hospital physician, who does not undertake an autopsy, or even sometimes when autopsies are undertaken, they can often be in error, and you really have to go to expert pathological identification.

Q In reference to the difficulties in measuring asbestos which you referred to, I take it that the difficulty in measuring, in your opinion, only grades of ambient air samples -- it would not be difficult with stacks?

A There are two points. It is difficult with ambient air, as I described, because of the identification problem. There is another difficulty not relating to identification, when one measures asbestos from a stack, and that is that the stacks have a number of fibers larger than -- longer than five microns, which can be a variable quantity depending upon the previous history of that air, the number of fibers longer than five microns in the factory air, the fraction of fibers longer than five microns. In fact, they would be much greater than those longer than five microns as it comes from the bag house. We have found that the fractions could vary at least ten times in circumstances where chrysotile asbestos is being applied. The variation is greater when you go to other asbestos fibers, but just looking at the use of that one fiber in occupational circumstances, one finds that variability, so that that criteria is a variable one. So hence,

it gives you an idea of what is there, but it is not a firm measure of total quantity of asbestos in the air.

Q Well, the standard, as indicated, would relate to fibers larger than five?

A That is right, but for every one larger than five, there are perhaps 100 smaller.

Q So you are saying the variability that would be unknown, would be the amount smaller?

A That is right. The number longer than five microns is not a constant. The fraction larger than five microns is not a constant.

Q So the type of iceberg, in your words, would only be shown --

A As the iceberg has a different density, so they are up and down.

Q And you would prefer, in the alternative, a "no visible emission" standard?

A With the requirement that the procedural requirements could be imposed, that you really must have all air in which asbestos is potentially present filtered prior to emission, and I have great confidence in fabric filters and actually, if one applies strictly in all circumstances rigid

procedural requirements, you could indeed reduce emissions to a level that would be completely compatible with what you were trying to do. There is no question that there is a great need to do this, the question is the effect of weighting, and it is a complicated issue, no question about it.

Q On what do you base your opinion that the standard of "no visible emissions" would actually result in lower than the numerical standard?

A Well, I have seen one factory in which there was a bag house that I didn't see anywhere near the bag house that there were emissions from the bag house. I have seen aerial -- let me finish with this first -- the bag house filtered the air that came from some of the equipment inside, where they had collectors near dusty operations. The filtration system in general for the plant was quite inefficient, and the general room air of the plant was filled with asbestos fibers. The exhaust for the general room air went up through that filter system, or went out the windows, and we have made measurements in that town and found that there was a very high level of asbestos that could be associated with that factory in the nearby

area, and yet that factory could even have a low -- I couldn't say for certain -- an efficient exhaust system. Moreover, one often finds in bag houses that breaks can occur, and if there are leaks, if they are inefficient, there is deposition nearby. I have seen aerial photographs of other factories -- a different one from that I just described, in which the exhaust had, nearby -- the area nearby the exhaust had extensive quantities of asbestos on the roof. The system, if sufficiently improved, that would not obtain, you would be so far improved over what now exists that I don't think anyone would have a problem, and making also -- implicit, in my judgment, and it is a subjective answer that I am giving, because I don't have measurements, is that when one makes comparisons between what I have seen on the roofs of spray asbestos, where you literally had blocks of Manhattan covered with the debris from spray insulation, the air levels measured a quarter of a mile away happened to be much, much less than I would have expected. They were of the order of 100, 200, 300 nanograms per cubic meter, when the wind was blowing in the right direction, and if that was the level that was measured from circumstances

where you literally had the stuff flying over the ground, then "no visible emissions" would certainly bring down by, I guess, a factor of a hundred, which gets you into the realm of ambient air, so that is partly the subjective basis for my answer, that the absence of any visible fiber outside the walls of the factory would give an ambient -- satisfactory ambient air level.

HEARING OFFICER LAWTON: Would you suggest a combination of the two, perhaps?

THE WITNESS: Yes, you could do that, but I was making the suggestion that if you do that, the combination would involve electronmicroscopy, where you would look at the total number of fibers. That would be a final recourse. You can use the -- you could even make a combination of all three, with various circumstances of implementation, where you use stack samples to get a measure of what is going on. They are a good indication of what is going on. If you have a leaky bag house, you will find it that way. The only point I am making is that to have that as a rigid, absolute measure might be a mistake, because there is uncertainty in that sense, because all of these things are full of uncertainty, and that

is what I really wanted to point out. Obviously, there are administrative and legal aspects that you have to take into account, and I don't want to say that you should do it this way, I just want to give you the information about the problems of the sampling of the different circumstances.

By Mr. Harker:

Q You mentioned a tear in the bag, the "no visible emission" standards, as proposed or suggested by the Federal Government, which would have to coupled with a requirement of certain technological procedures?

A Right.

Q And as far as enforcement, would it not seem to impose a greater important burden for a person to have to go to in checking the visible emissions to see if there was air in the bag, to check to see whether X, Y and Z are in place, as opposed to simply taking a stack measurement?

A I don't think it is hard to check to see if there is a tear in the bag, and that is an easier check than would be required in taking an air sample from the stack. It is also easier to see that exhaust systems are working, because you can just

take a measurement of the air flow in an exhaust system, where you put a manometer tube against the hole -- an opening in the duct, where again an air measurement is one you have to take and go back to the laboratory and analyze it. There may be some circumstances where one is easier and one is not. I don't see that the unannounced inspection system would impose a burden. It would have to be --

Q We will get some of the sub-micron fibers, and given the possibility that someone could simply have a full-time janitor sweeping up visible dust, would it seem likely that a "no visible emissions" standard might result in a great deal more emissions to the atmosphere?

A I really -- I don't think this would be fruitful. I can't say that in such and such a circumstance some factory will have a janitor walking around with a vacuum cleaner. I would say if they do that, it would be better having an official system. It would be non-productive, from the point of view of the factory. Perhaps you could find circumstances where such a procedure would not be ideal. I think conversely, you can find them on the other side.

One really has to use a lot of judgment in this problem. I think perhaps that might be the answer, that if the regulations can be written in such a way that there can be common sense applied by your control officers, that that would be the most desirable circumstance, because a knowledgeable patrol officer can walk through a situation and see exactly whether there is a problem or not. If he has the authority to require what he says is a problem abated, then you are in business, and if you also give him the authority to use some standard criteria, that is all right, too, but a rigid numerical criteria can also end up in a lot of hassling about measurements, and you can have people spending all their time doing these measurements, which is in some cases really not strictly valid.

Q Just to clarify one more point -- besides the tip of the iceberg problem that you mentioned in taking stack tests, as opposed to an ambient air test, that does not pose significant measurement problems?

A It doesn't pose significant identification problems. There will be other stuff there, but if you make the criteria that anything that is longer

than five microns and has a three-to-one length or ratio, is called an asbestos fiber, whether it is or is not, then you have a conservative factor on your side. The asbestos fibers under those circumstances can be defined as any fiber, and it won't be that disadvantageous as that which obtains in occupational measurements. Industrial hygienists that take these measurements don't have the ability to distinguish what is an asbestos fiber or not, and all are counted, even if many are not asbestos fibers.

MR. HARKER: Thank you.

HEARING OFFICER LAWTON: I believe there was a question from the audience. Would you identify yourself?

MR. DUELL: My name is Ward Duell, and I would just put together a couple of facts, and then ask a question.

EXAMINATION

By Mr. Duell:

Q You indicated that really the purpose is the long-term protection of the general population?

A That is what we are concerned with here, yes.

Q You indicated elapsed time for asbestosis and mesothelioma are both very long?

A That is correct.

Q Now, my question, then, is, do we have the old -- numbers of years ago -- base-line information to know that the asbestos levels in the air is increased, and do we have base-line information to know that the mesotheliomas of the general population is increasing? Is that base-line information available?

A I will answer as best I can. We really have no knowledge of past ambient air levels. We have one study through which some indirect measures involving quantitating of asbestos in lung tissue of autopsy specimens for individuals of the general population over different periods of time might give some answers to the question of whether there is significantly increased asbestos levels in the general population which is not exposed to asbestos, and also of individuals that have lived about an asbestos factory. This work is yet to be done. It is about to begin. I might mention here that at this time one finds that asbestos fibers in virtually everyone who comes through autopsy, or in the lungs

of people who come to an autopsy. If one looks for fibers, as opposed to asbestos bodies, which are the artifacts of inhalation of fibers, the question of whether mesothelioma is increasing in the general population has not been well studied. One of the studies in which I have engaged is concerned with the incidence of mesothelioma in the general population. I can only say on very crude data which involves -- the data that I am giving you only involves that which appears on death certificates, so that additional cases would probably be missed. There has been nearly a two-fold increase between 1968 and 1970 in New Jersey in the general population as causes of death. I don't think all of this is increased diagnostic ability.

Q Would you say, then, that this reinforces your concept of, as you indicated it, not waiting to prove that harm has been done, but prove it not harmful? Is this the --

A I feel very strongly that there is enough evidence of disease at low levels, and all hints of that disease point to an increasing amount, so that prudence certainly dictates that we act now.

Q So even without the base-line information,

you still have enough in-put to make that as a firm conclusion?

A Yes.

HEARING OFFICER LAWTON: Dr. Nicholson, thank you very much.

REEXAMINATION

By Mr. Harker:

Q Dr. Nicholson, I have one other comment or question. We have a section of regulations dealing with non-asbestos spraying.

A Yes.

Q And we would require certain procedural safeguards to be taken during such spraying, or prior to insulation. Do you have any data on the toxicity of some of these non-asbestos fibers, and if you have a comment on those regulations?

A My comment on that proposed regulation that I made on my prepared testimony is that those are quite appropriate. I feel strongly that that section is indeed correct. I mentioned we have no definitive knowledge on human populations. There have been in some cases some studies made where factory workers at a given point in time have been

looked at, but such studies can be fraught with difficulties, because most workers are healthy, so that one has to take a population and follow it many years to see if there are problems. There is a hint in some cases under some circumstances, that synthetic mineral fibers in animals have produced some kind of disease. We have seen three types of extruded silicates injected into the trachea of animals in an experiment, in which two gave rise to no noticeable effect, and the third gave rise to, in this case, mesothelioma in one of the animals. Another study by a physician -- someone at New York University, I believe -- found that some types of extruded silicates give rise to cancer when it is injected -- I believe it was injected, rather than inhaled, in conjunction with other material. The other material was the urethane foam.

Q Again, here, would you permit an intensive preliminary investigation on the showing of biological injury?

A That would have to be absolute information -- it would be very hard information to get in a human population. The problem is that quite often one finds that the results in animals are not

extrapolatable to humans. The only data I gave you is with animals, and it is fraught with all the difficulties if a very small number studied, and the differences between the physiology of animals and that of humans. If one had a population that one knew the exposure of and completely characterized -- let me take that back. I don't think you can do that, in essence, because while you could define the population exposed to prescribed materials and follow that population for 20 or 30 years to see the disease that occurs, that still would not be sufficient for all other materials, because, for example, in fiberglass alone, there are 70 different types of fiberglass, and if one goes to mineral wool or rock wool, as it is commonly termed, which is produced differently from fibrous glass, the varieties there are extensive. The type which uses material from steel mills, which contains many trace metals, and the trace metals it contains depends upon the slag -- it would be really impossible to do an experiment in the different possible forms to which people would be exposed. Initial experiments are possible, but not very practical. Again, you just take ordinary precautions.

The cost of the precautions you describe here might increase the cost of the job ten percent over that which would be if they were not that closely controlled. This is a cost that society should bear. It is passed on to society in the use of the buildings -- the buildings cost more, and the rent is a little higher, but I think it is a completely justified cost.

HEARING OFFICER LAWTON: Mr. Dumelle?

REEXAMINATION

By Mr. Dumelle:

Q Mr. Nicholson, I want to ask you one more question.

In the air pollution studies we have become aware of an urban factor, so that when you check on smoking and you take non-smokers in cities and non-smokers in rural areas, you have a higher incidence in urban areas, and nobody knows what this other factor is, and the only theories I have heard about it is that it possibly is related to benz(a)pyrene, and from that we get studies from others saying that a non-smoker in Chicago or New York is really breathing the equivalent of one or

two packs of cigarettes in terms of benz(a)pyrene. Is asbestos supposed to have an absorptive quality? Is it possible that asbestos is either absorbing benz(a)pyrene, or absorbs sulfur dioxide, and that this is part of the synergism?

A That could be the case. We really don't know the mechanism of the synergism. We don't know the mechanism alone. One possibility is that which you suggest, but there are no hard data that would prove it as yet.

Q Are there any studies on this particular aspect, to your knowledge?

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A There is one study in which benz(a)pyrene in asbestos were given to some hamsters in some circumstances which I forget -- I think it was intratracheal injections that were given, and I don't know how the benz(a)pyrene exposure was handled -- in which the combination was worse than when either one separately was administered, but I am a little vague on this. The data I am trying to remember are in the 1965 Asbestos Monograph of the New York Academy of Sciences, and you can refer to it -- I don't know the specific numbers.

Q Thank you.

HEARING OFFICER LAWTON: Is there a question from the audience?

EXAMINATION

By Mr. Sieg:

Q I am Mr. Sieg from Caterpillar Tractor Company. I just want to be sure I understood you properly.

You said that an ordinary ambient air sample has enough mixed fibers in it -- it is not always easy to identify the asbestos fiber?

A That is right. There are fibers present in ambient air. Some of these are fiberglass, there are diatoms, there are organic fibers, and largely those -- we find a fantiastic variety of garbage in ambient air.

Q Then I was wondering, your comment on the ambient air sample around toll booths, and so on, you are as positive of the three to four times there?

A / Yes. I perhaps could mention briefly the procedures of the use of microscopy here. This is made ambiguously on the basis of the quality. First, you disperse all samples in a low-temperature activated oxygen atmosphere, get rid of the organic material, and

you disperse in aerosols or grind by various procedures all the material there to break up the mineral residue and make the dispersal such as to see that it is not. Then looking at the residue with a 40,000-times magnification on the electron microscope, the number is determined by measuring the volume of fibers that correspond to the morphology of crystals, and that is very unique. The identification is made on that basis. There are other methods made using the microscope in which a beam of electrons strike it, and you look at the characteristic X-rays from that particle. That is what was done with the ambient air samples.

Q The last part, if you have a weight figure for what is in a cubic meter around the toll booth --

A The amounts that one finds in the ambient air varies. It varies depending upon the city, as I indicated here, from a fraction of a nanogram up, in some cases, something exceeding 50 nanograms per cubic meter. So there is a variable background there.

FROM THE FLOOR: I know you are planning to break for lunch, but my presentation won't take very long, and it will relate directly to what Dr. Nicholson was saying.

HEARING OFFICER LAWTON: The next witness is Mr. Romer. Do you have any transportation problem that continuing your presentation would create? In other words, if we break for 45 minutes --

MR. ROMER: And return approximately one o'clock or so?

HEARING OFFICER LAWTON: Yes, if this gentleman is only taking ten minutes, we could break, and you would definitely be on. I normally don't feel we need a full hour, and you could start at quarter to one. If that is compatible with your program --

MR. ROMER: Yes.

HEARING OFFICER LAWTON: Because I know there are slight problems that are sometimes created.

Now, is there anybody else from out of the city that has transportation problems that this type of program would create?

DR. FUNDSACK: I am Fred Fundsack.

HEARING OFFICER LAWTON: Yes?

DR. FUNDSACK: I think I am scheduled to testify after Mr. Romer. That would be satisfactory, but I would like to be able to testify after he testifies.

HEARING OFFICER LAWTON: Yes. Well, you are scheduled to do so. All right, we will hear from this gentleman now. On the basis of past experience, when a person says he is going to talk for ten minutes, it never turns out to be the case. I would hope you break that precedent.

MR. GOLINKEN: I will make it as brief as I can.

P. GOLINKEN,

thereupon gave his statement under oath, was examined and testified in narrative form as follows:

THE WITNESS: I really hadn't planned to testify at all, but I represent the Insulation Contractors, Heat and Frost Insulation Contractors.

HEARING OFFICER LAWTON: Would you state your name?

THE WITNESS: My name is Golinken. I am from the Chicago Asbestos Company, and we are contractors, insulation pipe and boiler and duct insulation pipe work, and the reason I was anxious to make these statements at this time was that some of the things that Dr. Nicholson has pointed out very directly apply to what I have to present, which is not about

asbestos.

Briefly, I will say that we agree primarily, and I have no defense for the use of asbestos or, in fact, we are doing something in our industry locally here to try and eliminate the use of asbestos in the functions we use it for, which is primarily as coatings and finishes. We are, for example, using other types of jackets, and other things which would eliminate the use of asbestos on construction work, where we have used it.

Now, that doesn't go into the asbestos spray problem, and we are not really concerned with that primarily. I think I would like to point out, though, that we are concerned with the use of fiberglass duct lining and fiberglass ducts which has grown very greatly in usage in new construction work in this area. We are against it for the same reason that asbestos is being eliminated. We feel, and we know that fiberglass particles are being introduced into the air through the duct systems that are using fiberglass liners or fiberglass ducts to transmit the air. This air is going into confined spaces where people are being subjected to

whatever harmful effects there might be.

I should state that while these particles are not toxic, at least to the extent we have been able to find out, and therefore it is not in the same realm of danger as asbestos, they are a dangerous cause of -- either cause or aggravate various lung or respiratory conditions, and therefore do present a danger to people who have to breathe the air, where these fibers are introduced.

Some of the dangers that Dr. Harwood discussed would apply, therefore, to the fiberglass particles that are coming through all of these diffusers that allow air into any confined space. We have conducted some research on this matter locally, and we have determined very conclusively that there are particles of fiberglass being transmitted and brought into the air stream when fiberglass liners are used in the ducts, or when fiberglass ducts are used. This, of course, all occurs after any filtration systems that are used in air transmission systems, so it comes directly into the space where the people breathe the air.

By the way, the quantity of the fiber transmission seems to be greatly increased after a

system has been in effect for a number of years, where the air has been distributed for many years. We haven't been able to establish just when this begins to occur, or just how it occurs. There is some deterioration, we do know that, and we are going to conduct some further research on it.

There have been some regulatory provisions introduced to prevent the use of fiberglass duct lining in certain locations, but we feel that the same logic which has caused this kind of regulation should be extended, because it applies to other areas as well.

The Hill-Burton Act, which has some governing effect on new construction of hospitals, has prohibited the use of fiberglass duct liners on any air systems which serve critical areas such as the operating rooms or nurseries, and similar types of spaces. The State of Indiana has virtually eliminated the use of fiberglass duct liners on any hospital construction.

We feel that that same kind of precaution should be used in other locations where the public will be subjected to any long-term exposure of these particles, the schools, offices, theatres, public

places, and so on.

The only other note I would like to add is that the only really functional -- necessary functional requirement that a duct liner acts to present is in reducing sound transmission. It has an acoustical value. It is also used thermally and that is, of course, where we feel it should be eliminated, because we can thermally insulate on the exterior and eliminate any potential harmful effects on the interior of the ducts. The sound transmission can be effectively -- I should say the elimination of sound transmission by the use of a liner can be done by judicious use, rather than lining the whole system. We have also conducted some research there, and we find that the last few feet before a diffuser, just lining in those locations, can accomplish just as much as if the entire duct system were lined. Also, there are ways of inserting some kind of a mechanical protection, such as a metal mesh, on the inside of a liner, and that reduced substantially any potential fiber transmission or deterioration of the liner. That is all I have to present.

Maybe there is a question or two from the Board members?

MR. DUMELLE: No.

HEARING OFFICER LAWTON: Thank you very much.

THE WITNESS: Okay.

HEARING OFFICER LAWTON: We will receive into the record Dr. Harwood's presentation in two parts, as Exhibit No. 20, and Dr. Nicholson's statement as Exhibit 21.

(The documents above referred to were thereupon marked Board Exhibits 20 and 21, for identification, and were received in evidence.)

HEARING OFFICER LAWTON: We will adjourn now until 12:45, at which time we will hear from Mr. Romer and Mr. Fundsack.

(Whereupon, an adjournment was taken until 12:45 o'clock p.m. of the same day.)

BEFORE THE
ILLINOIS POLLUTION CONTROL BOARD

In the matter of:

ASBESTOS REGULATIONS, CONTROL
OF SPRAY INSULATION

}
}
} No. R 71-16

Field Museum,
Chicago, Illinois
October 15, 1971
12:45 o'clock p.m.

BEFORE:

Mr. Samuel T. Lawton, Hearing Officer.

PRESENT:

Mr. Jacob D. Dumelle, Board Member
Mr. Timothy Harker, Administrative Assistant
Mr. Walter Romanek, Administrative Assistant.

Members of the Press
Members of the Public.

HEARING OFFICER LAWTON: We are ready to resume the hearing. Mr. Romer?

HAROLD ROMER,
called as a witness herein, having been first duly sworn, was examined and testified in narrative form as follows:

HEARING OFFICER LAWTON: Would you be good enough to identify yourself, tell us your present association and technical background, please?

THE WITNESS: Mr. Chairman, ladies and gentlemen, my name is Harold Romer, and I am Assistant Commissioner for Engineering Research and Development of the New York City Department of Air Resources. I was formerly a Chief Engineer and Director of Environmental Engineering in the New York City Department of Health, and a licensed professional engineer and a delegate of the American Academy of Environmental Engineers. I am also a professor of environmental engineering in the Environmental Sciences and Engineering Departments of the Graduate School of C. W. Post College on Long Island.

Some of the testimony I have prepared, I am sure is going to be somewhat redundant with that

of Bill Nicholson, and of course, that is not very surprising, in view of the fact that we worked very closely together. But I do have some special emphasis on the problems of engineering, which I feel more proficient to speak about.

It is essentially because of the very disturbing medical statistics that the New York City Environmental Protection Administration and the Department of Air Resources have been so concerned and have become, literally and figuratively, so completely immersed in the sprayed asbestos problem.

With your permission, Mr. Chairman, I will quote the one or two references which are not only indicative, but also provide the punch merited by the facts. The following material is abstracted from a 1969 United States Public Health Service Report, "Preliminary Air Pollution Survey of Asbestos -- A Literary Review":

"A large fraction of the people in the United States have been exposed to asbestos, including the following:

"(a) Approximately 100,000 workers using asbestos in their occupations.

"(b) Approximately 3,500,000 construction workers exposed indirectly to asbestos as they work with asbestos products or near those who handle asbestos products.

"(c) Approximately 50 million Americans who possibly have 'asbestos bodies' in their lungs."

In November, 1970, in a court case against the City of New York, we called upon Dr. Irving Selikoff to provide expert medical testimony. Dr. Selikoff, a consultant to our agency, is a renowned researcher and pioneer in the field of asbestos diseases. He is also Director of Environmental Science Laboratories at the Mt. Sinai School of Medicine in New York City. The following quotes are abstracted from his testimony:

"In New York, in 3,000 consecutive autopsies at Elmhurst Hospital in Queens, the V. A. Hospital in the Bronx and Mt. Sinai Hospital in Manhattan, examination of lung tissues have shown that asbestos bodies consist in 45 percent of all cases. Smaller particles of asbestos, seen only by electronmicroscopy, were found to be commonly present in almost all cases examined.

It now appears that virtually all New Yorkers' lungs contain asbestos."

Continuing with the quotes:

"Among asbestos workers, the incidence of death from lung cancer among smokers is about 20 times as high as for non-smokers. If a person is both an asbestos worker and a cigarette smoker, his chance of dying from lung cancer is 92 times the chance of a man who neither smokes cigarettes nor works with asbestos."

These statistics were quoted by Dr. Nicholson:

"Recently, I conducted an investigation with respect to asbestos-related conditions among the members of Local 12 (New York City) and Local 32 (Newark) of the International Association of Heat and Frost Insulators and Asbestos Workers, one of the building trade unions."

Continuing with the quotes:

"As of April 30, 1970, 403 of the original 632 union members in our study were dead, 72 of lung cancer. Currently, deaths among members of Local 12 in New York include one in five of lung

cancer, one in ten of mesothelioma, one in ten of asbestosis, and one in ten of gastrointestinal cancer."

If you work this out mathematically, the incidence of cancer turns out to be something like 40 percent of those who work in the industry in that particular union.

Continuing with the quote:

"Asbestos -related cancers occur not only in occupational exposed workmen, but may occur among people living or working in an area where asbestos fibers are disseminated. Mesothelioma, for example, which is invariably fatal, has occurred among people living in the contaminated households of asbestos workers as well as among people living within a half-mile of major asbestos sources such as asbestos factories, mines, and mills. Investigations by Wagner in South Africa, Newhouse in London, Leiben in Pennsylvania, Bohlig in Germany, and others, reveal a substantial health hazard to such persons. In London an examination of the records in one hospital revealed that of 76 recorded mesothelioma cases, 31 had worked in an asbestos

factory, nine of the remaining 45 had lived in the household of an asbestos worker, and 11 simply lived within a half-mile of an asbestos plant."

There will be more emphasis on that. Those comments were mine, not Dr. Selikoff's.

"My group at Mt. Sinai has investigated the homes of asbestos workers and has found the air the contaminated by asbestos particles carried home on the clothing of the workers. We have also seen cases of mesothelioma among relatives of asbestos workers and people who lived near an asbestos-using facility."

That is the end of the quotes.

The consumption of asbestos in the United States has assumed large proportions and its use is increasing at a rapid rate. The 1968 consumption in the United States was equal to 25 percent of the total world production. Annual world production in 1930 was 500,000 tons; in 1967, four million tons of asbestos were produced.

During the past ten years, asbestos-containing material has been used extensively in sprayed form at an increasing rate, to protect steel structures

from fire. In 1970, approximately three million pounds of asbestos were used to fire-proof structures. It is particularly with reference to this use of sprayed asbestos that I wish to address my remarks.

In the United States, almost 90 percent of the asbestos used is of the chrysotile fiber type that easily fragments into fibrils and the fibrils are 200 to 400 Ångstroms in diameter and often 2,000 Ångstrom units or more in length, and for your information, 200 Ångstrom units equals a millionth of an inch, approximately. Thus, one billionth of a gram of chrysotile asbestos could represent a million fibrils. Chrysotile asbestos fibrils can only be identified by the use of the electronmicroscope.

Seventy percent of the asbestos in this country is used in the construction industry.

From the published medical reports and epidemiological studies it would be fair to state that the individual, invisible asbestos fibrille represents a built-in "time bomb" set aeons ago when the fibrous silicate crystals cooled and began to grow within the fractures of the serpentine rock. The mechanism can be activated when these fibrils

are inhaled or ingested, albeit it may take 20-25 years to wreak its havoc in the form of excessive cancer incidence rates, including the invariably fatal pleural and peritoneal mesotheliomas.

It is because of the relative indestructibility of the asbestos fibril and its inherent potential for harm, as well as the unsatisfactory experience of the New York City Department of Air Resources in its efforts to control asbestos spraying operations, that the City of New York urges the banning of the spraying of any substance containing asbestos on in or upon a building or other structure during its construction, alteration or repair.

Because of our recognition of the intense health hazards of asbestos exposure, the Department of Air Resources on April 13, 1970, issued interim regulations, known as Commissioner's Orders, prescribing containment and the clean-up methodologies. This was the first such set of rules anywhere pertaining to the regulation of sprayed asbestos insulation. This was the catalyst that triggered regulatory agencies throughout the country to adopt similar procedures and the subsequent banning of

sprayed asbestos by several cities. We are pleased that both the States of Illinois and New York have proposed the prohibition of this dangerous practice throughout these states. I understand that the Federal EPA is also considering the banning of sprayed asbestos.

A challenge to the power of the Commissioner of the Department of Air Resources to regulate spraying operations using asbestos-containing materials for fireproofing and insulation at construction sites arose in the November 1970 case of LaMonica versus Jerome Kretchmer, Administrator of the Environmental Protection Administration. The State Supreme Court ruled in favor of the City and denied the injunction requested for the plaintiff, and declared the Commissioner's orders and the Air Pollution Control Code to be constitutional.

Dr. Selikoff, in testimony during the LaMonica-Kretchmer case, although the bulk of his remarks centered on the medical effects of asbestos inhalation, stated, "In Manhattan ambient levels far removed from construction sites are now $20-60 \times 10^{-9}$ grams per cubic meter of air, up to six times more than in, say, Staten Island, or

Ridgewood, New Jersey. Indeed, the ambient air levels about construction sites are even several times higher than these background levels. At these levels the average person might inhale ten to fifteen million asbestos fibrils in one 24-hour day.

The April 1, 1971 issue of the New York Times reported that Federal EPA began publishing a list of air pollutants which, in the opinion of Administrator William D. Ruckelshaus "May cause, or contribute to, an increase in mortality or an increase in serious irreversible or incapacitating reversible illness." Mr. Ruckelshaus "named three such pollutants -- asbestos, beryllium and mercury."

Our efforts at controlling the emission of asbestos from spray operations have not been successful. On numerous occasions, emissions were such that heavy asbestos snowfalls were observed and accumulations of asbestos as deep as three inches at curbs and other vertical rises occurred within a radius of several blocks of the spraying site.

Since there are 25 to 40 skyscraper asbestos insulation-spray operations in progress at

any one time, it would take virtually our entire field staff to provide adequate surveillance for this one function. Even if this were feasible, which it is not, I strongly question the ability of the industry to prevent the emission of asbestos into the outside air under actual day-to-day conditions of construction.

As usual, court actions resulting from the issuing of many summonses, have had but limited deterrent value. The adjournments were endless, and the fines when finally imposed, were only a minor overhead to the contractor. The administrative hearings held by the Department of Air Resources to show cause why an asbestos-spraying operation should not be halted, have been very time-consuming though, temporarily, of some value.

It should be made clear that the Commissioner's orders, promulgated on April 13, 1970, were intended to provide a workable methodology which could be implemented immediately by the construction industry. To provide for methodology which would insure positive containment of this carcinogenic material, would have resulted in the complete cessation of the construction on all skyscrapers in our city.

Our experience clearly indicates that even without present methodology, even with conscientious follow-up by the contractor, asbestos does, in lesser or greater quantities, can be found in almost any part of the building under construction. In industry, where asbestos products are manufactured, clean-room procedures often are employed, including an exhaust system and a filter bag house placed directly above or below such processes as cutting operations to preclude the likelihood of asbestos dust reaching the face of the worker or entering the workroom or the outside air. This positive type of control is required when handling asbestos, if confidence in the methodology and protection of the public and worker are to be assured.

Visible emissions, prohibited by our Code are difficult to observe, especially when spraying occurs on the other floors of skyscrapers, unless there is a veritable snowstorm of asbestos into the outside air. Perhaps more important than the visible emissions, however, are the fine asbestos particles which are not visible to the naked eye, but which are more hazardous because they can penetrate

the lungs' smallest alveoli. Again, I note that a billionth of a gram of chrysotile asbestos may contain a million fibrils of asbestos. Sampling for such particulates is not only extremely difficult at upper stories, but also time-consuming and expensive. By the time laboratory results would become available, the spraying procedure of this specific area sample would be long completed.

Even if a methodology were developed which could protect the ambient air from sprayed-asbestos particles, a problem, for which no solutions are currently known, would dictate the banning of sprayed asbestos. I am talking about demolition dust. It is difficult to visualize the demolition of a magnificent 30, or 40, or 50-story structure. Nevertheless, based upon the construction and demolition history of buildings in New York City, we can anticipate that a significant that a magnificent percentage of these "old-fashioned structures" will be demolished 30, 40 or 50 years hence.

The present state of the art is such that there is reasonable assurance the City of New York will be coated with asbestos when these buildings are demolished. We hope to develop ways and means

to cope with demolition dust problems, however, there is no certainty of success, particularly because of the inherent difficulties and high costs.

Last, and certainly not least, is the unbelievable hazard to workers employed in spraying asbestos. The tremendously high incidence of lung cancer, 100 percent fatal mesotheliomas and other carcinogenic causes of death experienced by asbestos insulation workers cry out against the use of sprayed asbestos.

With the knowledge that it was, at best, a most difficult task adequately to supervise all construction sites using sprayed asbestos insulation, and the time-consuming administrative hearings, and with the problem of multiple adjournments and meaningless fines in court, as well as the preponderance of medical evidence as to the pathogenicity of asbestos, the Department of Air Resources embarked on a campaign to encourage manufacturers to eliminate asbestos and develop suitable substitute fireproof materials.

At present, I can report that our Buildings Department has approved of three sprayed non-asbestos

fireproofing materials for specific assemblies, and a fourth material is well on its way to probable approval. Furthermore, at the request of the Department of Air Resources, the Health Research Council of the City Department of Health is evaluating the health aspects of these substitute materials.

On August 25, 1971, our City Council, at the recommendation of the New York City Environmental Protection Administration and Department of Air Resources, enacted into law the prohibition "No person shall cause or permit the spraying of any substance containing asbestos in or upon a building or other structure during its construction, alteration, or repair." The cities of Chicago, Philadelphia and Boston also have banned the use of sprayed asbestos insulation.

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The New York City Environmental Protection Administration and the Department of Air Resources have great concern, not only with sprayed asbestos, but also with all sprayed insulating materials. On August 25, 1971, our City Council enacted into law provisions relating to the methods of enclosure and clean-up when spraying any insulating material.

The same problem of emissions into the

open air we experienced with sprayed asbestos-containing insulation are being and doubtlessly will continue to be experienced with non-asbestos sprays, as long as the present methodology of temporary enclosure is practiced. The use of tarpulins for enclosure is not satisfactory on a continuing basis.

I have proposed several alternatives. Fireproofing of peripheral columns, spandrels, et cetera by non-spray means, such as boxed beam with lathe and plaster, drywall encasement, concrete encasement, et cetera. Spraying of peripheral steel is a major source of visible emissions into outer air. Design structures so that skin and windows can be installed before any spraying is done. In any insulation spraying operation, install exhaust system and bag house to ameliorate the astonishingly bad condition under which spray-workers now operate and to reduce emissions outside the building.

In my many consultations with architects, engineers, builders and their associations, there is general agreement that there are virtually insurmountable problems associated with insulation

spraying.

There are many available alternatives to spraying, however, each is more costly than spraying. Reported costs for the various alternatives differ widely, depending upon the builder one speaks with. Some average numbers indicate that for high-rise structures the cost of spray insulation is three-quarters of one percent to one percent of the total cost. What does "total cost" mean? Some mean "bricks and mortar" and others include bricks and mortar, foundation, land costs, architectural fees, financing charges, profit, et cetera. Costs for drywall encasement vary from two to three percent of "total cost", boxed beam with lathe and plaster from two to three percent of "total cost", and concrete encasement as high as five percent.

If one of the less costly approved alternatives were selected, the increase in cost would be in the area of one and three-quarters percent. No one wants to spend more than he is required, however, this additional cost does not appear prohibitive, and above all, will protect both public and worker.

We have been encouraging and working closely

with product manufacturers to develop competitive non-asbestos insulating materials and methodologies. Among these are application of insulation at the steel fabrication mill, insulation in rolls and strips which can be applied to the steel adhesives, application by troweling, intumescent mastics, insulation blankets wrapped around steel members on the site, et cetera. For reasons of confidentiality, I cannot go into additional details at this time. Some of these alternatives should be ready within six or twelve months. We welcome any new ideas and stand ready to be of assistance in bringing new products to fruition.

May I add at this point that on October 1, 1971, mineral wool blankets were approved in New York City for the protection of columns, and I consider this a major break-through in getting away from the spraying of insulation. If only the peripheral columns were blanketed with these insulation blankets, this would be a great step forward.

I would like to quote from an article by Ira Hooper, Modern Steel Construction, the issue for the first three months of 1971. It is a rather interesting quote:

"With all the current emphasis on the systems approach, it is curious that a practical factory assembled enclosure has not yet been developed for the fire protection of steel beams. In addition to obtaining the usual advantages of factory production, the problem of air pollution would be avoided. The pre-fabricated units could consist of two sides and a bottom that enclose the beam and are attached to the floor slab by stud wells or power-actuated fasteners. Or the units could be hung from studs welded to the bottom flange while the sides are braced by studs welded to the web."

What he is saying is that the systems approach has not been employed in this industry, and I quite agree with him. Now, we have a special problem which was touched upon before, particularly during the presentation by Dr. Nicholson, and the questions by the Board, namely that the State and Federal Labor Departments have set up the working atmosphere dust standard of not more than five fibers per cc, five microns or larger. What does this mean in view of our recent experience? A study in which

we are now engaged produced air analyses producing no fibers five microns or larger. However, all particulates, asbestos fibrils, were submicron. The concentration of the submicron fibrils were approximately 200 per cc. This is 40 times greater than the industrial standard for fibers. I wish to emphasize we are talking about fibrils, the small submicron particles and the fibers which are generally above five microns. It is important to be cognizant of this difference. By definition, the air met industrial quality standards since there were no fibers five microns or larger. Normally, industrial air analyses do not include examination for submicron particles. A sample of air having a zero count of fibers five microns or larger, would be considered excellent. Obviously the standard must be reexamined, especially since the submicron particulates are highly respirable and present a greater hazard to health.

The practical problems of performing electronmicroscopy, X-ray defraction, et cetera, examination of air samples for the positive identification of asbestos fibers, and for the identification of other insulation fibers less than five microns in length are recognized. Accordingly,

it is suggested that the method proposed by the Illinois Pollution Control Board, namely phase contrast microscopy at 430 times magnification to identify fibers five microns or larger, be utilized routinely provided that electronmicroscopy, X-ray defraction, et cetera, be employed at regular intervals of time to determine whether the ratio of the less than five microns fibers to fibers five microns or larger has increased over previous experience. Since medical evidence indicates that very small fibers may be the most dangerous, it is further recommended that consideration be given to revision of the air quality standard wherein fibers of any length be counted rather than only fibers of five microns or larger in length.

I call this paragraph a "caveat". There are more than 3200 manufactured products in this country which contain asbestos. In my judgment, control of the ubiquitous asbestos fiber should be given a high order of priority if the public's health is to be protected. The scope of the asbestos problem can be recognized by enumerating a few of the common uses: Textiles, and incidentally, recently, women's coat fabric was found to contain asbestos.

You may have read about this, and when the coat was shaken, a high concentration of asbestos was to be found in the vicinity of the coat or the person wearing the coat; yarns, cord and braiding; sealing and packing material; gaskets; filter-paper, used in filtering injectable drugs; coverings, coatings, and jacketings; auto parts; protective walls and curtains; furnace and drying oven linings; asbestos cement, such as water and sewer pipes, corrugated sheets, interior walls, ventilating shafts, air conditioning assemblies; thermal insulation; road surfaces; friction materials such as brake linings and clutch linings; asbestos plastics such as floor tiles and molded insulation, also used in aircraft industry; synthetic snow sprayed by small children on artificial Christmas trees from aerosol cans; and finally, asbestos can be found in talcum powder dusted liberally on baby bottoms and inhaled with the nursery air.

I would like to make the following recommendations: I urge the Illinois Pollution Control Board to ban immediately the spraying of asbestos-containing insulation and fireproofing in or upon a building or other structure during its construction, alteration or repair; also prohibiting the sale of

motor vehicles manufactured after January 1, 1975 if brake linings contain asbestos. I urge the Illinois Pollution Control Board to closely control other commercial uses of asbestos in construction work, the demolition of asbestos-containing structures, the manufacture and processing of asbestos-containing products, the transportation, storage and handling of asbestos-containing products, and the disposal of asbestos-containing wastes.

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I further urge the Illinois Pollution Control Board to control the spraying of any insulating material not otherwise prohibited, in or upon a building or other structure during its construction, alteration or repair. Thank you.

HEARING OFFICER LAWTON: Thank you very much. Mr. Dumelle, do you have any questions?

EXAMINATION

By Mr. Dumelle:

Q I have a question, Mr. Romer, about the five micron length. What we are really talking about, are we really talking about particle counts, the particles below five microns, as also being dangerous because of being more respirable? Is

there some number or some threshold that we can talk about, or are we saying asbestos or no asbestos?

A I would prefer not to try to answer that question, because as an engineer I lack the expertise in terms of the epidemiological response, and I would not feel qualified to answer that question. However, it would seem to me on the basis of my extended contact with this problem, that total attention has been paid to fibers in general, or dust particulates in general in the environment as well as the outside environment. Certainly, no attention has been paid to the submicron particulates, because of the problems of detection, and I had mentioned before, and I urge -- in fact, I dare say that in the many talks that I have given, that their attention has been called to this item by myself and others, and I anticipate that the next direction will be that of submicron particulates as well as micron-sized particulates.

MR. DUNELLE: Thank you.

EXAMINATION

By Mr. Harker:

Q Mr. Romer, are you prepared to say that the five micron limit, which is generally a standard used, has no basis in medicine, but really is based in the -- the practicality of the five micron size?

A From what I understand in the practice and literature I have read, that --

Q Am I correct, that New York City has regulations which would require certain procedural safeguards for the demolition of any structure, regardless of whether it contains asbestos or not?

A Yes, sir.

MR. HARKER: Thank you.

HEARING OFFICER LAWTON: Thank you very much, Mr. Romer.

THE WITNESS: Thank you.

HEARING OFFICER LAWTON: I think it is appropriate at this time to express the gratitude of the Board, to the Institute for Environmental Quality, for providing the witnesses we have heard this morning, and the preparation of these papers that have been received in the record. Again, we express the Board's appreciation to Dr. Harwood and Dr. Nicholson and Mr.

Romer.

The next witness is Mr. Pundsack of the Asbestos Information Association.

FRED. L. PUNDSACK,

called as a witness herein, having been first duly sworn, was examined and testified in narrative form as follows:

THE WITNESS: My name is Dr. Fred L. Pundsack, and I am vice president of research and development for the Johns-Manville Corporation. I am an inorganic chemist with a Ph.D. degree from the University of Illinois, and I have been involved in asbestos research since 1952. I am here today representing the Asbestos Information Association, an organization sponsored by nine leading manufacturers of asbestos products to provide information on asbestos and its health implications.

In the event that my presentation prompts questions relating to environmental control technology or the medical aspects of the asbestos situation, I have asked three experts in these fields to accompany me here today to assist in answering questions of a highly technical or medical nature. They are Dr.

Joseph L. Goodman, Associate Professor, Department of Preventive Medicine, Medical University of South Carolina, and Medical Director, Raybestos-Manhattan Corporation; Mr. Isaac H. Weaver, Corporate director for Environmental Control for the Raybestos-Manhattan Corporation; and Mr. George L. Swallow, Manager of Occupational Environmental Control, Johns-Manville Corporation.

Before proceeding, I would like to express the appreciation of the Asbestos Information Association for the opportunity to testify on the proposed asbestos regulations.

Asbestos has numerous important applications in our modern industrial society. Among these, it provides built-in protection against fire and deterioration in scores of common products in daily use. Through the years it has saved countless lives and billions of dollars in property damage by preventing or checking the spread of fires. Asbestos-containing acoustical products make a valuable contribution to noise abatement. Products containing asbestos are used, among other applications, in the construction of schools, houses, theaters, factories, office and other public buildings, and in the insulation of

furnaces, boilers and electrical equipment.

The asbestos industry within the State of Illinois plays an important role in the economic life of the state and its people. The member companies of the Asbestos Information Association own and operate a total of ~~eight~~ plants within the boundaries of the state. Some of the product lines produced at these plants include: asbestos-cement pipe, packings, gaskets, friction materials -- including both conventional and disc brake linings -- asbestos-cement sheet products, asbestos roofing and siding shingles, insulations, and many others. A total of 3,118 Illinois residents are employed at these manufacturing facilities. Last year, these employees received a total of \$24.4 million in wages, salaries and fringe benefits. Service and goods purchased within the state, such as water, electricity, telephone service, raw materials and freight amounted to more than \$17 million in 1970. In addition, state, county and local taxes, amounting to nearly a million last year, helped to pay for new schools, roads and other benefits to the state and its citizens. The value of goods produced at these manufacturing locations last year totalled \$70.5 million, and approximately \$14.5 million worth of

asbestos-containing products were sold in Illinois in 1970.

As you can see, the economic contribution of the asbestos industry in the State of Illinois is by no means small. And the figures I have just given you represent only those companies that are members of the Asbestos Information Association. There are also within the state many other concerns -- both large and small -- whose livelihood depends in some measure on the use of asbestos or asbestos-containing products. Such concerns would include many segments of the construction industry, companies engaged in the manufacture of asbestos-containing products, companies which use finished asbestos-containing products in the manufacture of other products, and many others. The economic value to the state derived by the use of asbestos by these other concerns is difficult to gauge, however it would certainly double or triple the figures I gave you earlier with regard to the contribution of the member companies of the Asbestos Information Association.

The asbestos industry has recognized for many years that there are occupational health hazards associated with the excessive inhalation of asbestos

dust over long periods of time. As a result, the industry, beginning in the late 1920s, has sponsored and cooperated in a wide variety of scientific research projects designed to identify these hazards and to eliminate them wherever they may exist, not only in asbestos mines, mills and manufacturing plants, but also among fabricators and applicators of finished asbestos products. Today, the asbestos industry, either as individual companies or through the Quebec Asbestos Mining Association's sponsorship of the Institute of Occupational and Environmental Health in Montreal, is supporting medical research at such highly regarded institutions as McGill University in Montreal, Tulane University in Louisiana, St. Luke's Hospital in Cleveland, the Environmental Sciences Laboratory at Mount Sinai Hospital in New York, the Industrial Health Foundation in Pittsburgh, the University of California at Berkley, Farleigh-Dickinson University in New Jersey, the University of Pittsburgh, the Medical College of South Carolina, and a number of others, both here and overseas. In addition, the industry has cooperated extensively in various investigations into the asbestos-health conducted by agencies of the Federal Government,

including the U.S. Public Health Service, the Federal Environmental Protection Agency, the National Institute of Occupational Safety and Health, and others. In the industry's own research laboratories, scientists and engineers are also engaging in work related to health, developing safer packaging and handling techniques for loosely bound asbestos products and also developing and improving techniques for the measurement and control of asbestos emissions both in the work-place and in the ambient air.

As a result of these activities, over the years the industry has amassed a substantial amount of medical knowledge and technological know-how in the asbestos-health area.

Basically, the known facts about asbestos-related disease can be summed up as follows:

First, asbestos-related health risks are basically confined to the occupational and para-occupational setting.

Second, the effects of excessive inhalation of asbestos are both time and dose related. This means that asbestos-related diseases may develop, generally, only after the inhalation of substantial amounts of asbestos dust over a substantial period of time.

And third, there is presently no evidence of hazard to the general public from exposure to the minute amounts of asbestos that may be present in community air.

Because the general purpose of the regulations under discussion here today is to prevent the emission of hazardous quantities of asbestos fibers into the air that the general public breathes, I will restrict my further comments on health to this area. It is very important to realize in any discussion of a possible public health hazard from asbestos exposure that the only scientific studies we have to go on are those of groups of people in occupational and para-occupational environments. There is no basis for assuming that data on occupational health risks associated with heavy, long-term asbestos exposure can be applied to the general public without taking into consideration the vast differences in exposure between the two.

Despite the fact that the only studies in existence are of occupational and para-occupational populations, a careful analysis of the data available can give us some general information about the question of a possible public hazard from asbestos. The

substance of this information is that diseases associated with occupational exposure to asbestos are dose related and that the doses below which no measurable increase in disease occurs are far higher than any dose to which the general public is exposed. This evidence is based on a number of epidemiological studies -- three of the most prominent being one by Dr. J. Corbett McDonald of 10,421 past and present asbestos mining and milling workers in Quebec, a second by Dr. M. L. Newhouse of 4,500 men who were employed in a British asbestos factory between 1933 and 1964 and the third a study by Drs. Knox, Doll and associates of 878 workers in an asbestos textile factory, also in Great Britain.

Dr. McDonald, in his study, divided his subjects into eight categories, based on length of employment and severity of dust exposure. He noted that only in the highest time-exposure category, comprising five percent of the total, was there any increase in deaths from respiratory and cardiac diseases, including lung cancer. Taken as a group, the Quebec asbestos workers had a mortality rate from all causes lower than the level expected in the general population.

Dr. Newhouse in her study in Great Britain found that workers who had low and moderate occupational exposure to asbestos dust showed a rate of deaths from diseases of the lung, including cancer of the lung and pleura, that were comparable with that of the general public. On the other hand, another study by Dr. Newhouse of mesothelioma which was referred to earlier and which is a relatively rare cancer of the lining around the lungs or abdomen, in the London area showed a small number of cases among people who lived in close proximity to an asbestos textile plant and also among people living in the household of an asbestos worker, who presumably brought home quantities of the material on his clothes from work. The intensity of exposure experienced by these so-called para-occupational populations in London is difficult to gauge, but there is every reason to believe that they were in fact higher than the low and moderate occupational levels in factories that have been shown to produce no excess of mesothelioma. These studies do, however, point up the need for proper control of emissions of asbestos from factories so that neighboring areas will not suffer para-occupational exposures to asbestos.

The Knox-Doll study, like the Newhouse and McDonald studies, confirmed the fact that low to moderate levels of asbestos exposure will not produce an excess of disease. It should always be remembered that these low to moderate levels in occupational settings inside factories were certainly higher than any to which the general public in this country is exposed.

In addition to these studies, there are several studies of the effect of various doses of asbestos on laboratory animals which also indicate that there are dose levels below which no measurable disease effect is observed. Dr. William Smith of Fairleigh-Dickinson University studied mesothelioma cancers in hamsters, and his data show that below certain specific doses of asbestos, no cancers were observed to develop over the entire natural life span of the animals. Dr. Merl Stanton of the National Institute of Health, Bethesda, Maryland, conducted a somewhat similar study with rats and observed the same type of dose-related response.

The most well-known of the American epidemiological studies related to asbestos exposure is that made by Dr. I. J. Selikoff of 632 members of

the Heat Frost and Asbestos Insulation Workers Union in the New York-New Jersey area. These men, for the most part, are journeymen-craftsmen who work on construction sites and apply insulation to boilers, steam and hot water pipes, heating ducts, et cetera, during the construction of a building or a power plant. They may also be involved in tearing off old insulation. When one construction job is completed, they move to another construction job.

The occupational environment to which they may be exposed involves not only asbestos but silica and particulate materials. In addition they are exposed to a variety of fumes from organic solvents and adhesives with which they work. In short, they have a widely varied and, in the past at least, a relatively uncontrolled occupational dust and fume exposure.

Dr. Selikoff found that the death rate from lung cancer was extremely high in this group of 632 workers, but this very high mortality rate occurred almost exclusively among those workers who were cigarette smokers.

In general, the high mortality rate of insulation workers found by Selikoff are much higher

than those found in the studies of larger groups of people who work in factories producing asbestos-containing products and in mines and mills processing asbestos. Why insulation workers' rate of certain disease is so much higher than all other groups is a question as yet unanswered. It may be that this group of insulation workers represents a very high occupational dust exposure when the wide variety of dust to which they are exposed is taken into account.

Unfortunately, as is so often the case with diseases that take from 10 to 40 years to develop, reliable data as to the past occupational exposure of these men to asbestos and other potentially hazardous dust and fumes in their work are not available.

As a result, at the present time we have little information as to what other factors may have influenced their high rate of disease except that those insulation workers who smoked cigarettes developed lung cancer at a rate far higher than the general population who smoked.

In 1970, with a grant from the National Air Pollution Control Administration, The National Academy

of Sciences called together a blue-ribbon panel of asbestos-health experts to draft a document on the problem that would serve as the medical basis for the Federal asbestos emission standards. Among those asked to serve on the panel were Dr. Clark Cooper of the University of California, Dr. Lewis Cralley of the Public Health Service, Dr. George Wright, Medical Director of St. Luke's Hospital in Cleveland, Dr. Irving Selikoff, of Mt. Sinai, and others.

The result of their study was this fifty-three page document I have in my hand. As a conclusion to this part of my presentation, I would like to read a few pertinent excerpts from the report, which constitutes the most up-to-date evaluation of the asbestos-health problem that is available.

On page 20, the report states, "We cannot extrapolate from the mortality experience of those who are directly and indirectly exposed in their employment to the general public who have had moderate or slight exposure from ambient air. There is evidence to suggest a gradient of effect from direct occupational, to indirect occupational, to family and neighborhood situations, in all of which

dust concentrations are probably high by comparison with most community air. This suggests that there are levels of asbestos exposure that will not be associated with any detectable risk. What those levels are is not known, but there is no evidence that persons in the general population -- without occupational, household or neighborhood exposures -- have any increased risk of neoplasm, even though there may be ferruginous bodies or fibers in their lungs."

In the conclusion and recommendation section of the report for the National Academy of Sciences, the following statements are made:

"At present, there is no evidence that the small numbers of fibers found in most members of the general population affect health or longevity. Asbestos is too important in our technology and economy for its essential use to be stopped. But, because of the known serious effects of uncontrolled inhalation of asbestos minerals in industry ... it would be highly imprudent to permit unrestricted additional contamination of the public environment with asbestos."

I believe that these statements from the report adequately express the position of the asbestos industry with regard to the health hazards of asbestos. We do not believe that at present the general public is in danger from the minute amounts of asbestos that exist in the ambient air. However, we also believe that it is prudent and feasible to establish reasonable emission controls that will protect the public and ensure that hazardous levels of asbestos will not develop in the community air in the future.

With this position in mind, I would now like to discuss the asbestos regulations proposed for the State of Illinois. To begin with, I would like to commend the Illinois Pollution Control Board for its efforts to ensure a safe environment for the State and all its citizens. Your credentials as a progressive yet fair control board are well known and respected in environmental circles throughout the nation.

While the asbestos industry as represented by the Asbestos Information Association is of the position that there is presently no hazard to the general public from the minute amounts of asbestos existing in the community air, we nevertheless are in

favor of regulations designed to accomplish one or more of the following three objectives:

One, to ensure the health and safety of those occupationally exposed to asbestos fiber.

Two, to prevent possible hazardous amounts of asbestos fiber from escaping into the ambient air, and

Three, to eliminate, where possible, the nuisance of even small amounts of asbestos fiber from escaping into the ambient air. While this third point is not directly related to health because of the small amounts of fiber involved, we wholeheartedly support the position that the environment should be as clean as possible.

For these reasons, we support most of the items in the proposed regulations. However, we do have several specific comments and suggestions to make regarding the regulations that we hope will be of help to the Board in arriving at a final document that will not only be effective but also practical and reasonable. I will discuss the sections upon which we wish to comment in the order in which they appear in the regulations.

Our first suggestion is with regard to Part II:

General Requirements, Section 201. This section calls for the obtaining of a permit to engage in any activity which could discharge asbestos fiber into the environment. We feel that this section is not specific enough with regard to which activities would be covered. As you are probably aware, in the vast majority of asbestos-containing products used in the country, the asbestos is locked in place with cement, plastics or other binding materials, and does not constitute a source of emissions to the atmosphere. Asbestos-cement pipe and vinyl-asbestos floor tile are examples of such products. Other products and activities do constitute possible emission sources, and under the regulation would require a permit. If it would be of help to the Board, the Association I represent would be most willing to prepare a list of those products in common use where the asbestos is in a non-locked-in or loosely-bound state and thus constitute a possible emission source which would require a permit.

As to Section 201, Sub-section B, requiring a satisfactory course of health instruction for employees in those industries involving the handling of loose or loosely-bound asbestos products, I would like to point out that the various trade associations

in the asbestos industry have over the years produced a number of safety practices manuals and booklets dealing with various sections of the industry. The Asbestos Information Association would therefore recommend for your consideration as instructional material for use under Section 201, Sub-section B, the safety practices booklets which I have submitted to the Board with my presentation.

Under Part V, Section 501, it is our opinion that Subsection B, which requires the enclosure of walls before toppling during demolition, is both impractical and unnecessary. We know of no suitable method for enclosing the walls of a structure during demolition. Even more important is the fact that there is, in reality, very little asbestos used in the construction of a modern medium-sized or high-rise structure. In addition, dust counts taken during the demolition of a building in Easton, Pennsylvania, which did contain significant quantities of asbestos products, showed very little dispersion of fiber into the surrounding air, both upwind and downwind of the demolition site. This is due to the fact that most asbestos-containing products in which the fiber is locked-in with cement, plastic or other

binding materials do not readily release fiber in the atmosphere, even during demolition. Subsection A of Section 501 requires the prior removal before toppling of boiler and pipe insulations -- two asbestos-containing products that might release dust from improper demolition. Since this will remove any loosely-bound asbestos products from the structure, we believe enclosure of the walls to be unnecessary, and recommend that simple wetting of the walls to be toppled would be sufficient to control the dispersion of dust.

With regard to Part VI, Section 601 of the regulations, we would like to recommend for your consideration a completely new approach to the control of asbestos emissions from manufacturing operations, and that has been referred to in some of the earlier testimony. As presently written, the regulations call for the establishment of a numerical emission standard of .5 fibers per cubic centimeter of air from any manufacturing operation, and .05 fibers per cc of air at the boundary line of the plant or factory.

We would recommend that Section 601 of the regulations be rewritten so as to substitute a control practice standard for the present numerical emission

standard. The reasons for this recommended approach are varied, but are primarily based on the lack of specific data with regard to safe non-occupational levels and the difficulty of accurate and meaningful emission sampling techniques at very low fiber concentrations.

The Federal Environmental Protection Agency studied this problem very carefully in preparing its national emission standards on asbestos, and arrived at the conclusion that a numerical standard was impractical at this time. In this regard, I would like to read a few excerpts from a document prepared by the EPA as justification for this viewpoint.

The document states that ideally, national emission standards for asbestos would be established on a concentration basis related to health effects and would provide an ample margin of protection to the public health regardless of the number of sources in a given geographical area. There are many practical considerations, the report goes on, which prohibit the establishment of a numerical standard at this time.

Foremost of these problems is the lack of

a data base. This base is lacking from both the health-effects standpoint for non-occupational exposure and the emissions standpoint. The majority of data available on asbestos health effects is related to occupational exposure and cannot be readily extrapolated to non-occupational levels.

A little later on, the report states that "A control practice approach to reducing asbestos exposure levels offers a practical alternative to numerical emission limits. A major advantage of a control practice standard would be realized in enforcement. Inspectors could be easily trained to certify compliance with control practice regulations."

The report goes on to describe some of the many difficulties involved in asbestos sampling and analysis, especially with regard to emissions into the ambient air, and concludes that present techniques are not only time consuming and expensive, but that they also tend to distort the concentration of asbestos in the sample being analyzed and are hence not meaningful in establishing the actual amount of asbestos being emitted into the atmosphere.

The National Academy of Sciences Committee report on asbestos health, which I quoted from earlier, agrees with the EPA position on numerical standards. It states, "Because of methodologic and other uncertainties, it is not yet feasible to base control on numerical ambient air quality standards."

I will not go into detail on the specifics of a control practice standard since they are primarily of an engineering nature, however, I would like to submit for your consideration the recently published asbestos emission standards of the Environmental Protection Agency, which contains sufficient information to set up a similar system for the State of Illinois, if you choose.

Section 602 of Part IV of the regulations deals with the discharge of asbestos-containing waste into the sewage systems or waters of the state. We believe that this section is unnecessary. In the first place, the water pollution control systems in use in most asbestos manufacturing plants across the state effectively remove the majority of asbestos wastes from the effluent before discharge into state waters.

Secondly, sewage plants also remove asbestos fibers from waste water during purification. In reality, the high dilution of small quantities of asbestos fibers in water is as effective and safe a method for the disposal of asbestos wastes as could be devised. There is no evidence that the swallowing of small amounts of asbestos constitutes any health hazard whatever.

In addition, studies conducted by Johns-Manville have found small amounts of asbestos fiber in samples of water tested from sources all over the United States, including well and spring water. The reason for this is quite simple. Asbestos-bearing serpentine rock is to be found in a majority of the states of the union, including Illinois, and the simple process of water flowing over and through these rocks erodes a certain amount of asbestos fiber.

Thus, we have all been drinking tiny amounts of asbestos in our water since birth, without any discernable adverse effects.

In Part VII, Section 701 of the regulations, we would recommend the addition of the word "visible" in the next to the last line, so that the amended section would read: "No product which may emit

asbestos-fiber during its transportation shall be transported unless such product is enclosed in such a manner as to preclude the 'visible' emission of asbestos fiber into the ambient air." The problem here is once again with analysis techniques and enforcement. The EPA has gone to a "no visible emissions" standard in circumstances similar to this one, and we would recommend to the Board that this also be applied in Illinois. This would also be useful in other sections of the proposed regulations where measurement and enforcement would tend to be extremely difficult if not impossible.

Our final comment is with regard to Part VII, Section 702, which would prohibit the use of asbestos in the brake lining of vehicles manufactured after January 1, 1975, and sold for use within the State of Illinois. We believe that on the basis of the existing scientific evidence such a prohibition is completely unnecessary. In 1968, this question was investigated thoroughly by the United States Public Health Service, and a report on these investigations by Jeremiah R. Lynch, entitled "Brake Lining Decomposition Products" was published in the Journal of the Air Pollution Control Association. The report stated

that "Except in all but the most extreme driving conditions, only a very small fraction of the 30 to 50 percent asbestos present in a brake lining escapes into the atmosphere as free fiber." Lynch found that the average amount of free fiber released was less than one percent of the fiber present in the brake lining composition. The percentage was higher only under conditions that would have resulted in brake failure. Lynch further reported that the majority of the fiber in linings was converted into an inert non-fibrous material by the heat of friction, and concluded that the use of asbestos-containing brake linings was "an inconsequential health factor in urban air pollution." His conclusion is reasonable in light of the fact that hundreds of millions of cars using asbestos-containing brake linings have been stopping and starting in this country for more than half a century, and yet there has been no epidemic of asbestos-related disease among the general public nor have significant quantities of asbestos been demonstrated to exist in the community air.

I think in this regard it is worth noting that Dr. Nicholson's testimony this morning actually served to confirm this. His measurements of ambient

air of the toll booths would certainly be in an area where , if he was ever going to have high concentrations, from brake linings, he would have it there. He actually found them only in the range of three to five nanograms per cubic meter, and this kind of level is one one is apt to find in almost any part of the United States on any given day.

I would also like to point out that these measurements of three, four or five nanograms per cubic meter are subject to an error of two, three or four or more as far as reproducibility is concerned.

While we consider this evidence to be conclusive, if the Board is still concerned about asbestos emissions from brake linings, you will be interested to know that the Federal Environmental Protection Agency has contracted with the Bendix Corporation to do additional tests in this area. This study is due for completion in June of 1972, and we would recommend that the Board, at the very least, await the outcome of the EPA study before making a final decision on asbestos-containing brake linings. It is our belief that the Bendix study will confirm the conclusion reached by Lynch as to the

safety of asbestos-containing brake linings.

This concludes my presentation on the proposed regulations. I have submitted for your consideration copies of all the medical papers, reports, booklets and documents that I referred to in my presentation. I would once again like to express the appreciation of the Asbestos Information Association for the opportunity to appear here this morning.

Although this does conclude my testimony, with your permission I would like very much to have Dr. Goodman, who is with me, take about one minute to present some information on animal studies on brake lining dust which is very pertinent to the brake lining discussion we just had.

HEARING OFFICER LAWTON: No, that will be fine. Don't go away, I am sure there will be questions relating to your presentation.

JOS~~PH~~ L. GOODMAN,

called as a witness herein, having been first duly sworn, was examined and testified as follows:

THE WITNESS: Gentlemen, Dr. Lawton, I am also on the Commission of Labor as a medical adviser in occupational safety and health, and I am concerned about the increased use of mineral fibers.

HEARING OFFICER LAWTON: Would you state your full name?

THE WITNESS: Joseph L. Goodman, M.D. I am an associate professor at the Medical University of South Carolina in private practice of industrial medicine, and I am associated with the Labor Department in this capacity of medical adviser.

We have found at the University of South Carolina and Dr. Paul Gross, who is the scientific director of the Industrial Hygiene Foundation, and also an associate professor at the Medical University of South Carolina, has instilled brake lining dust into the trachia of hamsters and guinea pigs for a period of one year. He used iron-free brake lining dust, because iron interferes with the pattern of the chrysotile asbestos. So introducing

the brake lining dust into the animal for a period of one year, these animals were sacrificed after one year. There was absolutely no anatomical defect noted. There was some inflammatory reaction that one finds when a foreign body is introduced in any animal of this nature. The trachea was not disturbed, the bronchial tubes were not disturbed, and the alveolus, which is the area in which the oxygen is transferred, is not disturbed at all, so all the mechanism of the lung was in its proper proportion. The inflammation was then reversible in the animals that were not sacrificed. Dr. Gross concluded that brake lining dust was biologically inert. The assumption, of course, is that there was no fibrosis at all in any of these animals that were sacrificed, and that were examined under the microscope, under the electron microscope, by the way. We are not dealing with fibrous material. The brake lining is then converted to phosphorite plus many other materials, and the injection of this phosphorite into this animal caused no adverse reaction whatsoever. The conclusion, then, one would draw is that the release of a small percentage of asbestos, even though it was included in the

introduction of this material, caused no anatomical change in the bronchial tubes, caused no change in the respiratory tract, and was therefore biologically inert, and a very safe material, causing no difficulty to this organism. We also found it of interest that the ingestion in the lungs of human beings, which is now in progress at the University of South Carolina, it was found that everybody has asbestos fibers in their lungs, but in the main, they are less than half a micron in size, and yet Dr. Gross and Dr. Eninger, who are very excellent pathologists, were unable to correlate any pathological findings at all in the lungs of these humans, and this was using the electron microscope to determine the size of the fibers, and they came out with an amount of somewhere between three million and six million per individual, and yet there was no pathology to be found, no cancer, no tumors, no fibrosis -- there was no phagocytosis, and you would find phagocytosis, which is the swallowing up of the foreign body in the hamsters and guinea pigs of the brake lining dust, which is a natural body reaction, but this is as far as I would like to state.

We find that Dr. Webster, who is the Chairman of the Council of the United Kingdom Leukemiosis

Research, made the study and made an important point that if all of the asbestos in the lungs of human beings is in this size, and they cannot find any pathological changes in the lungs, therefore, as in the brake lining dust, it is of this magnitude, and it would also be of a safe quality. Thank you very much.

HEARING OFFICER LAWTON: Thank you.

Perhaps Dr. Pundsack might return to the podium, since we will undoubtedly have some questions.

FRED L. PUNDSACK,
thereupon resumed the stand, and, having been previously duly sworn, was examined and testified further as follows:

EXAMINATION

By Hearing Officer Lawton:

Q In your presentation you indicated to the Board you would furnish certain materials, without specifying, and I would like to say on behalf of the Board, we would like to receive everything you have offered to furnish.

A All right.

Q I believe in particular you referred to a

distinction between what you call locked-in and non-locked-in.

A Locked-in and loosely bound.

Q Yes, we will be happy to get that.

A We will be happy to furnish it.

Q Anything else that you have offered to furnish, we would be pleased to get. Also, where you make the suggestions on modification of certain specific sections of the regulations, we would be happy to get your specific proposals in that respect.

A All right.

Q You have indicated where you feel that the particular section might be improved to the extent it is compatible with your own organizational structure. If you would care to make drafts of a particular section, we will be pleased to get that.

A We will be happy to.

Q You distinguish between locked-in and non-locked-in products or products in which the asbestos is locked in or not locked in, as the case may be, and suggest that where it is locked in, perhaps we don't have to use the same precautions. If those particular items are processed or cut or

shaped or anything done with them, don't they have the potential of taking on the attributes of emissions that we are trying to guard against, even if you start with them being locked in?

A I say in most of the cases of the locked-in materials, particularly asbestos cement, there is very little fiber. Even on cutting, it is released in a form that is respirable or not surrounded by an agglomerated cement. In the case of vinyl asbestos, floor tile, cutting the floor tile to fit it into rooms or something, certainly is not in our opinion apt to release any significant quantities.

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gt56

Q If you would care to amplify that point and submit it to the Board --

A I will be very glad to prepare a list of both what we consider loosely and tightly bound, or assumptions.

Q We would like to see how the attributes are -- how if it is processed, it might not develop into that.

While today may not be the most appropriate time to go into details, I know the Board would be interested in amplification of what you consider the control practice standard that you have alluded to in certain aspects, and you have indicated that the Federal EPA might be developed that will be available, but I am sure the Board wants to know more detail, and in that regard particularly, if you or your organization could submit something by way of a suggested regulation, it would be extremely helpful.

A We would be very glad to.

Q I appreciate that that may require a more detailed exposition than you are prepared to make at this time, but this record will remain open to receive things, and we will be very anxious to get that.

gt57

Now, in a more philosophical sense, I suppose, which undoubtedly has occurred to others, is asbestos or are asbestos particles degradable?

A Well, the statement is frequently made, of course, that asbestos is indestructible. As you are probably aware, asbestos is a general name that actually applies to a number of different minerals that have somewhat different properties, but 90 percent of most of the asbestos used is a variety called chrysotile. It is not indestructible. It begins to decompose at a temperature around 500 degree Centigrade, and it changes into a completely different material.

It is also attacked by acid. Chemically, chrysotile is much like magnesium hydroxide, milk of magnesia, and it actually reacts with acid much the same way, and again transforms into a material which is no longer asbestos.

Q I suppose my question was somewhat prefatory to what is really troubling me, what the approach should be and what our responsibility as a state agency should be. You point out, which I suppose is unquestionably supported statistically, that the highest danger are occupation or site-oriented, and

gt58 we in our regulations do address ourselves to that problem, but it seems to me that if asbestos, recognizing that there are many varieties, is only degraded by the type of impact which you suggest, which isn't going to occur in the ambient air under most circumstances, are we not in danger of a rather major build-up over a period of time that would move from the occupation or site orientation to a more general comprehensive danger, and if that is the case, don't we, as a Board, have a responsibility addressing ourselves to that as well as the more obvious and limited aspects of the problem?

A Well, you certainly have posed an interesting philosophical question, which goes to the heart of your responsibility.

Q I don't see how you can do that. An engineer might not be able to.

A Well, let me say this, as I hope I tried to point out in the discussion, the differences in levels of concentration that we are talking about in the occupationally exposed population in the factory, even the very low occupationally exposed, is many, many, many orders of magnitude greater than

gt59

the kind of ambient air concentrations that have been discussed here today, in nanograms per cubic meter. We are talking of levels of concentration a million fold more different. I think also from the point of view of building up in the ambient air, there is a natural occurrence mechanism, actually -- asbestos does not remain forever suspended in air. In other words, rain, for example, agglomerates very fine particles of material, washes it out, it is carried into streams and finds its way, perhaps eventually, into the ocean and is deposited there.

So it isn't as though you have a closed-in system, in which everything you put into the air remains there forever. This is analogous for example, in some respects, to the fact that everything you breathe in -- there has been some discussion of the minute sized fibers, and perhaps they are more respirable than the 5 micron and up fibers.

Actually, the question is not whether it is respirable, but whether or not it is respirable and stays in the lungs. Actually, most of the material breathed into the lungs is cleansed out and in fact, very small fibers tend not to be retained. They tend to be inhaled and expired on the following

gt60

breath.

Q Of course, you point up another related problem, that if it is cleaned from the air by water, then it might get into the drinking water and we have another problem in that regard. You suggest that the regulation relating to sewage treatment or discharge into sewer systems might be unnecessary because the industries using asbestos will have their own abatement procedures, and so do sewage treatment plants -- if that is the case, why would there be any objection to having this as a requirement?

In other words, if it is being done, what is objectionable about requiring it to be done?

A I think that it becomes a meaningless situation, and it is also one, then, that becomes very difficult to establish a standard on.

Q Of course, that is inherent in our --

A If you establish a zero standard, for example, this in many cases is a standard which is really impractical as far as achievement is concerned.

Q We are all troubled by this recurring problem of when slight buildup in the body becomes enough of a buildup to have some detrimental consequences. We encountered this in the mercury regulations,

gt51

I think, in that respect, while we were limiting our concern to water aspects of this, and that was the problem. I know that there likewise will be a concern for the Board in this category.

A We are troubled, obviously, in properly defining what is safe and what is not safe. I have an additional report on the McDonald study, which covers the 10,500 workers in asbestos, which was not available at the time we prepared the material to submit to you, and which does reach a conclusion from the statistical data available to them, that in the case of the workers studied at a dose level of 12 fibers per cc, this is in an occupational setting, and an occupational type of disease -- his conclusion is that there is no increased incidence of lung cancer in the population at that particular dose level.

Now, if you would like, I would be glad to submit that.

Q Yes, we will certainly be pleased to get everything, not only what you suggested in your comments, but anything that occurs to you, knowing what our problem is, and knowing the areas we are concerned with.

gt62

Mr. Dumelle, do you have any questions?

EXAMINATION

By Mr. Dumelle:

Q I think it is along the line that Mr. Lawton brought up, and that is the business of the threshold, if any. Dr. Nicholson states on page 6 of his statement, "Thus, we have no dose-response information on low-level asbestos exposure. We only have knowledge of a potential risk of cancer, at exposures much below occupational ones.

Obviously, additional research and continued surveillance are highly desirable."

So my question would be, what is your industry group doing to collect such low-level dose-response information, for example, like studying toll booth collectors, or people who are exposed to low levels, but perhaps a little higher than the general population?

A Well, let me go back in the statement. First, I have to say that I do not agree that there is not some information available, medical published information available on the effect of low and moderate doses. Now, this may become a matter of how do you define a low or moderate dose, but certainly the

gt63

studies I cited by McDonald in Canada, Newhouse in London, England, and the others, do have data on moderate and occupational setting exposures, and they are able to establish an incidence of disease, or no incidence of disease, so that in that sense there is information on low level exposures to asbestos dust, these low levels being much, much higher than the general public.

To answer another question you posed about toll booth collectors, which would be a very interesting study to do, except it might be obscured by fumes and carbon and exhaust emissions -- we have not done a study on that. However, we have in our studies of population groups done relatively low exposures, and we do hope to have additional information along this line to either supplement the Knox-Dahl studies, the Newhouse studies, and the McDonald studies.

MR. DUMELLE: Thank you.

EXAMINATION

By Mr. Harker:

Q Do you think there is a medical difference between chrysotile fibers and crocidolite?

A I can beg off and say I am not medically

qualified to answer the question.

There are some great superior effects observed with some of the asbestos fibers -- there is some evidence I have seen published that the so-called blue crocidolite fiber does have a more toxic effect than the chrysotile asbestos.

Again, I would like to say that I don't think this is the case, because there is a very peculiar situation in South Africa which was touched on very briefly by Dr. Nicholson this morning, and that is that in many cases -- quite a few cases of mesothelioma observed in one of the chrysotile mining regions of Africa, but not in -- crocidolite, not in the other crocidolite mining range of Africa, and the exact explanation for this is somewhat moot now.

It has been suggested that part of this is really related to the average diameter of the fiber from these two different deposits, and one of the fibers is actually more apt at a given dose level to reside in the lungs and not be cleansed than is the other. It is a very difficult question.

Q The British have established .2 fibers

gt55 per ce for air-borne crocidolite, without worrying about the difference. What do you think is the motivation for the American and British setting a numerical standard, as opposed to asking the course you recommended, "no visible emissions," when what you say is true, that there is no dose-time relationship?

A I think it is a matter of philosophy in how the approach is raised. The "no visible emissions" received considerable discussion this morning. Our feeling is that if you utilize the best emission control technology and engineering available to you, which at the moment would be a fabric bag house filter, and if you coupled that kind of an engineering control approach with "no visible emissions," you do in effect have a very low, very tough standard. You have one which is easy for an inspector to determine whether there is visible emissions, and you would have one which makes it easy for him to determine whether the bag house, for example, is actually operating.

All the inspector has to do is open the door of the bag house, and if it is not operating properly or has a hole in it, it is quite obvious,

gt66

because there is a deposit of dust on the outside of the bag house.

Q On page 6 -- I think you have taken some of the questions that I had and answered them. Let me mention this, the general public you used throughout your paper, and again I take it you clarified that by saying you are not opposed to the view that there are data in support of the belief that moderate or even low to moderate levels of exposure can cause disease?

A No, I have no question in an occupational type of setting, and this I think goes with your really approaching the question of the neighborhood exposure surrounding an asbestos factory.

Q You are using the word "general public" and that would include anybody that lived in the neighborhood as far as we are concerned, but your use of them would be including them as being exposed to any danger, and I was unclear on that point.

You were talking about Dr. Selikoff's data. I think you are saying that they are things involved -- there are things to which those men were exposed which we don't know about, which perhaps goes to the synergistic effect?

gt67

A We certainly know they are exposed in the course of their occupation or work to a wide variety of dust, asbestos-containing material being one of those, and we also know that the observations that were made on those 632 men are the result possibly to a large extent of the kind of exposures that they have suffered 25 or 30 years ago, and not necessarily the exposures today.

We know -- for example, in an insulation worker, in examination of the lungs, you will frequently find that there is more free crystalline silicate in the lungs than there is asbestos fiber. So what I am saying is they do have a very mixed type of thing.

Q That seems to go to the question -- perhaps it goes to the question of synergism, which seems to me to be a very important to us, to determine whether a person in one area or one industrial vicinity would be exposed to the other factor, which might work well in combination with asbestos, as opposed to whether he would not be exposed in another circumstance. That would seem to actually speak to enhance Dr. Selikoff's data, and whatever we might take of it.

gt68

A Except that I think you must remember that that is a study of 632 workers, and that if you have ever been on the sites that those workers for the most part, have a very, very high industrial exposure.

Q In other words, you are looking at a group that has a very high industrial exposure. You talked about low and moderate exposures in the studies you cited. What are those levels, and how do they relate to what Dr. Selikoff said?

A We have no published information on what the levels are in Dr. Selikoff's study. We can provide information in the papers that I have referred to, which indicate how the estimates of high, low, moderate, and so forth, exposures were arrived at in the Newhouse and the McDonald studies.

In the McDonald study, the actual fiber counts, since they have been taken and recorded, which goes back to about 1949, were actually taken on about 5,000 different job categories, and then estimates were made prior to the 1949 data being available. McDonald then weighted the relative dustiness of the environment. At that time, counts were being taken in terms of millions of particles

per cubic foot, and if a man were exposed from work environment, that was rated at 5,000,000 particles per cubic foot for a period of 20 years, and then McDonald would assign a dust exposure of 100. If he worked for ten years in that environment that had a dust count of 10,000,000 particles, he was assigned 100 for the ten years, and a 100 for the 20 years that he -- he would give him a dust index of 200, and he studied workers whose dust indices were 600, that was, the dust level exposures, and broke them down in terms of 500, 600 over 600, and that is a fairly complete study.

Q How did they compare to Mr. Selikoff's -- is there any --

A We don't have any criteria which permits us to determine that.

Q You used the word that you have every reason to believe that the Selikoff subjects were exposed to high or very high levels -- I am trying to find the basis for that conclusion.

A I hope I didn't say they were exposed to higher. I said that first the Selikoff study is unique in that there is a higher mortality rate in that sample than in any other samples of which

we are aware, and I hope I have suggested that perhaps one explanation of that actually the workers studied by Dr. Selikoff have a very high dust exposure.

Q That is what I am driving at. What is the basis of that conclusion, as opposed to the conclusion that the McDonald people had, perhaps lower or moderate levels?

A Well, the McDonald group -- McDonald found that in 5 percent of the samples which had a dust index of 600, he did get an increase in lung cancer.

Q You had no basis, then, for determining or saying the Selikoff group had a high level of dust exposure?

A No, other than as a possible explanation for his results.

Q I would like to talk about one of your suggestions about the proposed rates, that even though there is no medical data to conclude the need for such, that we restrict uses of asbestos which might be a nuisance, you said, small uses of asbestos, which might be a nuisance.

How can asbestos be a nuisance without

being a health hazard?

A I think in any case in which a very small visible emission would be coming from a plant, that might not necessarily constitute an actual health hazard in perhaps being down and dissipating into a nanogram per cubic meter category, but if it is preventable, I see no reason to have any kind of emission from a factory.

Q Do you have any data to support the conclusion that no asbestos fiber, or perhaps very little asbestos fiber is emitted during the handling program, cutting, trimming and fitting of the locked-in products? The reason the conclusion Mr. Lawton alluded to is, is there any data?

A I think we can provide you with data on the industry which would be typical of so-called locked-in materials.

Q Do you have any similar data perhaps to support the conclusion that asbestos from demolition of a building is not an emission source? You cite the measurements taken in eastern Pennsylvania. What was the count in that study?

A That demolition was on the way of a tenth of a fiber per cc at the time of demolition. We

would be very glad to submit these data.

Q Do you know the wind direction and speed?

A We took it both up and downwind. We do have the wind velocity. I don't happen to recall it at the moment.

Q Is there a clear dose-time relationship ever for occupational exposures?

A I believe that there is, and that the data support this. There are also animal studies that might seem to indicate the time-dose response.

Q What do you think the Japanese data on the ingestion of asbestos particles or the possible ingestion of asbestos fibers, as being related to the higher incidence of stomach cancers in Japan?

A I have not had an opportunity to study that. I really prefer not to comment.

Q Well, the only data that I have seen on the effects of ingestion as opposed to inhalation have been this Japanese data, the data of cancers produced in the stomach, perhaps due to a certain amount of ingestion during occupational exposure. Wouldn't occupational exposure, or wouldn't ingestion during work be actually a great deal less if they are inhaled, and speak to perhaps the low level of

exposure which might cause abdominal cancer?

A I would say that in many, many cases, in an occupational exposure to asbestos that is air-borne, that most of the asbestos that is inhaled into the lungs and which is not exhaled, but which tends to reside in the lung wall is actually cleared from the lungs by the normal clearing mechanism, which brings the material up to the top of the throat, and you actually swallow it, so actually any data or effect on air-borne asbestos constitutes a very high exposure in terms of material ingested, so anyone that is breathing in an occupational environment ingests a great deal of asbestos in the clearing mechanism of the lungs.

Q You related that point and said you believe the smaller fibers would be the ones tending to be dispelled from the lungs.

Do you have data on that?

A We can submit some information that is available in the literature in terms of the respirability of various sizes of fiber. I cannot, and certainly would not mean to imply that I know that the small fibers are or are not in this fashion. I think that Mr. Romer referred to the fact in his

talk that the small sizes are more respirable and more dangerous. Frankly, I know of no medical evidence that this is so, and if Mr. Romer had medical evidence to which he can refer, I certainly would appreciate it.

Q What percentage -- do you know the percentage of wash-out that occurs annually of any fiber or particle?

A I am not sure I understand you.

Q When you talked about wash-out in answering Mr. Lawton's question --

A You mean cleansing it?

Q Wash-out or -- what is that percentage then, or is it constant, year after year?

A I don't have any data on it. My guess would be that it is probably fairly constant

Q In that sense, if it were constant, would an increase, an annual increase in the amount of asbestos emitted to the atmosphere tend to move toward an eventual increase in the overall ambient air level and perhaps an actual high level?

A I would doubt very much that it would. I think that we are dealing with such huge numbers here in terms of the ability of the environment

to clean out the relatively small amounts of asbestos fiber that is involved --

Q But it could increase that, given the increase in emissions?

A If there were a tremendous increase in emissions annually, certainly it is possible that it might possibly on a long-term -- possibly on a short-term basis.

Actually, I think there is no large increase in the amount of emissions of asbestos. Much of the increase in the uses of asbestos to which people refer is primarily in the locked-in products.

MR. HARKER: That is all I have. I wanted to ask Mr. Goodman another question.

HEARING OFFICER LAWTON: Thank you.

JOSEPH L. GOODMAN,
thereupon resumed the stand, and having been first duly sworn, was examined and testified further as follows:

EXAMINATION

By Mr. Harker:

Q Is your test on the rats, or was it with guinea pigs --

A This was published in the proceedings at

Johannesburg in 1969 on page 68, and it is a **pulmonary-ferruginous bodies and their origin**, and it is a published report, it was about breakdown of dust, but this is one of the many --

Q The percentage of the fiber which was inhaled by the animals or inserted, was that asbestos?

A They said less than one percent.

Q Less than one percent?

A And that was of a magnitude of less than half a micron, of electron microscopy proportions.

Q Does the fact that -- in addition, perhaps the amount was less than -- of some people -- is the fact that the test took place only one year of significance that we should be concerned with?

A I think a year is long enough to determine the results as far as pathology is concerned. We have other studies, incidentally, of a similar nature, the injection of benz(a)pyrene and chrysotile into the pleural cavity, with no carcinoma occurring into the pleural cavity of hamsters, in studies done over a period of ten years off and on with different animals. But this study, I think the length of time is sufficient. It was considered sufficient by Dr. Webster and Dr. Ian Gross, who are

very principal investigators in the field.

Q Did they find asbestos bodies?

A Yes, they found asbestos bodies. They found all of us had asbestos bodies. If you burn leaves, you get asbestos bodies. The reason why they are is so that they can get to the core to see whether any of the asbestos bodies are there, but even with the chrysotile, one percent being there, Dr. Gross states unequivocally that the material is biologically inert.

MR. HARKER: Thank you.

THE WITNESS: I would like to make one comment about the fiber content in the lung itself, and when it is dissected at the medical university, it contained from 3 to 6 million fibers of less than half a micron, and there was no pathology in these lung sections, and this study is being done.

There is also a study being done in reference to cancer chains themselves of sputum. We are instituting a study to try to determine changes in the cancer cell before it appears in the lung, and we have an interest in this subject, and we appreciate your time.

MR. HARKER: Thank you very much.

HEARING OFFICER LAWTON: Mr. Zabourin, do you care to make any comment?

IVAN ZABOURIN,

having been first duly sworn, was examined and testified in narrative form as follows:

THE WITNESS: Mr. Chairman, ladies and gentlemen. I did not expect to have this occasion. I really appreciate your giving me this occasion to say a few words.

I am from Canada, I am a lawyer on the Queen's Council. I have been representing for 35 years, the Johns-Manville interests in Canada, and most of the asbestos industry for 30 years, from the Atlantic to the Pacific, and we produce in Canada, 90 percent of the world's chrysotile needs in asbestos, the U.S.S.R. being excluded.

I have had occasion over those years to attend world symposia of study. In the last three months, I have been with Dr. Selikoff in Finland, I have also been with him in Romania on different occasions, and I have followed very closely these study periods.

I have been in Russia and Siberia, where much, if not more chrysotile is mined than anywhere

else in the world, and I must say that in view of the fact, I submit to you, sir, that we produce from our friendly country of Canada, friendly to you, north of the boundary, the greater proportion of the world's needs in chrysotile asbestos, and that the greater proportion of this production of ours is used and processed by the U.S.A., just as this industry in Canada is in the main owned by American interests.

I can say that it is somewhat a common venture. This explains my *raison d'être* to appear here today.

The last time I came here was to celebrate somewhat a victory over Chicago, because I graduated at McGill -- when I was there, you used to play hockey quite often with our alma mater, sir, Dartmouth, and Mr. Harker studied law where I studied law.

But adverting to the asbestos problem, per se, I would say that in a couple of minutes, if you will allow me, that it seems to me that there is a curve from left to right in the assessment in the appreciation of the various incidences of response of the human being to this fiber of asbestos.

You, sir, asked a minute ago to Dr. Fundsack,

well, what of crocidolite in England? Well, I heard Dr. Selikoff tell me in Helsinki from the podium to a question put from the audience, that crocidolite shouldn't have been indicted any more than any other asbestos fiber in England, where it was ruled out.

I was in Africa with Dr. Selikoff in Johannesburg, where these crocidolites were involved. I say this because this scientific problem which is that of the study of asbestos and the biophysical properties, the incidences of toxicology, immunology, bacteriology, pathology, and all the fields of medicine are such that it will take quite a number of years before the problem is ironed out, but in the meantime, no turn to substitutes should be even thought of, because I assure you in my word, there is no scientist in the world who will tell you that asbestos should be banned. They will all tell you that it is controllable and should be controlled.

I have it in writing from Dr. Selikoff of June of last year. I have it from many other pathologists the world over. I don't disagree openly with my friend Bill Nicholson, who like me, is a physicist versed in the law. We both have

these common factors, that we don't lose patience because neither of us are pathologists or doctors in medicine.

Now, our experience in Canada, Dr. Pundsack has told you about, goes back to 35 years of observation. Our preventive medicine and engineering has been taking place since 1930. To my personal knowledge, in 1935, in the Corbett-McDonald McGill University study which has been going on for years, it discloses that the incidence is not any more prevailing of cancer in our miners exposed to asbestos than in the general population.

This confirms another study in the same vein that had been conducted way back in 1952 or '53 by the Cyronac Laboratory, which is really a school of profound study in the field of asbestos, a school which lasted for some 20 years, and of which today Dr. George Wright of St. Luke's Hospital, is, I would say, the prolongation -- who is quite a man of authority, and I say, Dr. Selikoff is undoubtedly a man for whom I have profound esteem, and a scientist of note.

His research is immense, and his task is gigantic, but considering the experience that we

have had in Canada; our exposure, I think, confirms the proposition which Dr. Pundsack was advancing a few minutes ago, that it is somewhat -- it seemed that this was stemming from your question, Mr. Chairman, that it is a question of response -- dose-response threshold limits.

This problem has been and is being analyzed. I am not personally conversant enough with the data to discuss it before you, but I am convinced that the study is not terminated. So with these few remarks, I also am a member of the Committee for Canada, that has been for some years for the nation investigating these properties of asbestos, and I was just two days ago with our Air Pollution Program, Dr. Winthrop, from Canada, discussing the aspects that I am discussing with you.

I would say in termination that of the studies that go back to 1930 in Canada in asbestos, we have had some 16,000 films examined by a committee of radiologists picked from a few nations, and few persons of eminence in the field of radiology.

I think we are in a position to say to you that our picture is not a bad one, indeed, and shows that the backlog is really what we are now

seeing coming up, where these heat and frost insulation workers' figures come up. We shouldn't be surprised, because I feel that we are dealing here with periods of intense exposure.

The sprayer -- we have here a contractor who spoke this morning -- they have intense exposures which we haven't today. In mining as well as the textile industry that I know -- and I don't profess to know them all -- on the continent, the dose-relationship today is not that which it was 25 or 30 years ago, and at no time was it comparable to that which existed in the sprayers' occupational field.

What I have been saying to you was really the summation of three persons two weeks ago in Bucharest.

HEARING OFFICER LAWTON: Excuse me a moment. Dr. Nicholson, don't go away.

THE WITNESS: At that symposium, Dr. Hillson, Dr. Wright, as well as Dr. Selikoff were speakers, and I say that their views rather coincide with what I express, in short, that asbestos is controllable and is not to be barred. I thank you, Mr. Chairman.

HEARING OFFICER LAWTON: Thank you very much,

come back and see us again.

THE WITNESS: Very kind of you, sir.

HEARING OFFICER LAWTON: Dr. Harwood, I believe you have a question that you would like to pose to Dr. Nicholson?

WILLIAM J. NICHOLSON,
resumed the stand, and having been previously duly sworn, was examined and testified further as follows:

EXAMINATION

By Dr. Harwood:

Q The question I have that I would like to check -- I might have misheard what Dr. Nicholson said with regard to the exposure levels in the ambient air. Now, I have a paper written by Dr. Nicholson in which he quotes figures in the New York ambient air of between 10 and 16 nanograms per cubic meter.

A That is Manhattan, isn't it?

Q Right, with various parts of Staten Island and Manhattan. I might have misheard this morning. You mentioned a figure of 3 near the toll booth.

HEARING OFFICER LAWTON: Could you speak up? We have to hear you, so will you speak into the

microphone?

THE WITNESS: In making measurements of the ambient air, there are many factors that give rise to error, and on any single measurement, the error of that measurement could be, as Dr. Pundsack pointed out, a factor of two or three. We have found in the study that I mentioned of the analysis of 100 -- in this case, it was about 200 samples from the Environmental Protection Agency, that on repeat samples, the average deviation from all duplicate measurements in the men was about 40 percent, so there is a wide variability.

So that in any case, you can have this error. The measured values that we have obtained in New York City in Manhattan, and were quite significant -- were significantly higher than those in other circumstances. I don't -- could I see the report? Do you have a copy of it? Because I don't remember the exact values. I am sorry, you have a report that has some very preliminary data, and the levels that we obtained in New York City that went into the final publication of this were somewhat lower.

Of the levels in Manhattan, they did go

up to in the tens of nanograms. The levels in the outlying burroughs were more often less than 10 nanograms. I don't have the final data. The problem is that as one starts the experiment, you end up with various errors that you correct as you go along. This is the first measurement that has ever been done of ambient air levels, and the measurements were true.

The improved measurements had more levels, particularly in the outlying burroughs, levels of the order of those that I saw in a well-ventilated entrance of the Long Island Expressway, the entrance to the tunnel.

The question is, how representative were our very few measurements to the actual problem? The answer is that all they do is indicate that you don't get tremendous quantities nearby. The vagaries of wind direction, of sample processing, could indicate that it is a greater problem than our preliminary measurements, which totalled no more than a dust sample would indicate.

The point I am making is that we do see the emissions from brakes. They are sufficient to indicate that at a given location, you would

find spreading, but the main conclusion is that one needs a great deal more work in this area to define it more accurately.

HEARING OFFICER LAWTON: Thank you very much. Does that complete --

THE WITNESS: That completes my answer to that.

HEARING OFFICER LAWTON: Are there other people present -- unless you feel there is something that ought to be on the record -- I want to give everybody an opportunity to be heard --

DR. HARWOOD: This will take a moment.

HEARING OFFICER LAWTON: All right.

DR. HARWOOD: The fact is that Mr. Thompson of the EPA has published a paper, and it was presented in June of this year in Canada, and he gave us values of .5 to 15 nanograms per cubic meter, and I would like to ask Dr. Nicholson if he thought these were more reasonable values.

DR. NICHOLSON: I have not.

DR. HARWOOD: The importance of it is that it ties in with the evidence I presented in the report that I gave to the Board on the figures for asbestos from brake linings in the urban atmosphere.

DR. NICHOLSON: In this report, there is the

point source level of 2,000 nanograms per cubic meter. The highest I remember our measuring was around 400 nanograms per cubic meter. It is the same ball park.

DR. HARWOOD: I am really more interested in the urban --

DR. NICHOLSON: I can give the urban values, and here I have submitted those -- the range of urban values that we found. These are data that I have considerable confidence in. It was after one year's experience in the sample -- that was that of -- let me read the table of our analysis of 187 quarterly samples.

61 were less than 4.5 nanograms per cubic meter or less. 103 were between 0.1 and 4.9. 12 were between 5.0 and 9.9, inclusive. 8 were from 10 to 19.9. 2 were 20 to 49. I believe the dose was 38 and 40 respectively, and one was 95. So that we had a few that were beyond this range of urban values. These were values in the -- in public buildings that were used -- that is outside public buildings that were used by the National Air Pollution Control Association monitoring of it.

They were taken by the Environmental

Administration, sent to us for analysis, and that was the range of the values. The ones in which we found high values appeared to come from point sources prior to distribution, and had a large number of large fibers present.

HEARING OFFICER LAWTON: Thank you very much. Is there anyone present who wishes to make a comment or be heard?

MR. DUELL: I would like to make a comment concerning the discussion.

HEARING OFFICER LAWTON: All right, would you come up, please?

MR. DUELL: It is only a thought that I have.

We have heard the discussion between the two people here. The essential thing that needs to be answered, does the material escaping from brakes have an effect on people, and I would suggest that one thing that might be looked at is the operating engineer, particularly those that operate cable equipment, because they are in the outdoor air, essentially, and they sit and work amongst brakes all of their working career. That was just a thought.

If people are going to do some investigating or checking, one might look to the health of this

group of people.

HEARING OFFICER LAWTON: Thank you very much.

DR. NICHOLSON: May I make a comment on occupational levels that we have found?

HEARING OFFICER LAWTON: Yes.

DR. NICHOLSON: The question arose as to what were the levels of asbestos that were experienced by individuals in different circumstances. There are two studies that have been made of the work environment of insulation workers, and one of these was published by Leroy Wellser, and Dr. Clark Cooper, and the general result from that study, when samples were taken in all different types of work and were rated according to the time that an insulator would be engaged in that type of work, was that the average exposure to asbestos workers, as measured in the last few years, as about 7 fibers long than 5 microns per cc, it might have been 6 or 8, but the figure was very close to 7.

That is very similar to the values that we have found when measuring the work environment of the asbestos workers in New York City, and the New Jersey area. That is the same area in which the friend of Dr. Selikoff referred to. I might

just read a half dozen values at some average levels obtained: Mixing of cement and applying it -- this indicates mixing of cement can give rise to a high level of about 50 fibers per cc, perhaps, while mixing takes place, but that is 2 minutes out of the hour's job. The rest of the hour, the man spends applying it to the pipe, and the average over his entire work ranges from 3 to 5, depending upon ventilation conditions when you apply the material to pipes. It varies between 5 to 12, depending upon the ventilation conditions.

If you are cutting materials and then applying them, it might be around 8. We also have an average of around 7 before the time that the insulator works with his asbestos, as to his average exposure in the past. Some of these exposures may have been higher, they may have spent more time working with asbestos then now. It is difficult to imagine that even in the past, the average exposure of an asbestos worker would often exceed 10 fibers per cc.

The only time you get very high exposures is the rip-out of old insulation, and they do that very little. Occasionally, some men -- not occasionally,

but aboard ship, and in naval installations, where that is more common, some of these men for short periods of time during the War, often engaged in shipboard insulation work. Other than that, their exposures are relatively low compared to some of the factory exposures that took place in the past.

One comment, again, with respect to light exposures, measured particles of dust -- as you know, when you blow up the side of a hill to extract the ore in the mine, much of the particulate material is not asbestos, and exposures measured in terms of particles per cc cannot easily be related to fibers per cc, and again, it is the same problem. You have an index of exposure that varies considerably, depending upon the circumstances of work.

HEARING OFFICER LAWTON: Thank you.

Is there anybody else who wishes to be heard or make a statment? Yes, sir?

MR. ZABOURIN: I just wanted to say that in the U.S.S.R., the picture we formed from our mines involved more than 25,000 workers producing more chrysotile asbestos than we did in Canada.

HEARING OFFICER LAWTON: Thank you very much.

Not hearing any indication of anyone wishing

to speak or make a statement, I will declare this portion of the hearing closed.

We will meet again in Waukegan at the Public Library on the 19th of October, which I believe is next Tuesday, and we will hear at that time, representatives of the Illinois Chamber of Commerce, the Illinois Environmental Protection Agency, Caterpillar, Incorporated, and the National Insulation Manufacturing Association.

Any of you who have not testified but would like to get anything into the record, please send whatever you care to to me, Samuel T. Lawton, Jr., Pollution Control Board, 189 West Madison. We will keep the record open for three weeks.

The statements and exhibits of Dr. Pundsack will be marked and received as Composite Exhibit No. 23.

(The document above referred to was thereupon marked Composite Exhibit No. 23, for identification, and was received in evidence.)

HEARING OFFICER LAWTON: I will declare this portion of the hearing closed. Thank you all for coming.

(Whereupon, an adjournment was taken until Tuesday, October 19, A.D., 1971 at 10:00 o'clock a.m.)

STATE OF ILLINOIS }
COUNTY OF C O O K } SS:

HOLLY ESKANDANI hereby certifies that she is the Certified Shorthand Reporter who reported in shorthand the proceedings had in the above-entitled matter, and that the foregoing is a true and correct transcript of said proceedings.



Certified Shorthand Reporter