

BEFORE THE ILLINOIS POLLUTION CONTROL BOARD
OF THE STATE OF ILLINOIS

IN THE MATTER OF:)
)
PROPOSED MTBE GROUNDWATER) R01-14
QUALITY STANDARDS AMENDMENTS:) (Rulemaking - Water)
35 ILL. ADM. CODE 620)

NOTICE OF FILING

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Clerk of the Board
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Service List

PLEASE TAKE NOTICE that I have filed today with the Clerk of the Illinois Pollution Control Board the **TESTIMONY OF RICHARD P. COBB, P.G.** with Exhibits by the Illinois Environmental Protection Agency, a copy of which is herewith served upon you.

Respectfully submitted,

by: Stephen C. Ewart
Stephen C. Ewart
Deputy Counsel
Division of Legal Counsel

DATE: February 16, 2001

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GROUNDWATER) R01-14
QUALITY STANDARDS)
AMENDMENTS:) (Rulemaking Water)
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TESTIMONY OF RICHARD P. COBB, P.G.

The Illinois Environmental Protection Agency hereby prefiles the attached TESTIMONY of RICHARD P. COBB, P.G. This testimony will be presented by Mr. Cobb at the Illinois Pollution Control Board hearing to be held on March 1 and 22, 2001.

Illinois Environmental Protection Agency

By: Stephen C. Ewart
Stephen C. Ewart
Deputy Counsel
Division of Legal Counsel

DATED: February 16, 2001
1021 North Grand Avenue Northeast
P.O. Box 19276
Springfield, IL 62794 -9276
217/782-5544

**TESTIMONY OF RICHARD P. COBB, P.G.
FOR THE PROPOSED MTBE GROUNDWATER QUALITY STANDARDS
R01 - 14**

QUALIFICATIONS/INTRODUCTION

My name is Richard P. Cobb and I am Manager of the Groundwater Section of the Illinois Environmental Protection Agency's ("Illinois EPA") Bureau of Water. For further detail on my qualifications I have enclosed a copy of my Curriculum Vitae in Exhibit I. This testimony, the statement of reasons, and exhibits included with this testimony describe the basis for the proposed amendments to the groundwater quality standards. The Illinois EPA is proposing a preventive notice and response level, and Class I, and II groundwater standard for Methyl Tertiary-Butyl Ether ("MTBE"). In addition we are proposing amendments to the compliance determination section.

Illinois EPA is proposing these amendments consistent with the Illinois Groundwater Protection Act ("IGPA") policy and program statement; in accordance with the requirements in Section 8 of the IGPA; and in response to the Illinois Pollution Control Board's ("Board") request to continually update the groundwater standard

BACKGROUND

Community water supplies ("CWS") in Illinois routinely sample for volatile organic chemicals as a result of Safe Drinking Water Act monitoring requirements. Under Illinois' CWS Laboratory Fee Program, analyses for MTBE have been reported as a part of standard laboratory methods since 1994. Therefore, we have been receiving SDWA compliance samples that are taken at the entry point to a community water supply distribution system. These are also referred to as ("finished water samples"). Since 1994 26 CWS have been impacted by MTBE contamination. Another factor to consider is that these are finished water samples and they are collected after treatment. Thus, the contamination level in the source water could be higher. In addition, there is also the potential risk to other potable wells, including private, semi-private and non-community water supply wells.

The Illinois EPA has evaluated each of these 26 CWS with MTBE detects as shown in Figure 1. The monitoring conducted at over 1,200 CWS participating in the program (just over 1,100 of these facilities are groundwater dependent) has resulted in 26 facilities with detections of MTBE. Four CWS have had to discontinue use of wells as a result of MTBE contamination:

- Oakdale Acres Subdivision (and two other small subdivisions served by private wells), located in Kankakee County, had to discontinue use of their wells and connect to a nearby CWS;
- Roanoke located in Woodford County has had to shut down wells due to high levels of MTBE;
- East Alton located in Madison County has had to use one of their wells as a hydraulic containment well with treatment and discharge to surface water to protect their well-field from a MTBE plume with a concentration exceeding 1,000 parts per billion (“ppb”); and
- The community of Island Lake had to take a well out of service as a result of elevated levels of MTBE.

Maps of each of these communities has also been prepared showing: the CWS; the type of aquifer being used; CWS well depth; MTBE and BETX concentrations; the location of potential contamination sources surveyed by Illinois EPA staff under the IGPA well site survey requirements; the location of reported leaking underground storage tank sites, the setback zone established under the IGPA; and, if delineated, the recharge area of the well(s). These maps are contained in Exhibit II.

CWS Facilities With MtBE Detections

CWS Name	County
T&C MOBILE ESTATES	Adams
BELVIDERE	Boone
HARDIN	Calhoun
GERMANTOWN	Clinton
GRAFTON	Jersey
OAKDALE ACRES SUBD	Kankakee
SOUTH ELGIN	Kane
MANTENO	Kankakee
CRYSTAL LAKE	Lake
ISLAND LAKE	McHenry
MC HENRY	McHenry
MARENGO	McHenry
BETHALTO	Madison
SAYBROOK	McLean
NOKOMIS	Montgomery
PRAIRIE DU ROCHER	Randolph
RUSHVILLE	Schuyler
NORTH PEKIN	Tazewell
MARQUETTE HTS	Tazewell
CREVE COEUR	Tazewell
ROCK FALLS	Whiteside
CLEARVIEW SBD V	Will
LOVES PARK	Winnebago
ROANOKE	Woodford



Legend

- # Active CWS Facility w/MtBE Detections
- % Active CWS Facility w/Closed Wells Due To MtBE Detections
- \$ Closed CWS Facility w/MtBE Detections

Figure 1. Community Water Supplies with MTBE Detections

MTBE is an organic chemical, specifically an ether. Ethers, especially those of low molecular weight such as MTBE, are significantly soluble in water¹. MTBE in drinking water can be detected by the senses of taste and smell at extremely low concentrations of 20 to 40 ppb². MTBE is primarily manufactured and isolated for use as a fuel additive. It is used in gasoline to increase the octane rating, in effect causing the fuel to burn more completely and therefore create less pollution in the exhaust. MTBE was used in small amounts from the late seventies primarily in California to help curtail the air pollution problems due to hydrocarbon emissions in large urban areas. In recent years, however, its use has spread throughout the country in response to increased air pollution control laws. MTBE is raising increasing concerns because it is being found in many water supply wells across the country^{3,4}

Some states such as California and Maine have taken the initiative to regulate or ban MTBE use within its borders. With increasing detection at fairly high levels in community and private water supply wells, MTBE has been raised as a contaminant of concern for its possibility to cause cancer and its disagreeable taste and odor².

MAJOR ISSUES

Solubility and Dispersal - MTBE is a high solubility for an organic compound. When in an organic solution such as gasoline, a high percentage of MTBE can transfer into water that is in contact with the organic phase. Once in the aqueous phase, MTBE

¹ United States Environmental Protection Agency, Office of Underground Storage Tanks. April 1988. Cleanup of Releases from Petroleum USTs: Selected Technologies. EPA/530/UST-88/001.

² Drinking Water Advisory: Consumer Acceptability Advice and Health Effects Analysis on Methyl Tertiary-Butyl Ether (MTBE), U.S. Environmental Protection Agency Office of Water, December 1997.

³ Squillace, P.J., D.A. Pope, and C.V. Price, March 1995, Occurrence of the Gasoline Additive MTBE in Shallow Ground Water in Urban and Agricultural Areas, U.S. Geological Survey, Fact Sheet FS-114-95.

⁴ United States Environmental Protection Agency. 1999. Blue Ribbon Panel on Oxygenates in Gasoline, Executive Summary, Executive Summary and Recommendations (including a statement by Carol Browner on the Findings)

can disperse in the water, and migrate at the same rate as the water in underground aquifers.⁵

Environmental Fate - MTBE is readily broken down in the presence of high UV such as direct sunlight. In its pure form, on the surface, or in shallow surface water, it volatilizes rapidly or is broken down by sunlight with sufficient time⁵. Natural degradation of MTBE in groundwater, however, is not as effective. The primary method of attenuation for MTBE in groundwater is through dispersion. Biodegradation is also not an effective method of natural breakdown of MTBE in a groundwater setting. MTBE is resistant to natural forms of degradation. According to research by the United States Geological Survey (“USGS”) biodegradation rate constants for MTBE are estimated to be several orders of magnitude lower than for other gasoline components such as benzene and toluene.^{6, 1}

MTBE vs. BTEX - Detections of MTBE in groundwater can often be traced to above ground bulk terminals and underground petroleum storage tanks (“USTs”), both of which have been leaking fuel materials to the groundwater surface. With releases or leaks of petroleum products, two components of concern often detected are MTBE and BTEX (benzene, toluene, ethylbenzene, and xylenes). BTEX plumes are very organic in nature, tend to “float” on the surface of groundwater, and the soluble components principally BTEX dissolve in the water layer. MTBE has a much higher solubility index than the BTEX components of petroleum products^{1,7,8}. Therefore, a larger proportion of MTBE is expected to be in the water layer, relative to the proportional amounts of BTEX in the water layer.

⁵ Squillace, P.J., J.F. Pankow, N.E. Korte and J.S. Zogorski. September 1997. Review of the Environmental Behavior and fate of Methyl Tert-Butyl Ether, Environmental Toxicology and Chemistry, Vol. 16, no.9.

⁶ Moran, M.J., J.S. Zogorski, and P.J. Squillace, 1999, MTBE in Ground Water of the United States – Occurrence, Potential Sources, and Long Range Transport, Proceedings of the Water Resources Conference, American Water Works Association.

⁷ Landmeyer, J.E., Chapelle, F.H., Bradley, P.M., Pankow, J.F., Church, C.D. and P.G. Tratnyek, 1998, fate of MTBE Relative to Benzene in a Gasoline-Contaminated aquifer (1993-1998). Ground Water Monitoring Review, Fall Issue, pps. 93-102.

¹ USEPA, Office of Underground Storage Tanks. April 1988.

⁸ Buxton, H.T., J.E. Landmeyer, and A.L. Baehr, 1997, Interdisciplinary Investigation of Subsurface Contaminant Transport and Fate at Point- Source Releases of Gasoline Containing MTBE, United States

Petroleum Plumes as MTBE Reservoirs - MTBE is both soluble in organic as well as aqueous liquid phases. It is more soluble, by roughly an order of magnitude, in the organic phase. When releases or leaks of petroleum products containing MTBE float on the surface of the groundwater, the petroleum plume may act as an MTBE reservoir allowing MTBE to dissolve into the water layer so long as MTBE concentration are available in the organic phase of the petroleum plume. Thus, in considering the treatment of MTBE, the remediation must remove the original petroleum plume containing MTBE as a reservoir of the MTBE while any necessary MTBE treatment is taking place for a CWS at the entry point of its distribution system. Without attending to the petroleum plume as an MTBE reservoir, the treatment of MTBE at a CWS may become a lengthy process. The recharge of the groundwater with MTBE from the original petroleum plume can occur for long periods of time. The half-life of MTBE is listed as between 4 months and 2 years ^{7, 8}.

MTBE is a Progressive Problem - As discussed earlier, MTBE has a very long residence time in groundwater. The source of MTBE contamination is often leaking USTs. With many known and unknown aging USTs still in the ground and potentially leaking, the increasing contribution of MTBE to groundwater seems inevitable. Since MTBE resists breakdown, any addition of MTBE to groundwater will most likely increase the concentration of MTBE detected in the downstream aquifer at some time in the future. ^{2,3,4, 5,10}

Geological Survey and Oregon Graduate Institute of Science and Technology, Petroleum Hydrocarbon Conference Proceedings.

⁹Keller, A. A., O.C. Sandall, R.G. Rinker, M.M. Mitani, B. Bierwagen, and M.J. Snodgrass, 1998, Cost and Performance Evaluation of Treatment Technologies for MTBE- Contaminated Water, Bren School of Environmental Science and Management and Department of Chemical Engineering, University of California Santa Barbara.

¹⁰ RFG/MTBE Findings and Recommendations, August 1999, Northeast States for Coordinated Air Use Management.

CURRENT TREATMENT METHODS COMPARED

Natural Attenuation / Biodegradation - Scientific studies have been performed that show natural attenuation of MTBE in groundwater is negligible. MTBE is considered persistent, or recalcitrant, in groundwater and degrades very slowly by natural chemical or biological degradation. With the recent introduction of MTBE into the underground environment, sufficient microbial organisms do not exist in most natural settings to degrade MTBE^{7,8}. Acidic chemical breakdown of MTBE can occur, but at lower pH levels than typically observed in nature. A study by Lawrence Livermore National Laboratory in California determined that very limited evidence exists that natural attenuation of MTBE is occurring in the field.¹²

Chlorination / Sodium Hypochlorite - The typical chlorination process used to disinfect drinking water supplies has been shown to have no noticeable effect on MTBE concentrations.¹¹

Ultraviolet Irradiation - High-energy ultraviolet light can be used in a similar manner as chlorine to disinfect drinking water supplies. The UV light disrupts DNA function and is designed to effectively kill all organic life in the water stream. However effective this method is on microbial life in the potential drinking water, it is ineffective on MTBE. Experiments performed at the University of California Davis confirmed that there was no evidence of MTBE degradation in water upon exposure to UV light emitted by a low-pressure mercury lamp.¹¹

Reverse Osmosis (RO) - This process utilizes a semi-permeable membrane, which allows only small particles to pass through. For instance, reverse osmosis has been used to filter salinity (salts) out of seawater to provide fresh drinking water for areas with extreme water supply problems. For large pumping rates, this method can be very expensive, depending on the constituents in the water. To date, most membrane technologies are not applicable to volatile organic chemicals. Little information is

¹¹ Chang, P. and T. Young. Reactivity and By Products of methyl Tertiary Butyl Ether Resulting from Water Treatment Processes, Department of Civil and environmental Engineering University of California Davis. (http://srp.uedavis.edu/mtbrept/vol5_5.pdf)

available concerning removal of MTBE using RO filtration. Ultimately, the high equipment cost, maintenance, and filter replacement costs would cause this method to lose cost-effectiveness. These systems are expensive even for home use, which in most cases is purification of already treated water. Even under those conditions, filters must be replaced periodically. For the cleansing of raw water for a CWS, filter replacement costs would make this method impractical unless the source water influent into the treatment system was fairly clean to start with and the flow of water through the system was moderate to low⁹.

Granular Activated Carbon (GAC) - Most concentrations of organic chemicals in a water phase are effectively reduced when treated with GAC. With MTBE, however, GAC is not as effective treatment medium due to the limited adsorption capability of GAC for MTBE. When used alone, removal of MTBE by GAC is not considered cost effective for treating the large volumes of water used by a CWS. Cost prohibitively large units or multiple pass GAC systems may be necessary to reduce the levels of MTBE to desired concentrations. GAC will also be reduced in its efficiency to remove MTBE if the influent water contains TDS, metals or especially organics. If benzene or other organic chemicals are present with MTBE, MTBE adsorbed on a GAC filtration unit could be dislodged by the benzene or other organic compound sending a large spike of MTBE through the treatment system. To protect from such an occurrence would require careful monitoring of the GAC system when GAC is used as a primary method of treating MTBE. Such monitoring of the GAC system will also increase costs. Studies have shown that MTBE may be treated cost-effectively with GAC only at low concentrations. GAC may be useful and cost effective as a means of secondary treatment as a polishing step following some other forms of MTBE removal⁹.

Air Stripping - Air stripping one of the most cost-effective approaches for removing VOCs from groundwater. Since MTBE is a volatile organic chemical with a moderately high vapor pressure, one would expect it to be susceptible to air stripping. MTBE, however, is not an efficiently air stripped under moderate conditions due to its high solubility in water and its low Henry's Law constant. The high solubility of MTBE

¹² Hoppel, A.M., E.H. Beckenbach, and R.U. Halden, 1998, An Evaluation of MTBE Impacts to California Groundwater Resources, Lawrence Livermore National Laboratory, University of California.

requires the construction of much larger air stripping units than constructed for conventional VOCs, which would impart higher capital and operating costs for MTBE treatment. However, if the temperature of the influent water containing MTBE contaminants can be raised significantly at reasonable cost, the size of the stripping unit can be reduced with the same or similar removal efficiency⁹.

In various field studies, MTBE has been air stripped effectively, but it requires very high air to water ratios, the use of influent water heating to facilitate volatilization, and the use of a packed tower with appropriate media. In one study, at 44:1, 75:1, 125:1, and 200:1 ratios of air-to-water the following removal efficiencies were achieved, respectively: 44%, 51%, 61%, 93-99%. At such high air-water ratios, however, the media in the stripping tower can become clogged with precipitating scale and freezing problems can occur in cold months. One study found that heating the influent water from 10°C to 27°C increased the efficiency of removal by a factor of two. This would require pre-heating the water, which would add additional cost. The cost of air stripping is approximately one-half that of GAC, but this does not include treatment of the resulting gas stream containing the MTBE vapors. If the facility is in an air pollution non-attainment area and cannot release MTBE into the atmosphere, treatment of the gas stream will be required. This will roughly double the cost, thus decreasing the cost-effectiveness of air stripping as a treatment option⁹.

If MTBE vapor treatment is not necessary, packed tower air stripping may be coupled with GAC treatment and air/water stream heating as a cost-effective method of reducing concentrations of the contaminant. Currently, this appears to be the most cost-effective method of treatment compared to other proven methods⁹.

⁹ Keller, A. A., et al., 1998.

TREATMENT SUMMARY

With the limited field-tested data available for most recently researched methods of MTBE treatment, few viable options exist that have wide applicability and are cost-effective. It is important to note that for traditional technologies such as GAC or air stripping, the average costs for treating MTBE-contaminated water is 40-80% higher than treating waters containing benzene or other organic chemicals. Air stripping is the lowest cost technology for high flow rates (100-1000 gpm), if no air treatment is required. Air treatment can be required. Hollow fiber membranes are the lowest cost technology for low flow rates (10-100 gpm), if no air treatment is necessary (which is normal at low flow rates). GAC will be most cost-effective at all flow rates if air treatment is required and the influent water has low levels of other organic chemicals. If air treatment is required and high levels of other organic compounds are detected, air stripping is more cost-effective than GAC at flow rates of 100 or greater. Advanced oxidation processes ("AOP") are in all cases more expensive than the alternative technologies, and there are sufficient uncertainties at this point with respect to by-products of AOP to warrant further study of this technology before accepting full utilization. At high flow rates, however, AOP may become cost-effective compared to other technologies, pending further full-scale field tests. Various forms of biodegradation may in fact soon take precedence over some these methods, but at this time there is not enough field study completed to warrant full implementation.

Most sources claim that treatment options for MTBE in groundwater should be conducted on a case-by-case basis. Each well may have different sets of parameters with respect to other wells. Factors such as pH, pumping rate, facility design, water hardness, inorganic levels, level of MTBE contamination, and the level of interference by other organic contaminants will differ by well or treatment application point.

**UNITED STATES ENVIRONMENTAL PROTECTION AGENCY' BLUE
RIBBON MTBE PANEL FINDINGS**

On November 30, 1998, Carol Browner, Administrator of the United States Environmental Protection Agency ("U.S. EPA") appointed a Blue Ribbon Panel of leading experts to investigate concerns raised by the discovery of MTBE, a gasoline additive, in some water supplies. According to the report produced from the Blue Ribbon Panel, U.S. EPA recommended that:

Recommended a comprehensive set of improvements to the nation's water protection programs, including over 20 specific actions to enhance Underground Storage Tank, Safe Drinking Water, and private well protection programs.

Review of the Blue Ribbon Panel recommendations and findings supports inclusion of a groundwater standard for MTBE⁴.

**SDWA UNREGULATED CONTAMINANT MONITORING REQUIREMENT
FOR MTBE**

U.S. EPA recently adopted new revisions to the Unregulated Contaminant Monitoring Regulation ("UCMR") under the SDWA. MTBE is one of 13 chemicals included in this regulation. One of the Blue Ribbon Panel recommendations consisted of accelerating the UCMR for MTBE prior to the implementation date of January 1, 2001¹³.

⁴ USEPA. 1999.

¹³ Federal Register 40 CFR Part 9, 141 and 142. September, 17 1999. Revisions to the Unregulated Contaminant Monitoring Regulation for Public Water Systems, Final Rule. Vol 64, No. 180.

**ILLINOIS EPA'S PROPOSAL TO AMEND THE GROUNDWATER QUALITY
STANDARDS**

Section 620.310(a)(3)(A)(i) Preventive Response Activities

This subsection has been amended to include a preventive response level MTBE based on its taste and odor threshold. Exhibit III details information on the taste and odor threshold for MTBE.

Section 620.410(b)

This subsection has been amended to include a Class I: Potable Resource Groundwater Standard for MTBE. This standard is based on a draft Illinois EPA health advisory, developed pursuant to 35 Ill. Adm. Code 620.605, and a review of what other states are doing. Exhibit IV details information on the health advisory information for MTBE.

Section 620.420(b)

This subsection has been amended to include a Class II: General Resource Groundwater Standard for MTBE. In the original regulatory proceeding, R89-14(B), the Class II: General Resource Groundwater standard for organic constituents was based on the capability of treatment technology to achieve the Class I standard. The treatment of MTBE is very difficult once it has dissolved into the groundwater.

The Henry's law coefficient for MTBE is very low making it difficult to remove. Granular activated carbon is also not effective because MTBE does not readily adsorb. Thus, the Class II standard is also proposed at 0.070 mg/l.

Section 620.505(a)(5)

This subsection has been amended to not exclude compliance points that are valid for determining groundwater quality, and in certain instances may be existing potable water supply wells.

CONCLUSION

This concludes my testimony. I will be happy to address any questions.

L:/epa3188/docs/regulatory/IGPA/MTBE/MTBEtest, February 15, 2001

EXHIBIT I – Curriculum Vitae of Richard P. Cobb

CURRICULUM VITAE OF RICHARD P. COBB, P.G.

I. Personal

A. Present Position: Manager, Groundwater Section, Illinois Environmental Protection Agency

II. Education

- 1981 B.S. Illinois State University (Geology)
- 1984 Illinois State University (Hydrogeology and Engineering Geology)
- 1986 United States Geological Survey National Training Center (Geochemistry for Groundwater Systems)
- 1986 Illinois State University Graduate Geohydrology Program (Hydrogeology of Waste Disposal Sites)
- 1987 Illinois State University Graduate Geohydrology Program (Hydrology of Glacial Deposits in Illinois)
- 1992 United States Geological Survey (MODFLOW and MODPATH groundwater modeling)
- 1994 24 Hour Occupational Health & Safety Training
- 1995 Illinois State University Graduate Geohydrology Program (Computer Modeling of Groundwater Systems)

III. License

Licensed Professional Geologist 196-000553, State of Illinois, expires 3/31/2001

IV. Certification

Certified Professional Geologist 7455, Certified by the American Institute of Professional Geologists 4/88

Certified Total Quality Management Facilitator
Certified by Organizational Dynamics Inc., 5/92

V. Summary of Experience

More than twenty years of experience of working as a professional geologist in hydrogeology, environmental geology and petroleum geology. Twelve years of diversified, interdisciplinary experience as a senior manager, junior manager of a technical hydrogeology unit, and lead worker for Illinois' statewide groundwater protection program. Three years of experience as a consulting well site geologist for major and independent oil companies conducting petroleum exploration and development in Arkansas, Kansas, Louisiana, Montana, North Dakota, Oklahoma and Utah. Two years of undergraduate teaching assistant experience for several geology courses.

VI. Summary of Computer Skills

I use the following computer programs: WordPerfect, 8.1, Microsoft Word 2000, QuattroPro, FoxPro, Power Point, Freelance Graphics, ARC VIEW II, Aqtesolv, SURFER, WHPA, DREAM, AQUIFEM, MODFLOW, MODPATH, and Visual MODFLOW.

VII. Professional Representation

- A. Illinois Environmental Protection Agency (Agency) liaison to the Governor appointed Groundwater Advisory Council (GAC).
- B. Agency representative on the Interagency Coordinating Committee on Groundwater (ICCG).
- C. Agency representative on the Senate Working Committee on Geologic Mapping.
- D. Agency representative on the State Certified Crop Advisory Board, and chairman of the ethics and regulatory subcommittee established in association with the American Society of Agronomy/American Registry of Certified Professionals in Agronomy, Crops and Soils.
- E. Chairman of the Agency Geographic Information System Users Group.
- F. Member of the Agency Cleanup Objectives Team from 1988 to 1993 that established soil and groundwater cleanup objectives on a site-by-site basis.
- G. Member of technical work group that developed Illinois groundwater quality standards regulations.
- H. Project leader for a special Agency work group that utilized vadose zone and solute transport modeling to develop soil cleanup objectives under different hydrogeologic settings for the leaking underground storage tank program.
- I. Agency representative on a special subcommittee of the ICCG charged with the development of a State Pesticide Management Plan for the protection of groundwater.

- J. Member of Agency task group involved with developing the siting criteria for a low level radioactive waste site in Illinois.
- K. Environmental regulatory representative from Illinois on the Fresh Water Foundation's Groundwater Information System (GWIS) project in the great lakes basin.
- L. Agency representative on four priority regional groundwater protection planning committees designated by the Director to advocate groundwater protection programs at the local level.
- M. Representative on the Groundwater Subcommittee of the National Section 305(b) Report, of the Clean Water Act, Consistency Workgroup.
- N. Bureau of Water representative on the Agency's Locational Data Policy Workgroup.
- O. Bureau of Water representative on the Agency GIS Steering Committee.
- P. Member of the Ground Water Protection Council's Wellhead Protection Subcommittee.
- Q. Elected Co-Chair of the Groundwater Division of the GWPC on September 1997. GWPC is a national, not for profit organization whose members are interested in the protection of the nation's ground water supplies. The mission of the GWPC is to promote the safest methods and most effective regulations regarding comprehensive ground water protection and underground injection techniques. GWPC's meetings, workshops, seminars, and symposia provide forums, educational resources, open communication, and active participation by its members. GWPC's membership includes local, state, and federal governments, citizen groups, industry, academia, and other parties interested in responsible protection and management of ground water resources.
- R. Chairman of Illinois' Source Water Protection Technical and Citizens Advisory Committee.
- S. United States Environmental Protection Agency National Ground Water Report work group member. One of 10 state representatives serving on a work group sponsored by U.S. EPA headquarters charged with development of a national report to be submitted to the U.S. Congress on the status and needs for groundwater protection programs across the country. January 1999 to present.
- T. Northeastern Illinois Planning Commission Water Supply Task Force member. The purpose of this task force is to assist the Commission in the development of a Strategic Plan for Water Resource Management. March 1999 to present.
- U. GWPC/U.S. EPA Futures Forum Work Group providing input on source water protection for the next 25 years. January 1999 to present.

V. GWPC/ASDWA work group providing input into the U.S. EPA Office of Ground and Drinking Water Strategic Plan for Source Water Protect. June 2000.

W. VIII. Professional Affiliation

National Groundwater Association
Illinois Groundwater Association
Association of Groundwater Scientists and Engineers
American Institute of Professional Geologists
The Society of Sigma Xi
Ground Water Protection Council

IX. Chronological Experience

9/92-Present Title: Manager of the Groundwater Section in Bureau of Water at the Illinois Environmental Protection Agency. I also serve periodically as Acting Manager for the Division of Public Water Supplies. My primary responsibilities include development and implementation of Illinois statewide groundwater quality protection, USEPA approved wellhead protection program, and source water protection program. My responsibilities include development and implementation of Illinois statewide groundwater quality protection, USEPA approved wellhead protection program, and the source water assessment and protection program for surface and groundwater public drinking water supplies. These duties include extensive coordination with federal, state and local stakeholders that include the Governor appointed Groundwater Advisory Council, the Interagency Coordinating Committee on Groundwater, four Priority Groundwater protection planning Committees, Illinois Source Water Protection Technical and Citizens Advisory Committee and through being co-chair of the GWPC Ground Water Division. Additionally, work with the Bureau of Water permit and Mine Pollution Control Program staff to develop source water protection, groundwater monitoring and aquifer evaluation and remediation programs. I have also served as a primary Agency witness at Illinois Pollution Control Board proceedings in the matter of groundwater quality standards, technology control regulations, and water well setback zone exceptions. Furthermore, I have served as an Agency witness in enforcement matters.

7/91-9/92 Title: Acting Manager of the Groundwater Section in Bureau of Water at the Illinois Environmental Protection Agency. My responsibilities include continued development and implementation of Illinois statewide groundwater quality protection and USEPA's approved wellhead protection program. Additionally, work with the Bureau of Water permit and Mine Pollution Control Program staff to develop groundwater monitoring and aquifer evaluation, remediation and/or groundwater management zone programs. I also served as a primary Agency witness at Illinois Pollution Control Board proceedings in the matter of groundwater quality standards and technology control regulations. Additionally, serve as an Agency total quality management (TQM) facilitator, and TQM trainer.

Manage a statewide regulatory compliance program for activities located within setback zones and regulated recharge areas of potable water supply wells.

7/88-7/91 Title: Manager of the Hydrogeology Unit, Groundwater Section in the Bureau of Water. Manage a staff of geologists and geological engineers that apply hydrogeologic and groundwater modeling principals to statewide groundwater protection programs. Oversight the development, integration and application of Geographic Information System, global positioning system, geostatistical, optimization, vadose zone, solute transport, groundwater flow and particle tracking computer hardware/software programs for groundwater protection and remediation projects.

Provide administrative support to the Section manager in coordination, planning, supervision, grant application and management, regulatory and legislative development in relation to the statewide groundwater quality protection program. Establish soil and groundwater cleanup objectives on the Agency Cleanup Objectives Team.

7/85-7/88 Title: Environmental Protection Specialist in the Groundwater Section of the Illinois Environmental Protection Agency. Lead worker and senior geologist in the development and implementation of Illinois statewide groundwater quality protection program.

3/81-12/83 Title: Consulting Well Site Geologist for Geological Exploration Consultants of Denver Colorado. Worked as a consulting well site geologist in petroleum exploration and development for major and independent oil companies. Responsible for the geologic oversight of test drilling for the determination and presence of petroleum hydrocarbons. Prepared geologic correlations and performed analysis of geophysical logs, drilling logs and drill cuttings. Supervised and analyzed geophysical logging. Made recommendations for conducting and assisted with the analysis of drill stem tests and coring operations. Provided daily telephone reports and final written geologic reports to clients.

1/79-3/81 Title: Undergraduate Teaching Assistant for Illinois State University Geology Department. Responsible for teaching and assisting with lecture sessions, lab sessions, assignment preparation and grading for petrology, stratigraphy and geologic field techniques.

X. List of Rulemaking or Cases in Which Expert Witness Experience Has Been Gained

IN THE MATTER OF: GROUNDWATER QUALITY STANDARDS (35 ILL. ADM. CODE 620), R89-14(B) (Rulemaking). Subject: I served as the principal Illinois EPA witness recommending adoption of this Agency proposal. R89-14(B) was adopted by the Board.

IN THE MATTER OF: GROUNDWATER PROTECTION: REGULATIONS FOR EXISTING AND NEW ACTIVITIES WITHIN SETBACK ZONES AND REGULATED RECHARGE AREAS (35 ILL. ADM. CODE 601, 615, 616 and 617), R89-5 (Rulemaking). Subject: I served as the principal Illinois EPA witness supporting adoption of this Agency proposal. R89-5 was adopted by the Board.

IN THE MATTER OF: GROUNDWATER QUALITY STANDARDS (35 ILL. ADM. CODE 620), R93-27 (Rulemaking). Subject: I served as the principal Illinois EPA witness recommending amendments of new constituent standards in this Agency proposal.

IN THE MATTER OF: PROPOSED REGULATED RECHARGE AREAS FOR PLEASANT VALLEY PUBLIC WATER DISTRICT, PROPOSED AMENDMENTS TO (35 ILL. ADM. CODE 617), R00-17 (Rulemaking). Subject: I served as the principal Illinois EPA witness supporting adoption of this Agency proposal.

IN THE MATTER OF: NATURAL GAS-FIRED, PEAK-LOAD ELECTRICAL GENERATION FACILITIES (PEAKER PLANTS), R01-10 (Informational Hearing) Subject: I served as a supporting Illinois EPA witness to discuss the impact of peaker plants on groundwater.

IN THE MATTER OF: PROPOSED AMENDMENTS TO TIERED APPROACH TO CORRECTIVE ACTION OBJECTIVES (35 Ill. Adm. Code 742), (R00-19(A) and R00-19(B)) (Rulemaking). Subject: I served as a supporting Illinois EPA witness recommending inclusion of MTBE in this Agency proposal.

STATE OIL COMPANY vs. DR. KRONE, McHENRY COUNTY and ILLINOIS EPA, PCB 90-102 (Water Well Exception). Subject: This case involved obtaining an exception from the owner of a non-community water supply well for placing new underground gasoline storage tanks within the 200 foot setback zone of well. I served as the principal witness for Illinois EPA on this case. The Board granted the exception with conditions.

SHELL OIL COMPANY vs. COUNTY of DuPAGE and THE ILLINOIS ENVIRONMENTAL PROTECTION AGENCY, PCB 94-25 (Water Well Setback Exception). Subject: A new underground gasoline storage tank was seeking an exception from the Illinois Pollution Control Board in relation to a private drinking water supply well setback zone. The DuPage County and the Illinois EPA held that the tank would be a significant hazard and opposed the exception. I served as the principal Illinois EPA witness. Shell withdrew the petition from the Board after hearings were held.

People ex rel. Ryan v. STONEHEDGE, INC., 288 Ill.App.3d 318, 223 Ill.Dec. 764, 680 N.E.2d 497 (Ill.App. 2 Dist. May 22, 1997). Subject: State brought Environmental Protection Act action against company engaged in business of spreading deicing salt, alleging that salt stored on company's industrial property leaked into area's groundwater supply, thereby contaminating it. The Circuit Court, McHenry County, James C. Franz, J., granted company's motion for summary judgment. State appealed. The Appellate Court, Colwell, J., held that: (1) wells existing before Illinois Water Well Construction Code was enacted are not "grandfathered" in as being in compliance with Code, so as to be automatically subject to testing for groundwater contamination, and (2) fact issues precluded summary judgment on

claim arising from alleged deposit of at least 50,000 pounds of salt in pile within 200 feet of two existing water supply wells. Affirmed in part and reversed in part; cause remanded.

People vs. AMOCO OIL COMPANY and MOBIL CORPORATION, Case no. 90-CH-79, Tenth Judicial Court, Tazewell County, Illinois. Subject: Groundwater contamination resulting from releases at above ground bulk petroleum storage terminals resulting in violation of Illinois' Groundwater Quality Standards Regulations (35 Illinois Administrative Code 620). I served as the principal Illinois EPA witness on this case. The case was settled with a penalty of \$125,000 and the requirement of a comprehensive corrective action program.

People vs. STONEHEDGE INC. Case no. 94-CH-46, Circuit Court of the 19th Judicial Circuit, McHenry County. Subject: This case involved a violation of the potable well setback zone provisions of Section 14.2 of the Illinois Environmental Protection Act. Stonehedge Inc. placed a salt pile of greater than 50,000 pounds within the 200 foot setback of multiple private drinking water supply wells. I served as an Agency principal witness. Stonehedge Inc. was found to be guilty of violating the setback prohibition in this case and was assessed a penalty of \$1,500 and attorneys fees of \$4,500.

SALINE VALLEY CONSERVANCY DISTRICT vs. PEABODY COAL COMPANY, Case No. 99-4074-JLF, United States District Court for the Central District of Illinois. Subject: Groundwater contamination from the disposal of 12.8 million tons of coarse coal refuse, slurry and gob. Witness for the Illinois EPA. This is an on-going case.

XI. Honors

Sigma Xi 4/81

Superior Performance Award 1/86

Superior Performance Award 11/87

Certificate of Commendation for Groundwater Protection Programs 4/92

Certificate of Appreciation for work on the Agency's Cleanup Objectives Team 4/93

Certificate of Appreciation for participation as an Agency TQM facilitator 4/93

Certificate of Appreciation for participation on a total quality action team 4/93

Certificate of Appreciation for participation in the Governors Environmental Youth Corps Program 4/93

Director's Commendation Award for participation in the development of the City of Pekin, Il. Groundwater Protection Program and commitment to the protection of Illinois groundwater. 7/95

Certificate of Appreciation for outstanding contribution to the development of the Ground Water Guidelines for the National Water Quality Inventory 1996 Report to Congress from the United States Environmental Protection Agency Office of Ground Water and Drinking Water. 8/96

Groundwater Science Achievement Award from the Illinois Groundwater Association for outstanding leadership and service in the application of groundwater science to groundwater protection in Illinois and in the development of the wellhead protection program and pertinent land-use regulations. 11/97

Certificate of Appreciation from the Ground Water Protection Council for distinguished service, remarkable dedication, valuable wisdom and outstanding contribution as a GWPC member, division co-chair and special committee member. 9/99

Drinking Water Hero Recognition by United States Environmental Protection Agency Administrator Carol Browner at the 25th Anniversary of the Federal Safe Drinking Water Act Futures Forum in Washington D.C. 12/99.

Certificate of Recognition from United States Environmental Protection Agency Region V Administrator Fred Lyons for outstanding achievements in protecting Illinois' groundwater resources. 12/99

XII. PUBLICATIONS

A. Legislation and Legislative Development Documents

Co-Author

A Plan for Protecting Illinois Groundwater, Illinois Environmental Protection Agency, January 1986. 65 p.

Groundwater in Illinois: A Threatened Resource, A Briefing Paper Regarding the Need for Groundwater Protection Legislation, Governors Office and Illinois Environmental Protection Agency, April 1987. 34 pp.

Illinois Groundwater Protection Act, Public Act 85-0863, September, 1987. 68 pp.

B. Regulations

Co-Author

Groundwater Quality Standards (35 Ill. Adm. Code 620), November, 1991. 79 pp.

Groundwater Protection: Regulations for Existing and New Activities within Setback Zones and Regulated Recharge Areas (35 Ill. Adm. Code 601, 615, 616 and 617), December, 1991. 132 pp.

Principal Author

Maximum Setback Zone Rules For Community Water Supply Wells (35 Ill. Adm. Code 671), February 1988. 50 pp.

Minimal Hazard Certification Rules (35 Ill. Adm. Code 670), February, 1994. 21 pp.

Amendments to the Groundwater Quality Standards Regulation, (35 Ill. Adm. Code 620), February 1994.

Regulated Recharge Area Regulation for Pleasant Valley Public Water District, (35 Ill. Adm. Code 617), under development.

Maximum Setback Zone Regulation for Illinois American Water Company-Peoria, (35 Ill. Adm. Code 618), under development.

C. Groundwater Quality and Hydrogeology

Principal Author

Cobb, R.P., and Sinnott, C.L., 1987. Organic Contaminants In Illinois Groundwater. Proceedings of the American Water Resources Association, Illinois Section, Annual Conference, Champaign, IL, April 28-29, p. 33-43.

Clarke, R.P., and Cobb, R.P., 1988. Winnebago County Groundwater Study. Illinois Environmental Protection Agency. 58 pp.

Cobb, R.P., et al, 1992. Pilot Groundwater Protection Needs Assessment for the City of Pekin. Illinois Environmental Protection Agency. 111 pp.

D. Groundwater Protection Program Documents

Principal Author

Buscher, W.E., and Cobb, R.P., 1990. Maximum Setback Zone Workbook. Illinois Environmental Protection Agency. 62 pp.

Cobb, R.P., 1990. Illinois Groundwater Protection Program: A Biennial Report. Interagency Coordinating Committee on Groundwater. 53 pp.

Cobb, R.P., Buscher, W.E., and A. Dulka, 1991. Illinois Approved Wellhead Protection Program Submitted to the United States Environmental Protection Agency Pursuant to Section 1428 of the Safe Drinking Water Act. Illinois Environmental Protection Agency. 44 pp.

Cobb, R.P., 1992. Illinois Groundwater Protection Program: A Biennial Report. Interagency Coordinating Committee on Groundwater. 118 pp.

Cobb, R.P., 1994. Illinois Groundwater Protection Program: A Biennial Report. Interagency Coordinating Committee on Groundwater. 118 pp.

Cobb, R.P., 1994. Briefing Paper and Executive Summary on the Illinois Groundwater Protection Act and Groundwater Protection Programs with Recommendations from the Illinois Environmental Protection Agency Regarding the Siting of a Low Level Radioactive Waste Site. Presented to the Low Level Radioactive Waste Task Force on December 9, 1994 in Champaign-Urbana.

Cobb, R.P., 1994. Measuring Groundwater Protection Program Success. In the proceedings of a national conference on Protecting Ground Water: Promoting Understanding, Accepting Responsibility, and Taking Action. Sponsored by the Terrene Institute and the United States Environmental Protection Agency in Washington D.C., December 12-13, 1994.

Cobb, R.P., Wehrman, H.A., and R.C. Berg, 1994. Groundwater Protection Needs Assessment Guidance Document. Illinois Environmental Protection Agency. +94 pp.

Cobb, R.P., and Dulka, W.A., 1995. Illinois Prevention Efforts: The Illinois Groundwater Protection Act Provides a Unified Prevention-Oriented Process to Protect Groundwater as a Natural and Public Resource, The AQUIFER, Journal of the Groundwater Foundation, Volume 9, Number 4, March 1995. 3pp.

Cobb, R.P., 1995. Integration of Source Water Protection into a Targeted Watershed Program. In the proceedings of the GROUND WATER PROTECTION COUNCIL'S Annual Ground Water Protection Forum in Kansas City Missouri.

Cobb, R.P., 1996. A Three Dimensional Watershed Approach: Illinois Source Water Protection Program. In the proceedings of the GROUND WATER PROTECTION COUNCIL'S Annual Ground Water Protection Forum in Minneapolis, Minnesota.

Cobb, R.P., and W.A. Dulka, 1996. Discussion Document on the Development of a Regulated Recharge Area for the Pleasant Valley Public Water District. Illinois Environmental Protection Agency. pp 28.

Cobb, R.P., 1996. Illinois Source Water Protection Initiatives-Groundwater Perspective. In the proceedings of the American Water Works Association's Annual Conference and Exposition in Toronto Canada . pp 585- 594.

Cobb, R.P., 1996. Illinois' Groundwater Protection Program: A Biennial Report. Interagency Coordinating Committee on Groundwater. 93 pp.

Cobb, R.P., and Dulka, W.A., 1996. Illinois Community Examines Aquifer Protection Measures. American Water Works Association Journal. p10.

Cobb, R.P., McMillan, W.D., and K.E. Cook. 1996. Drinking and Groundwater Sections of Illinois Water Quality Report (Section 305(b) Report.

Cobb, R.P., 1996. Illinois' Core Comprehensive State Groundwater Protection Program Application. Illinois Environmental Protection Agency. 159 pp.

Cobb, R.P., 1998. Illinois Source Water Assessment and Protection Program Application. 180 pp.

Cobb, R.P., et al. October 1999, Ground Water Report to Congress, United States Environmental Protection Agency.

Co-Author

Clarke, R.P., Cobb, R.P. and C.L. Sinnott, 1988. A Primer Regarding Certain Provisions of the Illinois Groundwater Protection Act. Illinois Environmental Protection Agency. 48 pp.

Kanerva, R.A., Clarke, R.P. and R.P. Cobb 1988. An Issues / Options Paper for Comprehensive Water Quality Standards for Groundwater. Interagency Coordinating Committee on Groundwater. 25 pp.

Kanerva, R.A., Clarke, R.P. and R.P. Cobb 1989. Discussion Document for Comprehensive Groundwater Quality Standards. Interagency Coordinating Committee on Groundwater. 25 pp.

Dulka, W.A., and R.P. Cobb, 1995. Grassroots Group Forges Groundwater Protection Law. American Water Works Association, Opflow, Vol. 21 No. 3. 2pp.

E. Geology

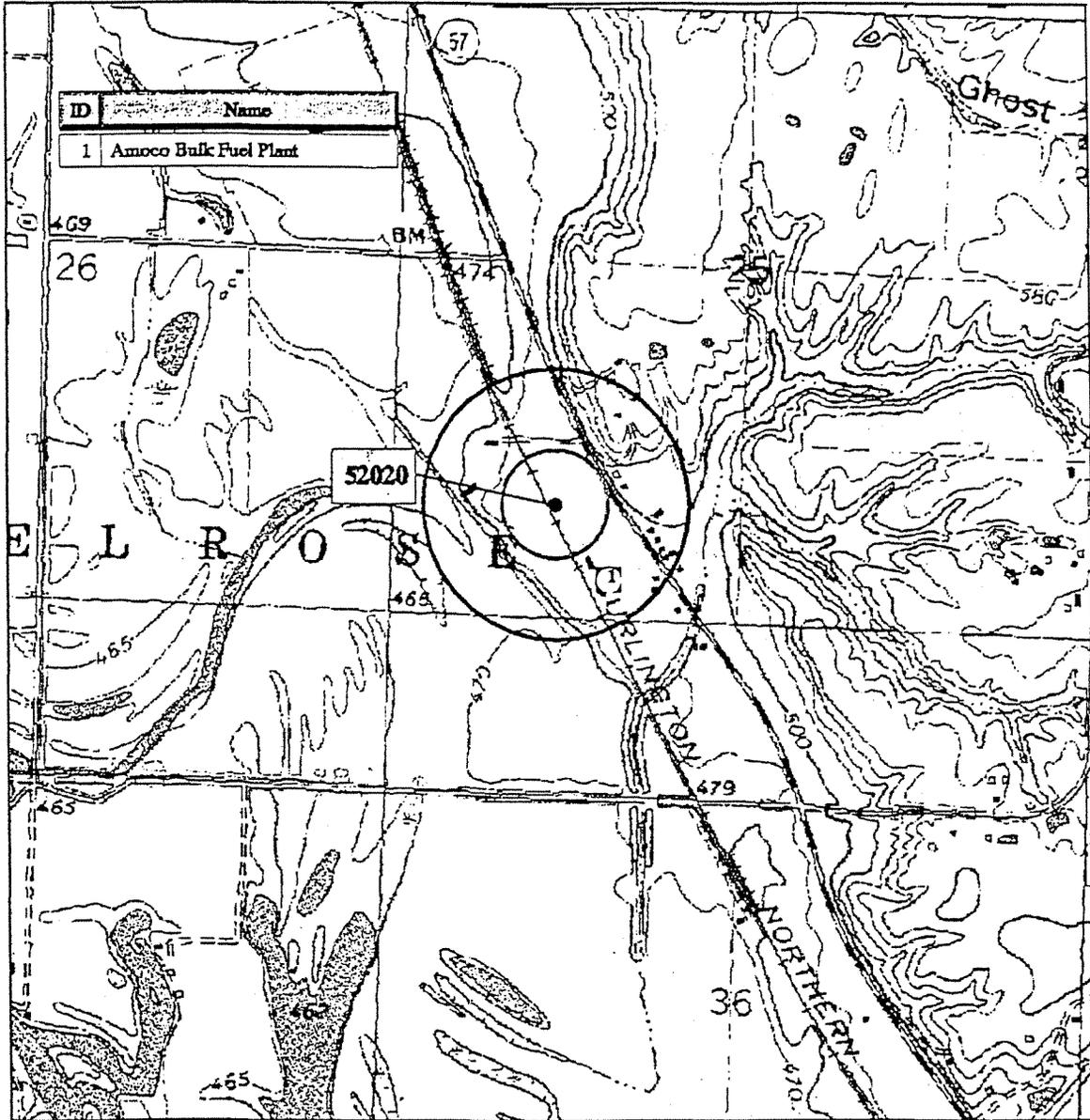
Principal Author

Cobb, R.P., 1980. Petrography of the Houx Limestone in Missouri. Transactions of the Illinois Academy of Science Annual Conference, Illinois Wesleyan, Bloomington, IL..

EXHIBIT II –Maps of Community Water Supplies with MTBE Detections

T&C Mobile Estates (0015815)

Potential Source and Detection Data



ID	Name
1	Amoco Bulk Fuel Plant



Illinois EPA

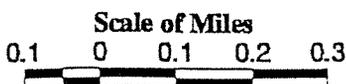
TAP	Well	5-Digit ID	Depth
01	Well #1	52020	84

Legend

CWS Wells

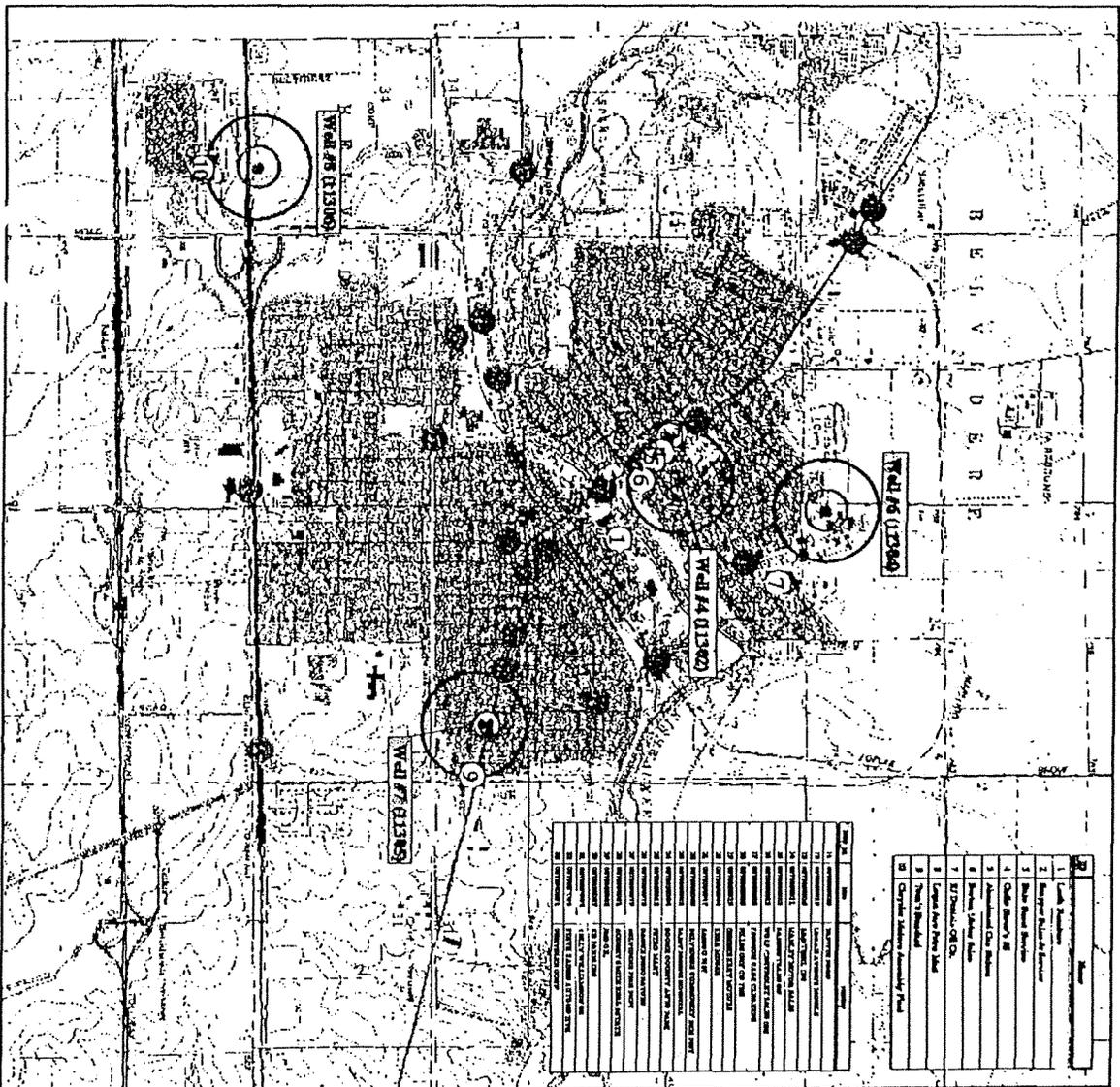
- Confined Aquifer
- Unconfined Aquifer
- LUST Site
- ② Above or Below Ground Fuel Storage
- Existing or Potential Maximum Setback Zone
- Minimum Setback Zone
- 5-Year Recharge Area

TAP	Chemical	Date	Level	MCL
01	methyl tert-butyl ether	04/18/95	3.00	none
01	methyl tert-butyl ether	07/17/95	1.30	none
01	methyl tert-butyl ether	10/17/95	1.00	none
01	methyl tert-butyl ether	10/28/96	1.00	none
01	methyl tert-butyl ether	01/29/97	2.00	none
01	methyl tert-butyl ether	09/22/97	1.00	none
01	methyl tert-butyl ether	11/17/98	1.20	none
01	toluene	07/30/94	0.60	1000



Source Information
 USGS Topo Map DRG obtained from Illinois DNR.
 Sampling Data from Illinois EPA Compliance & Assurance Section.
 Source ID performed in 1989 by Illinois EPA Groundwater Section.
 All results and MCL's in ug/l.

Belvidere (0070050) Potential Source and Detection Data

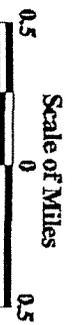


Illinois EPA

Legend

CWS Wells

- Confined Aquifer
- Unconfined Aquifer
- LOST Site
- ② Above or Below Ground Fuel Storage
- Existing or Potential Maximum Setback Zone
- Minimum Setback Zone
- 5-Y ear Recharge Area



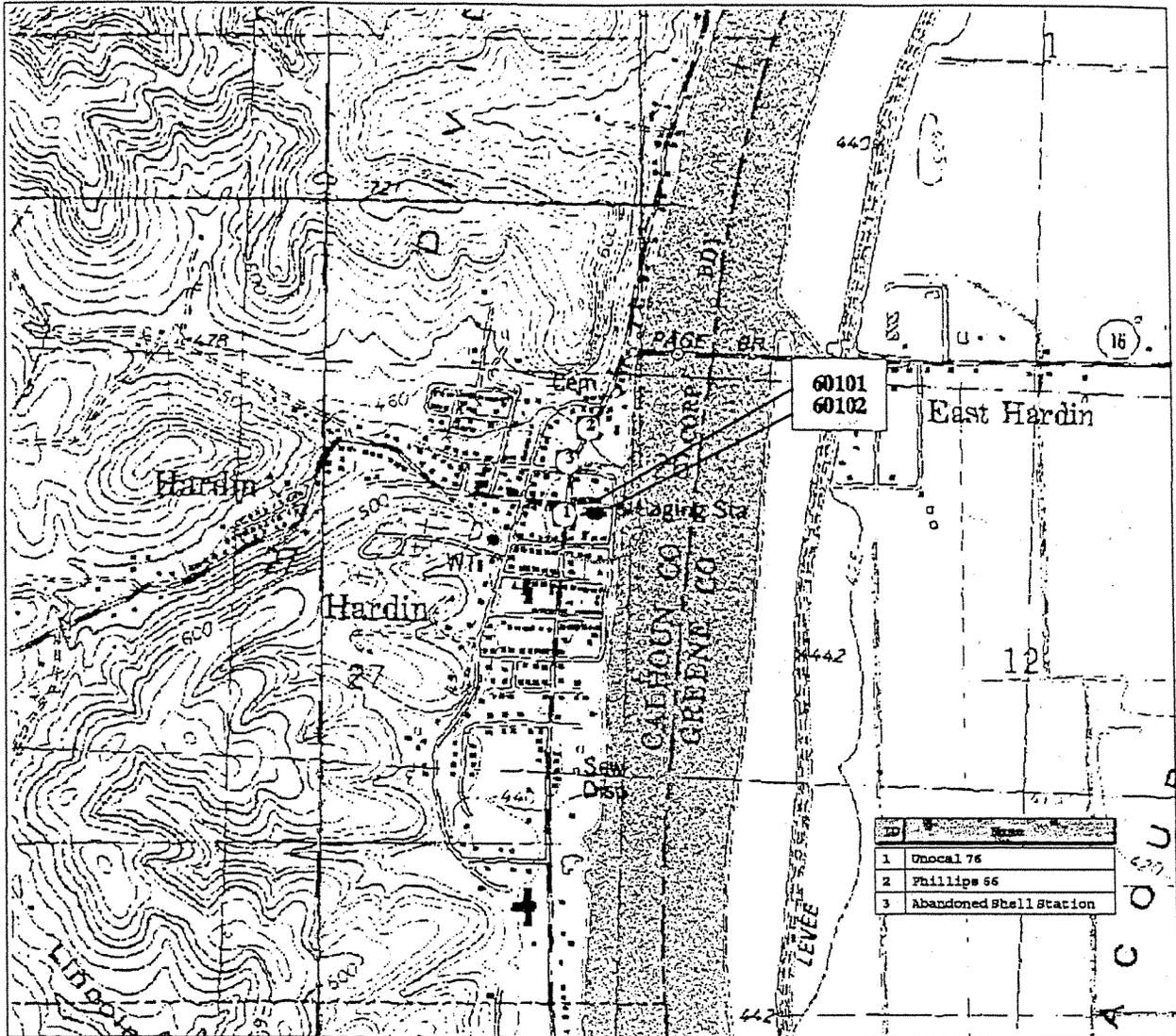
W-1	W-2	W-3	W-4	W-5	W-6
W-1	W-2	W-3	W-4	W-5	W-6
11302	11304	11305	11306	11306	11306
1800	802	989	1593	1593	1593

Chemical	Level	W-1	W-2	W-3	W-4	W-5	W-6	Depth	W-1	W-2	W-3	W-4	W-5	W-6
methy/tert-butyl ether	3.00	02	02	02	02	02	02	10417/95	none	none	none	none	none	none
methy/tert-butyl ether	1.00	02	02	02	02	02	02	04/22/96	none	none	none	none	none	none
methy/tert-butyl ether	9.00	02	02	02	02	02	02	1/27/98	none	none	none	none	none	none
methy/tert-butyl ether	7.00	02	02	02	02	02	02	02/09/98	none	none	none	none	none	none
methy/tert-butyl ether	12.00	02	02	02	02	02	02	01/17/98	none	none	none	none	none	none
methy/tert-butyl ether	10.00	02	02	02	02	02	02	9/17/98	none	none	none	none	none	none
benzene	1.20	02	02	02	02	02	02	01/20/90	\$0	\$0	\$0	\$0	\$0	\$0
benzene	5.00	02	02	02	02	02	02	10/11/90	\$0	\$0	\$0	\$0	\$0	\$0
benzene	5.00	04	04	04	04	04	04	10/11/90	\$0	\$0	\$0	\$0	\$0	\$0
benzene	2.40	02	02	02	02	02	02	01/08/92	\$0	\$0	\$0	\$0	\$0	\$0
benzene	4.70	04	04	04	04	04	04	01/08/92	\$0	\$0	\$0	\$0	\$0	\$0
benzene	3.00	05	05	05	05	05	05	01/08/92	\$0	\$0	\$0	\$0	\$0	\$0
benzene	1.80	02	02	02	02	02	02	04/09/92	\$0	\$0	\$0	\$0	\$0	\$0
benzene	1.70	04	04	04	04	04	04	01/23/93	\$0	\$0	\$0	\$0	\$0	\$0
benzene	4.30	02	02	02	02	02	02	04/22/94	\$0	\$0	\$0	\$0	\$0	\$0
benzene	0.60	06	06	06	06	06	06	01/09/97	10000	10000	10000	10000	10000	10000
benzene	0.80	04	04	04	04	04	04	01/27/98	10000	10000	10000	10000	10000	10000

Source Information
 USGS Topo Map DRG obtained from Illinois DNR.
 Sampling Data from Illinois EPA Compliance & Assessment Section.
 Source ID performed in 1988 by Illinois EPA Groundwater Section.

Hardin (0130200)

Potential Source and Detection Data



Illinois EPA

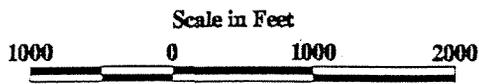
TAP	Well	5-Digit ID	Depth
01	Well #1	60101	50
01	Well #2	60102	64

Legend

CWS Wells

- Confined Aquifer
- Unconfined Aquifer
- Above or Below Ground Fuel Storage
- LUST Sites
- Minimum Setback Zone
- Existing or Potential Maximum Setback Zone
- 5-Year Recharge Area

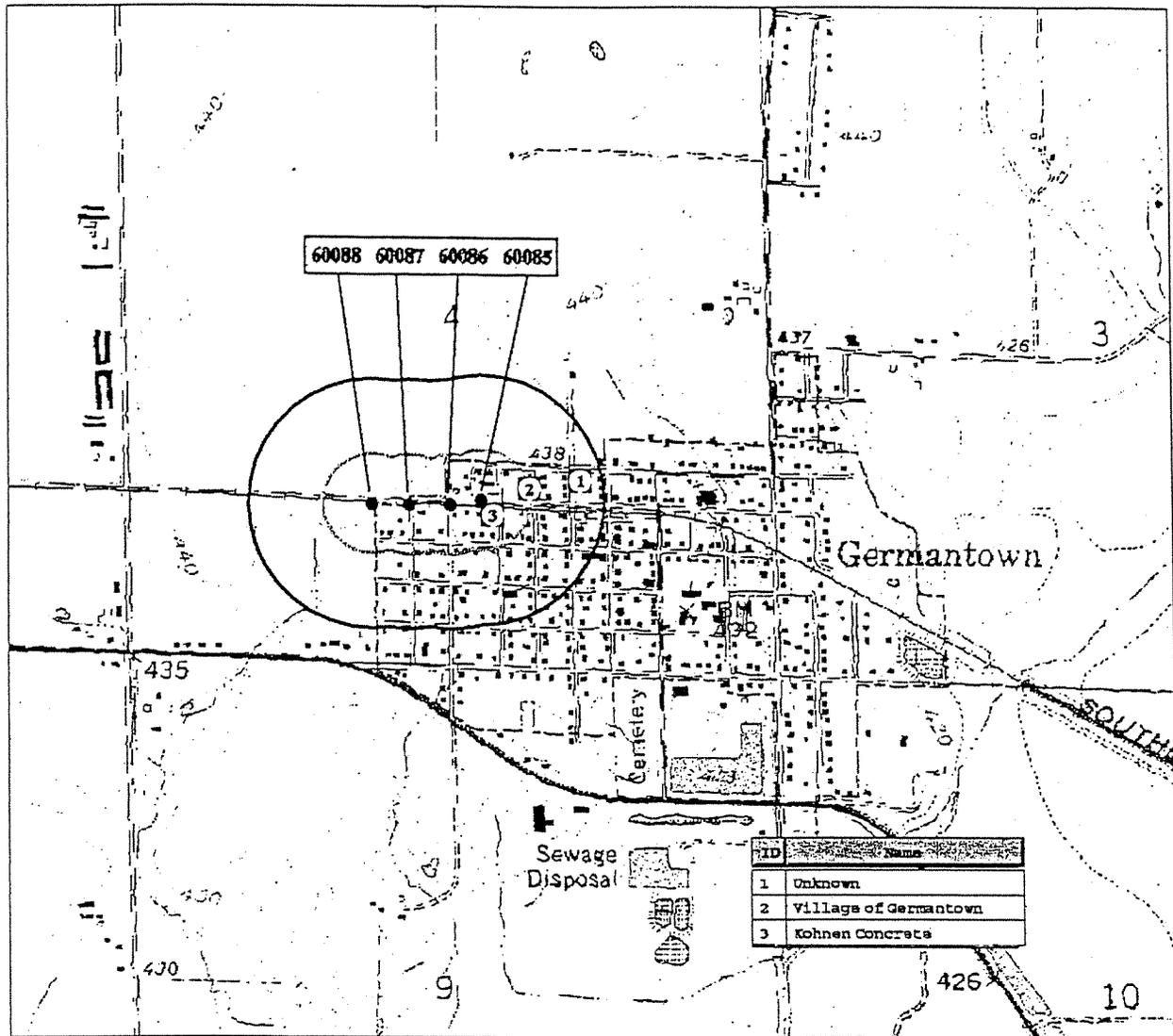
TAP	Chemical	Date	Level	MCL
01	METHYL TERT-BUTYL ETHER	07/30/96	2.00	none
01	METHYL TERT-BUTYL ETHER	10/28/96	1.00	none
01	METHYL TERT-BUTYL ETHER	01/29/97	1.00	none
01	XYLENE	01/29/97	7.20	10000
01	METHYL TERT-BUTYL ETHER	04/23/97	1.00	none
01	METHYL TERT-BUTYL ETHER	07/22/97	1.00	none
01	METHYL TERT-BUTYL ETHER	10/21/97	1.00	none
01	METHYL TERT-BUTYL ETHER	07/27/98	1.00	none
01	METHYL TERT-BUTYL ETHER	11/23/98	1.00	none



Sources Information
 USGS Topo Map DRG Obtained from Illinois DNR.
 Sampling Data from Illinois EPA Compliance & Assurance Section.
 Sources ID performed in 1988 by Illinois EPA Groundwater Section.
 All results and MCL's reported in ug/l.

Germantown (0270350)

Potential Source and Detection Data



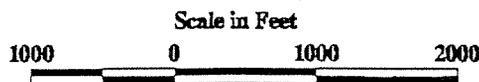
Illinois EPA

Legend

- CWS Wells
- ⊕ Confined Aquifer
- Unconfined Aquifer
- ② Above or Below Ground Fuel Storage
- ② LUST Sites
- ▭ Minimum Setback Zone
- ▭ Existing or Potential Maximum Setback Zone
- ▭ 5-Year Recharge Area

STATE	WELL	5-Digit ID	Depth
01	Well #1	60085	29
01	Well #2	60086	29
01	Well #3	60087	29
01	Well #4	60088	29

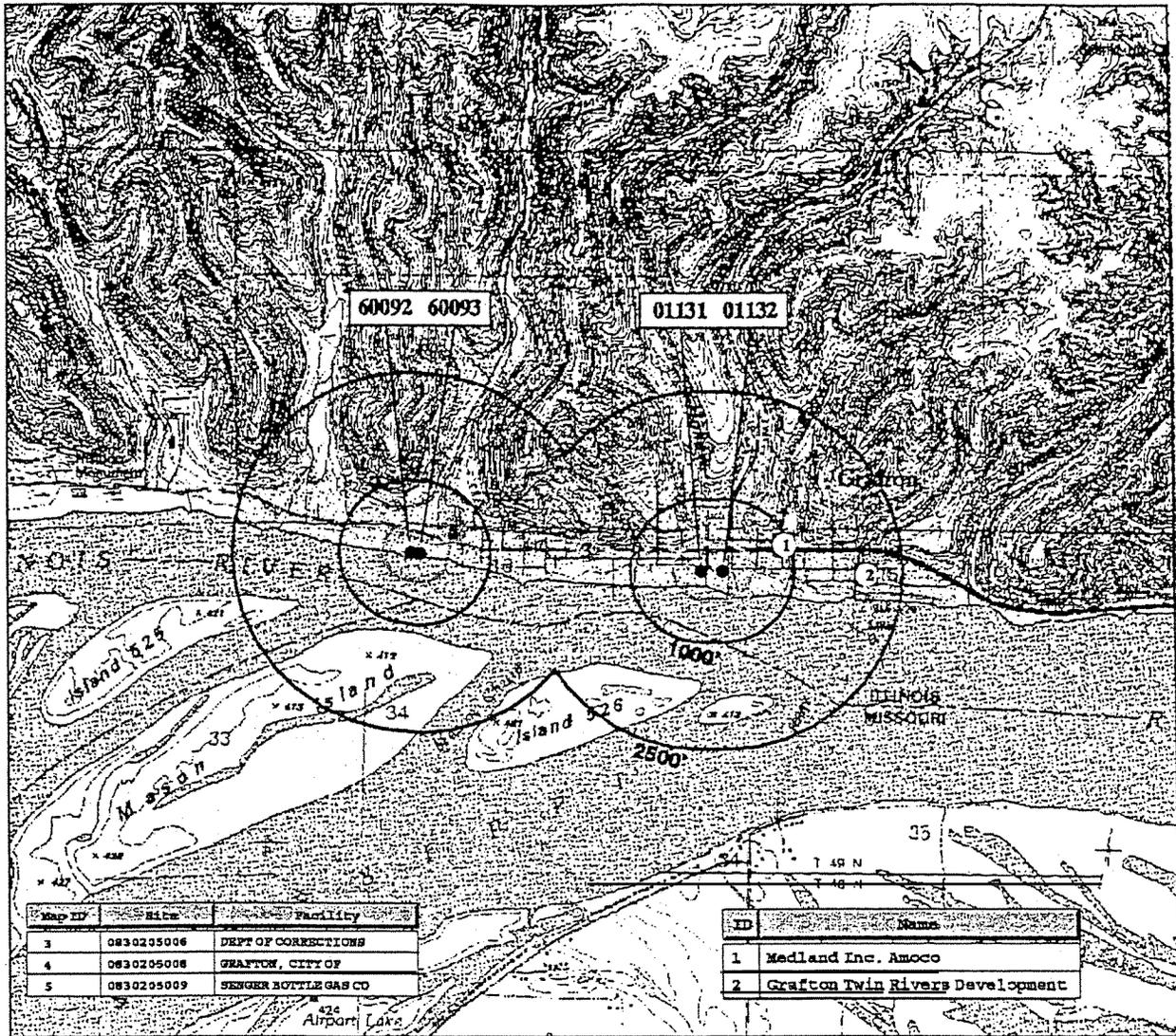
WELL	Chemical	Date	Level	MCL
01	METHYL TERT-BUTYL ETHER	02/10/98	5.00	none
01	METHYL TERT-BUTYL ETHER	07/08/97	2.00	none
01	ETHYLBENZENE	12/14/98	1.90	700
01	XYLENE	12/14/98	8.10	1000



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Source Information
 USGS Topo Map DRG Obtained from Illinois DNR.
 Sampling Data from Illinois EPA Compliance & Assurance Section.
 Source ID performed in 1988 by Illinois EPA Groundwater Section.
 All results and MCL's reported in ug/l.

Grafton (0830200) Potential Source and Detection Data

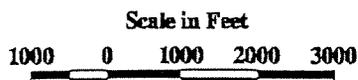


Illinois EPA

Legend	
CWS Wells	
	Confined Aquifer
	Unconfined Aquifer
	Above or Below Ground Fuel Storage
	LUST Sites
	Minimum Setback Zone
	Existing or Potential Maximum Setback Zone
	5-Year Recharge Area

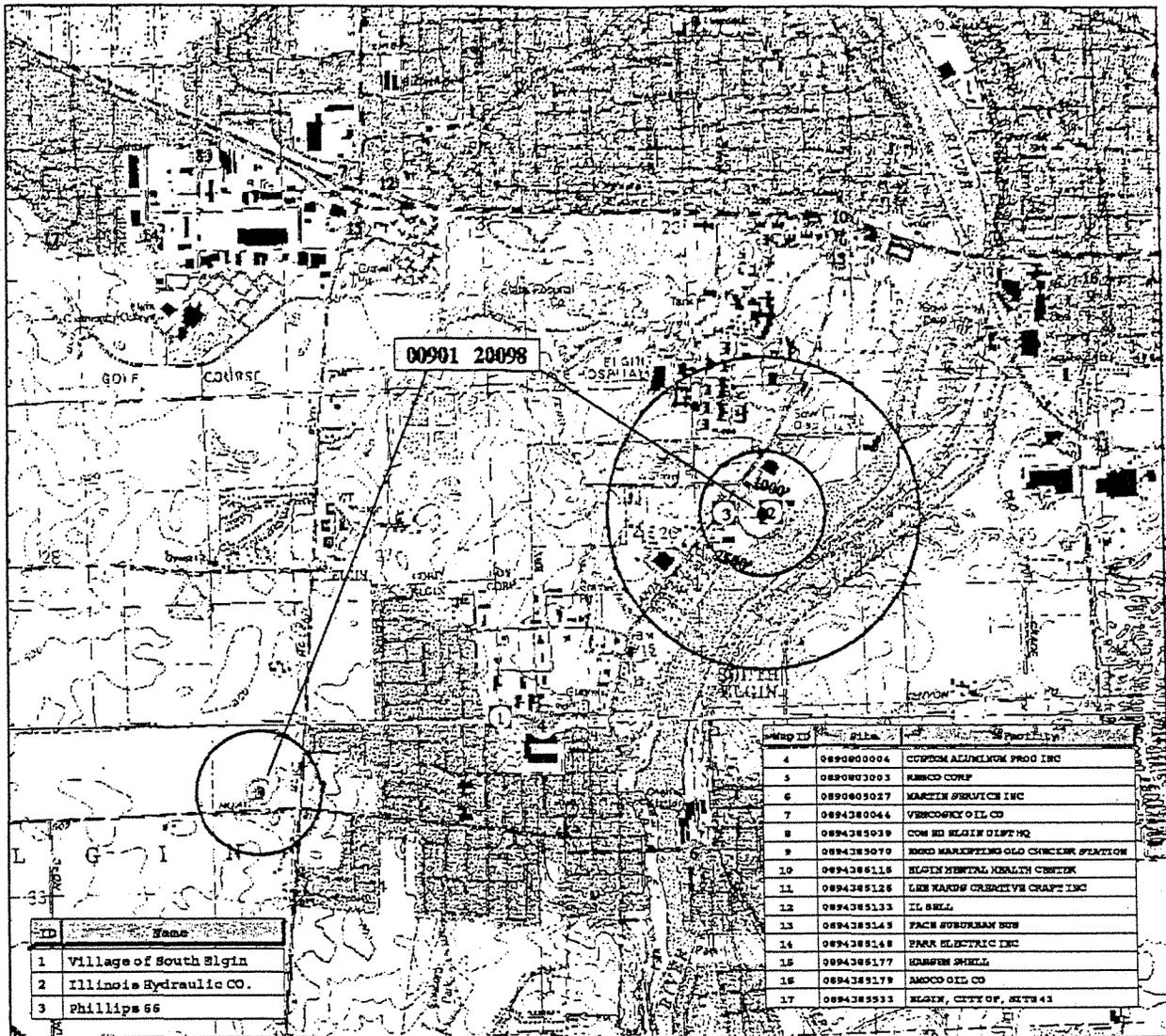
TRP	Well	9-Digit ID	Depth
01	Well # 2	60092	56
01	Well # 3	60093	63
xx	Well # 1	01131	F
xx	Well # 2	01132	F

TRP	Chemical	Date	Level	MCL
01	METHYL TERT-BUTYL ETHER	03/11/97	1.00	none
01	METHYL TERT-BUTYL ETHER	04/16/97	1.00	none
01	METHYL TERT-BUTYL ETHER	10/19/98	2.00	none



Source Information
USGS Topo Map DRG Obtained from Illinois DNR.
Sampling Data from Illinois EPA Compliance & Assurance Section.
Source ID performed in 1998 by Illinois Rural Water Association.
All results and MCL's reported in ug/l.

South Elgin (890800) Potential Source and Detection Data



Illinois EPA

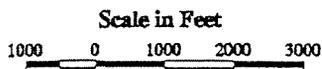
Legend

CWS Wells

- ☉ Confined Aquifer
- Unconfined Aquifer
- ② Above or Below Ground Fuel Storage
- ⊕ LUST Sites
- Minimum Setback Zone
- ▭ Existing or Potential Maximum Setback Zone
- 5-Year Recharge Area

TAP	Well	5-Digit ID	Depth
03	Well #5	20098	68
05	Well #7	00901	1300

TAP	Chemical	Date	Level	MCL
03	XYLENE	08/02/95	0.60	10000
03	METHYL TERT-BUTYL ETHER	10/17/96	2.00	none
05	XYLENE	10/23/97	0.60	10000

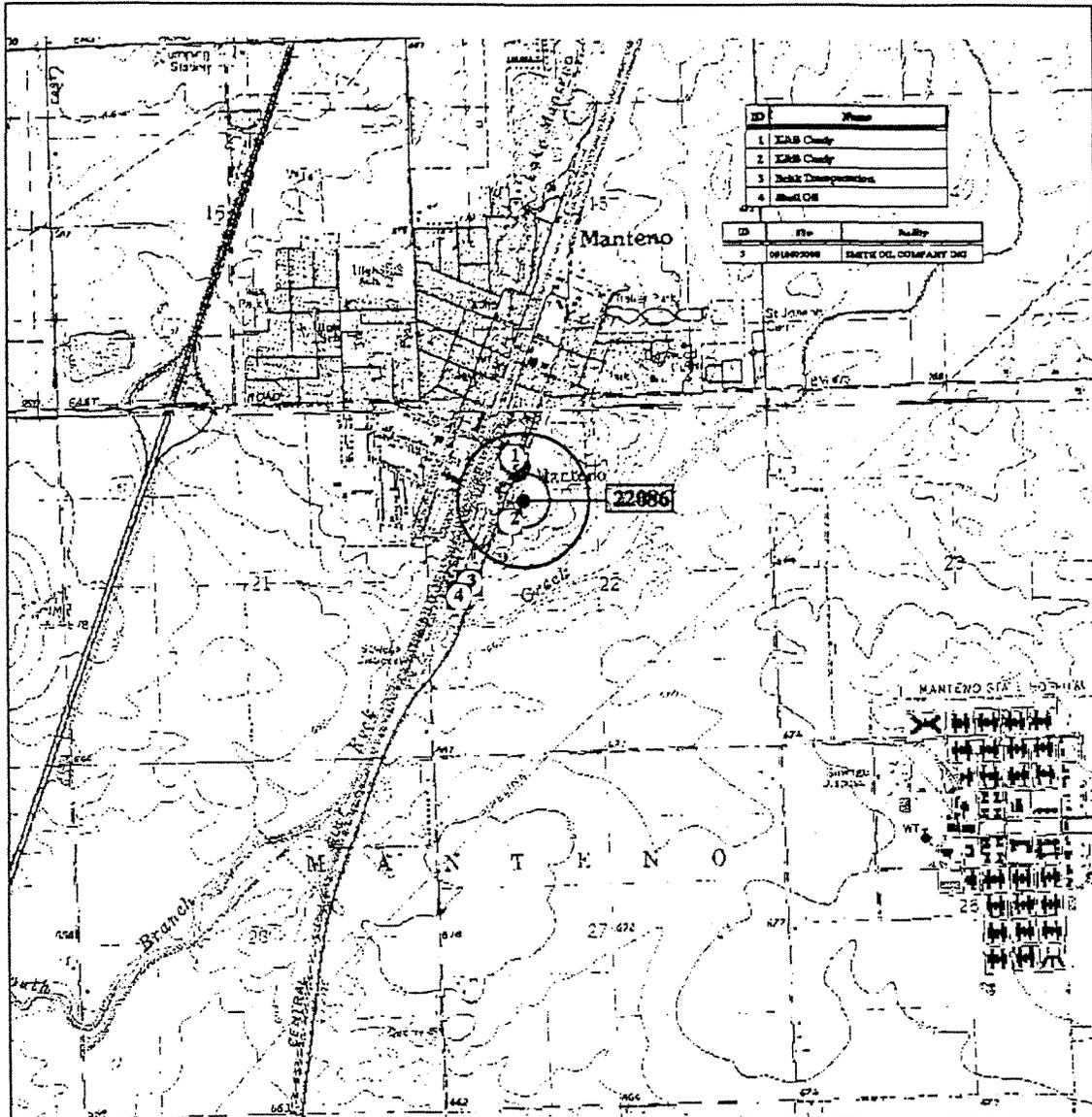


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Source Information
 USGS Topo Map DRG Obtained from Illinois DNR.
 Sampling Data from Illinois EPA Compliance & Assurance Section.
 Source ID performed in 1997 by Illinois EPA Groundwater Section.
 All results and MCL's reported in ug/l

Manteno (0910600)

Potential Source and Detection Data



Illinois EPA

Chemical	Level	Tap	Date	Mcl
methyl tert-butyl ether	1.00	04	10/14/98	none

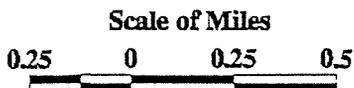
Legend

CWS Wells

- Confined Aquifer
- Unconfined Aquifer
- LUST Site
- ⊕ Above or Below Ground Fuel Storage
- Existing or Potential Maximum Setback Zone
- Minimum Setback Zone
- 5-Year Recharge Area

TAP	Well	5-Digit ID	Depth
01	Well #4	22086	04

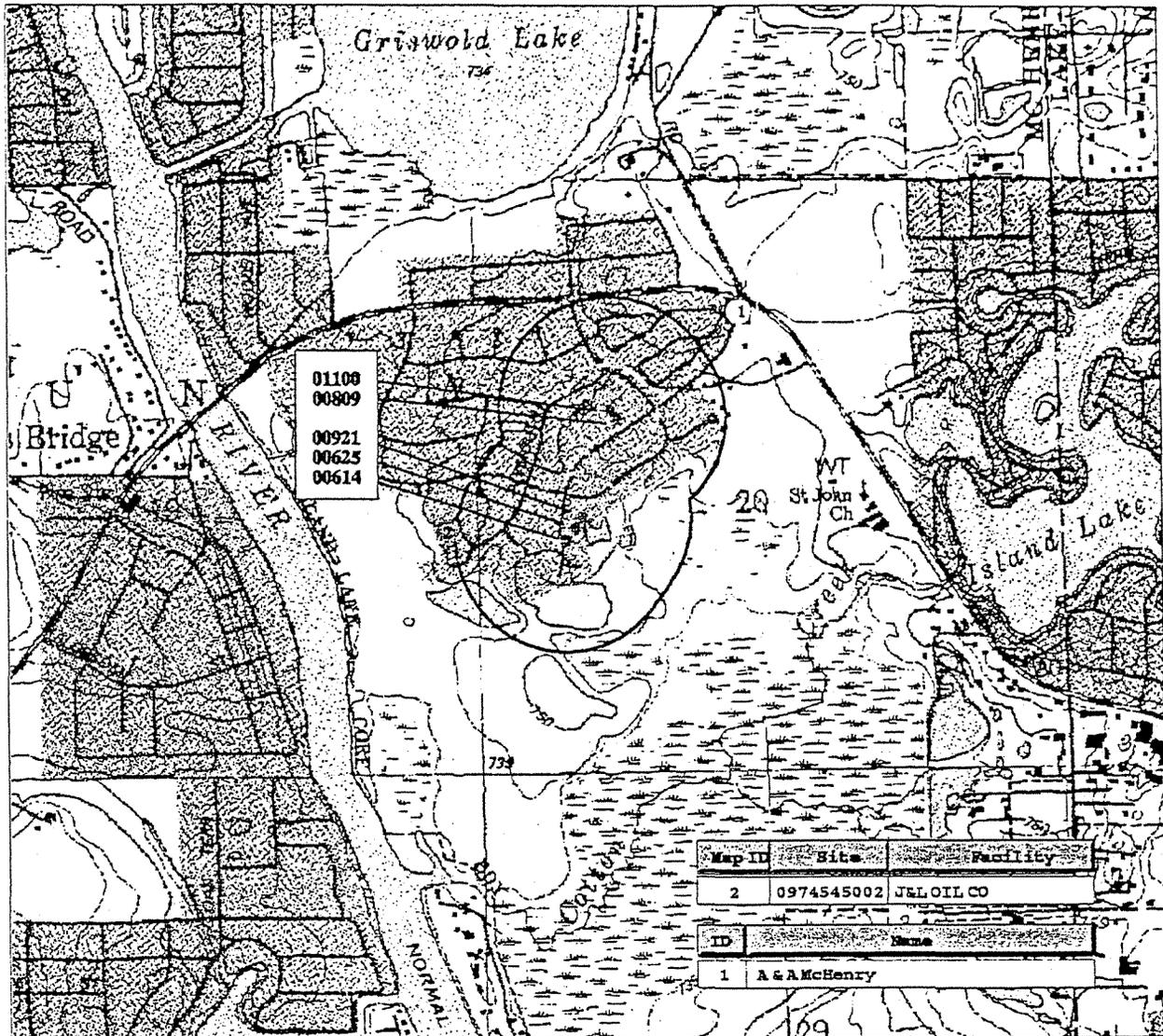
Source Information
 USGS Topo Map DRG obtained from Illinois DNR.
 Sampling Data from Illinois EPA Compliance & Assurance Section.
 Source ID performed in 1992 by Illinois EPA Groundwater Section.
 All results and MCL's in ug/l.



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Island Lake (0974540)

Potential Source and Detection Data



Map ID	Site	Facility
2	0974545002	J&L OIL CO
ID	Name	
1	A & A McHenry	

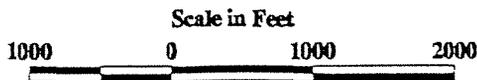


Illinois EPA

Legend	
CWS Wells	
	Confined Aquifer
	Unconfined Aquifer
	Above or Below Ground Fuel Storage
	LUST Sites
	Minimum Setback Zone
	Existing or Potential Maximum Setback Zone
	5-Year Recharge Area

TAP	Well	5-Digit ID	Depth
09	Well #7	00809	95
09	Well #4-6	00625	146
09	Well #4-10	00614	146
09	Well #8	00921	992
09	Well #9	01100	1310

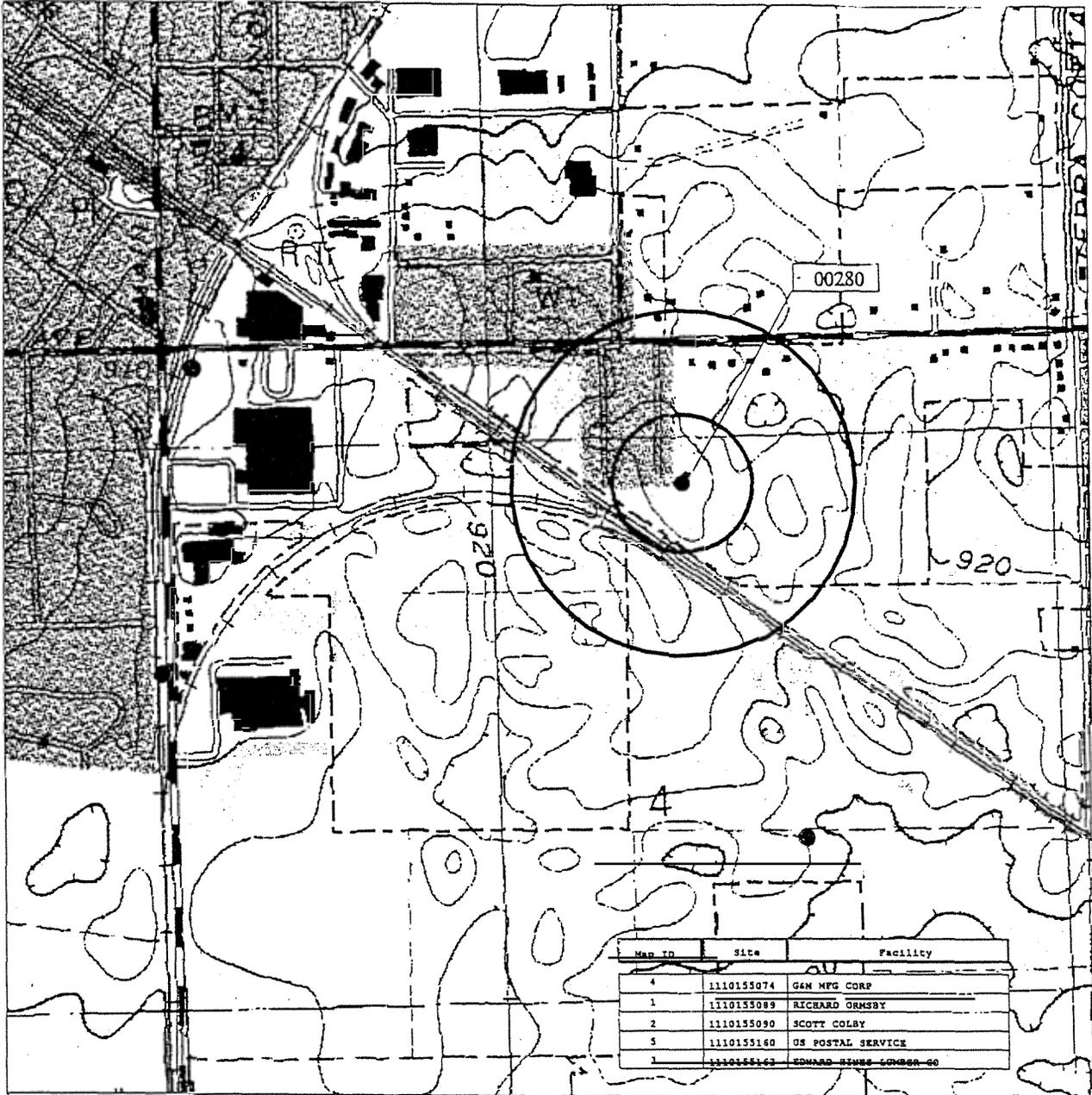
TAP	Chemical	Date	Level	MCL
09	METHYL TERT-BUTYL ETHER	12/09/93	50.00	none
09	METHYL TERT-BUTYL ETHER	01/25/94	37.00	none
09	METHYL TERT-BUTYL ETHER	02/15/94	50.00	none
09	METHYL TERT-BUTYL ETHER	05/25/94	20.00	none



Printed on Recycled Paper

Source Information
 USGS Topo Map DRG Obtained from Illinois DNR.
 Sampling Data from Illinois EPA Compliance & Assurance Section.
 Source ID performed in 1988 by Illinois EPA Groundwater Section.
 All results and MCL's reported in ug/l.

Crystal Heights Association (1115100) Potential Source and Detection Data



Map ID	Site	Facility
4	1110155074	G&N MFG CORP
1	1110155089	RICHARD ORMSBY
2	1110155090	SCOTT COLBY
5	1110155160	US POSTAL SERVICE
1	1110155163	EDWARD HINES-LONGER-OO



Illinois EPA

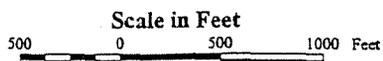
Legend

CWS Wells

- Confined
- Unconfined
- LUST Sites

□ Minimum Setback Zone

□ Existing or Potential Maximum Setback Zone



TAP	Well	5-Digit ID	Depth
02	Well #2	00280	320

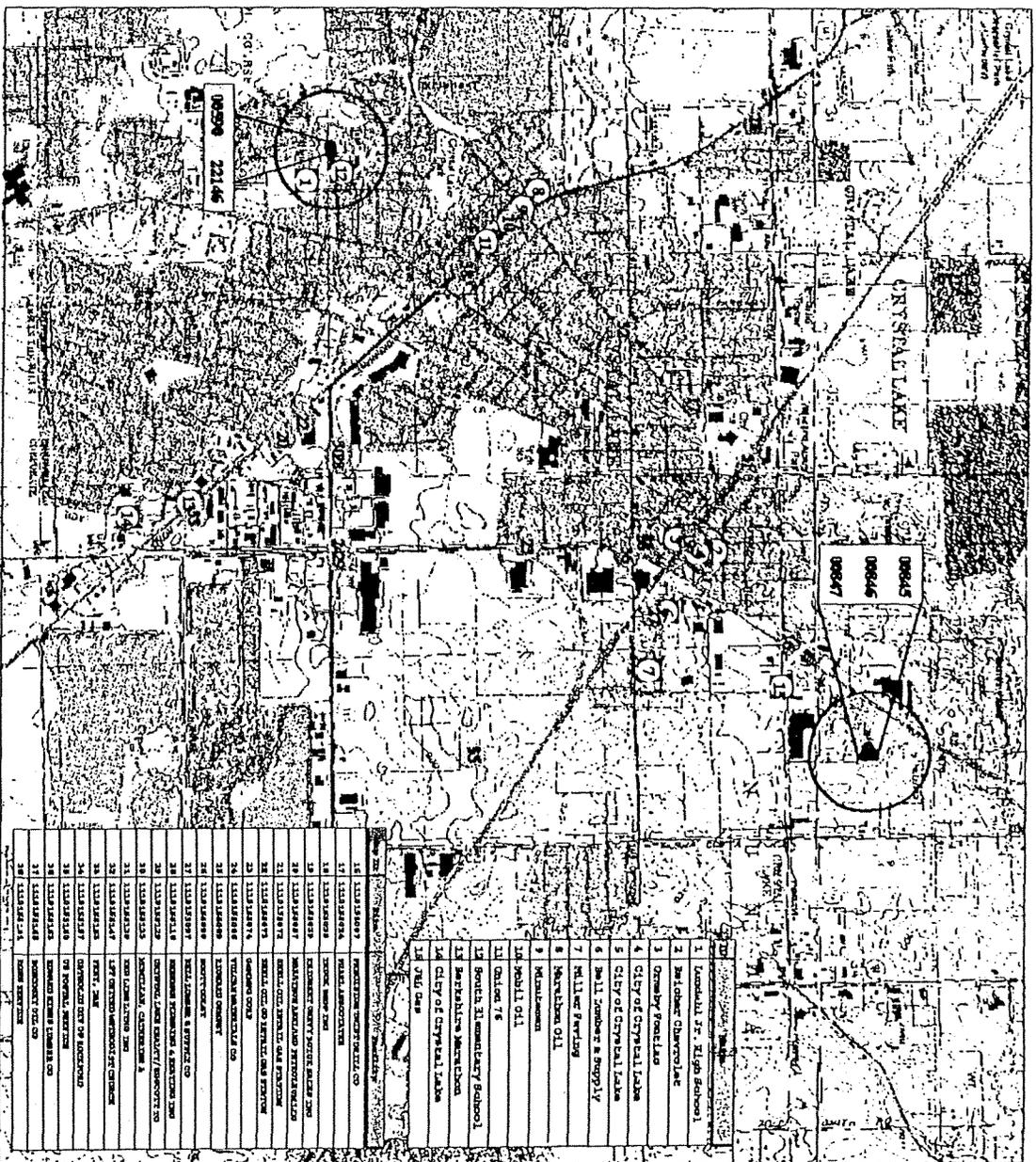
TAP	Chemical	Date	Level	MCL
02	METHYL TERT-BUTYL ETHER	03/06/95	4.00	none
02	METHYL TERT-BUTYL ETHER	05/16/95	12.00	none
02	METHYL TERT-BUTYL ETHER	02/25/97	8.00	none

Source Information

USGS Topo Map DRG Obtained Illinois DNR.
Sampling Data from Illinois EPA Compliance Assurance Section.
Source ID performed in 1988 by Illinois EPA Groundwater Section.
All results and MCL's reported in mg/l.

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Crystal Lake (1110150) Potential Source and Detection Data



Illinois EPA

Legend

- CWB Wells
- Cashed Aquifer
- Unconsolidated Aquifer
- Above or Below Ground Fuel Storage
- LDRI Sites
- Minimum Detention Zone
- Retaining or Potential Maximum Detention Zone
- 3-Year Recurrence Area

Scale in Feet



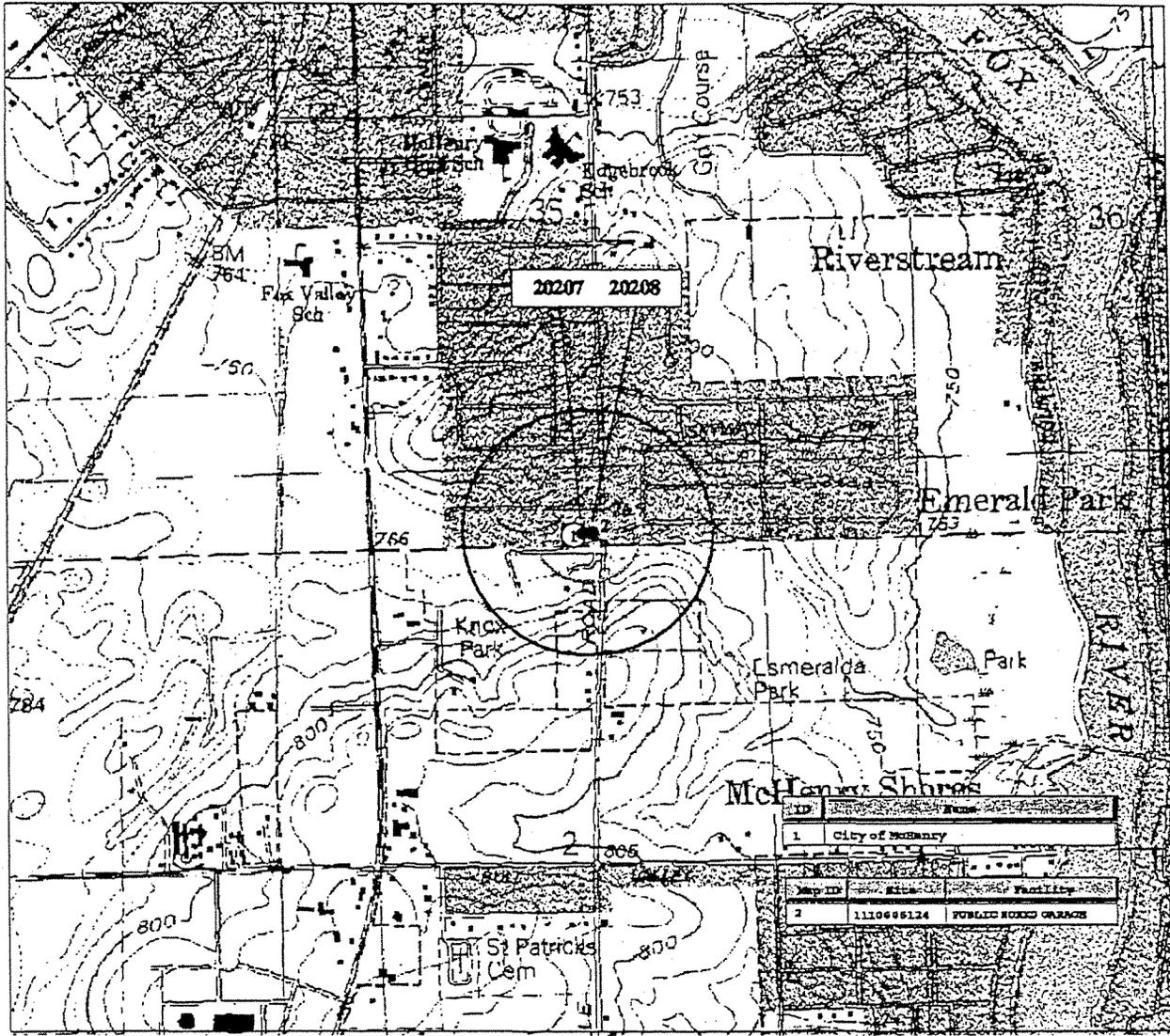
Source Information

USGS Topo Map DRG Obtained From Illinois DNR.
Geographic Data from Illinois EPA Compliance & Assessment Section.
Source ID performed in 1998 by Illinois EPA Compliance Section.
All results and MCL's reported in right.

Well ID	Detection Data
03 Well R6	22146 1395
03 Well R9	00590 205
06 Well R10	00945 258
06 Well R11	00946 237
06 Well R14	00947 243

Well ID	Detection Data
03 FRYDLEBENZEN	03/28/98 4.90 700
03 XYLENE	03/28/98 4.50 10000
03 METHYL TERT-BUTYL ETHER	10/13/98 1.00 10000
06 METHYL TERT-BUTYL ETHER	07/29/96 6.00 10000
06 XYLENE	07/29/96 0.80 10000
06 METHYL TERT-BUTYL ETHER	08/27/96 10.00 10000
06 METHYL TERT-BUTYL ETHER	10/21/96 3.00 10000
06 METHYL TERT-BUTYL ETHER	01/28/97 4.00 10000
06 METHYL TERT-BUTYL ETHER	04/14/97 5.00 10000
06 METHYL TERT-BUTYL ETHER	07/15/97 1.20 10000
06 XYLENE	10/29/97 3.00 10000
06 METHYL TERT-BUTYL ETHER	01/26/98 3.00 10000
06 METHYL TERT-BUTYL ETHER	04/20/98 3.00 10000
06 METHYL TERT-BUTYL ETHER	07/20/98 4.00 10000
06 METHYL TERT-BUTYL ETHER	10/13/98 3.10 10000
06 METHYL TERT-BUTYL ETHER	01/11/99 1.00 10000

McHenry (1110600) Potential Source and Detection Data



Illinois EPA

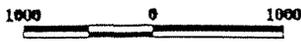
Legend

- CWS Wells
- Confined Aquifer
- Unconfined Aquifer
- Above or Below Ground Fuel Storage
- LUST Sites
- Minimum Setback Zone
- Existing or Potential Maximum Setback Zone
- 5-Year Recharge Area

TAP	Well	5-Digit ID	Depth
01	Well #2	20207	60
01	Well #3	20208	185

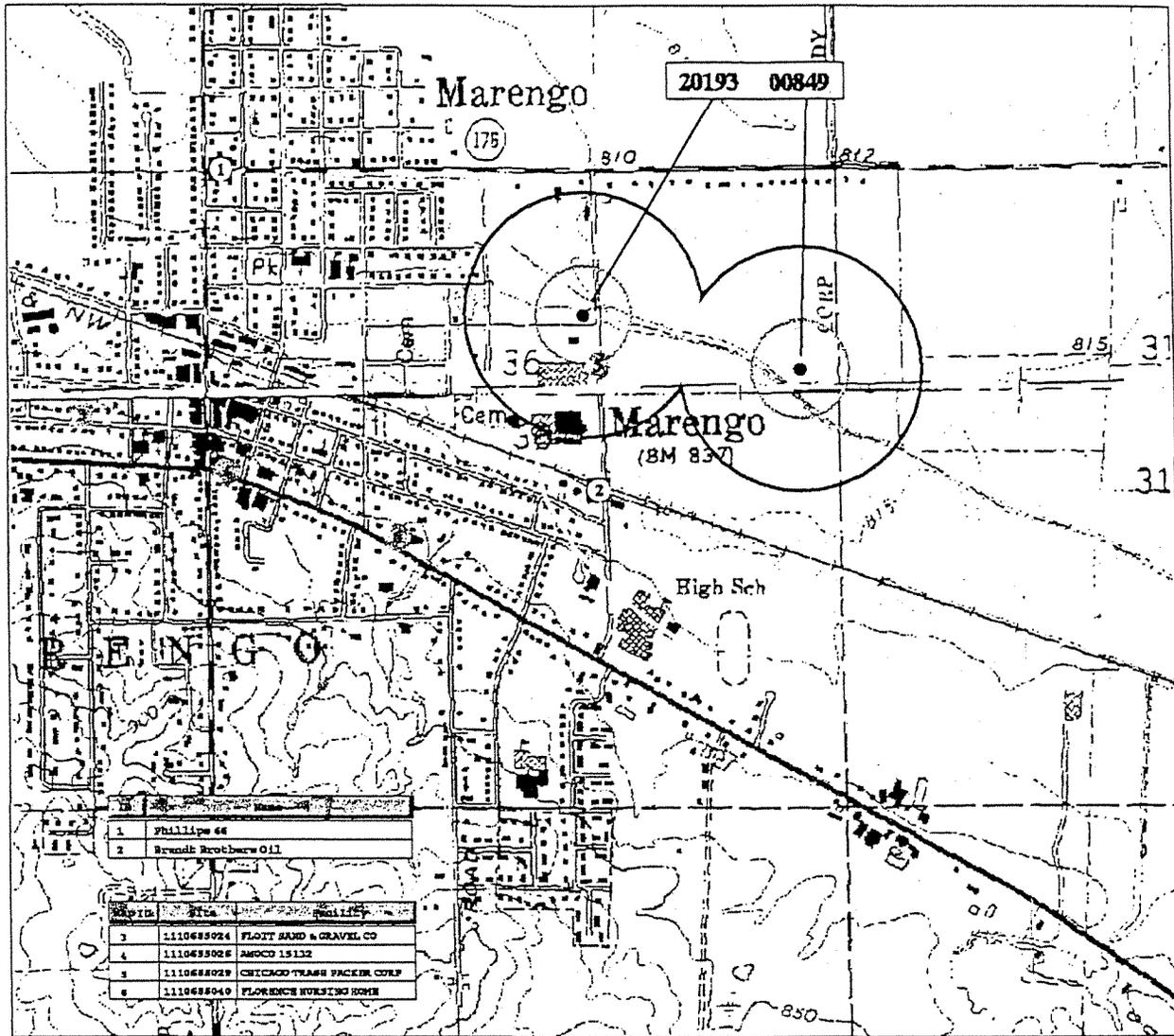
TAP	Chemical	Date	Colldata	Level	MCL
01	METHYL TERT-BUTYL ETHER	01/26/98	980125	1.00	nons

Scale in Feet



Source Information
USGS Topo Map DRG Obtained from Illinois DNR.
Sampling Data from Illinois EPA Compliance & Assurance Section.
Source ID performed in 1988 by Illinois EPA Groundwater Section.
All results and MCL's reported in ug/l

Marengo (1110650) Potential Source and Detection Data



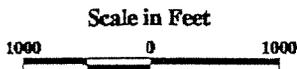
1	Phillips 66
2	Brands Brothers Oil
3	111065024 FLOTT SAND & GRAVEL CO
4	111065028 MSCO 15132
5	111065029 CHICAGO TRASH PACKER CORP
6	111065040 FLORENCE HUSKING HOME



Illinois EPA

Legend

- CWS Wells
- ☉ Confined Aquifer
- Unconfined Aquifer
- ② Above or Below Ground Fuel Storage
- ② LUST Sites
- ☐ Minimum Setback Zone
- ☐ Existing or Potential Maximum Setback Zone
- ☐ 5-Year Recharge Area



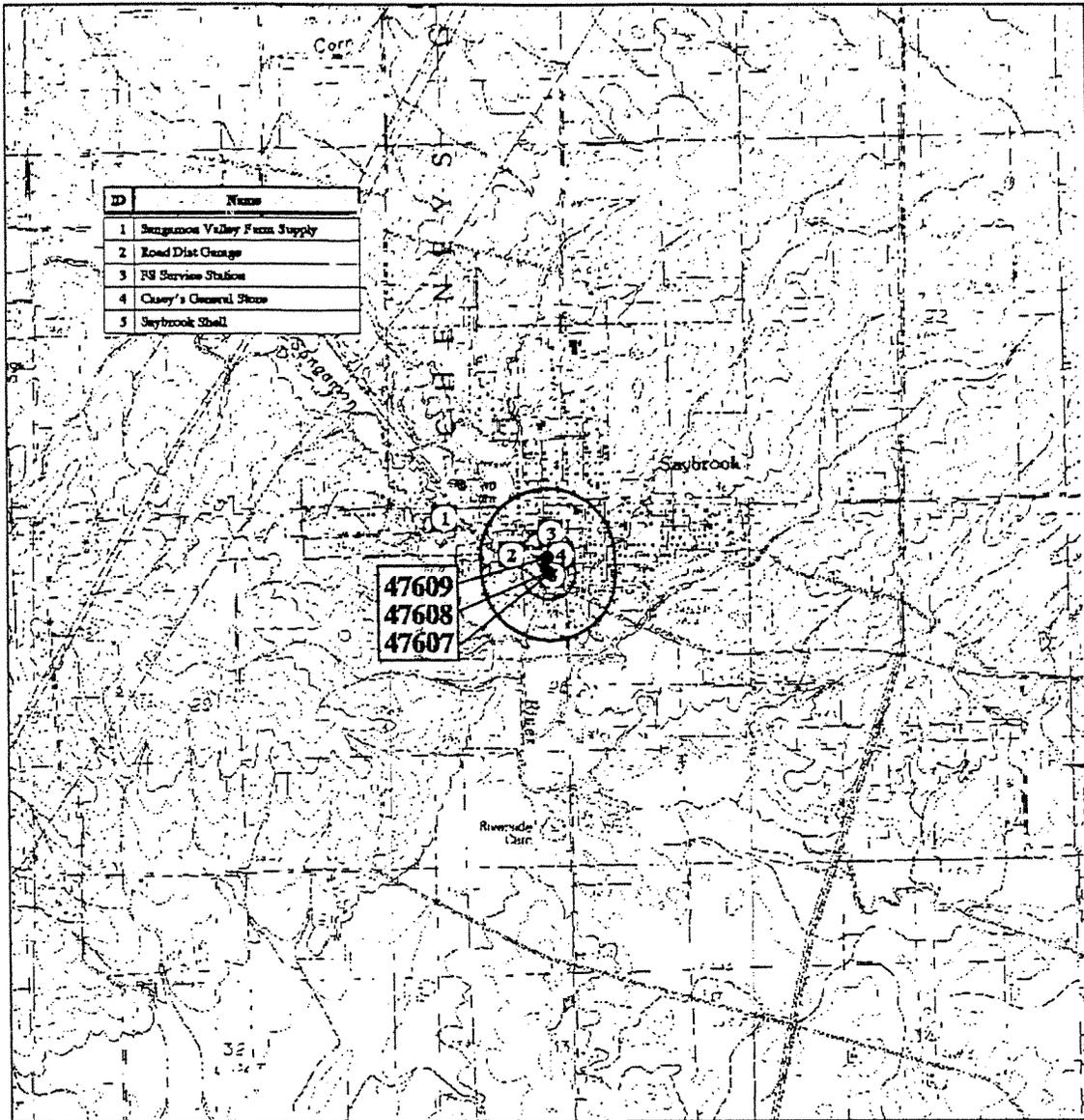
Source Information
USGS Topo Map DRG Obtained from Illinois DNR.
Sampling Data from Illinois EPA Compliance & Assurance Section.
Source ID performed in 1988 by Illinois EPA Groundwater Section.
All results and MCL's reported in ug/l.

Well	Well No.	Source ID	Depth
02	Well 96	20193	88
04	Well 97	00849	58

Well	Source ID	Date	Depth	Concentration
02	00849	01/17/89	8.97	5.0
02	00849	04/15/89	8.90	5.0
02	00849	07/10/89	8.74	5.0
02	00849	01/02/90	8.81	5.0
02	00849	04/02/90	8.20	5.0
02	00849	08/08/90	1.08	5.0
02	00849	09/19/90	5.20	5.0
02	00849	12/05/90	1.50	5.0
02	00849	05/21/91	8.30	5.0
02	00849	06/19/91	4.30	5.0
02	00849	09/19/91	3.60	5.0
02	00849	11/15/91	1.30	5.0
02	00849	06/01/92	8.88	5.0
02	00849	07/19/92	5.70	5.0
02	00849	03/12/96	3.08	ND
02	00849	06/17/96	1.68	ND
02	00849	09/10/96	2.08	ND
02	00849	10/08/96	4.08	ND
02	00849	12/08/96	2.08	ND
02	00849	03/12/97	6.08	ND
02	00849	08/18/97	3.08	ND
02	00849	07/20/97	4.08	ND
02	00849	12/02/98	3.08	ND
04	00849	06/17/96	1.38	ND

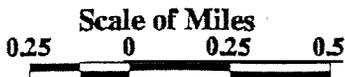
Saybrook (1130950)

Potential Source and Detection Data



Illinois EPA

Legend	
CWS Wells	
●	Confined Aquifer
●	Unconfined Aquifer
●	LUST Site
②	Above or Below Ground Fuel Storage
□	Existing or Potential Maximum Setback Zone
□	Minimum Setback Zone
□	5-Year Recharge Area

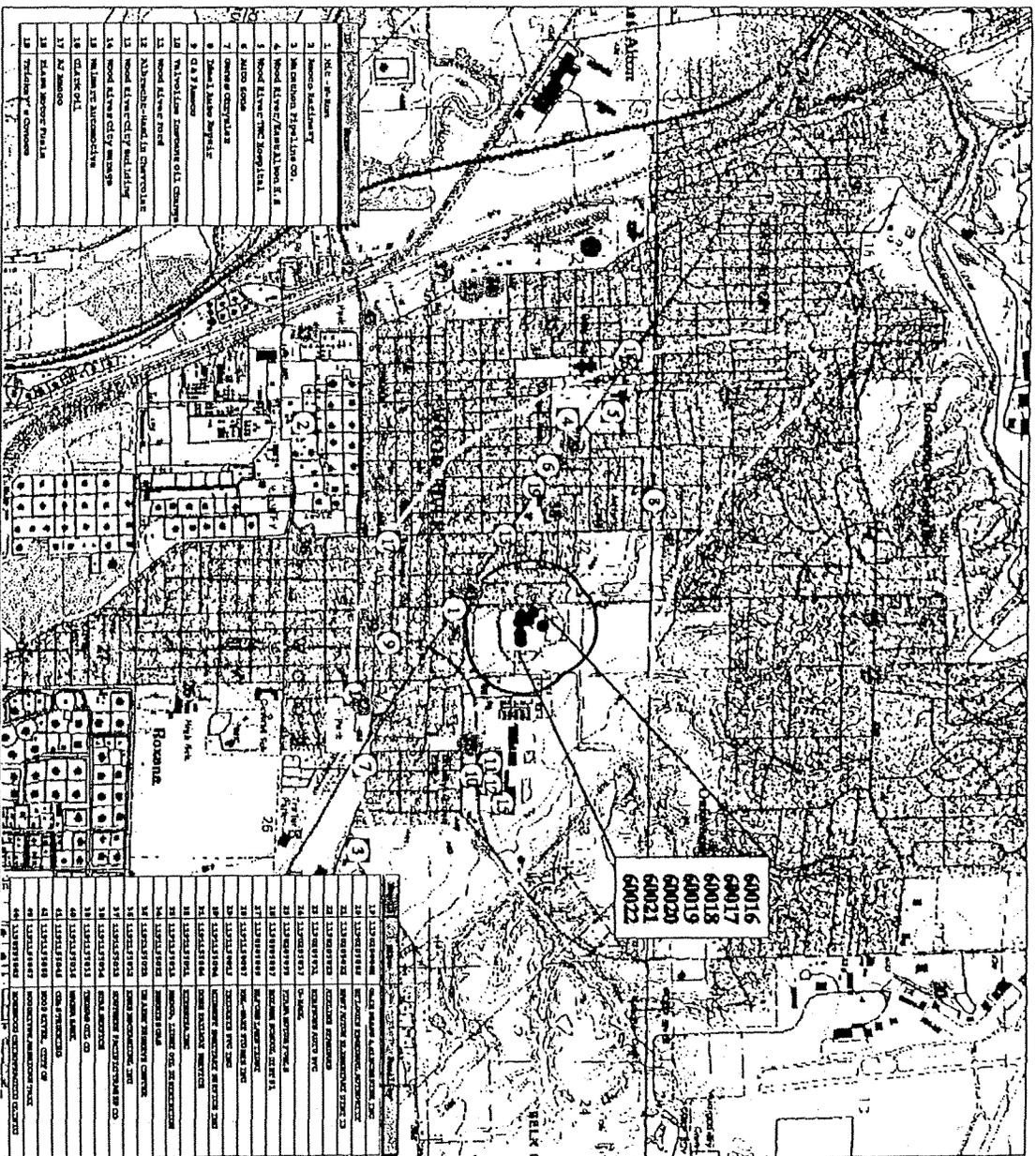


Source Information
 USGS Topo Map DRG obtained from Illinois DNR.
 Sampling Data from Illinois EPA Compliance & Assurance Section.
 Source ID performed in 1991 by Illinois EPA Compliance Section.

IAP	Well	Depth	5-Digit ID
01	Well #1	59	47607
01	Well #2	155	47608
01	Well #3	60	47609

Tap	Chemical	Date	Level	Met
01	methyl tert-butyl ether	3/26/96	3.00	none
01	methyl tert-butyl ether	10/21/97	4.00	none
01	benzene	3/22/94	2.90	5.0
01	benzene	9/19/94	4.00	5.0
01	benzene	6/27/95	1.00	5.0
01	ethylbenzene	9/19/94	21.00	700
01	ethylbenzene	6/27/95	0.70	700
01	ethylbenzene	6/30/96	3.70	700
01	ethylbenzene	3/31/98	0.80	700
01	ethylbenzene	6/23/98	3.30	700
01	ethylbenzene	9/29/98	3.80	700
01	toluene	9/19/94	0.80	1000
01	xylene	4/6/93	1.40	10000
01	xylene	9/19/94	9.90	10000
01	xylene	9/30/96	1.30	10000
01	xylene	6/23/98	2.20	10000

Bethalto (1190150) Potential Source and Detection Data



Illinois EPA

Legend

CWS Wells

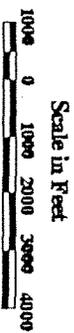
- Confined Aquifer
- Unconfined Aquifer

Above or Below Ground Fuel Storage

- 1 UST Slices
- 2 Michigan Seaback Zones

Excluding or Potential

- Maximum Seaback Zones
- 5-Year Recharge Area



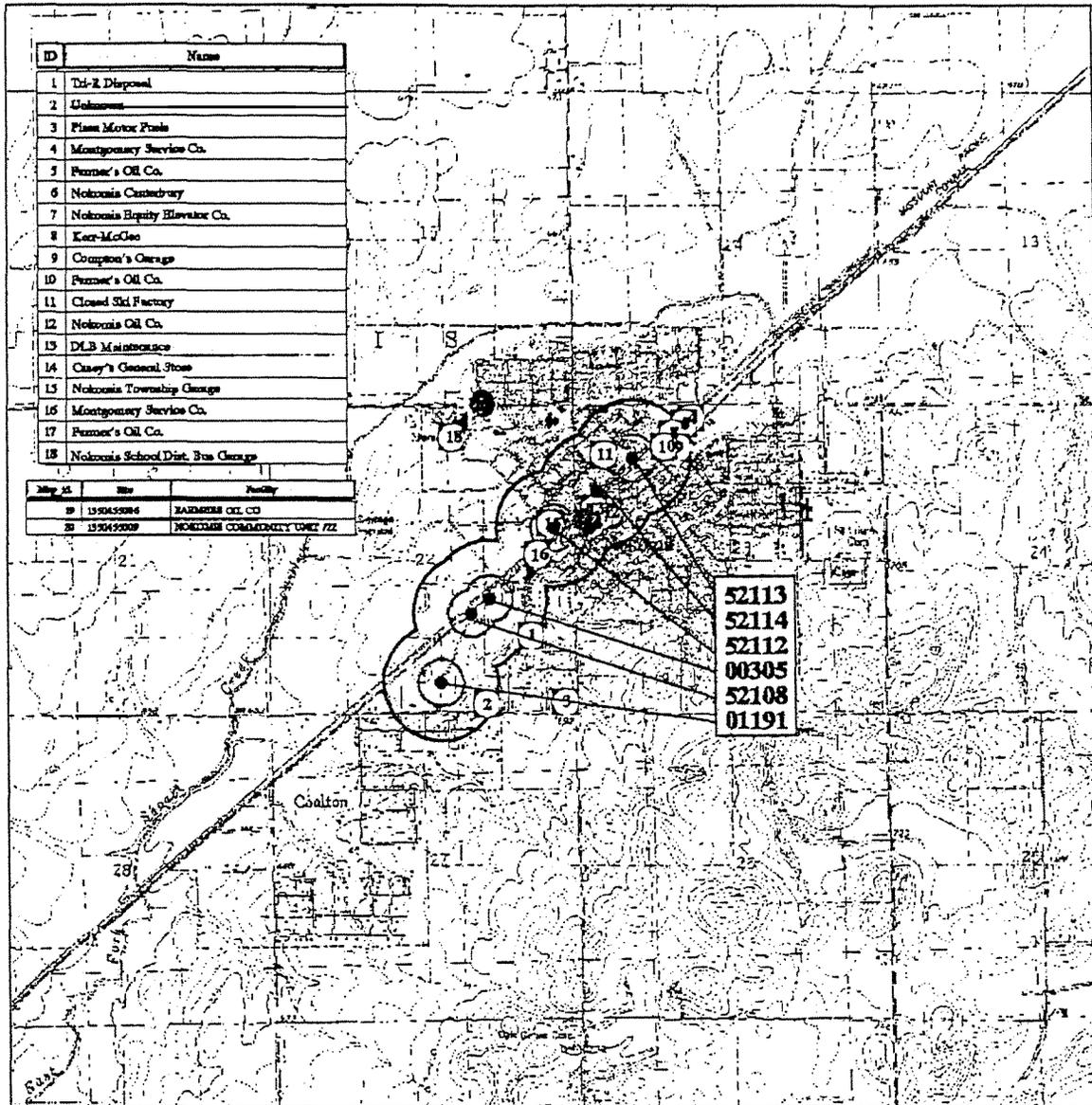
Source Information
URCS Topo Map DRG Obtained from Illinois DNR.
Sampling Data from Illinois EPA Compliance & Assessment Section.
Extended Source ID performed in 1996 by Metabolic Water Department.
All results and MCI's reported in ug/l.

Source ID	Name	Date	Distance
01	WALI #6	60018	35
01	WALI #7	60017	30
01	WALI #8	60018	31
01	WALI #9	60019	32
01	WALI #10	60020	38
01	WALI #11	60021	32
01	WALI #12	60022	36

Source ID	Name	Date	Distance
01	WALI #6	60018	35
01	WALI #7	60017	30
01	WALI #8	60018	31
01	WALI #9	60019	32
01	WALI #10	60020	38
01	WALI #11	60021	32
01	WALI #12	60022	36

Nokomis (1350450)

Potential Source and Detection Data

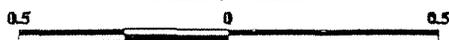


Illinois EPA

Legend

- CWS Wells**
- Confined Aquifer
 - Unconfined Aquifer
 - ⊕ LUST Site
 - ② Above or Below Ground Fuel Storage
 - Existing or Potential Maximum Setback Zone
 - Minimum Setback Zone
 - 5-Year Recharge Area

Scale of Miles



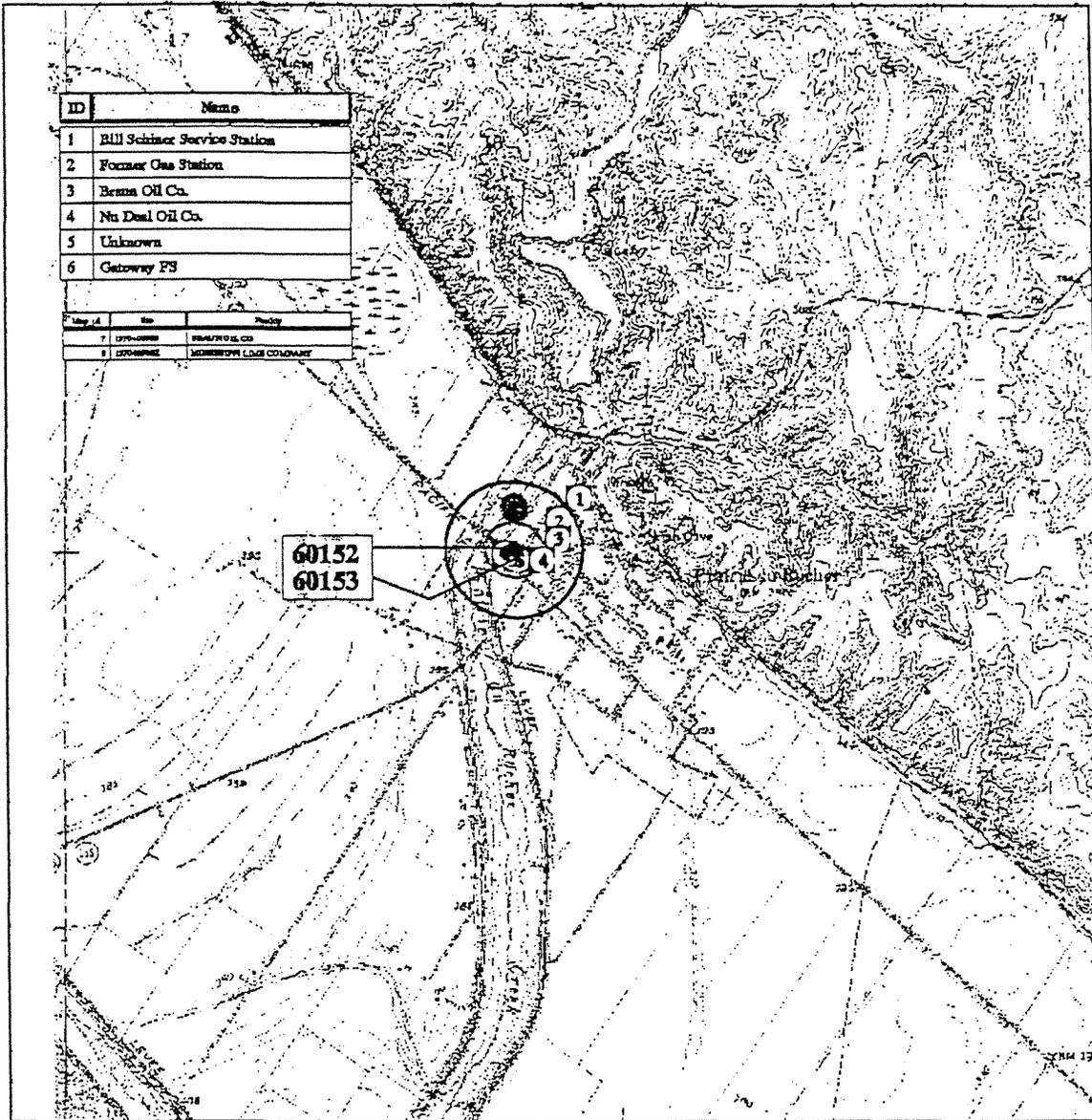
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TAP	Well	5-Digit ID	Depth
01	Well #4	52108	40
01	Well #8	52112	40
01	Well #9	52113	47
01	Well #10	52114	49
01	Well #11	00305	41
01	Well #13	01191	36

TAP	Chemical	Date	Level	MCL
01	methyl tert-butyl ether	3/26/96	1.00	none
01	methyl tert-butyl ether	3/11/97	1.00	none
01	benzene	6/20/95	0.60	5.0
01	benzene	12/26/95	1.00	5.0

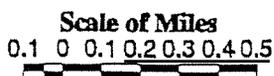
Source Information
 USGS Topo Map DRG obtained from Illinois DNR.
 Sampling Data from Illinois EPA Compliance & Assurance Section.
 Source ID performed in 1988 by Illinois EPA Groundwater Section.
 All results and MCL's in ug/l.

Prairie Du Rocher (1570400) Potential Source and Detection Data



Illinois EPA

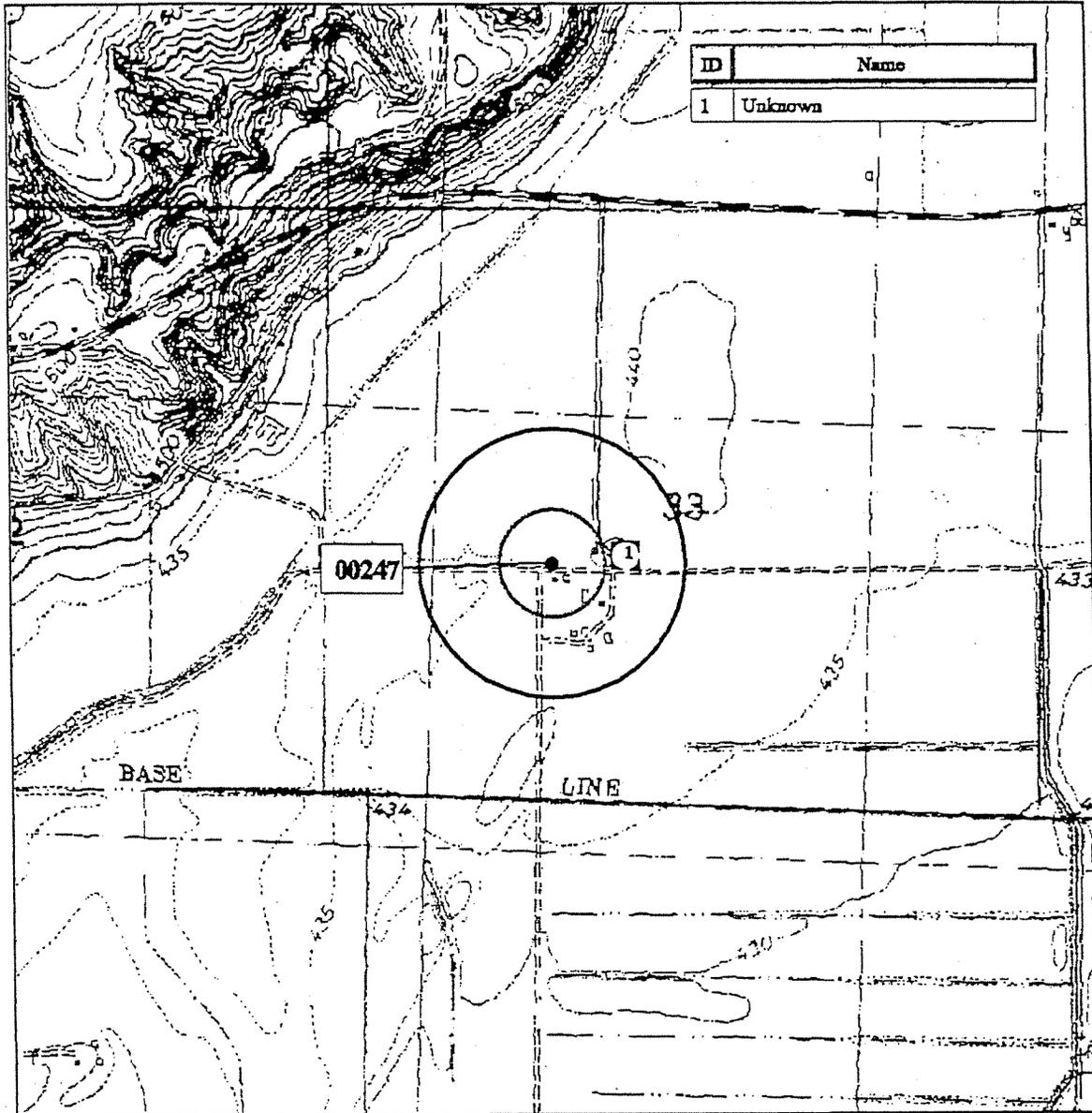
Legend	
●	Confined Aquifer
●	Unconfined Aquifer
⊙	LUST Sites
②	Above or Below Ground Fuel Storage
□	Existing or Potential Maximum Setback Zone
□	Minimum Setback Zone
□	5-Year Recharge Area



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Rushville (1690200)

Potential Source and Detection Data



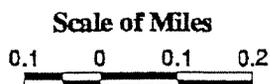
Illinois EPA

TAP	Well	5-Digit ID	Depth
03	Well #	00247	62

Legend	
●	Confined Aquifer
●	Unconfined Aquifer
●	LUST Site
②	Above or Below Ground Fuel Storage
□	Existing or Potential Maximum Setback Zone
□	Minimum Setback Zone
□	5-Year Recharge Area

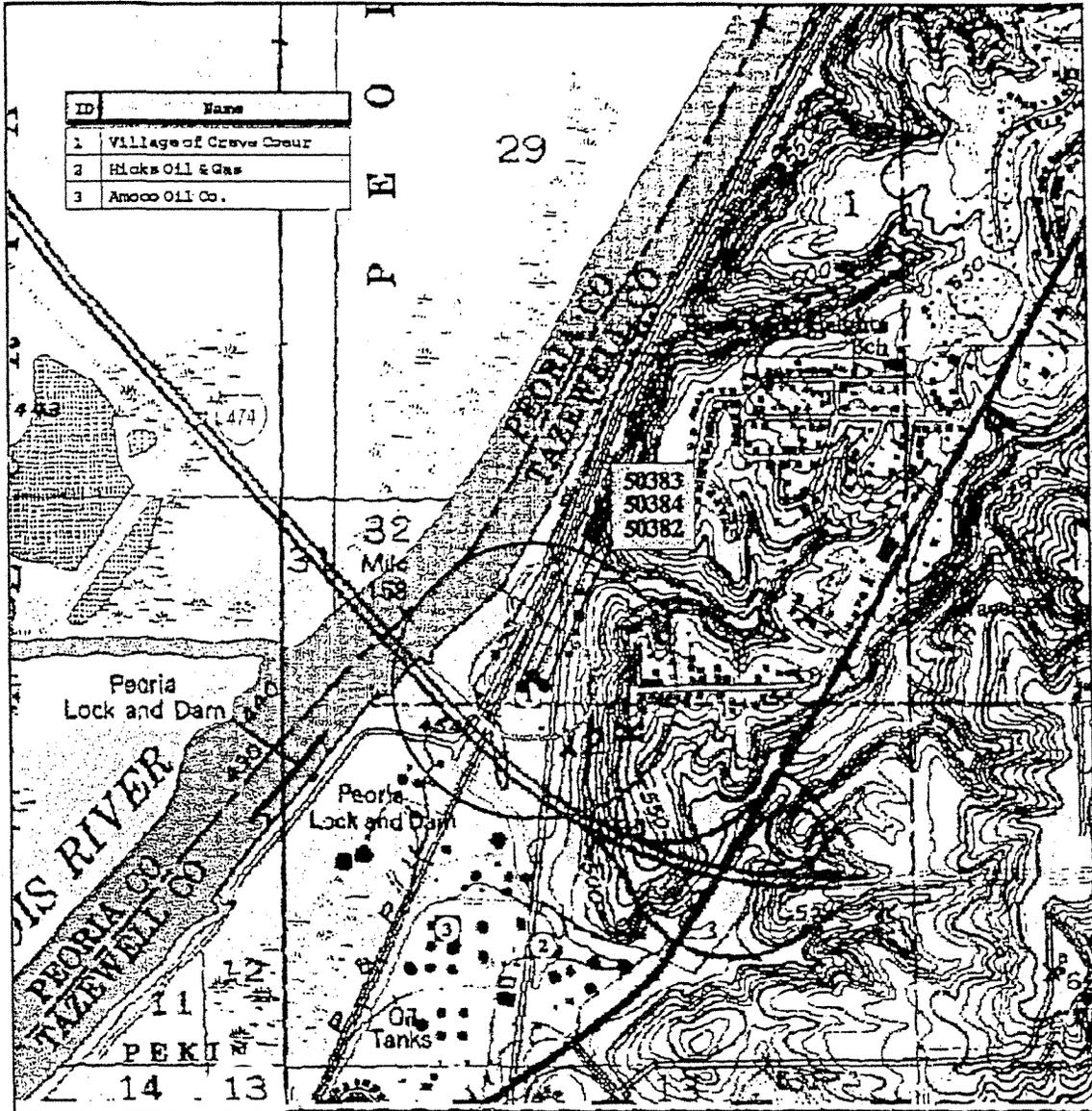
TAP	Chemical	Date	Level	MCL
03	methyl tert-butyl ether	08/07/96	2.00	none

Source Information
 USGS Topo Map DRG obtained from Illinois DNR.
 Sampling Data from Illinois EPA Compliance & Assurance Section.
 Source ID performed in 1990 by Illinois EPA Groundwater Section.
 All results and MCL's in ug/l.



Creve Coeur (1790100)

Potential Source and Detection Data

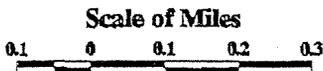


Illinois EPA

Legend

CWS Wells

- Confined Aquifer
- Unconfined Aquifer
- ② Above or Below Ground Fuel Storage
- Existing or Potential Maximum Setback Zone
- Minimum Setback Zone
- 5-Year Recharge Area



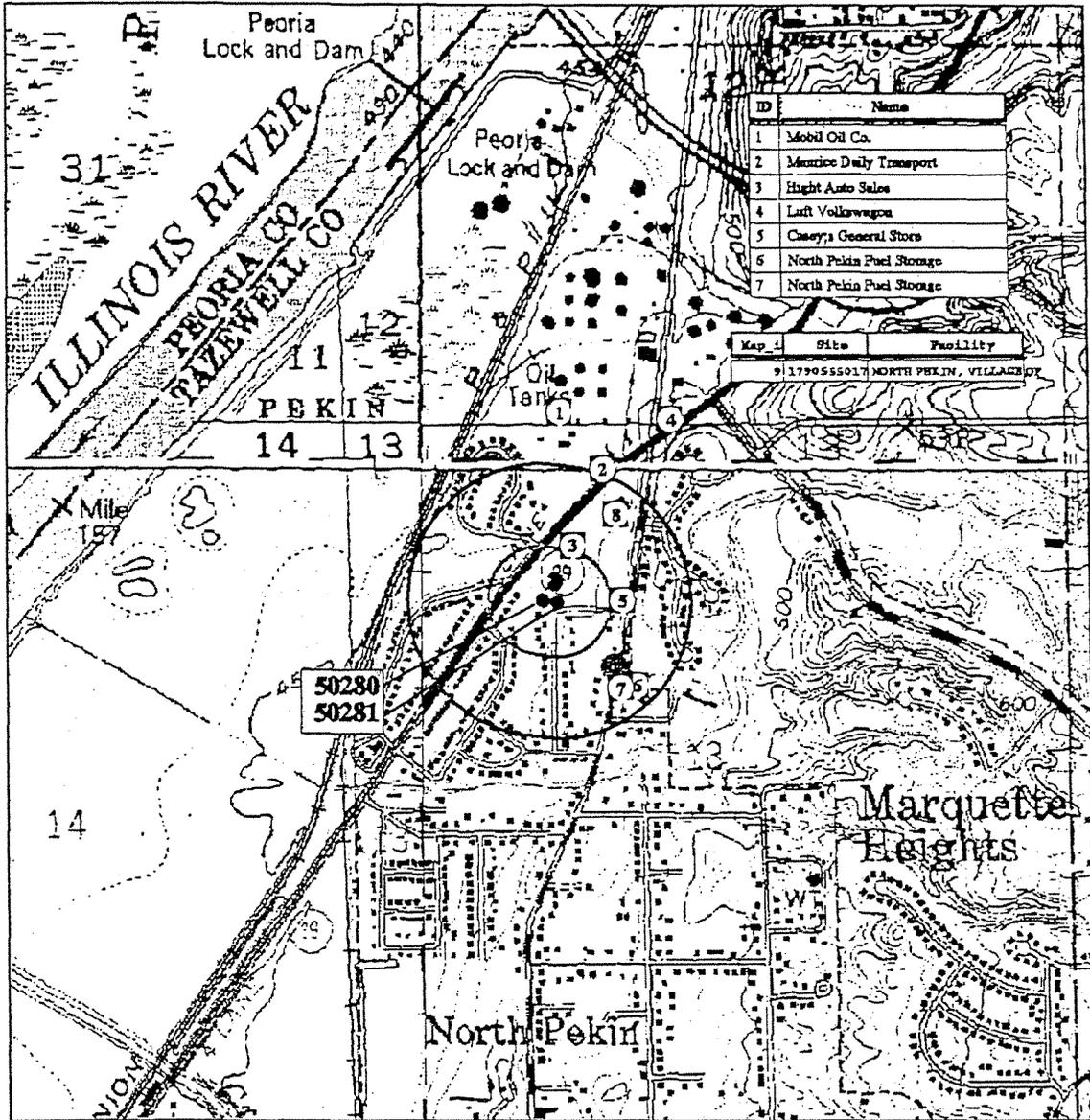
printed on recycled paper

TAP	Well	5-Digit ID	Depth
01	Well #1	50382	91
01	Well #3	50383	78
01	Well #4	50384	81

Well	Chemical	Date	Level	MCL
01	methyl tert-butyl ether	8/29/90	0.00360	none
01	methyl tert-butyl ether	9/26/90	0.00320	none
01	methyl tert-butyl ether	12/28/90	0.00370	none
01	methyl tert-butyl ether	1/19/91	0.00690	none
01	methyl tert-butyl ether	3/13/91	0.01000	none
01	methyl tert-butyl ether	4/23/93	0.00230	none
01	methyl tert-butyl ether	6/18/93	0.00280	none
01	methyl tert-butyl ether	9/24/93	0.00140	none
01	methyl tert-butyl ether	12/17/93	0.00120	none
01	methyl tert-butyl ether	1/14/94	0.00110	none
01	methyl tert-butyl ether	5/20/94	0.00110	none
01	methyl tert-butyl ether	6/30/93	0.00190	none

Source Information
 USGS Topo Map DRG obtained from Illinois DNR.
 Sampling data obtained from AMOCO.
 Source ID performed in 1989 by Illinois EPA Groundwater Section.
 All results and MCL's in ug/l.

Marquette Heights (1790400) Potential Source and Detection Data



Illinois EPA

Legend

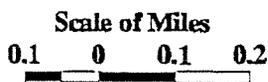
CWS Wells

- Confined Aquifer
- Unconfined Aquifer
- LUST Site
- ② Above or Below Ground Fuel Storage
- Existing or Potential Maximum Setback Zone
- Minimum Setback Zone
- 5-Year Recharge Area

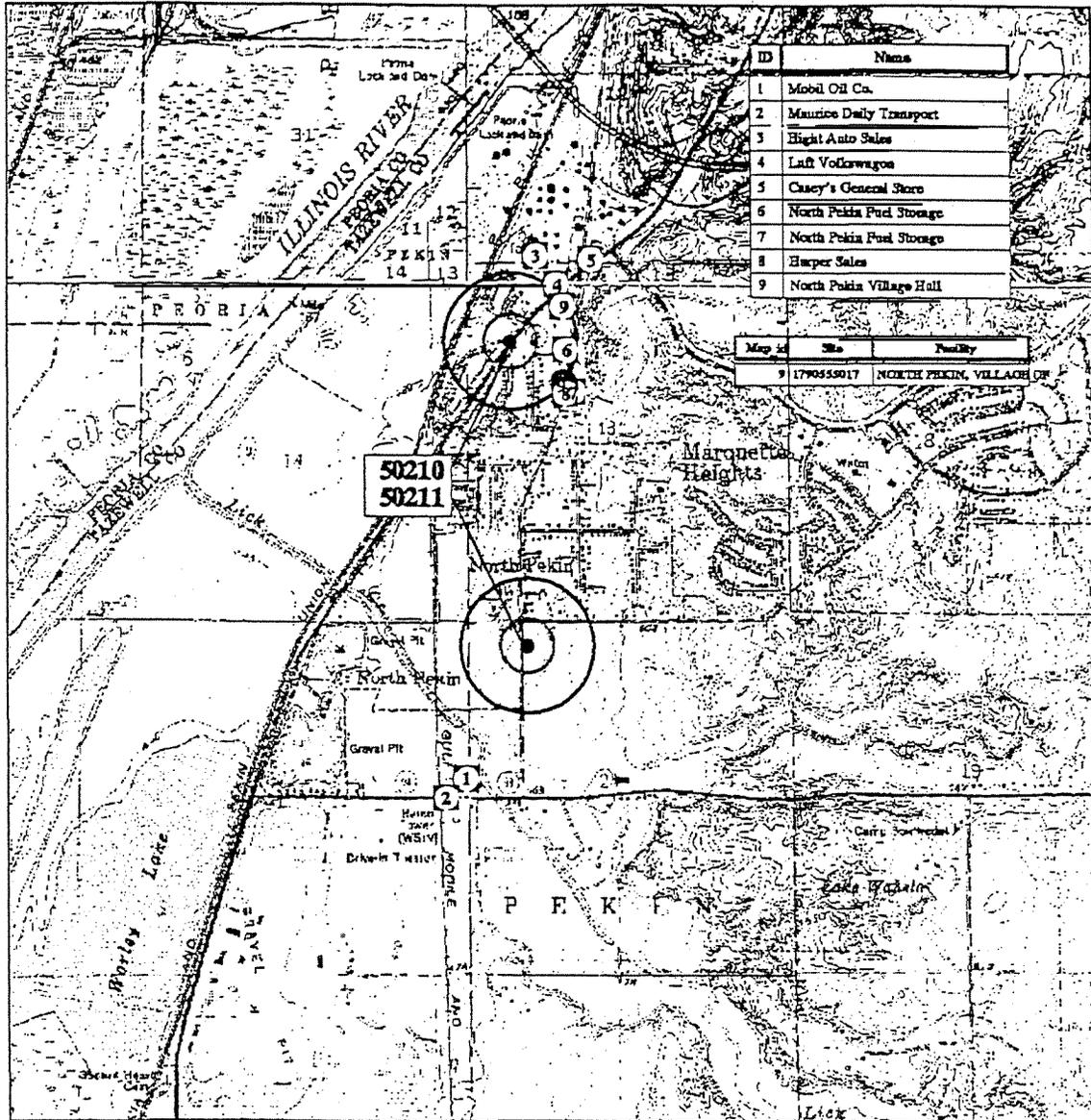
TAP	Well	5-Digit ID	Depth
01	Well #4	50280	95
01	Well #5	50281	94

Tap	Chemical	Date	Level	MCL
01	methyl tert-butyl ether	07/09/90	1.80	none
01	methyl tert-butyl ether	07/07/90	1.20	none

Source Information
 USGS Topo Map DRG obtained from Illinois DNR.
 Sampling Data from Illinois EPA Compliance & Assurance Section.
 Source ID performed in 1990 by Illinois EPA Groundwater Section.
 All results and MCL's in ug/l.



North Pekin (1790550) Potential Source and Detection Data



Illinois EPA

Legend	
●	Confined Aquifer
○	Unconfined Aquifer
⊙	LUST Site
②	Above or Below Ground Fuel Storage
□	Existing or Potential Maximum Setback Zone
□	Minimum Setback Zone
□	5-Year Recharge Area

TAP	Well	5-Digit ID	Depth
01	Well #2	50211	104
02	Well #1	50210	85

TAP	Chemical	Date	Level	MCL
01	methyl tert-butyl ether	07/23/90	0.40	none
01	methyl tert-butyl ether	07/23/90	0.90	none
01	methyl tert-butyl ether	07/23/90	0.80	none
02	methyl tert-butyl ether	07/09/90	1.10	none

Source Information
USGS Topo Map DRG obtained from Illinois DNR.
Sampling Data from Illinois EPA Compliance & Assurance Section.
Source ID performed in 1990 by Illinois EPA Groundwater Section.
All results and MCL's in ug/l.

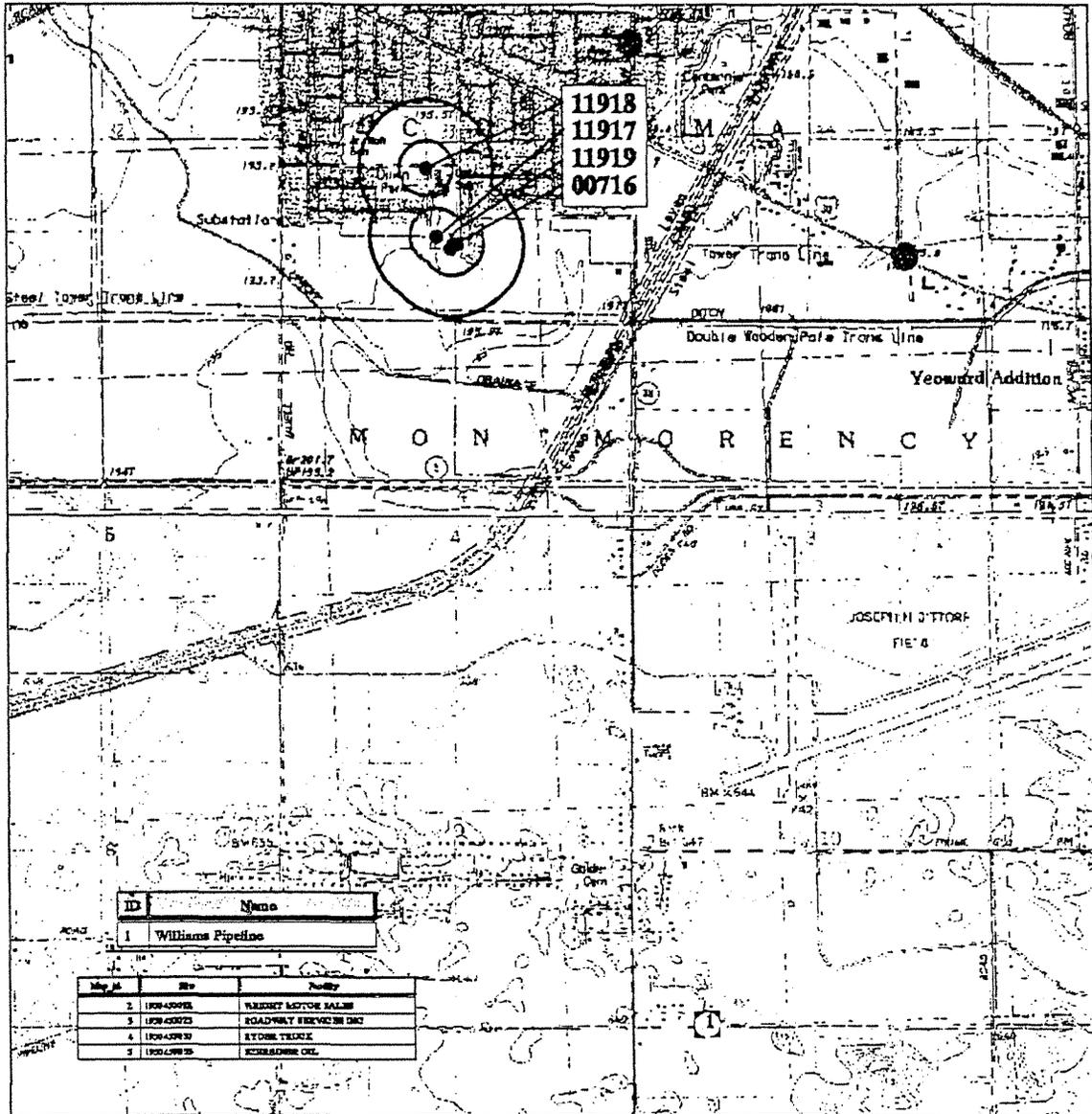
Scale of Miles



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Rock Falls (1950450)

Potential Source and Detection Data



ID	Name
1	Williams Pipeline

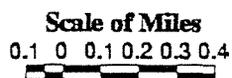
Map #	No.	Locality
2	195-0008	WALNUT LITCHIE SALON
3	195-0022	ROADWAY SERVICE INC
4	195-0032	STONE TRUCK
5	195-0035	RECHARGE OIL



Illinois EPA

Legend

- CWS Wells**
- Confined Aquifer
 - Unconfined Aquifer
 - LUST Site
 - ② Above or Below Ground Fuel Storage
- Existing or Potential
- Maximum Setback Zone
 - Minimum Setback Zone
- 5-Year Recharge Area



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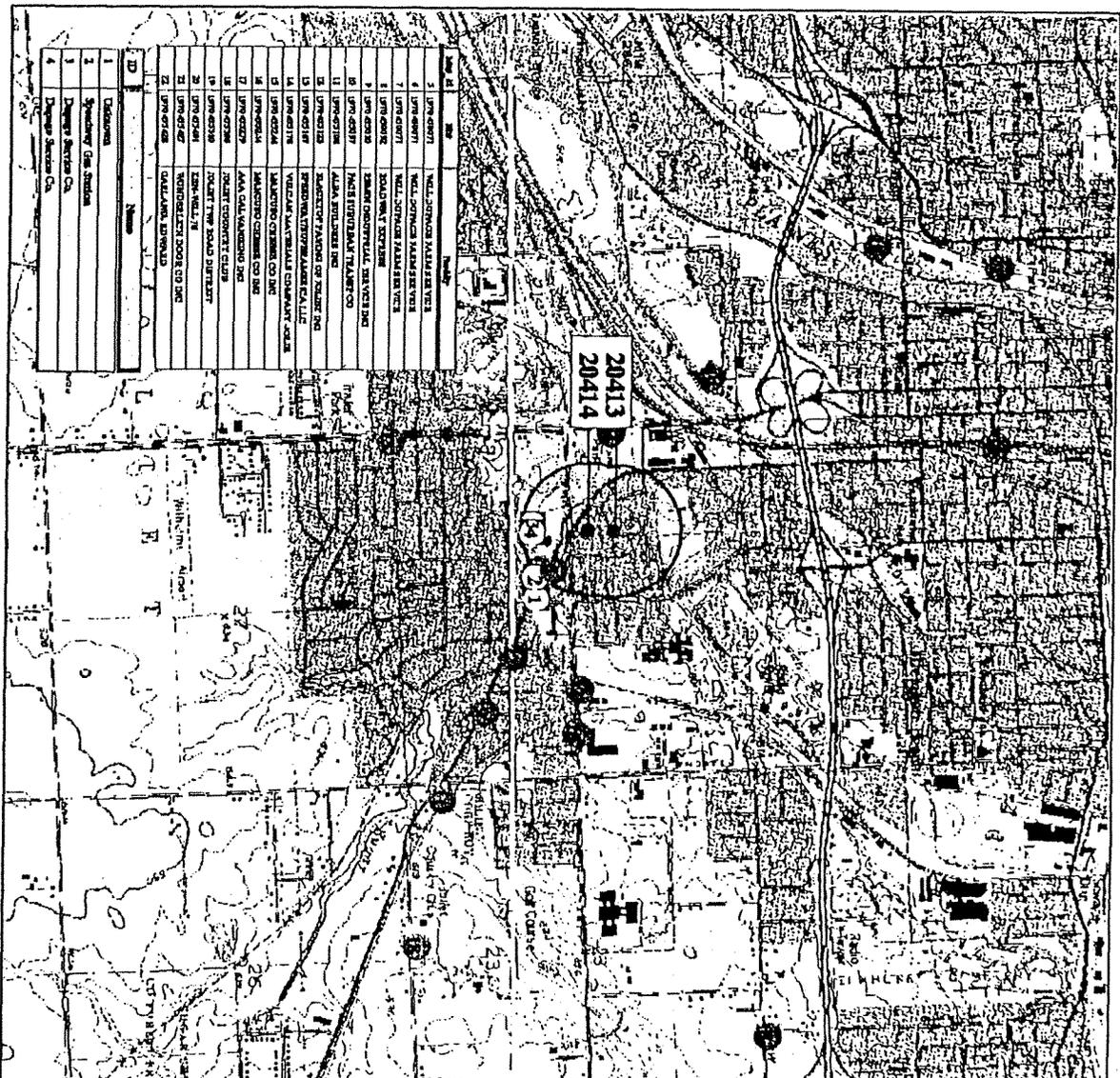
TAP	Well	5-Digit ID	Depth
01	Well #2	11917	136
01	Well #3	11918	70
01	Well #4	11919	131
01	Well #5	00716	

TAP	Chemical	Date	Level	MCL
01	methyl tert-butyl ether	11/20/95	4.00	none
01	methyl tert-butyl ether	02/05/96	3.00	none
01	methyl tert-butyl ether	02/03/98	1.00	none
01	methyl tert-butyl ether	07/20/98	1.00	none
01	xylene	02/01/94	1.20	10000
01	xylene	07/31/95	0.90	10000

Sources Information
 USGS Topo Map DRG obtained from Illinois DNR.
 Sampling Data from Illinois EPA Compliance & Assurance Section.
 Source ID performed in 1989 by Illinois EPA Groundwater Section.
 All results and MCL's in ug/l.

Clearview Subdy. (1975360)

Potential Source and Detection Data

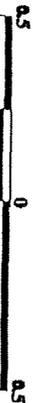


Illinois EPA

Legend

- CWS Wells
- Confined Aquifer
- Unconfined Aquifer
- LUST Site
- ② Above or Below Ground Fuel Storage
- Existing or Potential Maximum Saturated Zone
- Minimum Saturated Zone
- 5-Year Recharge Area

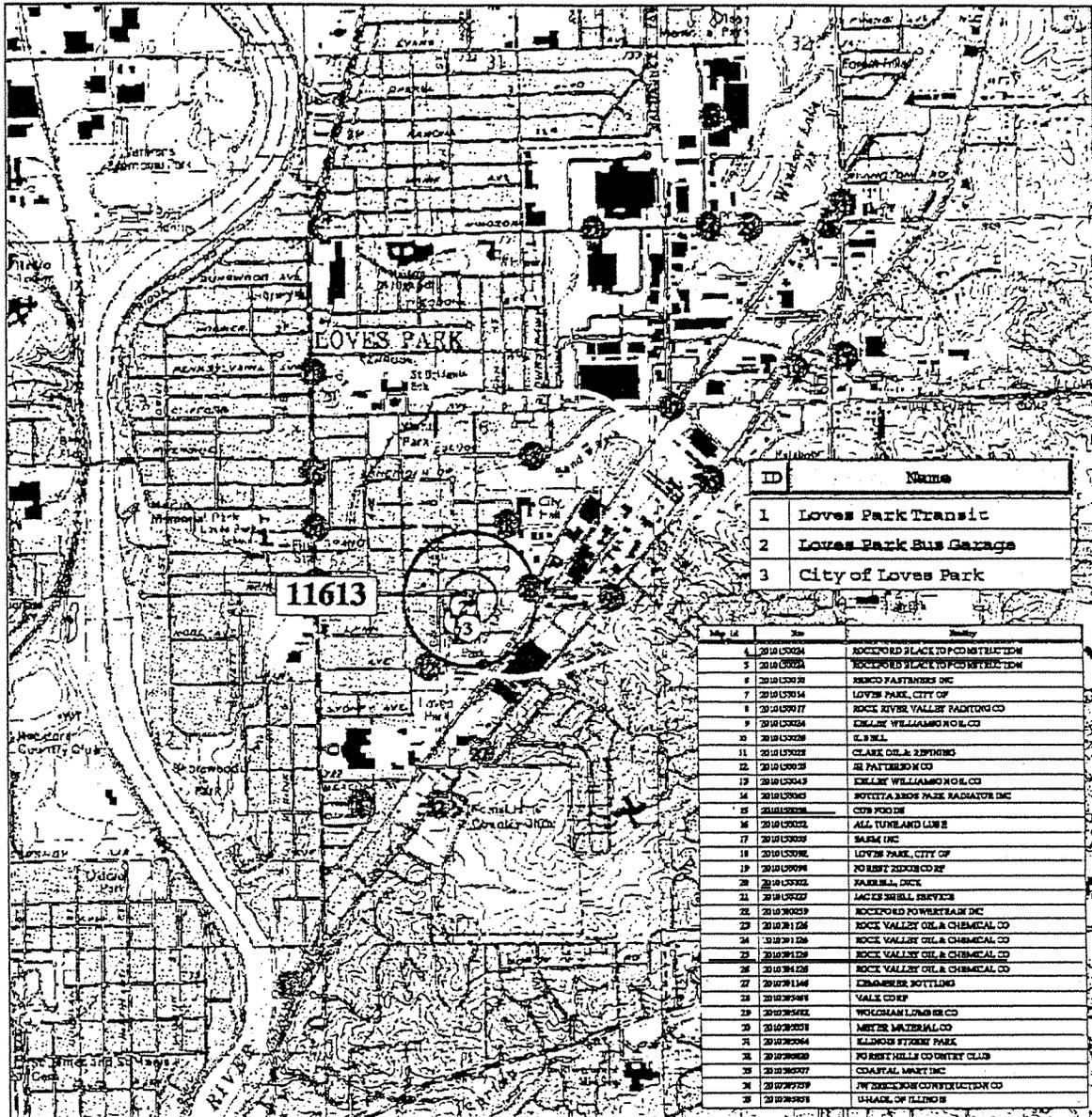
Scale of Miles



Source Information

USGS Topo Map DRG obtained from Illinois DNR.
 Sampling Data from Illinois EPA Compliance & Assurance Section.
 Source ID performed in 1995 by Illinois EPA Groundwater Section.
 All results and MCL's in ug/l.

Loves Park (2010150) Potential Source and Detection Data



Illinois EPA

Legend

CWS Wells

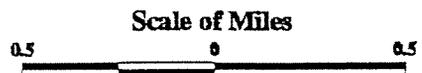
- Confined Aquifer
- Unconfined Aquifer
- ⊕ LUST Site
- ② Above or Below Ground Fuel Storage

Existing or Potential Maximum Setback Zone
 Minimum Setback Zone
 5-Year Recharge Area

TAP	Well	5-Digit ID	Depth
01	Well #1	11613	150

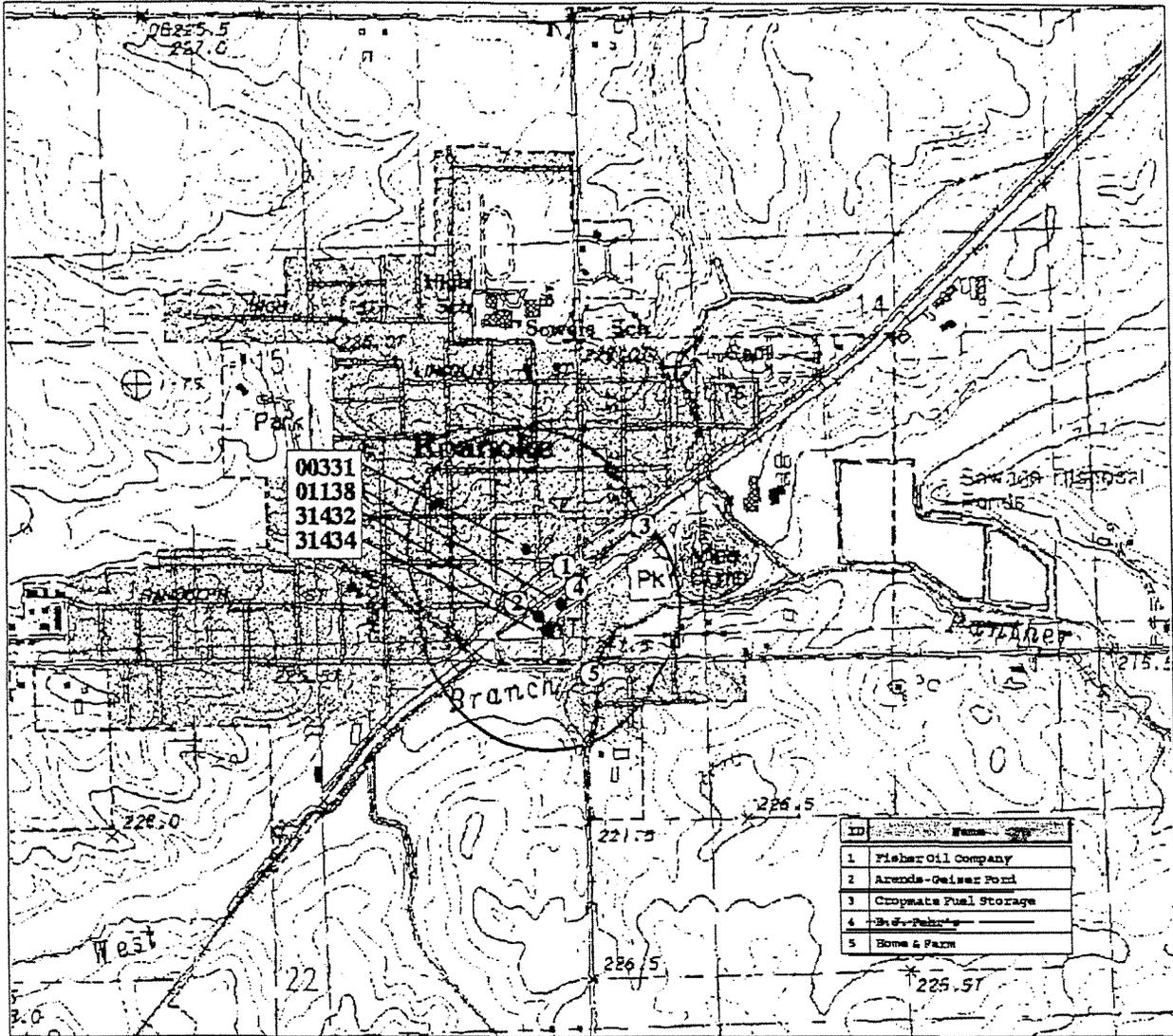
TAP	Chemical	Date	Level	MCL
01	methyl tert-butyl ether	06/23/98	1.00	none

Source Information
 USGS Topo Map DRG obtained from Illinois DNR.
 Sampling Data from Illinois EPA Compliance & Assurance Section.
 Source ID performed in 1991 by Illinois EPA Groundwater Section.
 All results and MCL's in ug/l.



Roanoke (2030550)

Potential Source and Detection Data



Illinois EPA

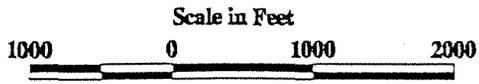
Legend

CWE Wells

- ③ Confined Aquifer
- Unconfined Aquifer
- ② Above or Below Ground Fuel Storage
- 2 LUST Sites
- Minimum Setback Zones
- Existing or Potential Maximum Setback Zones
- 5-Year Recharge Area

Tap	Well	5-Digit ID	Depth
02	Well #2	00331	121
02	Well #7	01138	47
02	Well #3	31432	52
02	Well #5	31434	50

TAP	Chemical	Date	Level	MCL
02	METHYL TERT-BUTYL ETHER	06/29/93	85.00	none
02	METHYL TERT-BUTYL ETHER	08/30/93	14.00	none
02	METHYL TERT-BUTYL ETHER	11/22/93	24.00	none
02	METHYL TERT-BUTYL ETHER	11/28/93	48.00	none
02	METHYL TERT-BUTYL ETHER	02/05/95	4.00	none
02	METHYL TERT-BUTYL ETHER	11/06/96	39.00	none



Source Information
 USGS Topo Map DRG Obtained from Illinois DNR.
 Sampling Data from Illinois EPA Compliance & Assurance Section.
 Source ID performed in 1998 by Illinois EPA Groundwater Section.
 All results and MCL's reported in ug/l.

EXHIBIT III – MTBE Taste and Odor Thresholds

EXHIBIT IV. - MTBE Health Advisory

**Drinking Water Advisory:
Consumer Acceptability Advice and Health
Effects Analysis on
Methyl Tertiary-Butyl Ether (MtBE)**

December 1997

U.S. Environmental Protection Agency
Office of Water
EPA-822-F-97-008

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LIST OF ABBREVIATIONS

DWEL	Drinking-Water-Equivalent-Level
HA	Health Advisory
kg	kilogram
L	liter
LOAEL	lowest-observed-adverse-effect level
MoE	margin of exposure
mg	milligram
MtBE	Methyl <i>tertiary</i> -butyl ether
MTD	Maximum Tolerated Dose
NOAEL	no-observed-adverse-effect level
OFW	odor free water
ppm	parts per million
μ g	microgram
TBA	<i>tertiary</i> -butyl alcohol
VOC	volatile organic compound

FOREWORD

EPA's Human Health and Criteria Division (HECD) of the Office of Water developed an Advisory document for methyl *tertiary*-butyl ether (MtBE). This document is a non-regulatory document that analyses the currently available cancer and non-cancer data on this contaminant, as well as studies on its organoleptic (taste and odor) effects. The document is not a mandatory standard for action; however, this Advisory supersedes any previous drafts of drinking water advisories for this chemical.

There are many uncertainties and limitations associated with the toxicity data base for this chemical. The animal tests available to date (1997) were not conducted by exposing the animals to MtBE in drinking water, but rather by inhalation exposure or by introducing MtBE in oil directly to the stomach several times a week. Although useful for identifying potential hazards, limitations of the reported studies do not allow confident estimates of the degree of risk MtBE may pose to humans from low-level drinking water contamination. The toxicokinetic models are also limited in helping to perform an adequate extrapolation from the inhalation data to actual oral exposure from drinking water intake. Additional research is needed to resolve these issues before a more complete health advisory can be issued. Therefore, given the needs of the States and Regions for an Office of Water (OW) position on MtBE contamination of drinking water, HECD developed this "Drinking Water Advisory: Consumer Acceptability Advice and Health Effects Analysis on Methyl *tertiary*-Butyl Ether (MtBE)".

MtBE is generally unpleasant in taste and odor. Studies have been conducted on the concentrations of MtBE in drinking water at which individuals can detect the odor or taste of the chemical. This Advisory recommends that keeping levels of contamination in the range of 20 to 40 $\mu\text{g/L}$ or below to protect consumer acceptance of the water resource would also provide a large margin of exposure (safety) from toxic effects.

The Advisory discusses the limitations of the current database for estimating a risk level for this contaminant in drinking water and characterizes the hazards associated with this route of exposure. This document has been peer reviewed both internally in the Agency and externally by experts in the field before its release to the public.

Note: In this Advisory, we use a risk characterization method called "Margin of Exposure (or safety)" which is different from traditional slope factors and reference doses (RfDs) as estimates of response to defined exposures. The "margin" is how far the environmental exposure of interest is from the lower end of the exposures at which animals or humans have shown some toxicity effect. The use of the margin of exposure approach is helpful in the following ways: 1. It allows for comparison of exposures associated with carcinogenic potential to those associated with non cancer health effects; 2. It provides the risk manager with a quick check to decide if the margin of exposure (safety) appears to be adequate even when mathematical extrapolation of

data from high to low dose cannot be done; and 3. It gives a better understanding of the degree of risk

associated with extrapolation of exposure data from animal studies to humans. For example, given the limited number of animals that usually can be used in experiments, they, at best, would detect a one in ten response (1×10^{-1}). A common procedure for carcinogens is to mathematically extrapolate from the exposure levels of animal tests to estimate risk at lower, environmental exposure levels. If the extrapolation is done as a straight line, a risk estimate of 1×10^{-6} generally corresponds to a margin of exposure of 100,000. If the true, but unknown, relationship is downward sloping, not a straight line, the risk at a 100,000 margin of exposure would be less than 1×10^{-6} and might be zero.

Health and Ecological Criteria Division
Office of Science and Technology
Office of Water

DRINKING WATER ADVISORY: CONSUMER ACCEPTABILITY ADVICE AND
HEALTH EFFECTS ANALYSIS ON
METHYL TERTIARY-BUTYL ETHER (MtBE)

EXECUTIVE SUMMARY

MtBE

MtBE is a volatile, organic chemical. Since the late 1970's, MtBE has been used as an octane enhancer in gasoline. MtBE promotes more complete burning of gasoline, thereby reducing carbon monoxide and ozone levels. Hence, MtBE is commonly used as a gasoline additive in localities that participate in the Winter Oxygenated Fuels program and/or the Reformulated Gasoline program to achieve or maintain compliance with the National Ambient Air Quality Standards. A limited number of instances of significant contamination of drinking water with MtBE have occurred due to leaks from underground and above ground petroleum storage tank systems and pipelines. MtBE, due to its small molecular size and solubility in water, moves rapidly into groundwater, faster than other constituents of gasoline. Public and private wells have been contaminated in this manner. Non-point sources, such as recreational watercraft, are most likely to be the cause of small amounts of contamination of surface waters. Air deposition through precipitation of industrial or vehicular emissions may also contribute to surface and ground water contamination. The extent of any potential for build-up in the environment from such deposition is uncertain.

This Advisory

The EPA Office of Water is issuing this Advisory to provide guidance for communities that may be exposed to drinking water contaminated with MtBE. The Advisory provides an analysis of current health hazard information and an evaluation of currently available data on taste and odor problems associated with MtBE contamination of water, as the latter affect consumer acceptance of the water resource. This Advisory does not recommend either a low-dose oral cancer risk number or a reference dose (RfD)¹ due to certain limitations of available data for quantifying risk. Guidance is given on the concentrations at which taste and odor problems likely would be averted, and how far these are from MtBE concentrations at which toxic effects have been seen

¹Reference Dose is defined as "an estimate (with uncertainty spanning approximately an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without appreciable risk of deleterious effects over a lifetime" (U.S. EPA, 1987).

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in test animals. (The measure used is called a "margin of exposure" or MoE. For instance, if a measured concentration is 100,000 times less than the range of observation of effects in test animals, the margin of exposure is 100,000.

Conclusion and Recommendation

This Advisory recommends that keeping levels of contamination in the range of 20 to 40 $\mu\text{g/L}$ or below to protect consumer acceptance of the water resource would also provide a large margin of exposure (safety) from toxic effects.

Taste and odor values are presented as a range, since human responses vary depending upon the sensitivities of the particular individual and the site-specific water quality conditions. These values are provided as guidance recognizing that water suppliers determine the level of treatment required for aesthetics based upon the customers they serve and the particular site-specific water quality conditions.

There are over four to five orders of magnitude between the 20 to 40 $\mu\text{g/L}$ range and concentrations associated with observed cancer and noncancer effects in animals. There is little likelihood that an MtBE concentration of 20 to 40 $\mu\text{g/L}$ in drinking water would cause adverse health effects in humans, recognizing that some people may detect the chemical below this range. It can be noted that at this range of concentrations, the margins of exposure are about 10 to 100 times greater than would be provided by an EPA reference dose (RfD) for noncancer effects. Additionally, they are in the range of margins of exposure typically provided by National Primary Drinking Water Standards under the Federal Safe Drinking Water Act to protect people from potential carcinogenic effects.

When adequate data become available, the Office of Water will publish another Advisory that includes quantitative estimates for health risks. This Advisory gives practical guidelines for addressing contamination problems and supersedes previous draft advisories. An Advisory does not mandate a standard for action.

Studies of MtBE Effects

There are no studies of effects on humans of long-term exposure to MtBE. All of the studies available for hazard assessment are laboratory animal studies.

Cancer effects. There are studies in rodents of the carcinogenicity of MtBE, as well as its metabolites, *tertiary*-butyl alcohol (TBA) and formaldehyde. The only oral cancer exposure study was conducted by Belpoggi and coworkers (1995). They gave MtBE to Sprague-Dawley rats (gavage in olive oil, at doses up to 1,000 mg/kg/day, 4 days per week for two years). Exposure caused a dose-related increase in the incidence of combined leukemia and lymphomas in the female rats and an increase in Leydig cell adenomas (benign testicular tumors) in the high-dose male rats. Use of this study to quantitatively assess risks from drinking water exposure has

limitations. There are potential differences in bolus versus drinking water exposures and possible vehicle (olive oil) effects. Moreover, there are few details on the actual reported tumor response data provided in the report. The lack of histopathological diagnoses and of individual animal data were reasons that the National Research Council panel recommended not using these tumor data in risk estimation until after a thorough peer review of this study.

There are two studies on the potential carcinogenicity of MtBE after inhalation exposure. Chun et al. (1992) administered MtBE to F344 rats at concentrations up to 8,000 ppm for 2 years. Exposure to MtBE caused an increase in the incidence of combined renal tubular adenomas and carcinomas, as well as Leydig cell adenomas of the testes in the male rats. The mild induction of α -2u-globulin by MtBE suggested that this protein may have played a role in male rat kidney tumorigenesis. The increase in the incidence of Leydig cell adenomas of the male rats in this study was not significantly different from the historical control value, although the difference from the concurrent controls was significant. Induction of Leydig cell tumors was also observed in Sprague-Dawley rats after oral exposure by gavage (Belpoggi et al., 1995) and lends support to the conclusion that the appearance of the tumor in both studies is treatment-related.

In the other inhalation study, Burleigh-Flayer et al. (1992) gave MtBE to CD-1 mice at concentrations up to 8,000 ppm for 18 months. This exposure was associated with a statistically significant increase in the incidence of hepatocellular carcinomas in male mice and of hepatocellular adenomas in female mice. The Chun et al. (1992) and the Burleigh-Flayer et al. (1992) studies currently cannot be used to calculate adequate hazard advisory values since we have no well-developed pharmacokinetic model for converting a chronic inhalation exposure of MtBE to an equivalent oral exposure. On-going work may support route-to-route extrapolation in the future.

The potential carcinogenicity of two metabolites of MtBE, TBA and formaldehyde has also been examined. In F344 rats, TBA has provided some evidence of carcinogenic activity in the males (but not in the female rats). In B6C3F1 mice, TBA exposure gave equivocal evidence of carcinogenic activity in male mice based on marginally increased incidence of thyroid tumors, and some evidence of carcinogenicity in female mice, based on an increased incidence of follicular cell hyperplasia and follicular cell adenomas of the thyroid gland. Data for carcinogenic activity is ambiguous for drinking water exposure to formaldehyde. A study by Soffritti et al. (1989) reported a dose-related increase in the incidence of leukemia and intestinal tumors in Sprague-Dawley rats. However, the experimental data presented in this publication was limited. Another drinking water study on formaldehyde by Til and coworkers (1989), using Wistar rats, found no evidence of carcinogenicity.

The carcinogenicity data support a conclusion that MtBE poses a potential for carcinogenicity to humans at high doses. The data do not support confident, quantitative estimation of risk at low exposure due to the limitations described above.

Noncancer toxicity. The collective evaluation of the reproductive and developmental studies of MtBE in animals indicate that inhalation exposure can result in maternal toxicity and adverse effects on the developing fetus (Bushy Run Research Center, 1991, 1989a, 1989b; Conaway et al., 1985). The fetal toxicity in the mouse developmental studies indicate that it may be more sensitive to inhalation of MtBE vapors than the rat or rabbit during gestation. However, it is possible to conclude that, at low concentrations, MtBE does not cause a developmental or reproductive hazard by inhalation in three different animal species. This also suggests that humans may not be at risk when exposed to very low concentrations of MtBE.

Effects on the kidney were observed in rats after oral and inhalation exposure to MtBE. The most pertinent noncancer toxicity data come from a 90-day oral exposure study in rats. The authors reported minimal effects on the kidneys at doses of 300 mg/kg/day and above (Robinson et al., 1990). In these animals, the MtBE was given once a day, as a bolus dose in corn oil. A single oral dose of MtBE in corn oil would not be considered representative of an intermittent exposure to MtBE that one would normally obtain from drinking water containing MtBE. In a longer term inhalation study, histopathological abnormalities were apparent (Chun et al., 1992). Uncertainties exist in quantifying risk from the oral data in the short-term study because of the bolus gavage dosing regime and the less-than-lifetime duration of the study. The uncertainty in extrapolating between routes affects the interpretation of the inhalation data.

The studies support a conclusion that MtBE can pose a hazard of noncancer effects to humans at high doses. The data do not support confident quantitative estimation of risk at low exposure.

Taste and Odor. Studies were conducted on the concentrations of MtBE in drinking water at which individuals respond to the odor or taste of the chemical. Human responses vary widely in this respect. Some who are sensitive can detect very low concentrations, others do not taste or smell the chemical even at much higher concentrations. Moreover, the presence or absence of other natural or water treatment chemicals can mask or reveal the taste or odor effects. Thus, variable preexisting water conditions around the country will increase variability in the acceptability of MtBE's presence in drinking water.

The studies have not been extensive enough to completely describe the extent of human variability, or to establish a population threshold of response. Nevertheless, the available studies allow a conclusion that keeping concentrations in the range of 20 to 40 micrograms per liter ($\mu\text{g/L}$) of water or below will likely avert unpleasant taste and odor effects, recognizing that some people may detect the chemical below this range.

Characterization Summary

Section 7.0 on hazard and dose response characterization summarizes the MtBE data. In this section, a table (Table 1) presents the margins of exposure comparing animal effects and human taste and odor data.

1.0 INTRODUCTION

The purpose of this Advisory is to support immediate needs for information by State and local drinking water facilities and public health personnel due to MtBE contamination of potable water. Ongoing research is anticipated to decrease some of the uncertainties in the current toxicity data as applied to the drinking water route of exposure. A Health Hazard Advisory value will be issued when the data base is improved to allow greater confidence in the toxicity conclusions. Nevertheless, there are sufficient data to give a general picture of the ranges of exposure that may raise concerns for people. In addition, the taste and odor of MtBE affect the potability of water at levels that provide an additional basis for assessment of quality and usability of water resources.

2.0 MtBE IN THE ENVIRONMENT

MtBE is used as an octane enhancer to replace lead in gasoline. It also promotes more complete burning of gasoline, thereby reducing carbon monoxide and ozone levels in localities which do not meet National Ambient Air Quality Standards (ATSDR, 1996; USGS, 1996). Almost all of the MtBE produced is used as a gasoline additive; small amounts are used by laboratory scientists (ATSDR, 1996). When used as a gasoline additive, MtBE may constitute up to 15% (v/v) of the gasoline mixture. MtBE production in the United States was estimated at 6.2 billion kilograms in 1994 and 21 billion kilograms in 1995 (NSTC, 1997 and 1996).

In the Clean Air Act of 1990 (Act), Congress mandated the use of reformulated gasoline (RFG) in those areas of the country with the worst ozone or smog problems. RFG must meet certain technical specifications set forth in the Act, including a specific oxygenate content. Ethanol and MtBE are the primary oxygenates used in the RFG program to meet the oxygen content requirement. MtBE is used in about 84% of RFG supplies. Currently, 32 areas in a total of 18 states are participating in the RFG program, and RFG accounts for about 30% of the gasoline nationwide. Studies have identified significant air quality and public health benefits that directly result from the use of oxygenated fuels. The refiners' 1995/96 fuel data submitted to EPA indicate that the national emissions benefits exceeded the required reductions. The 1996 Air Quality Trends Report showed that toxic air pollutants, such as benzene, a known carcinogen, declined significantly between 1994 and 1995. Early analysis indicates this progress may be attributable to the use of RFG. Starting in the year 2000, the required emission reductions are substantially greater, at about 27% for VOCs, 22% for toxics, and 7% for NOX.

About 40% of the U.S. population live in areas where MtBE is used (USGS, 1996). MtBE is a volatile chemical; therefore, in most areas, the major exposure to MtBE is from air. In some instances, drinking water sources may be contaminated. Leaking underground storage tank systems and pipelines for gasoline products are the cause of reported ground water contamination. According to the Toxic Chemical Release Inventory published in 1995,

approximately 3% of the MtBE released from industrial sources enters surface water or publicly-owned treatment plants (ATSDR, 1996). Surface waters can also become contaminated as noncombusted MtBE in gasoline is released into air and precipitated by rain and snow.

Unlike most gasoline components, MtBE is a small, highly water-soluble molecule. Therefore, it does not bind strongly to soils, but travels relatively rapidly to and through surface and underground water. In addition, MtBE appears to be resistant to chemical and microbial decomposition in water (ATSDR, 1996).

MtBE has been reported in ground water and drinking water derived from ground water. Based on monitoring data collected by the U.S. Geological Survey (USGS), it appears that wells most susceptible to contamination are shallow ground water wells in urban areas (USGS, 1996). There is limited MtBE drinking water occurrence information. The information available is insufficient to characterize the extent of drinking water contamination on a nationwide basis, because the samples collected are generally from locations with known or suspected contamination (NSTC, 1996).

In air, MtBE may represent 5-10% of the volatile organic compounds that are emitted from gasoline-burning vehicles, particularly in areas where MtBE is added to fuels as part of an oxygenated fuel program (ARCO, 1995). There are no reliable data on MtBE levels in food, but food should not be a significant source of exposure to MtBE. Limited data suggest that MtBE will not bioaccumulate in fish or food chains (ATSDR, 1996).

The recent report of the National Science and Technology Council (NSTC, 1997) provides extensive occurrence data for MtBE and other fuel oxygenates, as well as information on applicable treatment technologies. For additional information concerning MtBE in the environment, this report can be accessed through the NSTC Home Page via link from the Office of Science and Technical Policy (OSTP) at the following address:

Home Page at: http://www.whitehouse.gov/WH/EOP/OSP/html/OSTP_Home.html.

Information on analytical methods for determining MtBE in environmental media are compiled in the ATSDR Toxicological Profile (1996) for this chemical.

3.0 CHEMICAL AND PHYSICAL PROPERTIES

MtBE is an aliphatic ether. It is a colorless liquid with a characteristic odor. It has a low molecular weight (88.15 g/mole), high volatility (vapor pressure 245 mm Hg at 25° C), and high water solubility (40-50 g/L; ATSDR, 1996). In its liquid or gaseous state, it is expected to be readily absorbed into the blood stream. It is moderately lipophilic with a log K_{ow} of 1.24 (ATSDR, 1996), which will facilitate its absorption across the lipid matrix of cell membranes.

4.0 TOXICOKINETICS

There are no data on the absorption of MtBE in humans after ingestion; the uptake of MtBE via inhalation has been reported to be rapid (Cain et al., 1994; Prah et al., 1994; Johanson et al., 1995). In animals, absorption of MtBE administered by oral, intraperitoneal, or inhalation routes is rapid and extensive (Industrial Bio-Test Laboratories Inc., 1972a,b; Bio/dynamics, 1984; Savolainen et al., 1985; Bio-Res Lab., 1990a,b,c,d; Miller et al., 1997). The extent of dermal absorption in rats is slow and limited, but increases with increasing dose levels (Bio-Res Lab., 1990a,b).

The metabolism and elimination of MtBE and its metabolites also proceed rapidly regardless of the route of administration. After absorption, MtBE is demethylated to form TBA and formaldehyde by the *O*-demethylase of the microsomal cytochrome P-450 system (Brady et al., 1990). TBA is further metabolized to formaldehyde (in rodents) or conjugated with glucuronic acid to form TBA-glucuronide, which is excreted in urine (Cederbaum and Cohen, 1980; Williams, 1959). Other oxidative metabolites of TBA include 2-methyl-1,2-propanediol and alpha-hydroxy isobutyric acid (Bio-Res Lab., 1990b; Miller et al., 1997). Formaldehyde may be reduced to methanol or oxidized to formic acid, which is further biotransformed to carbon dioxide.

Since MtBE is rapidly absorbed into the circulation from inhalation and ingestion exposures, it is expected that MtBE is distributed to all major tissues. A large fraction of the MtBE in blood has a very short half-life of 10-30 minutes. The minor long-term exponential decay component in humans exposed to MtBE via inhalation suggests that a small amount of MtBE can deposit in the tissues (Prah et al., 1994; Johanson et al., 1995). Animal studies showed that 24-96 hours after single short-term exposures, the total residual levels in various tissues (brain, muscle, skin, fat, liver, and kidney) were, in general, low regardless of route of exposure (Industrial Bio-Test Laboratories Inc., 1972a, 1972b; Bio/dynamics, 1984; Savolainen et al., 1985; Bio-Res Lab., 1990a,b,c,d; Miller et al., 1997). Several investigators (Borghoff et al., 1996; Rao and Ginsberg, in press) are developing toxicokinetic models to derive concentrations in blood and brain for rodents and humans after short-term exposure. These models will be reviewed before being used for route-to-route extrapolation, especially when exposures are repeated or continuous.

4.1 Dosimetry: Route-to-Route Extrapolation

While there are few reports available on the effect of MtBE via ingestion, there are many on inhalation exposure. Attempts have been made to crudely extrapolate inhalation dose-response to an equivalent oral dose-response to offer a perspective on the possible oral hazard/risk suggested by the inhalation data given that the available direct oral data are so limited. In so doing, one must convert the inhalation dose to units of mg/kg-day, determine what assumptions are reasonable for extrapolating this to an equivalent oral exposure in mg/kg-day, and then

calculate a related oral potency (slope factor) using the calculated oral dose and the inhalation response.

There are several inherent uncertainties or limitations involved in the estimation of human equivalent oral dose from animal inhalation data. Factors that impact absorption from the lungs and thus dose include: 1) the physical properties of the chemical (e.g., aerosol or gas, including the particle size), 2) respiration rate and minute volume of the experimental animal, and 3) exposure conditions (continuous vs. intermittent exposures). Factors that impact the interspecies aspects of the conversion are: 1) allometric scaling between species to compensate for different body sizes, 2) differences in respiratory system structure and physiology, and 3) the qualitative and quantitative differences in absorption and biotransformation between species.

Another important uncertainty in the extrapolation is in establishing whether the parent toxicant or its metabolite(s) is responsible for the biological activity. The absorbed dose via inhalation exposure does not go through the same liver metabolism (the first-pass effect) as that via ingestion. Many chemicals (e.g. formaldehyde) produce different toxic and carcinogenic effects via different routes of exposure. This means that it is important to determine whether it is the parent compound or a metabolite that is responsible for the observed effects. Specific uncertainties and limitations in the toxicokinetic data for MtBE are discussed below.

Most of the absorption data on MtBE were collected following short-term inhalation exposure. Duration of exposure and the rate of respiration are two very important parameters which control the absorption of MtBE. During the exposure period, a state of equilibrium is established between the inhaled and exhaled air; therefore, the percent absorbed dose by inhalation is influenced by the pharmacological properties of the toxicant. For example, substances like MtBE with an anesthetic effect at higher dose will slow down the respiratory rate and, thereby, slow down the rate of absorption via the lungs into the blood. Accordingly, overall absorption of MtBE would be anticipated to be lower at a higher dose because of its effect on the central nervous system. There is not enough information to estimate the exact absorbed dose in long-term inhalation or oral exposure.

As already mentioned, via the inhalation route, MtBE enters the blood without passing through the gastrointestinal tract and the liver which is responsible for most of MtBE metabolism by way of the hepatic cytochrome P-450 system. To what extent MtBE metabolism is influenced by the gastrointestinal tract is not known. It is likely that differences in the metabolism between exposure routes do occur and affect toxicity. Using inhalation exposure to estimate the oral dose ignores potential first-pass effects in the liver. However, the uncertainties in the route-to-route extrapolation of dose for MtBE are mitigated by the fact that the metabolites qualitatively appear to be the same by differing routes, the distribution and excretion patterns are the same and the tissues in which toxicity, including carcinogenicity, have been reported overlap between routes.

4.2 NSTC's Extrapolation of Dose from Inhalation Exposure

A number of the studies utilized for this Advisory involved the inhalation route of exposure. At present, there is no appropriate toxicokinetic model to convert an applied inhalation exposure concentration to a dose in the target organ, although models are under development at CIIT (Borghoff et al., 1996) and the University of Connecticut (Rao and Ginsberg, in press). In the absence of a well-developed toxicokinetic model, the inhalation exposure concentrations were converted to dose values following the method used by the interagency task force on MtBE (NSTC, 1996; 1997). The NSTC (1996) conversion method assumes that for a given exposure concentration of MtBE, the adjusted external human equivalent dose would be the same from studies of any kind of animals, regardless of the species used. The calculation also assumes 100% absorption of MtBE, and appears to be a default value in the absence of reliable inhalation and absorption data.

The equation used for the dose conversion by the NSTC (1997) is presented as follows:

$$\text{Human Equivalent Dose (HED)} = \frac{C \text{ ppm} \times 10^{-6} \text{ ppm}^{-1} \times \text{MM} \times \text{RR} \times \text{EC}}{\text{MV} \times \text{BW}}$$

Where:

- C = Atmospheric concentration
- MM = Molar mass expressed in milligrams (88,150 mg for MtBE)
- MV = Molar volume at 20°C (24.04 L)
- RR = Human respiration rate (20,000 L/day)
- EC = Exposure condition (# hrs/24 hr) x (# days/week)
- BW = Average human body weight (70 kg)

The value of 10^{-6} ppm^{-1} in the equation is a unit adjustment factor that expresses the amount of the contaminant that is present in each unit of inspired air.

When the concentration of MtBE is 1 ppm, the exposure condition is continuous (24 hrs/day and 7 days per week), the EC is 1 and the HED is calculated as 1.05 mg/kg-day as follows:

$$\text{HED} = \frac{1 \text{ ppm} \times 10^{-6} \text{ ppm}^{-1} \times 88,150 \text{ mg} \times 20,000 \text{ L/day}}{24.04 \text{ L} \times 70 \text{ kg}} = 1.05 \text{ mg/kg/day}$$

In cases where exposures are conducted for 6 hrs/day and 5 days per week, the EC is equal to (6/24) (5/7) or 0.1786. Consequently, 1 ppm of MtBE is equivalent to 0.1875 mg/kg-day.

The Office of Water has presented the NSTC (1997) methodology for extrapolation of the inhalation exposure doses to oral doses in studies with MtBE in order to be consistent with the risk assessment values of those provided in the NSTC (1997) report. The limitations of the methodology generate significant uncertainties.

5.0 HEALTH EFFECTS DATA

5.1 Human Studies

There are very limited data on the effects of MtBE in humans by any route of exposure and no data are available for the oral route. In cases where 37 or 43 human volunteers were exposed to low levels of MtBE in air (1.39 or 1.7 ppm) for 1 hour (Cain et al., 1994; Prah et al., 1994), there was no significant increase in symptoms of eye, nasal, or pulmonary irritation when the results for periods of exposure to MtBE were compared to results from exposure to ambient air. There were also no significant effects on mood (determined by the Profile of Mood States test) or in the results from several performance-based neurobehavioral tests. In both studies, the females ranked the quality of the air containing MtBE lower than the control atmosphere. However, in the study by Cain et al. (1994), where the subjects were also exposed to an atmosphere containing a 7.1 ppm mixture of 17 volatile organic compounds (VOCs) that are frequent air contaminants in areas around gasoline stations, the air quality of the MtBE-containing atmosphere ranked higher than that with the VOC mixture.

The results from studies of neurological effects (headache, dizziness, disorientation, fatigue, emotional distress, etc.), gastrointestinal problems (nausea, diarrhea), and symptoms of respiratory irritation in individuals exposed to MtBE vapors through MTBE-containing fuels are inconclusive (Hakkola et al., 1996; Moolenaar et al., 1994; White et al., 1995). The three studies cited were different in their design and utilized slightly different parameters for monitoring effects. All studies evaluated exposure to a MtBE-gasoline mixture and not MtBE.

The studies by Hakkola et al. (1996) and White et al. (1995) compared the effects in two groups exposed to different concentrations of MtBE from treated gasoline because of their lifestyles. The moderately-exposed individuals either drove a gasoline delivery truck, worked in a gasoline station or worked on car repairs. The minimally-exposed individuals merely used a gasoline-powered vehicle to go to and from work or as part of their job. Hakkola et al. (1996) found that there were no statistically-significant differences between the signs and symptoms reported by 101 drivers of tanker trucks in Finland (where the gasoline contains 10% MtBE) and 100 milk truck drivers. Blood concentrations of MtBE or its metabolites were not monitored. In the study by White et al. (1995), the odds ratio was 8.9 (95% CI = 1.2-75.6) for the reporting of one or more symptoms when 11 individuals with blood MtBE levels of $>2.4 \mu\text{g/L}$ were compared with 33 individuals with lower levels. The odds ratio increased to 21 (95% CI = 1.8-539) when commuters were excluded from the population studied and 8 workers with blood levels of >3.8

$\mu\text{g/L}$ were compared to 22 individuals with lower blood MtBE levels. All individuals lived and worked in the area around Stamford, Connecticut.

A study in Alaska (Moolenaar et al., 1994) compared effects and blood levels of MtBE from a time period when oxygenated fuels were in use (Phase I) to those after the oxygenated fuels use had stopped (Phase II). The subjects were volunteers who were occupationally exposed to motor vehicle exhaust or gasoline fumes. Eighteen workers participated in Phase I and 22 in Phase II. Twelve of those that participated in Phase I of the study also participated in Phase II. A questionnaire was used to gather information on signs and symptoms and blood samples were collected for measurement of MtBE at the beginning and end of a typical work day. In Phase I, the median post-shift MtBE level was higher than the pre-shift value (1.80 vs. 1.15 $\mu\text{g/L}$). During Phase II, the values were more comparable (0.25 vs. 0.21 $\mu\text{g/L}$). Median post-shift blood measurements of TBA were higher during Phase I than in Phase II (5.6 vs. 3.9 $\mu\text{g/L}$).

Signs and symptoms that could be associated with MtBE exposure were reported more frequently during Phase I than Phase II (Moolenaar et al., 1994). During Phase I, 50% or more of the participants reported headaches, eye irritations and nose and throat irritations. Reporting of these symptoms occurred in less than 10% of the participants during Phase II. However, it is difficult to evaluate if psychosomatic factors and individual sensitivity had influenced these results. The volunteers may have chosen to participate because of their sensitivity to contaminants in the atmosphere.

Perfusion of MtBE through the bile duct and gallbladder was once used as a medical treatment for gallstones. During this procedure, some of the MtBE enters the blood stream and is distributed systemically. Effects reported in patients treated by this procedure included sedation, perspiration, bradycardia (slow heart beat) and elevation of liver enzymes (Allen et al., 1985; Juliani et al., 1985, and Wyngaarden, 1986). These signs cannot be attributed totally to MtBE because of the confounding effects of anesthesia and the infusion process itself.

5.2 Animal Studies

5.2.1 Noncancer Effects

5.2.1.1 Acute and Subchronic

Studies of the systemic effects of MtBE have been conducted in animals, but the majority involve inhalation exposure. Since this Provisional HA is mainly concerned with the effects of MtBE in drinking water, it will focus on oral toxicity studies. From an acute standpoint, MtBE is not very toxic. The oral LD_{50} in rats is 3.9 g/kg (3,900 mg/kg). Treated animals exhibit central nervous system depression, ataxia and labored breathing (ARCO, 1980).

In a two-week study, Sprague-Dawley rats (10/sex/dose) were dosed daily with MtBE in corn oil by gavage at 0, 537, 714, 1,071 or 1,428 mg/kg/day. At the highest dose, anesthesia was immediate, but recovery was complete within two hours. Although there was a dose-related decrease in body weight gain, it was significant only in females at the highest treatment regimen. Increases in relative kidney weights were noted in the males at 1,071 and at 1,428 mg/kg/day and in females at the 1,428 mg/kg/day dose. There were no gross lesions seen at any treatment level. Based on the increases in relative kidney weight, a No-Observed-Adverse-Effect-Level (NOAEL) of 714 mg/kg/day and a Lowest-Observed-Adverse-Effect-Level (LOAEL) of 1,071 mg/kg/day are established by these experiments (Robinson et al., 1990).

Sprague-Dawley rats (10/sex/dose) were treated orally with MtBE in corn oil for 90-days at 0, 100, 300, 900 or 1,200 mg/kg/day. Anesthesia was evident at the highest dose, but as in the 14-day study, full recovery occurred in two hours. There was a significant decrease in final body weight of females only at the highest level of treatment. The diarrhea seen in the treated animals was considered to be the consequence of the bolus dosing regime. In females, there were increases in relative kidney weights at 300, 900 and 1,200 mg/kg/day, while in males, increases were noted only at the two highest treatment levels. Reductions in blood urea nitrogen, serum calcium and creatinine were observed in males and a reduction in cholesterol in females was reported, but there were no clear dose-dependent results. Based on the alterations in kidney weights, a NOAEL and LOAEL of 100 and 300 mg/kg/day, respectively, are identified by this study (Robinson et al., 1990).

Sprague-Dawley rats (60 animals per sex, per dose group) were given 0, 250 or 1,000 mg/kg/day MtBE in olive oil via gavage, 4 days per week, for 104 weeks. This dosing regimen gives a 7-day time-weighted average daily dose of 0, 143 and 571 mg/kg/day. Survival appeared to be decreased in female rats after 16 weeks, but no statistical treatments on data were reported. There was no reporting of hematological, clinical chemistry or urinalysis parameters, or any indication as to whether or not these endpoints were evaluated. The authors did not observe any differences in food consumption or final body weights in the various groups. In addition, they did not report any noncancer histopathological changes (Belpoggi et al., 1995). Due to the limited scope, intermittent treatment schedule and scant data reporting in this study, it is not possible to set a NOAEL or LOAEL.

The subchronic data from the study by Robinson et al. (1990) were used to develop a DWEL for kidney effects from MtBE. The increase in kidney weights at doses of 300 mg/kg/day and higher was considered to be an adverse effect, since increases in organ weights are a marker for adverse organ effects (Weil, 1970). The diarrhea observed was considered to be a gastrointestinal complication of the gavage dosing. Based on the NOAEL of 100 mg/kg/day, a DWEL for kidney effects of 3,500 $\mu\text{g/L}$ can be derived for a 70 kg adult drinking 2 L of water per day, using an uncertainty factor of 1,000. The uncertainty factor reflects a 10 for the less-than-

lifetime duration of the study, a 10 for interspecies variability and a 10 for intraspecies variability.

Kidney toxicity was also observed in both males and females in the 2-year inhalation study in F344 rats by Chun et al. (1992) discussed in the section on cancer effects. In fact, EPA derived a Reference Concentration of 3 mg/m³, based on the kidney and liver effects of MtBE (U.S. EPA, 1993). These data support the conclusion that, after MtBE exposure, kidney toxicity is of concern. However, the use of the Robinson et al. (1990) study for evaluation of kidney effects has two significant uncertainties. One is that the study was for 90 days and not for a lifetime, and the second is the extrapolation of dose from a single daily bolus dose in corn oil to the continuous small doses from drinking water exposure. In general, it would be anticipated that a 90-day exposure period would tend to underestimate the toxicity, while the bolus dose would be more likely to overestimate the toxic response. However, the relative effects of these two factors are uncertain.

5.2.1.2 Reproductive and Developmental Studies

Reproductive Studies

Two inhalation studies in rats were available on the reproductive effects of MtBE. A two-generation reproduction study was conducted in Sprague-Dawley CD rats using target concentrations of 0, 400, 3,000 or 8,000 ppm of MtBE for 6 hours/day, 5 days/week for 10 weeks before mating, during mating, gestation and lactation days 5-21 (Bushy Run Research Center, 1991; Bevan et al., 1997b). Statistically-significant reductions in body weight and body weight gains in male and female F₁ and F₂ pups were noted with the 3,000 ppm and 8,000 ppm exposures during the latter periods of lactation. At 3,000 ppm, only transient body weight reductions were noted in F₁ males and females during their pre-mating period. At 8,000 ppm, pup survival was significantly reduced ($p < 0.01$) in the F₁ litters on lactation days 0-4 and in F₂ litters on postnatal day 4. Clinical signs of toxicity were noted in both generations at 3,000 and 8,000 ppm; this included hypoactivity and lack of startle reflex. Ataxia and blepharospasm (eyelid twitching) were observed at 8,000 ppm. At necropsy, increased liver weights were reported in the F₁ generation at 3,000 and 8,000 ppm in both sexes, although no histopathological effects were noted. The NOAEL and LOAEL for both parental and pup toxicity were 400 and 3,000 ppm, respectively.

A one-generation study (Biles et al., 1987) in Charles River CD rats was carried out with two matings, using target concentrations of 0, 300, 1,300 or 3,400 ppm of MtBE vapor for 6 hours/day, 5 days/week, prior to and during mating. Exposure was continued during 5-day mating intervals. In males, exposure continued until the end of the second mating to produce the F_{1b} litters. In females, exposure continued during the gestation period and lactation days 5 to 21, but not during the first 4 days of the lactation period. A NOAEL and a LOAEL may be

identified at 300 ppm and 1,300 ppm, respectively, based on pup viability in the F_{1b} litters. However, this study has limited usefulness in the evaluation of reproductive toxicity because of some noted flaws (e.g., the loss of one entire litter of 12 pups at birth in the mid-dose group remains unexplained).

Developmental Studies

Four inhalation studies were evaluated: one in rats (Conaway et al., 1985), two in mice (Conaway et al., 1985; Bushy Run Research Center, 1989a; Bevan et al., 1997a) and one in rabbits (Bushy Run Research Center, 1989b; Bevan et al., 1997a). The Conaway et al. studies in the rat and mouse were performed at target concentrations of 0, 250, 1,000 or 2,500 ppm of MtBE for 6 hrs per day on days 6 to 15 of gestation. Dams were sacrificed at gestation day 20 for rats and gestation day 18 for mice. The concentrations for the Bushy Run studies in mice and rabbits were 0, 1,000, 4,000 ppm or 8,000 ppm. Mice were exposed on days 6 to 15 of gestation and rabbits were exposed on days 6 to 18 of gestation. Mice dams were sacrificed on gestation day 18 and rabbits on gestation day 28.

In the rat study (Conaway et al., 1985), no effects were noted in rats at the highest dose tested, 2,500 ppm. Also, in the rabbit study (Bushy Run Research Center, 1989b; Bevan et al., 1997a), no developmental toxicity was noted at the highest dose tested, 8,000 ppm, but maternal toxicity was noted at 4,000 ppm and above.

For mice, in the Bushy Run study, maternal toxicity was noted at the two higher concentrations (4,000 ppm and 8,000 ppm). Also, fetal skeletal variations and reduction in fetal weight were noted at the higher doses. In the Conaway et al. (1985) mouse study, the most noted developmental effect was a dose-related increase in the incidence of skeletal malformations per litter with incidence of 7.4 percent in the control group compared to 11.5 percent, 16 percent and 22.2 percent in the 250, 1,000 and 2,500 ppm groups, respectively. These malformations included cleft palate, scrambled and fused sternebra and angulated ribs. Cleft palate occurred in two fetuses of one litter in the control group; one fetus in the 1,000 ppm group; two fetuses, each in a different litter of the 2,500 ppm group; and none in the 250 ppm group. There were also 17, 11 and 17.3 percent resorptions in the 250, 1,000 and 2,500 ppm groups, respectively, compared to 9 percent in control. Based on the incidence of skeletal malformations in these two mice studies, a developmental NOAEL in mice can be projected in the range of 250 ppm to 1,000 ppm.

The collective evaluation of the two developmental mouse studies discussed above reflects a NOAEL in the range of 250 to 1,000 ppm for developmental toxicity. The NOAEL of 400 ppm for parental toxicity in the rat two-generation reproductive study falls within the NOAEL range for developmental effects. These values are projected as equivalent to doses of 65.6 mg/kg/day to 262.5 mg/kg/day, respectively. Using these two values, the projected, no-effect-concentration

in drinking water for humans is in the range of 2.3 to 9.2 mg/L (2,300 to 9,200 $\mu\text{g/L}$). Since the NOAEL in the reproductive study is also 400 ppm, exposure to MtBE in drinking water within this concentration range should not cause reproductive or developmental toxicity in humans. This health range assumes that a 70 kg adult consumes 2 L of water per day. An uncertainty factor of 1,000 was applied to the NOAEL. This factor includes a 10-fold factor for interspecies variability, 10 for intraspecies variability, and 10 to account for acute exposure and the limitation associated with the conversion of the inhaled dose to an oral dose in the absence of adequate pharmacokinetic models. The conservative use of the 10-fold factor for acute exposure should provide an additional margin of protection for potential effects on the developing fetus.

5.2.1.3 Neurotoxicity Studies

Inhalation exposure of animals to high levels of MtBE is associated with depression of the central nervous system in the period immediately after exposure (Daughtrey et al., 1997). Symptoms observed in groups of 22 male and 22 female F344 rats in the hour after a 6-hour exposure to an atmosphere containing 4,000 or 8,000 ppm MtBE included labored respiration, ataxia, decreased muscle tone, abnormal gait, impaired treadmill performance and decreased hind-limb grip strength. These effects were not noted 6 and 24 hours after the cessation of exposure. There were no apparent effects from a single 6-hour exposure to 800 ppm MtBE.

Subchronic exposures of groups of 15 male and 15 female rats under the same daily exposure conditions used for the acute study gave no indication that the repetition of exposure exacerbated the acute central nervous system response (Daughtrey et al., 1997). There was a significant decrease in the absolute, but not the relative, brain weight in the high-dose group at the end of the 13-week exposure period. However, there were no significant changes in brain or peripheral nervous system histopathology that could be related to MtBE. These studies identified 800 ppm as a NOAEL and 4,000 ppm as a LOAEL for acute effects of MtBE on the central nervous system.

The 800 ppm NOAEL for acute neurotoxic effects is projected to be equivalent to a dose of 210 mg/kg/day. Using this value, the projected no-effect concentrations in humans is 7.35 mg/L (7,350 $\mu\text{g/L}$) for a 70 kg adult drinking 2 L/day water. An uncertainty factor of 1,000 was used for this calculation. The uncertainty factor includes a 10 for use of a frank effect, 10 for interspecies variability and 10 for intraspecies variability. The uncertainty factor does not include an adjustment for the short-term duration, because the daily repetition of exposure had no influence on the effects observed.

5.2.1.4 Mutagenicity Studies

Several studies were available to assess the mutagenicity of MtBE. With one exception, this chemical has not exhibited genetic toxicity in a variety of *in vitro* and *in vivo* mammalian and non-mammalian test systems. Positive results were noted in a mouse lymphoma assay in the presence of microsomal enzymes (ARCO, 1980). The only positive response is due to the formaldehyde produced from *in vitro* metabolism (Stoneybrook Laboratories Inc., 1993). The objective of the mutagenicity studies is to determine whether MtBE's carcinogenic activity is associated with positive *in vivo* genetic activity (McKee et al., 1997). The weight of evidence from the mutagenicity data summarized below indicated that MtBE is not mutagenic.

MtBE was negative in sex-linked recessive lethal test in the *Drosophila melanogaster* (Hazelton, 1989). It was also negative in the Ames assays using *Salmonella*, both with and without metabolic activation (ARCO, 1980; Life Science Research, 1989a).

Chromosome aberrations (ABS) or sister chromatid exchange (SCE) induction tests in Chinese hamster ovary cells were negative with or without activation (ARCO, 1980). MtBE did not cause mutations in cultured Chinese hamster V79 cells (Life Science Research, 1989b). Inhalation of MtBE at dose levels up to 8,000 ppm did not cause chromosomal aberrations in bone marrow cells of F344 rats exposed 6 hours/day for 5 days (Bushy Run Research Center, 1989c) or micronuclei in bone marrow cells of CD-1 mice exposed for 6 hours/day for 2 days (Bushy Run Research Center, 1993). MtBE was also negative for mutations at the *hprt* locus in lymphocytes of CD-1 mice (Ward et al., 1995).

No increase in unscheduled DNA synthesis was observed in the hepatocytes of CD-1 mice that were exposed to MtBE vapor concentrations of up to 8,000 ppm for 6 hours/day for two consecutive days (Bushy Run Research Center, 1994). It did not cause DNA damage in the primary rat hepatocyte culture test (Life Science Research, 1989c), nor was it clastogenic in a rat *in vivo* cytogenetic assay (ARCO, 1980).

5.2.2 Cancer Effects

5.2.2.1 Studies of the Carcinogenicity of the Parent Compound (MtBE)

There are three chronic/cancer studies of MtBE in two rodent species (two inhalation studies, one in mice and one in rats, and one gavage study in rats). High doses of MtBE were used in all of the carcinogenicity studies and in some cases they have exceeded the Maximum Tolerated Dose (MTD).

Gavage Study

When MtBE (99% pure) was administered orally to Sprague-Dawley rats (gavage in olive oil, at doses of 0, 250 or 1,000 mg/kg-day, 4 days/week for two years), no significant differences in food/water consumption or body weight gain were observed. The chemical caused a dose-related increase in the incidence of leukemia and lymphomas in females (2/58 in the controls, 6/51 in the low-dose group and 12/47 in the high-dose group) and an increase in the testicular interstitial Leydig cell adenomas in the high-dose males (18.3% vs. 3.3% in the controls and/or low-dose animals). Survival was decreased 15 and 20% in the low- and high-dose females, respectively after 9 to 12 months of treatment (Belpoggi et al., 1995). There are some limitations in the reporting of the data as discussed below (quoted from NSTC, 1997):

The Belpoggi et al. study was published in the peer-reviewed literature. However, no detailed technical report of the bioassay is available. Lacking a detailed report about the bioassay, the NRC panel (NRC, 1996) identified a number of issues and questions which reflects upon the risk assessment use of these data. The NRC noted that the morphological criteria used to classify histopathological findings for both the lymphoma-leukemia and interstitial cell tumor responses were not adequately described and that the study did not adequately address the impact on tumor outcomes or differences in survival between controls and dosed groups. NRC went on to say that 'because of the importance of this study for eventual use in risk assessment, the superficial reporting of the data and the nature of the observed lesions, the committee felt strongly that an independent in-depth review of the data, especially the pathology (microscopic slides) of the critical lesions is warranted (as was done with the inhalation studies) before the data are used for risk assessment'. While the NRC raised questions about survival differences and the tumor outcome, it should be noted that Belpoggi et al. included statistical analyses that adjusted for intercurrent mortality. Several attempts by the Interagency Oxygenated Fuels Assessment Steering Committee to arrange for a pathology review of the Belpoggi et al. study have not been successful, hence, the underlying concerns raised by NRC review cannot yet be resolved.

Inhalation Studies

In a report by Chun et al. (1992), F344 rats were exposed to 0, 400, 3,000, or 8,000 ppm MtBE by inhalation, 6 hrs/day, 5 days/week for 2 years. This study was recently published as Bird et al. (1997). Survival time was statistically and significantly reduced in the exposed male rats in a dose-related manner. The mean body weights of the 8,000 ppm group (both sexes) were reduced throughout the experiment. (The mean body weight was decreased 19% in the males at week 82 and 13% in the females at the end of the experiment). An increase in chronic, progressive nephropathy was observed in the exposed male and female rats. The combined incidence of

renal tubular adenomas and carcinomas² was increased significantly in the male rats exposed to the mid-dose (controls, 1/35; low-dose, 0/32; mid-dose, 8/31; high-dose, 3/20). The reduced survival rate of the high-dose group may have decreased the sensitivity of the test to produce a dose-related increase in tumors.

A study by CIIT (Prescott-Mathews et al., 1997) shows that MtBE caused a mild induction of α -2u-globulin nephropathy and enhanced renal cell proliferation in F344 male rats, suggesting that α -2u-globulin nephropathy may potentially play a role in male rat kidney tumorigenesis.

EPA (U. S. EPA, 1991) published three criteria for establishing whether α -2u-globulin is responsible for the kidney tumor in male rats: 1) increased number and size of hyaline droplets in renal proximal tubule cells of treated rats, 2) accumulating protein in the hyaline droplets is α -2u-globulin, and 3) additional aspects of the pathological sequence of lesions associated with α -2u-globulin nephropathy are present. EPA's policy states that if experimental data do not meet the criteria in any one of the three categories, the α -2u-globulin alone is not considered responsible for the renal tumor formation and the renal tumor may be used for risk assessment, both qualitatively and quantitatively. Based on the available data, EPA concludes that the first criteria has been met, but the second and third criteria have not been adequately satisfied.

The mechanism of action of MtBE kidney carcinogenesis in male rats is not fully understood at the present time. In this case, the identification of the full spectrum of α -2u-globulin-specific nephropathy is complicated by a background of chronic progressive nephropathy (CPN) in both male and female rats and the apparent absence of one or more key α -2u-globulin pathological factors. The apparent absence may be a true non α -2u-globulin consequence, it may be masked by CPN, or it may be that the mild induction is insufficient to elicit the full α -2u-globulin response. It is possible that other proteins related to α -2u-globulin may also be involved (HEI, 1996). Ongoing research on the potential role of α -2u-globulin accumulation in male rat kidney

²Renal Tumor Incidence of F344 Male Rats After Inhalation Exposure to MtBE (Chun et al., 1992)

Administered exposure (ppm)	Human equiv. Dose* (mg/kg-day)	Tumor incidence+	Survival-adjusted Tumor incidence
0	0	1/50	1/35
400	75	0/50	0/32
3000	562.5	8/50	8/31
8000	1500	3/50	3/20

+tumor type: combined renal tubular cell adenomas and carcinomas

* See section 4.2 NSTC's Extrapolation of Dose from Inhalation Exposure

may improve our understanding of the carcinogenesis of MtBE and its metabolite, TBA, in the kidney.

A statistically significant increased incidence of the interstitial testicular Leydig cell adenomas of the treated rats was detected in the Chun et al. (1992) study (32 in the controls, 35 in the low-dose, 41 in the mid-dose, and 47 in the high-dose). The increase in the incidence of Leydig cell adenomas of the male rats in this study (Chun et al., 1992; Bird et al., 1997) was not significantly different from the historical control value, although the difference from the concurrent controls was significant. The concurrent control incidence was 64% and the historical control values ranged from 64 to 98% in the same laboratory (Bird et al., 1997). (Leydig cell adenomas occur at a high spontaneous rate in the F344 strain of rats.) However, this type of tumor was also observed in another strain of rats, the Sprague-Dawley, upon oral exposure by gavage (Belpoggi et al., 1995). Since the Sprague-Dawley rat does not have a significant spontaneous background incidence for this type of tumor, the conclusion that the appearance of the tumor in both studies is MtBE treatment-related is more confident.

In a report by Burleigh-Flayer et al. (1992), CD-1 mice were exposed to 0, 400, 3,000 or 8,000 ppm MtBE by inhalation, 6 hrs/day, 5 days/week for 18 months. Mortality was increased and the mean survival time was decreased in the high-dose mice compared to controls. The body weight gain was also decreased in the 8,000 ppm group compared to the controls (a decrease of 16% and 24% for male and female mice, respectively), indicating that the high dose exceeded the MTD. A statistically-significant increase was found in the incidence of hepatocellular carcinomas in male mice and of hepatocellular adenomas in female mice exposed to 8,000 ppm of MtBE³. The hepatic tumors were only evidenced at the high dose. Since MtBE is generally negative in mutagenicity tests, and the hepatocellular tumors induced by MtBE in CD-1 mice were detected only in the high-dose animals where the dose exceeded the MTD, the authors of the study (Burleigh-Flayer et al., 1992; Bird et al., 1997) considered the mouse liver tumor finding not likely to be due to a direct-DNA acting phenomenon. The NAS panel (NRC, 1996) also

³Hepatocellular Tumors in Female Mice After Inhalation Exposure to MtBE (Burleigh-Flayer et al., 1992)

Administered exposure (ppm)	Human equiv. Dose (mg/kg-day)	Tumor incidence		
		Adenoma	Carcinoma	combined
0	0	2/50	0/50	2/50
400	75	1/50	1/50	2/50
3000	562.5	2/50	0/50	2/50
8000	1500	10/50	1/50	11/50

In the male mice, the combined hepatocellular tumor incidence for the control, low-, mid-, and high-dose groups are 12/47, 12/47, 12/46 and 16/37, respectively.

suggested that the non genotoxic, hormonally-related mechanisms are the most plausible explanation for the development of mouse liver tumors”

Based on short-term studies in mice at CIIT, Moser et al. (1996) speculated that endocrine modulations may play a role in the hepatocarcinogenic effect of MtBE. The CIIT studies include: a) inhalation exposure (approximately 8,000 ppm, 6 hrs per day, 5 days per week) of female B6C3F1 mice to MtBE for 3 or 21 days, resulting in an increased relative liver weight, increased P450 content and its activity, as well as a decreased relative uterus weight; b) gavage treatment of B6C3F1 mice with MtBE (1,800 mg MtBE/kg body weight/day for 3 days) resulting in increased estrogen metabolism in isolated mouse hepatocytes (Moser et al. 1996).

EPA has calculated three slope factors from the cancer studies which appeared in the NSTC (1997) document. These estimates of slope factors are not likely to underestimate risk for the general population. The ability to calculate such an estimate does not imply greater confidence in potential cancer hazard. True risk for most individuals in the population is likely to be lower and for some may even be nearly zero. Because there are uncertainties inherent in these values, they should be used cautiously.

The first slope factor is based on the Belpoggi et al. (1995) gavage study. Using the combined tumor incidence of lymphoma and leukemia in the female rats and a scaling factor of body weight raised to the 2/3 power, a slope factor of $4 \times 10^{-3} \text{ (mg/kg/day)}^{-1}$ can be calculated by the linearized, multistage model⁴.

The second slope factor is based on the Chun et al. (1992) data. Based on the combined renal tubular cell adenomas and carcinomas in the male F344 rats, using a scaling factor of body weight raised to the 2/3 power, a slope factor of 6×10^{-4} per ppm can be calculated by the linearized, multistage model. Additional understanding of the mode of action of this response could substantially alter these estimates or make them irrelevant.

The third slope factor is based on the Burleigh-Flayer et al. (1992) data. Based on the liver tumor incidence in the female CD-1 mice, using a scaling factor of body weight raised to the 2/3 power, a slope factor of 3×10^{-4} per ppm was calculated by the linearized, multistage model.

⁴Based on the Proposed Guidelines for Carcinogen Risk Assessment (FR 61, 17960, April 23, 1996), with the same tumor data, using a scaling factor of body weight raised to the 3/4 power, an LED_{10} of 35.6 mg/kg-day and a slope factor of $2.8 \times 10^{-3} \text{ (mg/kg-day)}^{-1}$ are obtained. The drinking water concentration will be 12 $\mu\text{g/L}$ for a risk of one in a million using this slope factor.

5.2.2.2 Studies of the Carcinogenicity of MtBE Metabolites

tertiary-Butyl Alcohol

F344 rats were exposed to TBA via drinking water at concentrations of 0, 1.25, 2.5 or 5 mg/mL for 2 years (the average delivered, daily doses of TBA were approximately 0, 85, 195, and 420 mg/kg-day for males and 0, 175, 330 and 650 mg/kg-day for females). There was some evidence of carcinogenic activity in male rats based on an increased incidence of renal tubular hyperplasia and renal tubular adenomas or carcinomas, and no evidence of carcinogenic activity in female rats (Cirvello et al., 1995; NTP, 1995). Compared to controls, the survival was significantly lower for the high-dose animals, especially in the males. Increased nephropathy was also noted in all treated animals.

B6C3F1 mice were exposed to TBA in drinking water at concentrations of 0, 5, 10 or 20 mg/mL for 130 weeks (the average daily delivered doses were 0, 535, 1035 or 2065 mg/kg-day for males and 0, 510, 1015 or 2105 mg/kg-day for females). There was equivocal evidence of carcinogenic activity in male mice, based on marginally increased incidence of thyroid tumors and some evidence of carcinogenicity in female mice, based on an increased incidence of follicular cell hyperplasia and follicular cell adenomas of the thyroid gland. Survival of males in the high-dose group was significantly lower than that of the control group. Thus, the National Toxicology Program (NTP) studies of TBA show no clear evidence of carcinogenicity in either species.

Formaldehyde

There is sufficient evidence of carcinogenicity in animals by the inhalation route (IARC, 1995). Inhalation exposure of F344 rats to formaldehyde for 2 years at 14.3 ppm induced squamous cell carcinomas of the nasal cavity in both male and female F344 rats (the doses were: 0, 2, 5.6 or 14.3 ppm, 6 hours per day, 5 days per week), but not in female B6C3F1 mice (same doses and exposure conditions) (Kerns et al., 1983). Lifetime inhalation studies of formaldehyde in Sprague-Dawley rats at 14 ppm (Sellakumar et al., 1985), and Wistar rats at 10 ppm (Woutersen et al., 1989) also produced nasal tumors.

By the drinking water route of exposure, the evidence of carcinogenic activity for formaldehyde is somewhat ambiguous. One lifetime drinking water study of formaldehyde in Sprague-Dawley rats at concentrations of 0, 10, 50, 100, 1,000 or 1,500 ppm showed a dose-related increase in the incidence of leukemia and intestinal tumors (Soffritti et al., 1989). Similar to the Belpoggi et al. (1995) study of MtBE (which was conducted by the same laboratory), the reporting of the study is somewhat limited and the pathology also lacks an independent review. Another 2-year drinking water study of formaldehyde using Wistar rats at doses ranging from 0, 1.2, 15 to 82 mg/kg/day for males and 0, 1.8, 21, to 109 mg/kg/day for females showed no evidence of carcinogenicity (Til et al., 1989).

6.0 ORGANOLEPTIC PROPERTIES

Water contaminated with MtBE may have an unpleasant taste or odor. These characteristics, often referred to as "organoleptic properties," cannot be used by EPA for developing primary drinking water standards, but are of concern and do play a role in the production of finished drinking water, as most U.S. citizens would not drink "unpleasing" water. Taste and odor may also alert consumers to the fact that the water is contaminated with MtBE and, therefore, were considered in the development of this Advisory.

Not all individuals respond equally to taste and odor because of differences in individual sensitivity. The taste and odor responses reported in observed individuals for MtBE are in the 15 to 180 $\mu\text{g/L}$ range for odor and the 24 to 135 $\mu\text{g/L}$ range for taste (NSTC, 1997, Young et al., 1996; API, 1993; Prah et al., 1994; Dale et al., 1997). The ranges are indicative of the variability in individual response. The lower ends of the range for both taste and odor are the lowest concentrations eliciting a response among 7 of 9 participants in a study by Young et al. (1996). In this study, the geometric mean for taste was 48 $\mu\text{g/L}$ and that for odor was 34 $\mu\text{g/L}$. Participants in this study were selected for their above average sensitivity to basic tastes and odors. In fact, 3 of the 7 participants detected the lowest odor concentration, while 4 of 9 participants detected the lowest taste concentration. The homogeneity in the response among the small group of female subjects, along with the geometric mean values support classification of the subjects as sensitive.

A study commissioned by the American Petroleum Institute (API, 1993) and conducted by TRC Environmental Corp. used 6-7 individuals "chosen to represent a normal distribution of olfactory sensitivity" to measure taste and odor thresholds of 97% MtBE in distilled water. Calculated threshold values were 39 $\mu\text{g/L}$ for taste, 45 $\mu\text{g/L}$ for odor detection, and 55 $\mu\text{g/L}$ for odor recognition. The intensity of the odor of MtBE was also reported to be greater in water than in air. The subjects described the taste of MtBE in water as "nasty", bitter, nauseating, and similar to rubbing alcohol.

In a study by Prah et al. (1994), the concentration of MtBE in distilled water that was identified as having an odor by 50% of the study participants (19 males and 18 females) was 180 $\mu\text{g/L}$. This value is regarded as the high end of the odor range even though it is a median response concentration. There were undoubtedly individuals who could only detect the odor of MtBE at even higher concentrations.

The Metropolitan Water District of Southern California recently conducted a study on the taste and odor thresholds and other characteristics of MtBE (Dale et al., 1997). They found that the range for the 60% probability (± 1 SD) of correct taste detection of MtBE in odor-free water (OFW) and untreated Colorado River water was 24 to 37 and 26 to 58 $\mu\text{g/L}$, respectively. The corresponding range for detecting the odor of MtBE in OFW was 43 to 71 $\mu\text{g/L}$. These tests

were conducted by having nine trained analysts undergo six "triangle tests" for several concentrations, in which each analyst determined the odd case when blindly presented with either two blanks and one spiked sample or one blank and two spiked samples. It cannot be determined from this small, non-representative sample what percentage of the general population would be able to detect MtBE in their drinking water at these concentrations. However, these taste and odor threshold data are consistent with those reported by Young et al. (1996) and API (1993). This study by Dale et al. (1997) found people more sensitive to taste than odor, which is consistent with the API (1993) findings for MtBE taste and odor thresholds, but in the opposite order to that found by Young et al. (1996). Collectively, these data support a range of 20 to 40 $\mu\text{g/L}$ as an approximate "threshold" for organoleptic responses. However, some subjects in this study were able to detect MtBE at much lower concentrations; thus, in a general population, some unknown percentage of people will be likely to detect the taste and odor of MtBE in drinking water at concentrations below 20 $\mu\text{g/L}$.

The study by Dale et al. (1997) went beyond simply measuring taste and odor thresholds. The investigators also asked four panelists to describe the taste and odor of MtBE in OFW at concentrations ranging from 2 $\mu\text{g/L}$ to 190 $\mu\text{g/L}$. At concentrations of 2-5 $\mu\text{g/L}$, the consensus judgment of the panelists was that the taste of MtBE in OFW could be described as "sweet." At concentrations of 21-190 $\mu\text{g/L}$, the characterization was either "solvent" or "sweet solvent." Similar characteristics applied at concentrations of 21-190 $\mu\text{g/L}$ for the odor of MtBE in OFW. The panelists were also asked to rate the intensity of the taste and odor, which can become "objectionable" at a sufficiently high intensity. The panelists considered the taste of MtBE in OFW objectionable at a concentration of approximately 50 $\mu\text{g/L}$ and the odor objectionable at approximately 90-100 $\mu\text{g/L}$. It is noted that these tests were conducted with non-chlorinated water at 25 degrees C. Chlorination would likely raise the thresholds for the taste and odor of MtBE in water, and higher temperatures (e.g., for showering) would likely reduce these thresholds.

It is not possible to identify point threshold values for the taste and odor of MtBE in drinking water, as the concentration will vary for different individuals, for the same individuals at different times, for different populations, and for different water matrices, temperatures, and many other variables. Nevertheless, it seems reasonable to offer a range of 20-40 $\mu\text{g/L}$ as advisory guidance for helping to ensure consumer acceptance of the taste and odor of MtBE in drinking water.

7.0 CHARACTERIZATION OF HAZARD AND DOSE RESPONSE

7.1 Hazard Characterization

There are very few data on human responses to MtBE. In controlled studies, there were no observable responses to short-term (1 hour) exposures to low concentrations of MtBE in air,

although women felt the air quality was substandard (Cain et al., 1994; Prah et al., 1994). Other short-term human studies of MtBE are of limited value, because they evaluated effects under conditions where MtBE was combined with simultaneous exposures to other chemicals, such as gasoline, medicines and/or anesthesia (Allen et al., 1985; Hakkola et al., 1996; Juliani et al., 1985; Moolenaar et al., 1994; White et al., 1995; Wyngaarden 1986). Studies of gasoline/MtBE mixtures are inconclusive, but suggest that MtBE-containing gasoline vapors may be irritating to eyes, the respiratory system and the nervous system (Hakkola et al., 1996; Moolenaar et al., 1994; White et al., 1995). There have been no long-term studies of human exposure to MtBE.

Rodent studies identify the kidneys, brain and developing fetus as sensitive to MtBE. The neurotoxicity data from inhalation exposures in rats (Daughtrey et al., 1997) showed transient CNS depression and decreased motor activity at high levels of MtBE (8,000 ppm). However, there are no data to support the hypothesis that MtBE dissolved in drinking water has adverse effects on the nervous system in humans.

The collective evaluation of the reproductive and developmental studies of MtBE in animals indicate that inhalation exposure can result in maternal toxicity and adverse effects on the developing fetus (Bushy Run Research Center, 1991, 1989a, 1989b; Conaway et al., 1985). The fetal toxicity in the mouse developmental studies indicate that it may be more sensitive to inhalation of MtBE vapors than the rat or rabbit during gestation. However, it is possible to conclude that, at low concentrations, MtBE does not cause a developmental or reproductive hazard by inhalation in three different animal species. This also suggests that humans may not be at risk when exposed to very low concentrations of MtBE.

Effects on the kidney were observed in rats after oral and inhalation exposure to MtBE. After short-term oral exposure, increases in kidney weights were noted (Robinson et al., 1990), while in a longer term inhalation study, histopathological abnormalities were apparent (Chun et al., 1992). The oral data from the short-term study are confounded by the bolus gavage dosing regime and the less-than-lifetime duration of the study, while the uncertainty in extrapolating between routes affects the interpretation of the inhalation data.

The use of inhalation data to project effects from the oral exposures is generally not desirable but, in the case of MtBE, there is qualitative similarity in the effects observed with both routes. However, when using the inhalation data to calculate a human equivalent dose for the risk assessment calculations, additional uncertainty is introduced by the mathematical conversion.

In animals, there are two chronic inhalation studies available, one in rats causing increased incidence of renal and testicular tumors (Chun et al., 1992) and one in mice inducing liver tumors (Burleigh-Flayer et al., 1992). By the oral route, there is one gavage study in rats producing a dose-related increase in leukemia and lymphoma in the females and an increase in testicular tumors in the males (Belpoggi et al., 1995). In addition, formaldehyde, a metabolite of MtBE, is

an animal carcinogen. By inhalation exposure, it induces nasal tumors in rats (Kerns et al., 1983). By the drinking water route of exposure, one study shows a dose-related increase in leukemia (Soffritti et al., 1989) and another study shows no evidence of carcinogenicity (Til et al., 1989). In addition, there is some suggestive evidence of carcinogenicity of TBA (another MtBE metabolite) – an increased incidence of renal tumors in rats and an increase in thyroid tumors in the female mice after drinking water exposure.

Most of the cancer studies of MtBE and its metabolites have limitations, such as high mortality among the treated animals, limited reporting of pathology and of historical tumor incidence, etc. In spite of the limitations, there are some consistent tumor findings for MtBE and its metabolites. This consistency contributes to the overall weight of evidence. A statistically-significant increase in interstitial Leydig cell adenomas of the testes was detected in the exposed rats after both inhalation (Chun et al., 1992; Bird et al., 1997) and gavage exposures (Belpoggi et al., 1995). In addition, the elevation of kidney tumors in male F344 rats treated with TBA (a metabolite of MtBE), via drinking water (Cirvello et al., 1995; NTP, 1995) supports the increase of similar tumors in male rats after exposure to MtBE by inhalation (Chun et al., 1992; Bird et al., 1997). The similarity in the finding of a dose-related increase in leukemia of rats (Sprague-Dawley, male and female combined) after exposure to formaldehyde (also a metabolite of MtBE) via drinking water (Soffritti et al., 1989) and the increase of leukemia/lymphomas in female rats (same strain) after exposure to MtBE via gavage (Belpoggi et al., 1995), suggests a possible involvement of formaldehyde in the leukemogenic effect of MtBE. However, issues remain unresolved related to these studies, which were conducted by the same laboratory. Both studies provided limited reporting and no information on historical incidence of leukemia⁵.

MtBE does not appear to be DNA reactive. The chemical has been tested in an array of both *in vitro* and *in vivo* systems, and the results have been negative overall. The possibility that the genesis of the rat kidney tumors involves the α -2u-globulin mechanism is being investigated, but, as yet, the evidence does not show that the mechanism accounts for the tumors satisfying all the EPA criteria (U.S. EPA, 1991). The observation of nephropathy and toxicity in association with tumorigenicity in the rat kidney suggests that a number of factors, possibly including the α -2u-globulin mechanism, may be at work. The observation of testicular tumors from MtBE and thyroid tumors from TBA suggest the need for examination of disruption of pituitary and thyroid

⁵Unlike NTP carcinogenicity studies, the histopathology diagnoses from the inhalation studies of MtBE in rats and mice have not been subject to a full peer-review. Also, there is a major difference between the oral and inhalation carcinogenicity studies of MtBE. Lengthy reports of the inhalation studies of MtBE in rats and mice were submitted to EPA. These reports (Burleigh-Flayer et al., 1990; Chun et al., 1992) provide significantly more information than what is contained in the published peer-reviewed literature (Belpoggi et al., 1995; Bird et al., 1997). Based on these reports, we can conclude that the inhalation studies were conducted in conformance with Good Laboratory Practices, while there is a lack of evidence to back up that the gavage study is also conducted in conformance with Good Laboratory Practices.

hormone function, as such disruption is not uncommon with these tumors (Hill et al., 1989; USEPA, 1997). It has been suggested that MtBE-induced mouse liver tumors also may be hormone-related (Moser et al., 1996; Bird et al, 1997).

Although MtBE is not mutagenic, a nonlinear mode of action has not been established for MtBE. In the absence of sufficient mode of action information at the present time, it is prudent for EPA to assume a linear dose-response for MtBE. Although there are no studies on the carcinogenicity of MtBE in humans, there are multiple animal studies (by inhalation and gavage routes in two rodent species) showing carcinogenic activity and there is supporting animal carcinogenicity data for the metabolites. The weight of evidence indicates that MTBE is an animal carcinogen, and the chemical poses a carcinogenic potential to humans (NSTC, 1997, page 4-26).

7.2 Characterization of Organoleptic Effects

There have been several studies of taste and odor response by humans. There is typically variation among individuals in these responses to a chemical, and this is the case for MtBE. The studies on MtBE have been of a few individuals each. Larger numbers of individuals might show the full distribution of sensitivity of humans which remains uncharacterized. Nevertheless, the existing studies were performed independently and show distributions that are consistent with one another. This lends confidence to the conclusion that sensitive individuals respond to odor and taste at about 20 to 40 $\mu\text{g/L}$.

Other influences on consumer perception and acceptance of the taste and odor of MtBE contamination of water are as yet uncharacterized. These include development of tolerance, exposure through food and beverage preparation or showering, and reaction to published reports of contamination. Moreover, the presence or absence of other natural or water treatment chemicals can mask or reveal the taste or odor effects. Thus, variable preexisting water conditions around the country will increase variability in the acceptability of MtBE's presence in drinking water.

7.3 Dose Response Characterization

There are no studies of long-term human exposure to MtBE; the pertinent data on potential adverse effects are from rodent studies. The available data do not provide sufficient information on the potential toxic effects from drinking water exposure and support only an uncertain view of the quantitative dose and response relationship. For quantitative assessment of adverse health effects from drinking water exposure, the preferred data would be from studies of effects of episodic oral exposure through water or food. For MtBE, the data are either from inhalation studies or from daily, high dose (bolus), gavage studies, using vegetable oil as a vehicle. Estimating drinking water dose equivalents based on inhalation studies or on bolus dosing studies introduces significant uncertainties.

The results of the Robinson et al. (1990) study, supported by the inhalation exposure data of Chun et al. (1992) provide adequate support for the conclusion that MtBE may exert adverse effects on the kidney. However, EPA does not have high confidence in the use of the Robinson et al. (1990) study, nor any other study presently available for quantitation of the potential noncancer or cancer effects of MtBE. Because of the lack of confidence in quantitative estimation of drinking water risk, this Advisory does not recommend either a low-dose oral cancer risk number or a low end RfD. Instead, the Advisory provides perspective by showing the margins of exposure between observations of the range of animal effects and water concentrations. Table 1 summarizes this margin of exposure information. A final health advisory will be written when the data base is improved sufficiently to allow greater confidence in the integration of data. Since the production of potable water is a prerequisite for its use, it is evident that the organoleptic (taste and odor) effects of MtBE should be considered. The available data (Prah et al. 1994; Young et al., 1996; Dale et al., 1997; NSTC, 1997) suggest that the lower range for the organoleptic effects of MtBE is 20 to 40 µg/L.

The values in Table 1 show the lower end of ranges of observation of effects in animals tested for cancer and noncancer responses. Table 1 also shows the MoEs (i.e., the ratios of the observed numbers to the sensitive range of human response to odor and taste (20 to 40 µg/L). The cancer LED₁₀ are based on analyses of the Belpoggi et al. (1995), Chun et al. (1992), and Burleigh-Flayer et al. (1992) studies as described in section 5.2.2.1 above. The noncancer NOAEL values are based on analyses recounted in section 5.2.1.: kidney effects in a subchronic gavage study on rats, reproductive/developmental effects from inhalation studies in rodents, neurotoxicity for frank, reversible effects in rats observed after short-term inhalation exposures. The ranges given for taste and odor represent the low ends of the reported values for organoleptic responses to MtBE in water discussed in section 6.0. These available data provide an estimate that the lower range for the organoleptic effects of MtBE is about 15 to 39 µg/L (taste and odor) from an empirical observation.

Values are rounded to one significant number, 20 and 40 (odor and taste), to avoid the appearance of precision that use of two significant numbers would give. Since characterization of the full distribution of sensitivity is not provided by available data, the numbers should be regarded as approximate, not precise. For the same reason, a range is presented. The data are used only to estimate sensitive range and should not be mistaken as defining thresholds of human response. In practice, the efforts of water suppliers to satisfy consumers on the acceptability of taste and odor of water, also will be influenced by considering the effects of other chemical in local waters.

7.4 Comparison of Margins of Exposure with Potential Environmental Concentrations and Guidance on Taste and Odor

Table 1 permits comparison of an observed environmental concentration with the observed effects levels for test animals to calculate a margin of exposure by dividing the environmental concentration into the value at the low end of the range for an effect displayed.

If the objective is to avoid unpleasant taste and odor, this Advisory recommends that a concentration in the range of 20 to 40 $\mu\text{g/L}$ likely will protect sensitive members of a population. At 20 $\mu\text{g/L}$, the margin of exposure is approximately forty thousand (40,000) for cancer effects and over one hundred thousand (100,000) for some noncancer effects. At 40 $\mu\text{g/L}$, the MoE is approximately twenty thousand (20,000) for cancer effects and sixty thousand (60,000) for some noncancer effects. In the case of noncancer critical effects, the lower end of the developmental NOAEL-range was used as the minimum effect level in the MoE calculation; the cancer value was calculated using the LED_{10} (95% lower bound of the dose for a 10% extra risk)⁶.

Comparison indicates that there are over four to five orders of magnitude between the 20 to 40 $\mu\text{g/L}$ range and concentrations associated with observed ranges of effects in animals. There is little likelihood that an MtBE concentration of 20 to 40 $\mu\text{g/L}$ in drinking water would cause adverse effects in humans, recognizing that some people may detect the chemical below this range. It can be noted that at this range of concentrations, the margins of exposure are about 10 to 100 times greater than would be provided by an EPA reference dose (RfD) for noncancer effects. Additionally, they are in the range of margins of exposure typically provided by National Primary Drinking Water Standards under the Federal Safe Drinking Water Act to protect people from potential carcinogenic effects.

⁶Based on the USEPA's recently proposed guidelines for carcinogen risk assessment (U.S. EPA, 1996), the rationale supporting the use of the LED_{10} is that a 10% response is at or just below the limit of sensitivity for discerning a significant difference in most long-term rodent studies. The NOAEL in most study protocols is about the same as an LED_5 or LED_{10} — the lower 95% confidence limit on a dose associated with a 5% or 10% increased effect. The MoE value for cancer was obtained by dividing the concentration equivalent to the LED_{10} (23 mg/kg/day equivalent to 805,000 $\mu\text{g/L}$) by 20 $\mu\text{g/L}$ to obtain a MoE of 40,200. The MoE for noncancer effects was obtained by dividing the concentration equivalent to the lower end of the NOAEL for the developmental toxicity range (65.6 mg/kg/day equivalent to 2,292,500 $\mu\text{g/L}$) by the environmental water concentration of 20 $\mu\text{g/L}$ to obtain an MoE of 114,625. The calculations assume a 70 kg body weight and 2 L/day water consumption.

Endpoint	Parameter	Concentration ⁴ $\mu\text{g}/\text{L}$	MoE compared to 40 $\mu\text{g}/\text{L}$	MoE compared to 20 $\mu\text{g}/\text{L}$
Noncancer	NOAEL			
Kidney		3,500,000	90,000	180,000
Neurological		7,400,000	185,000	370,000
Reproductive/Developmental		2,300,000 - 9,200,000	$\geq 60,000$	$\geq 120,000$
Cancer ²	LED ₁₀ ³			
Rat Lymphoma and Leukemia (gavage) in females		805,000	20,000	40,000
Rat Kidney Tumor (inhalation) in males		6,230,000	160,000	320,000
Mouse Liver Tumor (inhalation) in females		11,025,000	280,000	550,000

¹ The margins of exposure is calculated by dividing the NOAELs for noncancer endpoints or LED₁₀ for cancer effects by 40 $\mu\text{g}/\text{L}$ or 20 $\mu\text{g}/\text{L}$ which is the low end of the taste and odor threshold, respectively.

² The data from Belpoggi gavage study and the Chun and Burleigh-Flayer inhalation studies were used in the calculation. Air concentration of MtBE in ppm was converted to mg/kg-day by the NSTC method: 1 ppm = 1.05 mg/kg-day (NSTC, 1996, See also 4.2).

³ The LED₁₀ is defined as the 95% lower bound on dose for a 10% extra risk which was calculated by applying the tumor incidence data to the multistage model. As indicated by the NSTC (1996), a lifetime adjustment factor of 2.37 [i.e., $(24/18)^3$] was applied to the mouse liver tumor data to account for the short duration of the study (18 months instead of 24 months). In addition, as done by NTIS, the rat kidney tumor incidence in the highest exposure group was excluded from the risk analysis because this exposure group was terminated at 82 weeks (not 102 weeks) due to extremely high mortality.

⁴ The NOAEL and LED₁₀ were initially calculated in mg/kg-day and then converted to $\mu\text{g}/\text{L}$, assuming a body weight of 70 kg and a water consumption rate of 2 liters per day.

8.0 REFERENCES

Allen, M.J., Borody, T.J., Bugliosi, T.F., May, GR., LaRusso, N.F., and J.L. Thistle. 1985. Cholelitholysis using methyl tertiary-butyl ether. *Gastroenterology* 88:122-125.

API. 1993. American Petroleum Institute. Odor threshold studies performed with gasoline and gasoline combined with MtBE, EtBE and TAME. Washington, DC: API # 4592

ARCO. 1980. ARCO Chemical Company. Methyl tertiary-butyl ether: acute toxicological studies. Unpublished study for ARCO Research and Development, Glenolden, PA.

ARCO. 1995. ARCO Chemical Company. Methyl t-Butyl Ether (MtBE): A status report of its presence and significance in US drinking water. Presented to the Office of Water, U.S. Environmental Protection Agency. Presented by ARCO Chemical Company. June 8, 1995.

ATSDR. 1996. U.S. Department of Health and Human Services. Toxicological profile for methyl *tert*-butyl ether:

Belpoggi, F., Soffritt M., and C. Maltoni. 1995. Methyl-tertiary-butyl ether (MtBE) — a gasoline additive — causes testicular and lymphohaematopoietic cancers in rats. *Toxicol. Ind. Health* 11:1-31.

Bevan, C., Tyl, R.W., Neeper-Bradley, T.L., Fischer, L.C., Panson, R.D., Kneiss, J.J., and L.S. Andrews. 1997a. Developmental toxicity evaluation of methyl tertiary-butyl ether (MTBE) by inhalation in mice and rabbits. *J. Appl. Toxicol.* 17(S1):S21-S30.

Bevan, C., Neeper-Bradley, T.L., Tyl, R.W., Fischer, L.C., Panson, R.D., Kneiss, J.J., and L.S. Andrews. 1997b. Two-generation reproductive study of methyl tertiary-butyl ether (MTBE) in rats. *J. Appl. Toxicol.* 17(S1):S13-S20.

Biles, R.W., Schroeder, R.E., and C.E. Holdsworth. 1987. Methyl *tert*-butyl ether inhalation in rats: a single generation reproduction study. *Toxicol. Ind. Health.* 34:519-534.

Bio/dynamics, Inc. 1984. The metabolic fate of methyl tertiary-butyl ether (MtBE) following acute intraperitoneal injection. Project No. 80089. Unpublished report submitted to American Petroleum Institute, Washington, DC. 150 pp.

Bio-Res. Lab. 1990a. Bio-Research Laboratories. Pharmacokinetics of methyl tertiary-butyl ether (MtBE) and *tert*-butyl alcohol (TBA) in male and female Fischer-344 rats after administration of ¹⁴C-MtBE by iv, oral, and dermal routes. Report #38842. Senneville, Quebec, Canada: Bio-Research Laboratories.

Bio-Res. Lab. 1990b. Bio-Research Laboratories. Mass balance of radioactivity and metabolism of methyl tert-butyl ether (MtBE) in male and female Fischer-344 rats after administration of ^{14}C MtBE by iv, oral, and dermal routes. Report #38843. Senneville, Quebec, Canada: Bio-Research Laboratories.

Bio-Res. Lab. 1990c. Bio-Research Laboratories. Pharmacokinetics of methyl tert-butyl ether (MtBE) and tert-butyl alcohol (TBA) in male and female Fischer-344 rats after single and repeat inhalation nose-only exposure to ^{14}C -MtBE. Report #38844. Senneville, Quebec, Canada: Bio-Research Laboratories.

Bio-Res. Lab. 1990d. Bio-Research Laboratories. Disposition of radioactivity of methyl tertiary-butyl ether (MtBE) in male and female Fischer-344 rats after nose-only inhalation exposure to ^{14}C -MtBE. Report #38845. Senneville, Quebec, Canada: Bio-Research Laboratories.

Bird, M.G., Burleigh-Flayer, H.D., Chun, J.S., Douglas, J.F., Kneiss, J.J. and L.S. Andrews. 1997. Oncogenicity studies of inhaled methyl tertiary-butyl ether (MTBE) in CD-1 mice and F-344 rats. *J. Appl. Toxicol.* 17:(S 1): S45-S56.

Borghoff, S.J., Murphy, J.E., and M.A. Medinsky. 1996. Development of a physiologically based pharmacokinetic model for methyl tertiary-butyl ether and tertiary-butanol in male Fischer-344 rats. *Fundam. Appl. Toxicol.* 30:264-275.

Brady, J.F., Xiao, F., Ning, W.J., and C.S. Yang. 1990. Metabolism of methyl tertiary-butyl ether by rat hepatic microsomes. *Arch. Toxicol.* 64:157-160.

Burleigh-Flayer, H.D., Chun, J.S., and W.J. Kintigh. 1992. Methyl tertiary butyl ether: vapor inhalation oncogenicity study in CD-1 mice. Report 91N0013A. Export, PA: Bushy Run Research Center.

Bushy Run Research Center. 1994. Methyl tertiary-butyl ether: *in vivo*-*in vitro* hepatocyte unscheduled DNA synthesis assay in mice. Project ID 93N1316. Export, PA.

Bushy Run Research Center. 1993. Methyl tertiary-butyl ether: bone marrow micronucleus test in mice. Project ID 93N1244. Export, PA.

Bushy Run Research Center. 1991. Two-generation reproduction study of inhaled methyl tert-butyl ether in CD Sprague-Dawley rats. Final Report, August 13. Project ID 53-594. Export, PA.

Bushy Run Research Center. 1989a. Developmental toxicity study of inhaled methyl tertiary butyl ether in CD-1 mice. Final Report, July 20. TSCATS/403186. EPS/OTS No. FYI-OTS-0889-0689. Export, PA.

Bushy Run Research Center. 1989b. Developmental toxicity study of inhaled methyl tertiary butyl ether in New Zealand white rabbits. Final Report, May 12. EPA/OTS No. FYI-OTS-0889-0689. Export, PA.

Bushy Run Research Center. 1989c. Methyl tertiary butyl ether repeated exposure vapor inhalation study in rats: in vivo cytogenetic evaluation. Project Report 51-635. Export, PA.

Cain, W.S., Leaderer, B.P., Ginsberg, G.L., Andrews, L.S., Cometto-Muniz, J.E., Gent, J.F., Buck, M., Berglund, L.G., Mohsenin, V., Monhan, E., and S. Kjaergaard. 1994. Human reactions to brief exposures to methyl tertiary-butyl ether. (Unpublished data from John B. Pierce Laboratory, New Haven, Connecticut).

Cederbaum, A.I. and G. Cohen. 1980. Oxidative demethylation of t-butyl alcohol by rat liver microsomes. *Biochem. Biophys. Res. Comm.* 97:730-736.

Chun J.S., Burleigh Flayer, H.D., and W.J. Kintigh. 1992. Methyl tertiary ether: vapor inhalation oncogenicity study in Fisher 344 rats. Export, PA; Bushy Run Research Center; report 91N0013B.

Cirvello, J.D., Radovsky, J.E. Heath, D.R. Farnell and C. Lindamood, III. 1995. Toxicity and carcinogenicity of t-butyl alcohol in rats and mice following chronic exposure in drinking water. *Toxicol. and Ind. Health.* 11:151-165.

Conaway, C.C., Schroeder, R.E., and N.K. Snyder. 1985. Teratology evaluation of methyl tertiary-butyl ether in rats and mice. *J. Toxicol. Environ. Health.* 166:797-809.

Dale, M.S., Moylan, M.S., Koch, B., and Davis, M.K. 1997. MTBE: Taste and odor threshold determinations using the flavor profile method. Presented at the Water Quality Technology Conference, November 9-13, 1997. Denver, CO.

Daughtrey, W.C., Gill, M.W., Pritts, I.M., Fielding Douglas, J., Kneiss, J.J., and L.S. Andrews. 1997. Neurotoxicological evaluation of methyl tertiary-butyl ether in rats. *J. of Appl. Toxicol.* 17 (S1):S57-S64.

Hakkola, M., Honkasalo, M.L., and P. Pulkkinen. 1996. Neuropsychological symptoms among tanker drivers exposed to gasoline. *Occup. Med.* 46:125-130.

Hazelton. 1989. Hazelton Laboratories America, Inc. Mutagenicity test on methyl tertiary-butyl ether. *Drosophila melanogaster* sex-linked recessive lethal test. Study No. 1484-0-461. Kensington, MD.

Health Effect Institute report (HEI), 1996. The potential health effects of oxygenates added to gasoline. A review of the current literature. A special report of the Institute's oxygenates evaluation committee. Health Effects Institute, Cambridge, MA. In Interagency Oxygenated Fuel Assessment. 1996. Office of Science and Technology (OSTP) through the committee on Environment and Natural Science and Technology Council (NSTC).

Hill, R.N., Erdreich, L.S., Paynter, O.E., Roberts, P.A., Rosenthal, S.L., and Wilkinson, C.F. 1989. Thyroid follicular cell carcinogenesis. *Fundam. Appl. Toxicol.* 12:629-697.

Industrial Bio-Test Laboratories Inc. 1972a. Absorption, distribution, and excretion study with 2,2-MMOP in albino rats. (Unpublished data from Industrial Bio-Test Laboratories Inc., Northbrook, Illinois, to Sun Oil Company). IBT No. E200 (A).

Industrial Bio-Test Laboratories. 1972b. Absorption, distribution, and excretion study with 2,2-MMOP in monkeys. (Unpublished data from Industrial Bio-Test Laboratories Inc., Northbrook, Illinois, to Sun Oil Company). IBT No. E200 (B).

IARC. 1995. International Agency for Research on Cancer. Formaldehyde. In: IARC monographs on the evaluation of carcinogenic risks to humans: wood dust and formaldehyde. Lyon, France: IARC. 62:217-362.

Johanson, G., Nihlen, A., and A. Lof. 1995. Toxicokinetics and acute effects of MtBE and EtBE in male volunteers. *Toxicol. Lett.* 82/83:713-718.

Juliani, G., Gandini, G., Gabasio, S., Bonardi, L., Fascetti, E., and L. Gremo. 1985. Colelitolisi chimica transcutanea con metil-ter-butyl etere (MtBE). *La Radiol. Med.* 71:569-574.

Kerns K.D., Pavkov K.L., Donofrio D.J., Gralla E.J., and J.A. Swenberg. 1983. Carcinogenicity of formaldehyde in rats and mice after long-term inhalation exposure. *Cancer Res.* 43:4382-4392.

Life Science Research, Roma Toxicology Centre S.P.A. 1989a. Reverse mutation in *Salmonella typhimurium*, test substance: MtBE. Report No. 216001-M-03489. Rome, Italy.

Life Science Research, Roma Toxicology Centre S.P.A. 1989b. Gene mutation in Chinese hamster V79 cells, test substance: MtBE. Report No. 216002-M-03589. Rome, Italy.

December 1997

Life Science Research, Roma Toxicology Centre S.P.A. 1989c. Unscheduled DNA synthesis (UDS) in primary rat hepatocytes (autoradiographic method), test substance: MtBE. Report No. 216003-M-03689. Rome, Italy.

McKee, R.H., Vergnes, J.S., Galvin, J.B., Douglas, J.F., Kneiss, J.J., and L.S. Andrews. 1997. Assessment of the *in vivo* mutagenic potential of methyl tertiary-butyl ether. *J. Appl. Toxicol.* 17 (S1):S31-S36.

Miller, M.J., Ferdinandi, E.S., Klan, M., Andrews, L.S., Douglas, J.F., and J.J. Kneiss. 1997. Pharmacokinetics and disposition of methyl t-butyl ether in Fischer-344 rats. *J. Appl. Toxicol.* 17(S1):S3-S13.

Moolenaar, R.L., Hefflin, B.J., Ashley, D.L., Middaugh, J.P., and R.A. Etzel. 1994. Methyl tertiary butyl ether in human blood after exposure to oxygenated fuel in Fairbanks, Alaska. *Arch. Environ. Health* 49(5):402-409. (also CDC 1993a. Centers for Disease Control and Prevention. An investigation of exposure to methyl tertiary-butyl ether in Fairbanks, Alaska. Atlanta, GA: U.S. Department of Health and Human Services, National Center for Environmental Health. October 22, 1993.)

Moser, G.J., Wong, B.A., Wolf, D.C., Moss, O.R., and T.L. Goldsworthy. 1996. Comparative short-term effects of methyl tertiary-butyl ether and unleaded gasoline vapor in female B6C3F1 mice. *Fundam. Appl. Toxicol.* 31:173-183.

NRC. 1996. National Research Council. Toxicological and performance aspects of oxygenated motor vehicle fuels. Washington, DC: National Academy Press.

NSTC. 1996. National Science and Technology Council. Interagency assessment of potential health risks associated with oxygenated gasoline. National Science and Technology Council Committee on Environment and Natural Resources and Interagency Oxygenated Fuels Assessment Steering Committee.

NSTC. 1997. National Science and Technology Council Committee on Environment and Natural Resources. Interagency Assessment of Oxygenated Fuels.

NTP. 1995. National Toxicology Program. Toxicology and carcinogenesis studies of t-butyl alcohol (CAS No. 76-65-0) in F344/N rats and B6C3F1 mice (drinking water studies). Research Triangle Park, NC: National Institute of Health; Technical Report Series No. 436, NIH Publication No. 94-3167.

Prah, J.D., Goldstein, G.M., Devlin, R., Otto, D., Ashley, D., House, S., Cohen, K.L., and T. Gerrity. 1994. Sensory, symptomatic, inflammatory, and ocular responses to and the

metabolism of methyl tertiary-butyl ether in a controlled human exposure experiment. *Inhal. Toxicol.* 6:521-538.

Prescott-Mathews, J.S., Wolf, D.C., Wong, B.A., and S.J. Borghoff. 1997. Methyl tert-Butyl Ether Causes α 2u-globulin Nephropathy and Enhanced Renal Cell Proliferation in Male F344 Rats. *Toxicol. Appl. Pharm.* 143:301-314.

Rao, H.V. and G.L. Ginsberg. A Physiologically-Based Pharmacokinetic Model Assessment of Methyl t-Butyl Ether in Groundwater for a Bathing and Showering Determination. *Risk Anal.* (In press)

Robinson, M., Bruner, R.H., and G.R. Olson. 1990. Fourteen- and ninety-day oral toxicity studies of methyl tertiary-butyl ether in Sprague-Dawley rats. *J. Am. Coll. Toxicol.* 9:525-540.

Savolainen, H., Pfaffli, P., and E. Elovaara. 1985. Biochemical effects of methyl tertiary-butyl ether in extended vapor exposure in rats. *Arch. Toxicol.* 57:285-288.

Sellakumar A.R., Snyder C.A., Solomon J.J., and R.E. Albert. 1985. Carcinogenicity of formaldehyde and hydrogen chloride in rats. *Toxicol. Appl. Pharmacol.* 81:401-406.

Soffritti M., Maltoni C., Maffei, F., and R. Biagi. 1989. Formaldehyde: an experimental multipotential carcinogen. *Toxicol. Ind. Health.* 5:699-730.

Stoneybrook Laboratories Inc. 1993. Activated-mouse lymphoma (L5178Y/TK+/+) mutagenicity assay supplemented with formaldehyde dehydrogenase for methyl tertiary butyl ether. Status Report 65579. Princeton, NJ.

Til, H.P., Woutersen R.A., Feron, V.J., Hollanders, V.M.H., and H.E. Falke. 1989. Two-year drinking-water study for formaldehyde in rats. *Food Chem. Toxicol.* 27:77-87.

U.S. EPA. 1987. Reference Dose (RfD): Description and use in health risk assessments. Integrated risk information system (IRIS): Appendix A. United States Environmental Protection Agency. Washington, D.C. Integrated risk information system documentation, vol. 1. EPA/600/8-66/032a.

U.S. EPA. 1991. Alpha 2 μ -globulin association with chemically induced renal toxicity and neoplasia in the male rat. Risk Assessment Forum. United States Environmental Protection Agency. Washington, D.C. EPA/625/3-91/019F.

U.S. EPA. 1993. Assessment of potential health risks of gasoline oxygenated with methyl tertiary-butyl ether (MtBE). Office of Research and Development. United States Environmental Protection Agency. EPA/600/R.93/206.

U.S. EPA. 1996. Proposed guidelines for carcinogen risk assessment. United States Environmental Protection Agency. Federal Register 61(79):17960-18011.

U.S. EPA. 1997. Assessment of thyroid follicular cell tumors. Risk Assessment Forum. United States Environmental Protection Agency. Washington, D.C. EPA/630/R-97/002.

USGS. 1996. Occurrence of the gasoline additive MtBE in shallow ground water in urban and agricultural areas. United States Geological Survey Fact Sheet 114.95. October.

Ward, Jr., J.B., Daiker, D.H., Hastings, D.A., Ammenheuser, M.M., and M.S. Legator. 1995. Assessment of the mutagenicity of methyl-tertiary butyl ether at the HPRT gene in CD-1 mice. *Toxicologist* 15:79 (abstract).

Weil, C.S. 1970. Significance of organ-weight changes in food safety evaluation. In: Roe, F.J., ed., *Metabolic Aspects of Food Safety*. New York, NY, Academic Press, pp. 419-454.

White, M.C., Johnson, C.A., Ashley, D.L., Buchta, T.M. and D.J. Pelletier. 1995. Exposure to methyl tertiary-butyl ether from oxygenated gasoline in Stamford, Connecticut. *Arch. Environ. Health* 50(3):183-189. (also CDC 1993b. Centers for Disease Control and Prevention. An investigation of exposure to methyl tertiary-butyl ether among motorists and exposed workers in Stamford, Connecticut. Atlanta, GA: U.S. Department of Health and Human Services, National Center for Environmental Health. September 14, 1993.)

Williams, R.T. 1959. *Detoxication Mechanisms*, 2nd Ed., p. 67, John Wiley and Sons, Inc., New York. (As cited in Cederrbaum, A.I, G. Cohen. 1980. Oxidative demethylation of t-butyl alcohol by rat liver microsomes. *Biochem. Biophys. Res. Comm.* 97: 730-736.)

Woutersen R.A., van Garderen-Hoetmer A., Bruijntjes J.P., Swart A., and V.J. Feron. 1989. Nasal tumors in rats after severe injury to the nasal mucosa and prolonged exposure to 10 ppm formaldehyde. *J. Appl. Toxicol.* 9: 39-46.

Wyngaarden, J.B. 1986. New nonsurgical treatment removes gallstones. *JAMA.* 256:1692.

Young, W.F., Horth, H., Crane, R., Ogden, T., and M. Arnott. 1996. Taste and odor threshold concentrations of potable water contaminants. *Water Res.* 30:331-340.

NOTICE OF HEALTH ADVISORY FOR METHYL TERTIARY-BUTYL ETHER (MTBE)

Prepared by
Office of Chemical Safety
Illinois EPA
June 9, 1994

REASON FOR ACTION

As a result of routine monitoring of public water supply systems, the gasoline additive Methyl Tertiary-Butyl Ether (MTBE) has been detected at least in two public water supplies. Therefore, the Illinois Environmental Protection Agency (Agency) is announcing its intention to issue a health advisory, pursuant to 35 Illinois Administrative Code Part 620 Subpart F: Health Advisories, for Methyl Tertiary-Butyl Ether. According to Section 620.605 of Subpart F, the Agency shall issue a health advisory for a chemical substance if all of the following conditions are met:

- 1) A community water supply well is sampled and a substance is detected and confirmed by resampling;
- 2) There is no standard under Section 620.410 for such chemical substance; and
- 3) The chemical substance is toxic or harmful to human health according to the procedures of Appendix A, B, or C.

The Agency has determined that all three conditions have been met, prompting the issuance of this draft proposal for a health advisory. By this issuance, the Agency is opening a 30-day public comment period, until August 22, 1994, regarding this health advisory draft. Upon closing the public comment period, the Agency will consider all comments received and amend the health advisory if warranted. The final health advisory will then be published in the Environmental Register (the Illinois Pollution Control Board News) with responses to comments received. An abbreviated version of the final health advisory will also be published in local newspapers which serve communities in whose public water supply systems MTBE has been detected.

PROPOSED GUIDANCE LEVELS

Section 620.605 of Subpart F prescribes the methods for developing health advisories for carcinogens and noncarcinogens. Since the Agency has determined that there is insufficient evidence of the carcinogenicity of MTBE at this time (discussed in the attachment to this notice), the method for developing a health advisory for noncarcinogens was used. Briefly, this method specifies that the USEPA's maximum contaminant level goal (MCLG) is the guidance level, if available, or the human threshold toxicant advisory concentration (HTTAC) must be determined using the procedures contained in Appendix A of Section 620. USEPA has not published an MCLG for MTBE, therefore the Agency used the Appendix A procedures to calculate the HTTAC.

Appendix A specifies in prescribed order the toxicological data to be used in developing the HTTAC, ranging from a verified Reference Dose developed by USEPA to a laboratory animal study of subchronic duration in which only a lowest observable adverse effect level (LOAEL) has been determined. This preferred order reflects increasing uncertainty in the toxicological database regarding a chemical's potential to cause adverse health effects in humans, and is manifested in increasingly large safety factors which are applied to the data to calculate the HTTAC (maximum 10,000-fold safety factor).

In the case of MTBE, the Agency has selected the only study available in which the test animals were exposed by the oral route of exposure as the basis for the HTTAC. Among other findings, this 90-day subchronic study reported increases in serum cholesterol and persistent diarrhea in all doses tested, including the lowest dose of 100 mg/kg/d. As a result of using this subchronic study in which only a LOAEL was determined, the language of Subpart F specifies the application of safety factors totalling to 10,000 to the animal data, resulting in the HTTAC guidance level of 0.07 mg/l, or 70 parts per billion (ppb). The details of the derivation of the HTTAC are presented in the attachment to this notice.

At this point it is necessary to discuss an aspect of the evolving science of risk assessment which has a bearing on this notice. The Agency has been informed verbally by USEPA personnel that in most cases USEPA no longer favors the

calculation of acceptable exposure values for humans by using laboratory animal data divided by uncertainty factors totalling to 10,000. This preference will be included in a chapter in the book Essential Elements (in press; ILSI Press, 1994). Instead, USEPA now prefers to utilize uncertainty factors totalling to no more than 3,000. The Agency agrees with this approach in general, except in cases where the overall toxicity database for a chemical is very weak. In the case of MTBE, the database contains enough laboratory animal data to determine that there are not major toxicity gaps which would warrant the use of a 10,000-fold uncertainty factor. The Agency is therefore also using an overall uncertainty factor, of 3,000 to calculate a guidance level for MTBE. Use of a 3,000-fold safety factor with the same laboratory animal data described above results in a HTTAC guidance level of 0.23 mg/l, or 230 ppb. The details of the derivation of this HTTAC are also presented in the attachment to this notice.

Since there is no provision in the language of Subpart F for the use of a 3,000-fold uncertainty factor in the derivation of the HTTAC, the Agency is proposing to utilize HTTACs derived by both a 3,000-fold and a 10,000-fold uncertainty factor in the health advisory for MTBE. It is proposed that the HTTAC derived using the 10,000-fold uncertainty factor (70 ppb) be a precautionary health advisory concentration and the HTTAC derived using the 3,000-fold uncertainty factor (230 ppb) be the final health advisory concentration. The precautionary health advisory would be a level in a public water supply below which no action would be necessary and above which caution should be exercised by the public water supply (such as increased sampling of the water and identification of the potential source(s)), while the final health advisory would be a level above which the public water supply should begin actions to decrease the concentration or utilize an alternate water supply. The Agency is requesting comment on the use of this approach when a total uncertainty factor of 10,000-fold is utilized to calculate a health advisory.

SUPPLEMENTARY INFORMATION

Section 620.605 also specifies that the health advisory must contain a general description of the characteristics of the chemical substance and its potential adverse health effects.

General Description of MTBE

MTBE (Chemical Abstracts Service Number 1634-04-4), also known as 2-methoxy-2-methylpropane, is a colorless liquid with a disagreeable taste and odor. Its taste in water can be recognized at approximately 0.7 mg/l (700 ppb) (Connecticut DEP), although recent research suggests that some people may be able to detect its presence in the range of 0.25 mg/l and possibly as low as 0.04 mg/l (API, 1993). It has a high solubility in water, approximately 48,000 mg/l (von Burg, 1992). Because of this high solubility, it has a high propensity to move through soil with infiltrating rainwater and snowmelt and to potentially reach groundwater.

Its main use is as an octane booster in unleaded gasoline; it also has minor uses as an intermediate in the production of other chemicals, especially isobutene, and as a treatment to dissolve gallstones. Its use has been increasing recently due to requirements under the Clean Air Act Amendments of 1990 for metropolitan areas which are not in compliance with carbon monoxide standards to increase the percentage of oxygenated fuel in gasolines, especially in the wintertime. As a result, it has been estimated that approximately 20% of the gasoline sold in the United States contains MTBE, at levels ranging from 2% to 15% in the gasolines (Costantini, 1993).

Potential Adverse Health Effects of MTBE

Relatively few reports of adverse effects of MTBE on humans exist, and testing for the full range of possible health effects in laboratory animals has not yet been completed. Summaries of the acute, reproductive and developmental, and chronic toxicity data for MTBE are presented.

Acute Toxicity - Other than a single report in the medical literature of acute kidney failure due to leakage of MTBE during gallstone treatment (Ponchon, 1988), there is no information regarding the effects of short-term, high level exposure to MTBE in humans. The data from laboratory animal studies indicate that this chemical is not very toxic during brief exposures, with lethal doses in the range of 3,000-4,000 ppm by oral exposure (about one pint for an adult human) and 24,000-40,000 ppm (in air) by inhalation exposure (this would be within the explosive range in air) (Reese and Kimbrough, 1993; von Burg, 1992; USEPA, 1993). The toxic effect in both exposure types was central nervous system depression. MTBE does not appear to cause skin irritation except in cases of previously damaged skin, and eye irritation and opacity of the cornea has been reported (von Burg, 1992).

Reproductive and Developmental Toxicity - The reproductive effects of MTBE have been reported in three studies, and reproductive and developmental toxicity has been assessed in a fourth, using rats, mice, and/or rabbits. No significant effects were reported in two of the reproductive studies (Biles *et al.*, 1987; Conaway *et al.*, 1985), and the third reported effects on offspring (reduced body weight and reduced weight gain in rat pups, and slightly reduced pup survival) only at doses which were also toxic to the parents (Neeper-Bradley, 1991). Similarly, the reproductive and developmental study also reported offspring effects (reduced numbers of viable implantations and/or live births, reduced body weight, decreased ossification, and increased incidence of cleft palate in mouse pups) only at doses toxic to the adults (Tyl and Neeper-Bradley, 1989). This makes it difficult to say whether the effects on reproductive performance were truly an effect of MTBE on the offspring, or whether these effects resulted from the toxicity to the parents. Since the doses which showed these toxic effects were high (3,000-4,000 ppm), the potential for human reproductive effects at the much lower anticipated environmental exposure levels is extremely small.

Chronic Toxicity - There are no studies of the effects on humans exposed to MTBE for long periods, although anecdotal reports of increased complaints of headache, nausea, vomiting, eye irritation, and respiratory problems have surfaced recently in certain areas in conjunction with wintertime MTBE increases in gasoline. These complaints are the subject of on-going research.

There is only one 90-day subchronic study in laboratory animals exposed by the oral route, which was the study finally selected to derive the HTTAC by the Agency after following the procedures of Appendix A. This study is evaluated in depth in the attachment to this notice. There are several animal subchronic and chronic studies using the inhalation route of exposure, primarily evaluating the neurotoxic effects of MTBE. In one study (Greenough *et al.*, 1980) in which the maximum dose tested was 1,000 ppm for 6 hrs/day, 5 days/wk, for 13 weeks, no significant effects (other than anesthesia following dosing at high concentrations) were reported. In another study (Dodd and Kintigh, 1989), in which the maximum dose tested was 8,000 ppm (same dosing regimen), slight changes in blood chemistry, increased serum cortisone levels in both sexes, reduced weight gain, increased kidney, liver, and adrenal gland weights, and sporadic neurotoxic effects were seen at doses of 4,000 and/or 8,000 ppm. There is also a recently completed lifetime cancer bioassay in mice and rats (Burleigh-Flayer *et al.*, unpublished; Chun *et al.*, unpublished), the details of which are evaluated in the attachment to this notice.

FOR FURTHER INFORMATION, COMMENTS

Persons who wish to receive further information about this notice or who wish to provide comment on its contents are requested to contact:

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ATTACHMENT TO NOTICE OF HEALTH ADVISORY FOR
METHYL TERTIARY-BUTYL ETHER (MTBE)OVERVIEW OF THE KEY STUDIES

In the only oral study (Robinson *et al.*, 1990), rats were given 0, 100, 300, 900, or 1,200 mg/kg (ppm) by gavage. Rats given 1,200 ppm exhibited profound anesthesia after dosing throughout the study, but recovered after the dose within two hours and suffered no aftereffects. Body weight decreased with increasing dose, with the difference between treated and control rats being statistically significant at 1,200 ppm. Other measurements showing statistical significance included: decreased blood urea nitrogen (BUN) and serum creatinine (measures of kidney function) at all doses; increased serum cholesterol at all doses; increased kidney weight at 300 ppm and above; increases in several other organ weights at 900 ppm and above; and changes in blood parameters at 1,200 ppm. Microscopic examinations revealed effects only at 1,200 ppm, where degenerative changes in the kidneys of the male rats were noted. Finally, loose stools and diarrhea were seen at all doses throughout the study.

Viewing the results of this study, it would appear that the kidney is the target organ of MTBE. However, these results must be interpreted carefully. The decreases in BUN and serum creatinine probably have no adverse effect on the animals (decreased kidney function is often signaled by increases in these parameters), and may even indicate an increase in kidney function. The increased kidney weights seen at 300 ppm and above are not in themselves an adverse effect, only an indication of a possible adverse effect at even higher doses or longer exposure times. Finally, the microscopic changes seen at 1,200 ppm in males are often seen in male rats (and only male rats) exposed to certain organic chemicals, due to overproduction of a unique protein in the male rat kidney. Thus, it is not clear at this time whether MTBE is toxic to the kidney.

It would appear that a no observed adverse effect level (NOAEL) has not been determined by this study, since increased serum cholesterol and diarrhea were observed at all doses. Thus, the 100 ppm dose would be considered to be the lowest observable adverse effect level (LOAEL) for MTBE. The procedure for calculating a health advisory for drinking water in the groundwater quality standards (35 Ill. Adm. Code 620, Subpart F) gives preference to oral studies which determine a NOAEL or LOAEL, and this study may be considered to develop the health advisory for MTBE.

A lifetime inhalation cancer bioassay has recently been completed with mice and rats, but the results have not been published (Burleigh-Flayer *et al.*; Chun *et al.*). The Agency has been given summaries of the studies submitted to USEPA by the USEPA contact for MTBE. These results are briefly summarized, but since the studies are still undergoing review it must be realized that this information is preliminary.

Both species were exposed to 0, 400, 3,000, or 8,000 ppm in air. As in the oral study above, the male rats experienced an increased incidence of kidney degeneration. This became the leading cause of death in male rats, and resulted in early termination of the 3,000 and 8,000 ppm male groups. The other main cause of death in male rats was leukemia, seen in both the control and 400 ppm group. (In fact, the incidence in the control group was higher, 33/50, than in the 400 ppm group, 22/50.) Non-cancer effects of MTBE included symptoms of central nervous system depression in both sexes of rats at 3,000 and 8,000 ppm, but not at 400 ppm, and an increased incidence of kidney degeneration in male rats at 400 ppm. The only tumors which were related to MTBE exposure were tumors in the kidneys of male rats in the 3,000 and 8,000 ppm groups. These tumor types are also thought to be related to the overproduction of the male rat protein, and the significance of these results for humans is questionable.

In the mouse study, symptoms of central nervous system depression similar to those seen in rats were observed at 3,000 and 8,000 ppm. Increases in liver and kidney weights were also seen at these doses, and an increase in the number of liver cells (noncancerous), an indication of toxic effects on the liver, was reported at 8,000 ppm. The only tumors found in excess of controls were liver tumors in females in the 8,000 ppm group. However, the significance of this finding for humans is also questionable, since this tumor type is common in the strain of mouse used in this study, and is known to occur in controls at a relatively high rate.

In reviewing the results of these studies, it is difficult to say whether MTBE presents a carcinogenic hazard to humans. However, the noncancer effects may be relevant for determining a health advisory level for MTBE. In this regard, the rat study has produced a LOAEL of 400 ppm based on kidney effects in male rats (this dose may be a NOAEL given the questionable significance of this effect for humans), while the mouse study has produced a NOAEL of 400 ppm. The mouse

portion of this study may be considered to develop the health advisory for MTBE, once it has finished USEPA's review process.

DERIVATION OF THE HEALTH ADVISORY FOR MTBE

The first step in the derivation of a health advisory is to determine whether the chemical presents a carcinogenic hazard to humans. To date, there have been no investigations whether there is an increased incidence of cancer in humans associated with exposure to MTBE. As discussed above, there is some evidence that MTBE causes tumors in laboratory animals, but the types of tumors found in the rat and mouse cancer bioassays may not provide good evidence of a carcinogenic hazard to humans since these tumors may be species-specific responses with little or no relevance to humans. Furthermore, these studies are still undergoing review by USEPA and a final determination of the usability of the results for determining the carcinogenic hazard to humans has not been made. Therefore, the Agency has determined at this time that the derivation of the health advisory for MTBE will be based on the non-cancer effects of this chemical. This derivation may be changed in the future, depending on the USEPA's determinations, once the cancer bioassay data have been published and the weight-of-evidence for human carcinogenic potential has been determined.

In deriving a health advisory to protect against a health effect for which there is a threshold dose below which no damage occurs (i.e., noncarcinogenic effects), Section 620.605 specifies that USEPA's maximum contaminant level goal (MCLG), if available, is the health advisory concentration. USEPA has not published a MCLG for MTBE, therefore, the Agency must calculate the human threshold toxicant advisory concentration (HTTAC) as the health advisory concentration, using the procedures specified in Appendix A of Section 620.

Appendix A specifies in subsection (a) that the HTTAC is calculated as follows:

$$HTTAC = \frac{RSC \times ADE}{W}$$

Where:

- HTTAC = Human threshold toxicant advisory concentration in milligrams per liter (mg/l);
- RSC = Relative source contribution, the relative contribution of the amount of the exposure to a chemical via drinking water when compared to the total exposure to that chemical from all sources. Valid chemical-specific data shall be used if available. If valid chemical-specific data are not available, a value of 20% (=0.20) must be used;
- ADE = Acceptable daily exposure of substance in milligrams per day (mg/d) as determined pursuant to subsection (b); and
- W = Per capita daily water consumption equal to 2 liters per day (L/d).

Subsection (b) of Appendix A specifies that the ADE be calculated using, in specified order: USEPA's Verified Oral Reference Dose (an estimate of a daily exposure to a chemical which is expected to be without adverse effect for humans, including sensitive subgroups, for a lifetime of exposure); a NOAEL which has been identified as a result of human exposures; a LOAEL which has been identified as a result of human exposures; a NOAEL which has been determined from studies with laboratory animals; and a LOAEL which has been determined from studies with laboratory animals.

There is no Verified Reference Dose currently available from USEPA. As mentioned above, there is a paucity of studies on the adverse effects in humans exposed to MTBE. Thus, the Agency has determined that a NOAEL or LOAEL based on human exposures is not available at this time. Therefore, the ADE must be calculated from laboratory animal data. Of the studies reviewed by the Agency, the 90-day rat subchronic study and the cancer bioassay (noncarcinogenic effects) are the most appropriate animal studies for calculation of the ADE. It is then necessary to determine which study is the most valid for purposes of calculating the ADE.

Subsection (c) of Appendix A specifies criteria for establishing the validity of data from animal studies, leading to determinations of high, medium, or low validity. High validity studies are those using the oral route of exposure and which

meet specified criteria depending on the type of study, and are to be used preferentially if available. The rat 90-day subchronic study was conducted using the oral route, while the cancer bioassay was an inhalation study. Therefore, only the subchronic study could be a high validity study. However, the requirements for a high validity subchronic study include, among other things, a study using two species and determining a well-defined NOAEL. The 90-day rat subchronic study used only one species and only determined a LOAEL, as discussed above. Having no high validity study, the Agency must determine which of the two studies is most appropriate for calculating the ADE.

Subsection (c) goes on to specify that in order for a subchronic study in which a LOAEL is determined to be deemed a medium validity study, the study must satisfy all other standards for a high validity study. This is not the case for the 90-day rat subchronic study, since there was only one species tested. Similarly, in order for a study other than an oral exposure study to be deemed a medium validity study, the study must satisfy all other standards for a high validity study and use appropriate correction factors for conversion to the oral route. However, the requirements for a high validity cancer bioassay include, among other things, at least 25% survival at 18 months in mice and 24 months in rats. This was not the case in the cancer bioassay, since the male rats in the 3,000 and 8,000 ppm groups were terminated early due to excessive mortality. Thus, both candidate studies are defined as low validity studies, and the 90-day rat subchronic study is selected because exposure was by the oral route.

The determination of the ADE from the subchronic study is made using the language of subsections (b)(5) and (b)(6). Subsection (b)(6) specifies that for substances for which a NOAEL is not available, one-tenth of the LOAEL is substituted for the NOAEL in subsection (b)(5). Subsection (b)(5) specifies that if studies of low validity must be used, the ADE must be calculated using 1/1000 of the NOAEL. The overall result of the procedures in these two subsections is that the ADE is 1/10,000 of the LOAEL, times the average weight of an adult human, 70 kg:

$$ADE = \frac{100\text{mg/kg/d} \times 70\text{kg}}{10,000\text{kg/d}} = 0.7\text{mg/d}$$

At this point, the calculation of the HTTAC would proceed according to the formula listed above. However, the Agency has been informed by USEPA personnel that in most cases USEPA now prefers to calculate acceptable exposure values for humans by using laboratory animal data divided by no more than a 3,000-fold uncertainty factor; a 10,000-fold uncertainty factor would be used only where the overall toxicity database is very weak for a chemical. The Agency agrees with this emerging USEPA approach. Since the MTBE database contains enough laboratory animal research to indicate that there are not major toxicity data gaps which would warrant the use of a 10,000-fold uncertainty factor, the Agency is also calculating the ADE using a 3,000-fold uncertainty factor:

$$ADE = \frac{100\text{mg/kg/d} \times 70\text{kg}}{3,000} = 2.3\text{mg/d}$$

Finally, the determination of the HTTAC is straight-forward, since there are no chemical-specific data available for the RSC term:

$$HTTAC = \frac{0.20 \times 0.7\text{mg/d}}{2.0\text{d}} = 0.07\text{mg/l}$$

Or:

$$HTTAC = \frac{0.20 \times 2.3\text{mg/d}}{2.0\text{d}} = 0.23\text{mg/l}$$

The final step in determining the health advisory is to compare the HTTAC value calculated from the Appendix A procedures to the chemical's Practical Quantitation Limit (PQL). In the case of MTBE, no USEPA SW-846 analytical method specifies a PQL for this chemical. However, the Agency's Division of Laboratories has determined that a detection limit of 0.005 mg/l is appropriate for water samples. Therefore, the HTTAC value is above the detection limit.

The Agency has decided to issue a two-part health advisory. The precautionary health advisory concentration for Methyl Tertiary-Butyl Ether (MTBE) is 0.07 mg/l or 70 parts per billion in drinking water. People can be exposed to this concentration of MTBE in drinking water over a 70 year lifetime. Above this concentration, appropriate caution should be exercised by the Public Water Supply, such as increased frequency of sampling and identification of the MTBE source(s). The final health advisory concentration is 0.23 mg/l or 230 parts per billion in drinking water. Above this concentration, the Public Water Supply should begin actions to decrease the amount of MTBE in the system.

REFERENCES

- API, American Petroleum Institute. 1993. Odor Threshold Studies Performed with Gasoline and Gasoline Combined with MTBE, ETBE, and TAME. API Publication Number 4592.
- Biles, R. W., Schroeder, R. E., and Holdsworth, C. E. 1987. Methyl Tertiary Butyl Ether Inhalation in Rats: A Single Generation Reproductive Study. *Toxicol. Ind. Health* 3: 519-534.
- Burleigh-Flayer, H. D., Chun, J. S., and Kintigh, W. J. (unpublished). Methyl Tertiary Butyl Ether: Vapor Inhalation Oncogenicity Study in CD-1 Mice. Submitted to USEPA, Docket No.: OPTS-42098.
- Chun, J. S., Burleigh-Flayer, H. D., and Kintigh, W. J. (unpublished). Methyl Tertiary Butyl Ether: Vapor Inhalation Oncogenicity Study in Fischer 344 Rats. Submitted to USEPA, Docket No: OPTS-42098.
- Conaway, C. C., Schroeder, R. E., and Snyder, N. K. 1985. Teratology Evaluation of Methyl Tertiary-butyl Ether in Rats and Mice. *J. Toxicol. Environ. Health* 16: 797-809.
- Connecticut Dept. of Environmental Protection. (undated). Action Level for Methyl Tertiary Butyl Ether (MTBE) in Drinking Water. Prepared by H. V. Rao, C. J. Dupuy, and D. R. Brown, Connecticut Dept. of Health Services.
- Costantini, M. G. 1993. Health Effects of Oxygenated Fuels. *Environ. Health Perspectives Suppl.* 101 (Suppl. 6): 151-160.
- Dodd, D. E. and Kintigh, W. J. 1989. Methyl Tertiary Butyl Ether (MTBE): Repeated (13-Week) Vapor Inhalation Study in Rats with Neurotoxicity Evaluation (unpublished study). Union Carbide, Bushy Run Research Center for MTBE Committee. TSCATS 403189. EPA/OTS # FY1-OTS-0889-0689. Cited in Revised and Updated Drinking Water Quantification of Toxicological Effects for Methyl Tert-Butyl Ether (MTBE), Final Draft. USEPA; ECAO-CIN-D023, July, 1993.
- Essential Elements (in press). ILSI Press, Washington, D.C. 1994.
- Greenough, R. J., McDonald, P., Robinson, P., et al. 1980. Methyl Tertiary-Butyl Ether (Driveron) Three Month Inhalation Toxicity in Rats. Project No. 413038. Unpublished report submitted to Chemische Werke Höls AG, Marl, West Germany. 230 p. Cited in Revised and Updated Drinking Water Quantification of Toxicological Effects for Methyl Tert-Butyl Ether (MTBE), Final Draft. USEPA; ECAO-CIN-D023, July, 1993.
- Neeper-Bradley, T. L. 1991. Two-Generation Reproduction Study of Inhaled Methyl tert-Butyl Ether in CD Sprague-Dawley Rats (unpublished study). Union Carbide, Bushy Run Research Center. Cited in Revised and Updated Drinking Water Quantification of Toxicological Effects for Methyl Tert-Butyl Ether (MTBE), Final Draft. USEPA; ECAO-CIN-D023, July, 1993.
- Ponchon, T., Baroud, J., Pujol, B., Valette, P. J., and Perrot, D. 1988. Renal Failure during Dissolution of Gallstone by Methyl Tert-Butyl Ether. *Lancet* 2: 276-277.
- Reese, E., and Kimbrough, R. D. 1993. Acute Toxicity of Gasoline and Some Additives. *Environ. Health Perspectives Suppl.* 101 (Suppl. 6): 115-131.
- Robinson, M., Bruner, R.H., and Olson, G.R. 1990. Fourteen and Ninety-Day Oral Toxicity Studies of Methyl Tertiary-Butyl Ether in Sprague-Dawley Rats. *Journal of the American College of Toxicology* 9(5): 525-540.

Tyl, R. W., and Neepet-Bradley, T. L. 1989. Developmental Toxicity Study of Inhaled Methyl Tertiary Butyl Ether in CD-1 Mice. Project Report 52-526. Prepared by Bushy Run Research Center, Union Carbide Corporation for the Methyl Tertiary Butyl Ether Committee, Washington, D.C. Microfiche No. OTS0000689-1. Cited in Revised and Updated Drinking Water Quantification of Toxicological Effects for Methyl Tert-Butyl Ether (MTBE), Final Draft. USEPA; ECAO-CIN-D023, July, 1993.

USEPA. 1993. Revised and Updated Drinking Water Quantification of Toxicological Effects for Methyl Tert-Butyl Ether (MTBE), Final Draft. USEPA; ECAO-CIN-D023, July, 1993.

von Burg, R. 1992. Toxicology Update. Methyl Tert-Butyl Ether. J. Appl. Toxicol. 12: 73-74.



State of Illinois
ENVIRONMENTAL PROTECTION AGENCY

Mary A. Gade, Director

2200 Churchill Road, Springfield, IL 62794-9276

217/785-0830

November 4, 1994

G.A. Van Gelder, DVM, Ph.D., ABVT
Manager, Toxicology
Health, Safety and Environment
Shell Oil Company
One Shell Plaza
P.O. Box 4320
Houston, TX 77210

Dear Dr. Van Gelder:

This letter confirms the meeting to evaluate comments received regarding the Illinois Environmental Protection Agency's proposed Health Advisory for MTBE which we discussed over the telephone. The meeting is scheduled for November 14, 1994, beginning at 12:30. The room is available until 5:00 PM, if necessary. The meeting will be held in Room 031 on Floor 8, James R. Thompson Center, 100 W. Randolph, Chicago, Illinois, 60601.

I have enclosed an agenda for the meeting, a copy of the Health Advisory Section of the Illinois Groundwater Quality Standards, and a summary of the Agency's opinions on two key issues which have emerged from the comments.

I'm looking forward to a productive meeting. Please call (217/785-0830) if you have any further comments or questions.

Sincerely,

A handwritten signature in cursive script that reads "Tom Hornshaw".

Thomas C. Hornshaw, Ph. D.
Manager, Toxicity Assessment Unit
Office of Chemical Safety

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Attachment

MTBE Meeting Agenda

- | | |
|---------------|--|
| 12:30 - 12:45 | Introductions and Background |
| 12:45 - 1:45 | Key Issues (LOAEL vs. NOAEL, RSC) |
| 1:45 - 2:00 | Break |
| 2:00 - 3:15 | Other Issues (Tase/Odor Threshold, Uncertainty Factors, 2-Tier Vs. Single Advisory, Edits) |
| 3:15 - 3:30 | Wrap-up |

RESPONSES TO SIGNIFICANT COMMENTS REGARDING PROPOSAL FOR HEALTH ADVISORY FOR METHYL TERTIARY-BUTYL ETHER

The Illinois Environmental Protection Agency (Agency) has received three comments in response to the Notice of Health Advisory for Methyl Tertiary-Butyl Ether (MTBE), published in the Illinois Environmental Register No. 484, July, 1994. The comments were received from the American Petroleum Institute (API), the Methyl Tertiary Butyl Ether Task Force (Task Force), and Shell Oil Company (Shell). The comments cover several technical and typographical subjects, the most significant of which address the Agency's determination of a Lowest Observable Adverse Effect Level (LOAEL) versus a No Observable Adverse Effect Level (NOAEL) and the uncertainty factors which result from this determination, and the Agency's use of the default value of 20% as the Relative Source Contribution (RSC) term versus the use of an RSC derived from chemical-specific data in the calculation of the Health Advisory. The Agency's responses to these key issues are presented in this paper.

LOAEL vs. NOAEL

API and Shell disagree with the Agency's characterization of the diarrhea and elevated serum cholesterol reported at the 100 mg/kg dose in the Robinson et al. (1990) study as a LOAEL. In reviewing the results of this study, the Agency determined that the authors' reports that "treated rats in all dose groups also displayed diarrhea throughout the exposure period" and their findings that "females exposed to all dose levels exhibited significant increases in serum cholesterol" indicated that the study had not identified a No Observed Adverse Effect Level. This determination is an outcome of the evaluation of the validity of the candidate studies required by the Groundwater Quality Standards regulation when animal studies must be used to develop a Health Advisory. This evaluation was discussed briefly in the July, 1994 Notice, and will be expanded for explanation of the Agency's rationale.

Section 620. Appendix A(c)(1)(A)(iii), which identifies the elements necessary for High Validity Studies, requires:

Data from animal subchronic studies with a minimum of 3 dose levels and control, 2 species, both sexes, 4 animals per dose per sex for non-rodent species or 10 animals per dose per sex for rodent species, a duration of at least 5% of the test species' lifespan, and a well-defined NOAEL (emphasis added).

The Agency determined that the reports of diarrhea in all animals and elevated serum cholesterol in females in all dose groups could not be called a "well-defined NOAEL" for purposes of establishing High Validity for this study. Thus, the lowest dose tested, 100 mg/kg, was determined to be a LOAEL.

API and Shell have commented that the results of the study should not be interpreted in this manner. Both claim that the occurrence of diarrhea in treated animals is not well-documented

or described in the Robinson study, that diarrhea is a common observation in rats dosed with corn oil, and it is a questionable endpoint for extrapolation to low-dose lifetime health effects. Both also claim that the modest increases in serum cholesterol in the female rats are not indicative of a meaningful health effect, arguing that the authors' statistical evaluation incorrectly attributes a significant difference for the 300 mg/kg dose, that there is no compelling evidence for a dose response, that only the 900 mg/kg dose in males achieved values significantly different from controls, and that the increases are near the range of normal variability. Finally, API argues that the diarrhea and elevated serum cholesterol are not significant results, citing the authors' conclusions that the study indicated that dose levels below those which induce anesthesia (1200 mg/kg) do not result in significant pathophysiological changes.

The Agency remains unconvinced that the Robinson et al. study has identified a well-defined NOAEL. Regarding the occurrence of diarrhea, we have interpreted the authors' reports of diarrhea in "treated rats in all dose groups" to mean all groups receiving doses of MTBE, but not those receiving the vehicle control (corn oil). Thus, we believe that the diarrhea is likely to be treatment-related, at least in females; this belief is supported by the findings of the 14-day study also reported in this paper, in which "by the third day of dosing, all treated animals displayed loose stools which continued throughout the remainder of the exposure period." We have reviewed the National Toxicology Program's report on the lifetime cancer bioassays of gavage vehicles in male Fisher rats, which included corn oil, and find no mention of diarrhea as an effect of corn oil (NTP, 1994). Finally, we have relied on the experience of one of the Agency's Office of Chemical Safety toxicologists, who reports that, in over 8 1/2 years of experience in an industrial toxicology laboratory, the occurrence of diarrhea in rats in conjunction with corn oil vehicles was very infrequent (Morrow, 1994). While we cannot rule out the possibility that the diarrhea reported by Robinson et al. was vehicle-related, we continue to believe that this effect was a result of the MTBE exposure.

Regarding the elevated serum cholesterol findings, the Agency acknowledges that the statistical significance of the 300 mg/kg dose in female rats is questionable and possibly incorrectly reported, and that there is no obvious dose-response relationship among the female treatment groups even though all but the 300 mg/kg group is significantly greater than controls. However, we maintain that these results are potentially indicative of a real effect in the rats; it is possible (although unlikely) that the effect may plateau relatively quickly, such that the dose-response relationship is defined at doses below those tested in this study. Further, we again note that the results of the 14-day study reported in this paper also include elevated serum cholesterol in females of most treatment groups.

Regarding the biological significance of the diarrhea and elevated serum cholesterol and whether these endpoints are relevant for extrapolating to human health risks, the Agency maintains that such effects are relevant for use in developing the Health Advisory. While neither endpoint is relatively serious, diarrhea can be deleterious to the organism over time by contributing to dehydration, electrolyte imbalance, and/or poor nutritional status, and elevated cholesterol, while not in itself a biologically serious effect, is a caution for more serious effects over time. While the authors' concluded that dose levels below those which induce anesthesia do not result in significant pathophysiological changes, the Agency would be very uncomfortable using a dose which does not induce anesthesia as the basis for developing a Health Advisory. We continue

or described in the Robinson study, that diarrhea is a common observation in rats dosed with corn oil, and it is a questionable endpoint for extrapolation to low-dose lifetime health effects. Both also claim that the modest increases in serum cholesterol in the female rats are not indicative of a meaningful health effect, arguing that the authors' statistical evaluation incorrectly attributes a significant difference for the 300 mg/kg dose, that there is no compelling evidence for a dose response, that only the 900 mg/kg dose in males achieved values significantly different from controls, and that the increases are near the range of normal variability. Finally, API argues that the diarrhea and elevated serum cholesterol are not significant results, citing the authors' conclusions that the study indicated that dose levels below those which induce anesthesia (1200 mg/kg) do not result in significant pathophysiological changes.

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to believe that the 100 mg/kg dose, as a LOAEL, is the most relevant value to use in the development of the Health Advisory. This reasoning, plus the relative paucity of data regarding the ingestion of MTBE, argues for the continued use of the 3000-fold uncertainty factor as the most appropriate value for the final Health Advisory.

MTBE RELATIVE SOURCE CONTRIBUTION TERM

The comments of both API and the Task Force addressed the Agency's use of the default value of 20% as the Relative Source Contribution (RSC) term, which is specified in Section 620. Appendix A(a). (This is also a standard USEPA default assumption, used in risk assessments to account for all other exposures to a chemical other than direct ingestion in drinking water, such as through the diet, ambient air, the workplace, and volatilization from the household water supply).

Both comments cite a USEPA study (USEPA, 1993) which estimates the amount of MTBE exposure experienced by the general public during activities other than drinking water, such as working, outdoor exercise, refueling, driving, etc. This study is proposed to be used as chemical-specific data instead of the default value to account for exposures to MTBE other than via direct ingestion of drinking water. If this study is used to define the RSC term, the range of weighted annual MTBE ambient air concentrations of 0.04 - 0.07 mg/m³ would result in a RSC term of approximately 45% - 70% for drinking water exposures. Depending on the final determination of the RSC term, the Health Advisory (HA) for MTBE would then be in the range of 0.52 - 0.80 mg/l, instead of the proposed 0.23 mg/l using a 20% RSC term.

While the Agency agrees with the data presented in the USEPA study, it cannot agree that these data fully account for all other sources of MTBE contributing to a person's daily exposure. Use of only this study to account for inhalation exposures does not consider inhalation exposures which will occur in the home as a result of volatilization of MTBE from the household water supply during uses of the supply for purposes other than drinking. Since the Agency is not aware of studies evaluating such exposures, an evaluation of the indoor inhalation pathway was undertaken using data reported for trichloroethylene (TCE).

The transfer of volatile organic chemicals (VOCs), including TCE, from water to air has been studied by several investigators (Andelman, 1985; McKone, 1987; McKone and Knezovich, 1991). Of particular interest for this analysis are studies which measure the transfer of VOCs during showering since this activity is likely to be the greatest contributor to indoor VOC exposure due to the temperature, amount of water used, turbulent flow, and the relatively small volume of air in the bathroom. Therefore, the McKone and Knezovich study, which measures the evolution of TCE into a bathroom's air during operation of the shower, was selected for development of the transfer rate of MTBE to the air during showering. This study evaluated the effects of shower temperature and duration on the transfer efficiency of TCE from water to air, concluding that the transfer efficiency is $61 \pm 9\%$ and that inhalation exposures in the shower could be equivalent to an ingestion exposure

of from 1-4 liters per day.

Assuming that the transfer efficiency of any VOC for which transfer efficiency has not been measured is directly proportional to that of another VOC having a measured transfer efficiency, the transfer efficiency of MTBE from water to air can be estimated from the TCE data by comparing the overall mass transfer coefficients from water to air (K_{LA}) for both chemicals. McKone (1987) has shown that K_{LA} can be approximated by:

$$K_{LA} \approx \left[\frac{2.5}{D_{BL}^{2/3}} + \frac{RT}{HD_{BA}^{2/3}} \right]^{-1}, \text{ where}$$

D_{BL} = diffusion coefficient in water (m^2/s),

D_{BA} = diffusion coefficient in air (m^2/s),

R = universal gas constant, $0.0624 \text{ torr}\cdot m^3/\text{mol}\cdot K$,

T = temperature, $303K$ (air temperature in hot shower), and

H = Henry's law constant ($\text{torr}\cdot m^3/\text{mol}$).

The diffusion coefficients of TCE and MTBE were calculated according to methods recommended in Lyman (1982), assuming a water temperature of $37^\circ C$ and an air temperature of $30^\circ C$ to be representative of hot shower conditions. The calculated values for TCE and MTBE for D_{BL} are $1.094E-09 \text{ m}^2/s$ and $9.870E-10 \text{ m}^2/s$, respectively, and for D_{BA} are $9.40E-06 \text{ m}^2/s$ and $9.28E-06 \text{ m}^2/s$, respectively.

Substituting the calculated D_{BL} and D_{BA} values and Henry's law constants of $6.916 \text{ torr}\cdot m^3/\text{mol}$ for TCE and $4.484 \text{ torr}\cdot m^3/\text{mol}$ for MTBE into the overall mass transfer coefficient equation, values for K_{LA} were calculated to be $4.236E-07 \text{ m}^2/s$ and $3.950E-07 \text{ m}^2/s$ for TCE and MTBE, respectively. The ratio of the two K_{LA} values of 0.9325 , when compared to the measured TCE transfer efficiency of 61% , suggests an MTBE transfer efficiency of approximately 56.89% .

Once the transfer efficiency has been determined, an estimate of a resident's cumulative daily intake from showering (CDI_s) can be calculated for any VOC water concentration (C_w) using reasonable estimates of water use during showering and the volume of the shower, plus standard USEPA assumptions for body weight (BW , 70 kg) and breathing rate (BR , $20 \text{ m}^3/d = 0.014 \text{ m}^3/\text{min}$). For this exercise, it is assumed that the resident's shower duration (SD) is 10 min/d , the shower flow rate (FR) is 10 l/min , and the volume (V) of the shower is 2.3 m^3 . The CDI_s for any C_w is calculated from:

$$CDI_s = \left[\frac{(FR \times SD \times \text{Transfer Efficiency}) \times BR \times SD}{V \times BW} \right] \times C_w.$$

After substituting, the CDI_s for any C_w becomes:

$$CDI_s = (0.049 \text{ l/kg/d}) \times C_w.$$

This shower inhalation intake can be compared directly with the daily ingestion intake (CDI_i) of the VOC from drinking water for the same C_w by again employing standard USEPA

assumptions for BW (as above) and daily water intake (WI, 2.0 l/d). The CDI_I is calculated from:

$$CDI_I = \frac{WI}{BW} \times C_w,$$

which becomes after substitution:

$$CDI_I = (0.029 \text{ l/kg/d}) \times C_w.$$

These two CDIs are now directly comparable for any water concentration of MTBE. The ratio of CDI_S to CDI_I is 1.69, suggesting that the resident's daily showering contributes approximately 169% of the daily exposure to MTBE compared to the exposure due to ingestion alone. This is equivalent to an additional ingestion intake of (169% x 2.0 l/d), or 3.38 l/d.

An evaluation of other non-ingestion household water uses (cooking, toilet use, washing dishes and clothes, humidifier, etc.) is not as straightforward as the evaluation of shower exposures due to greater variability in the frequencies of the activities/uses. McKone (1987) estimates that the ratio of the indoor inhalation dose to the drinking water ingestion dose for VOCs ranges from 1.5 - 6.0 (includes showering and all other inhalation exposures). As estimated above, the ratio for showering alone is 1.69 for MTBE, which suggests that the ratio for all indoor inhalation exposures must be greater than 1.69. Assuming that the other indoor inhalation exposures are at least one-sixth to one-fifth the magnitude of the shower exposure, it can be assumed that these exposures' ratio to the drinking water ingestion exposure is at least 0.31, or 31% of the ingestion exposure. Thus, these exposures contribute at least the equivalent of 0.62 l/d of direct ingestion, and the total adjusted intake due to in-home water use for purposes of a chemical-specific RSC should be at least (3.38 l/d + 0.62 l/d + 2.0 l/d), or 6.0 l/d.

The data from USEPA (1993) can now be used to calculate the remainder of the resident's daily exposure to MTBE. This exposure is the result of ambient air exposures plus indoor air exposures which are not due to an MTBE-contaminated water supply (i.e., exposure to MTBE which originated from the ambient air and is then inhaled in the residence, workplace, and other buildings). These calculations have been completed using the USEPA data for a 6-month oxyfuel season, which predicts 0.04 mg/m³ and 0.07 mg/m³ as the Low and High annual average MTBE air concentration, and the standard USEPA assumption for breathing rate as above. The CDI (in mg/d) resulting from ambient air exposures (CDI_A) can be calculated from:

$$CDI_A = BR \times \text{Annual Average Concentration},$$

which results in estimates of 0.8 mg/d and 1.4 mg/d for the Low and High annual averages, respectively.

The final step in the development of a chemical-specific RSC is to apportion the contributions of the Acceptable Daily Exposure (ADE) of 2.3 mg/d of MTBE between ambient air and the home water supply. As calculated from the USEPA data, the ambient air exposures contribute between 0.8 mg/d and 1.4 mg/d of the 2.3 mg/d ADE. This leaves between (2.3 mg/d - 1.4

mg/d or 2.3 - 0.8 mg/d), or 0.9 mg/d to 1.5 mg/d to be contributed by the home water supply. As calculated above, the equivalent exposure intake value for the water supply is at least 6.0 l/d. Distributing the 0.9 mg/d to 1.5 mg/d portion of the ADE for home and water use into the adjusted exposure value of at least 6.0 l/d, the Health Advisory concentration for MTBE using chemical-specific RSC data can be no more than 0.15 mg/l to 0.25 mg/l. Since the value for the Health Advisory originally proposed by the Agency, 0.23 mg/l, falls within this range, the Agency proposes to adopt the Health Advisory as originally proposed.

REFERENCES

- Andelman, J.B. 1985. Inhalation exposure in the home to volatile organic contaminants of drinking water. *Sci. Total Environ.* 47:443.
- Lyman, W.J. 1982. *Handbook of Chemical Property Estimation Methods*. W.J. Lyman, W.F. Reehl, and D.H. Rosenblatt, Eds. (McGraw-Hill Book Co., NY).
- McKone, T.E. 1987. Human exposure to volatile organic compounds in household tap water: the indoor inhalation pathway. *Environ. Sci. Technology.* 21:1194.
- McKone, T.E. and Knezovich, J.P. 1991. The transfer of trichloroethylene (TCE) from a shower to indoor air: experimental measurements and their implications. *J. Air Waste Manage. Assoc.* 40:282.
- Morrow, Leslie D. 1994. Personal communication.
- NTP. 1994. Comparative Toxicology Studies of Corn Oil, Safflower Oil, and Tricaprylin in Male F344/N Rats as Vehicles for Gavage. National Toxicology Program, Technical Report Series, No. 426. U.S. Dept. of Health and Human Services, Public Health Service, National Institutes of Health. April, 1994.
- Robinson, M., Bruner, R. H., and Olson, G. R. 1990. Fourteen- and ninety-day oral toxicity studies of Methyl Tertiary-Butyl Ether in Sprague-Dawley rats. *J. Amer. Coll. Toxicol.* 9:525.
- USEPA. 1993. Assessment of Potential Health Risks of Gasoline Oxygenated with Methyl Tertiary Butyl Ether (MTBE). EPA/600/R-93/206. Office of Research and Development. November, 1993.

CERTIFICATE OF SERVICE

I, the undersigned, CERTIFY that I have served a copy of the **TESTIMONY OF**

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by depositing said document in the United States Mail in Springfield, Illinois on February 16, 2001.



Stephen C. Ewart