EXHIBIT 3

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Wednesday, August 15, 2012

Illinois Pollution Control Board
C/O John Therriault
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James R. Thompson Center
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Re: Docket No. PCB 2012-126

A Toxicologist's Perspective on Statements made about Potential Health Effects during the Hearing (August 1, 2012) on Ameren Energy Resources (AER) Request for Variance from the Illinois Sulfur Dioxide (SO2) Multi-Pollutant Standard (MPS)

Dear Members of the Illinois Pollution Control Board:

The purpose of this letter is to provide a toxicologist's perspective on the statements about potential health effects made in the hearing (August 1, 2012) on Ameren Energy Resources (AER) request for variance from the 2015 and 2017 Sulfur Dioxide (SO2) emission rate provisions of the Illinois Multi-Pollutant Standard (MPS). My knowledge of the statements made during the hearing comes from the transcript of the hearing, which was provided to me by Amy Antoniolli of Schiff Hardin LLP.

Discussions about the potential for adverse health effects provided in this letter are predicated on the *assumption* that there is a causal relationship between SO2 exposures and adverse health effects. However, most epidemiological evidence points toward particulate matter as a stronger causal agent for mortality and morbidity (i.e., effects other than mortality) than gaseous SO2. Because SO2 is usually found in association with particulate pollution, (SO2 is the precursor for fine sulfate particles) and because only limited evaluation of potential confounding (intervening variables that makes it appear that an effect is caused by a particular agent when the effect is actually due to something else) has been conducted in most epidemiological studies, separating the health effects of these two pollutants has been very difficult.

The Board should be aware that more studies than not have failed to find statistically significant associations between long-term and short-term SO2 concentrations and adverse health outcomes, indicating that the apparent associations may well be due to other factors. In fact, EPA has concluded that there is not a causal relationship between long-term SO2 exposure and respiratory effects or mortality. While EPA has concluded that the results of clinical studies in which humans are exposed to SO2 concentrations much higher than those found in ambient air support a causal relationship between respiratory morbidity and short-term exposure to SO2, nine of the 10 primary epidemiology studies attempting to correlate short-term exposure to asthma-related emergency room visits or hospitalizations relied upon

by EPA found either no association or very small positive associations. EPA further concludes that the evidence on short-term SO2 exposure is only *suggestive* of a causal relationship with mortality. Therefore, despite the dogmatism with which health effects from SO2 are asserted, when the studies providing the underlying support for such declarations are more closely examined, it becomes clear that the association between SO2 exposure and respiratory health effects and mortality is not nearly as certain as typically portrayed by the media, regulators and activists. Asthma is the health effect most commonly cited by these groups as associated with SO2 exposure, and they point to the rise in asthma in the US population as proof of effect. However, there are many theories about the rise in asthma over the past 30 years, and exposure to outdoor pollution is probably the least plausible explanation given that the air quality in Illinois and the nation as a whole has improved dramatically during the same time period over which asthma prevalence has increased, as discussed in more detail in the specific responses to public comments.

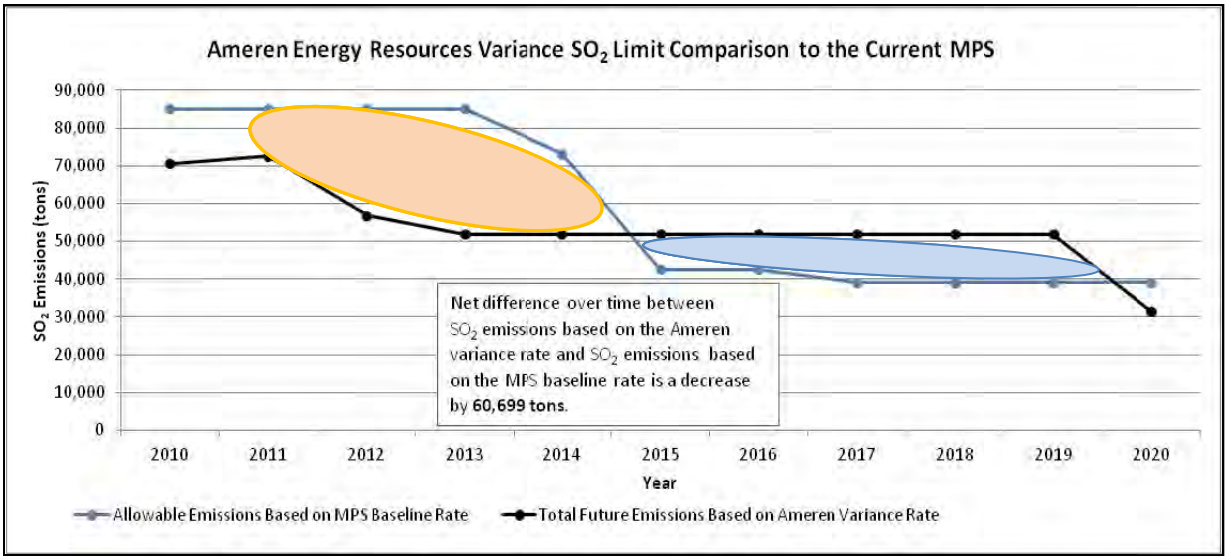
Although it can be universally agreed that respiratory effects such as asthma attacks and chronic obstructive pulmonary disease (COPD) represent adverse or harmful effects, simply establishing that a person who has possibly been exposed to SO2 in an environmental setting experienced bronchoconstriction or a reduced expiratory volume is not proof that the potential exposure caused the effect. This is because in any individual, these effects could have been caused by a number of different factors, for example, indoor or outdoor allergens, smoking or passive exposure to cigarette smoke, or viral pathogens. Likewise, the fact that a power plant may emit SO2 does not necessarily mean that off-site air concentrations will be high enough to elicit adverse health effects.

More details about research on the potential health effects of SO2 are provided in the specific responses to public comments.

Background

The MPS and the AER Requested Variance

The MPS specifies a 0.5 lb/MMBtu SO2 emission rate until 2014 when the required emission rate is decreased to 0.43 lb/MMBtu, followed by a decrease to 0.25 lb/MMBtu in 2015. Under the MPS, the SO2 emission rate is further decreased to 0.23 lb/MMBtu in 2017. AER has requested a variance from meeting the 0.25 lb/MMBtu SO2 emission rate required by the provisions of the MPS in 2015 and the 0.23 lb/MMBtu emission rate that is required in 2017. However, AER has proposed a mitigation emission rate of 0.35 lb/MMBtu that would take effect in 2013, resulting in AER taking action to reduce SO2 emissions earlier than required by the MPS. AER would comply with the 0.35 lb/MMBtu mitigation emission rate from 2013 to 2019, with the 2017 MPS emission rate of 0.23 lb/MMBtu being met by 2020. The figure below provides a comparison of AER's SO2 emissions under the provisions of the MPS and the SO2 variance.



The blue line represents AER's baseline emissions under the MPS and the black line represents emissions under the SO2 variance. As can be seen in the figure, AER's emissions under the SO2 variance are considerably lower than the MPS emissions between 2012 and 2014 and are slightly higher from 2015 to 2019. However, the area representing the difference between the MPS and SO2 variance emissions from 2012 to 2014 is larger than the area between the MPS and SO2 variance emissions from 2015 and 2019. Therefore, there is an overall net improvement (i.e., decline) in SO2 emissions under the SO2 variance proposed by AER. It should also be noted that AER has voluntarily operated at lower SO2 emission rates than allowable under the MPS since 2010, demonstrating its commitment to reducing emissions. If the reduced SO2 emissions starting in 2010 are taken into consideration, the positive difference between AER's SO2 variance emissions and those allowable under MPS is even larger.

Overall Net Health Benefit of the Variance

Over the course of the entire variance period, between now and 2020, the total tons of SO2 reduced in the atmosphere will be greater if the AER-requested variance is granted than if it is not. The source of reduction in SO2 emissions over the course of the variance period is two-fold: 1) AER will emit less SO2 via a mitigation rate representing a 19% to 30% reduction in the MPS allowable SO2 through 2014; and 2) AER has agreed to keep two of its coal-fired power plants shut down during the variance period, which will result in less coal being burned.

Assuming that the SO2 emissions from the AER coal fleet are at levels that could potentially result in off-site ambient concentrations high enough to cause adverse health effects under the provisions of the MPS, which is not a foregone conclusion, granting the variance would result in an overall net health benefit. For example, if a **hypothetical** risk relationship of one additional asthma-related emergency room (ER) visit per 100 tons of SO2 emitted is *assumed*, there will have been 77 fewer ER visits over the duration of the SO2 variance than

if AER complies with the MPS on schedule. Please note, however, that this assumed risk relationship is purely hypothetical, as risk relationships of this nature are typically expressed on a per capita per ton basis, taking into account the size of the potentially exposed population.

Health Effects Concerns Raised during the Hearing

While concerns about potential health effects associated with exposure to SO2 are understandable, there are misconceptions about what the scientific research is telling us, as described above and further elaborated upon below.

There appears to be a continuous spectrum of sensitivity to SO2, with some people being completely unaffected by concentrations that lead to severe bronchoconstriction in others. Asthmatics are particularly sensitive to the effects of SO2 and the effects are enhanced if SO2 reaches the lower regions of the lungs, which is more likely to occur during mouth breathing (as opposed to breathing through the nose) and exercise. Maximum effects of SO2 occur within a few minutes and continued exposure does not typically increase the response and the effects are generally short-lived and completely reversible (WHO, 2006).

Illinois Attorney General's Office

James Gignac, the Assistant Attorney General (pg 84 of Transcript) stated in the hearing that there is a harm that would occur, despite AER's calculations demonstrating an overall reduction in the tonnage of SO2 emissions over the 12-year period. The basis for his statements regarding potential harm is the increase in emissions that would be allowed between the years of 2015 and 2019 under the variance.

While there could, theoretically, be additional adverse health outcomes between the years of 2015 and 2019, there would be fewer between the years of 2012 and 2014 and because the magnitude of the emission reductions during the early years is greater than the increased SO2 emissions that will occur between 2015 and 2019, there would be fewer adverse health effects overall. Therefore, the health benefits of approving the variance requested by AER outweigh the health costs, resulting in an overall health benefit, when the entire period of the variance is taken into account. Assuming that one accepts that the current emissions pose a health threat, not approving the variance represents a tradeoff between greater reductions in health effects from 2012 and 2014 in exchange for smaller reductions in health effects between the years of 2015 and 2019.

As mentioned in the beginning of this letter, the above discussion is predicated on the assumption that the C-R (concentration-response) relationships reported in epidemiological studies indicate causal relationships between the SO2 exposures and adverse health effects. However, in most studies that have examined the potential for confounding by other co-pollutants (particulates, ozone, nitrogen oxides), the small associations observed between ambient SO2 concentrations and adverse health outcomes usually become null when two-pollutant models are used, indicating that the associations are stronger for the other pollutants or that there is no association with SO2 (EPA, 2008; EPA, 2009; Goodman et al., 2010). As a result, more studies than not have failed to find statistically significant associations between long-term and short-term SO2 concentrations and adverse health outcomes. According to the

EPA's own Integrated Science Assessment (ISA, EPA, 2008) prepared in support of the most recent SO2 National Ambient Air Quality Standards (NAAQS) review, "Overall, the epidemiologic studies do not provide sufficient evidence to infer a causal relationship between long-term exposure to SO2 and asthma, bronchitis, or respiratory symptoms". The EPA ISA also concluded that "The available epidemiologic evidence on the effect of long-term exposure to SO2 on mortality is inadequate to infer a causal relationship at this time" (EPA, 2008).

The EPA ISA does conclude that "the human clinical, epidemiologic, and animal toxicological data are sufficient to conclude that there is a causal relationship between respiratory morbidity and short-term exposure to SO2" and that "The evidence is suggestive of a causal relationship between short-term exposure to SO2 and mortality". However, of the 10 primary epidemiology studies attempting to correlate short-term (daily) SO2 exposures to adverse health outcomes relied upon by EPA, nine found either no association or very small positive associations between daily SO2 concentrations and asthma-related emergency room visits or hospitalizations. Among the studies for which weak positive associations were observed, conclusions were either: 1) based only on results from single-pollutant models (i.e., multiple pollutant models were not used, which are applied to determine confounding by co-pollutants); or 2) based on results from single-pollutant models that were not statistically significant in two-pollutant models.

Statistical significance is key to determining if exposure and effect are causally associated. Determining whether the effect is isolated, independent, or secondary to a known effect of exposure is also important because these types of effects may be the result of other factors not related to the exposure of interest (Goodman et al., 2010). Isolated effects occur in only a few test subjects and independent effects are those which occur in the absence of other effects expected via the same mechanism of action. The fact that these effects occur inconsistently and lack biological plausibility is an indication that they are more likely due to another factor or measurement error rather than exposure related. A test of statistical significance helps determine whether effects are caused by the exposure under study. These tests compare differences between exposed and non-exposed groups of test subjects as opposed to evaluating effects in independent individuals. If the difference between exposed and non-exposed groups is not statistically significant, the exposure is either insufficient to cause the effect under study or the study is not sufficiently powered, most likely due to having too few test subjects.

Only one of the 10 epidemiological studies (NYSDOH, 2006) correlating daily SO2 concentrations to adverse health outcomes relied upon by EPA in the latest NAAQS review found a marginally statistically significant association with increased SO2 levels in both single-and two-pollutant models. However, the authors of that one study acknowledged that correlations between co-pollutants made these results difficult to interpret.

Peoria Families against Toxic Waste

A representative for the Peoria Families against Toxic Waste, Tracey Cox, indicated that health experts understand and have pointed out that cumulative SO2 reductions do nothing to help communities with short-term pollution impacts and it is those short-term impacts that happen to children and the elderly population across the state.

It is not correct that cumulative SO2 reductions do nothing to abate short-term pollutant impacts. Long-term concentrations are not completely independent of short-term concentrations of the same pollutant. In fact, EPA has performed extensive evaluations to determine relationships between short-term and longer-term concentrations of various pollutants and has on occasion set a longer-term standard to limit the relative frequency with which shorter-term exposures will exceed a particular level. In addition, EPA's screening modeling guidance indicates that for a point source it can be assumed that the maximum daily average concentration is 0.4 of the maximum 1-hour and that the maximum annual concentration is 0.08 of the max 1-hr (http://www.colorado.gov/airquality/permits/screen.pdf). From this we can infer 24-hour to annual ratio of 0.4/0.08 = 5. Thus, it is widely accepted that long-term and short-term concentrations are related to one another. For this reason, the overall net reduction in SO2 provided by the variance in comparison to the MPS regulatory schedule is also expected to have an effect or reducing short-term exposures over the variance time period.

Sierra Club

A representative of the Sierra Club raised the issue of the increasing incidence of asthma in children that has been observed over the last several decades, indicating that in his job as a school social worker, he has seen the effects of SO2 in terms of asthma.

Nationwide, SO2 concentrations fell 32% between 1980 and 1990 alone. The distribution of asthma in other countries also fails to implicate SO2 or other pollutants as an aggravating factor. Some of the highest asthma mortality rates occur in Australia and New Zealand, which have excellent air quality. Asthma is more prevalent in rural areas of the Scottish highlands, which have some of the lowest ozone concentrations in the world, than in more urban and polluted parts of the United Kingdom, according to a recent report (Friebele, 1996).

Changes in the diagnostic coding of asthma and survey questions in self-reporting asthma questionnaires over the last 30 years have likely altered the diagnosis of asthma cases and caused changes in prevalence and incidence statistics. The International Classification of Diseases (ICD) provided by the World Health Organization (WHO) was revised in 1978 (9th revision) and 1990 (10th revision) resulting in a change to the coding of asthma. In the ICD 8, a patient with "asthmatic bronchitis" would have been coded under bronchitis, while in ICD 9 this same person would be coded under asthma (Marcus and Braman, 2010). One study that analyzed asthma patient records found an increase in patients with an asthma classification that had a history of smoking in the 1980s versus the 1970s. The cause of this difference was attributed to the change in classification of asthmatic bronchitis from a bronchitis heading to an asthma heading, resulting in asthmatic bronchitis patients now falling under the umbrella of asthma in the 1980s (Marcus and Braman, 2010). This change in coding may also influence the validity of epidemiology studies that look at hospital ER visits for asthma as potential indicators of an association between ambient pollutant concentrations and respiratory effects over years during which changes in the asthma definition has changed.

A large source of asthma surveillance data is compiled by the National Center for Health Statistics of the Centers for Disease Control and Prevention (CDC) under the National Health Interview Survey (NHIS). The NHIS questions used to evaluate asthma prevalence changed in 1997 and 2001, resulting in three separate types of questions that could impact asthma prevalence estimates from 1980 to 1996, 1997 to 2000, and 2001 to the present (National Heart and Lung Institute; www.nhlbi.nih.gov/health/prof/lung/asthma/surveil.htm). These changes prevent comparisons of reported asthma rates from 1980 to 1997 to the more recent data set from 1997 to 2001 and illustrate the potential variability in reported asthma prevalence depending on how asthma questions are phrased, and what sort of asthma information is requested (lifetime incidence versus episodes in the past 12 months, for example).

The increase in asthma cases may also be partially explained by factors relating to changes in healthcare access and physician perceptions. The diagnosis of asthma may have become more likely than a similar diagnosis of bronchitis or chronic obstructive pulmonary disease (COPD) among patients with similar symptoms. One study looked at healthcare data from Manitoba, Canada from 1980-1990 and found a statistically significant increase in asthma diagnosis above background increases found for other diseases with similar symptoms over that time period (Manfreda et al., 1993). The study attributes some of the increase to an increase in the likelihood of asthma diagnoses.

Another factor that may have contributed to the apparent increase in childhood asthma prevalence is that children spend much more time indoors today than they did 30 years ago. In addition to contributing to the development of asthma, exposure to various indoor air irritants can also exacerbate asthma symptoms. Cat, cockroach, and house mite dust allergens have all been causally linked to exacerbation of asthma symptoms in sensitive individuals, and environmental tobacco smoke exposure has also been causally linked to exacerbation of asthma symptoms in young children (Institute of Medicine, 2000).

Therefore, the suggestion by Mr. Cox that SO2 emissions (pg. 133 of transcript) are significant contributors to the rise in reported asthma cases (and other diseases) is not supported by evidence in the literature.

ELPC

Jennifer Cassel from the ELPC read a letter from Illinois health professionals opposing AER's variance request. The letter stated that "High levels of SO2 and NOx can exacerbate respiratory systems in at-risk individuals, including children and the elderly, including asthma and COPD attacks." (pg 144 of transcript). The operative word in this statement is "high" levels. There are many controlled human studies that have exposed healthy and asthmatic test populations to SO2 and that have measured small lung function decrements in the asthmatic population, particularly at higher than normal exertion levels. However, most fail to show a statistically significant response, and even in asthmatics (a sensitive subpopulation), responses are only seen at high concentrations on the order of 250 ppb (715 $\mu g/m^3$) over a 10 minute period (WHO, 2006). Peak exposures in the range of 4,000 ppb (11,440 $\mu g/m^3$) to 5,000 ppb (14,330 $\mu g/m^3$) are required for reductions in mean lung function in normal (non-asthmatic) individuals at rest. No significant changes in group mean

lung function in healthy individuals have been seen below short-term exposures of 1000 ppb (2860 µg/m^3), even with exercise (WHO, 2006).

To put the SO2 concentrations above into context, according to the Illinois EPA 2009 Air Quality Report (IEPA, 2010), the statewide average 24-hour SO2 concentration for 2010 was 39 µg/m^3 (15 ppb) and 45 µg/m^3 (17 ppb) in 2009. The statewide average 1-hour high in 2010 was 197 µg/m^3 (75 ppb), compared with 212 µg/m^3 (81 ppb) in 2009 and 335 µg/m^3 (128 ppb) in 2008. Therefore, not only have the air concentrations monitored in Illinois been well below levels demonstrated to cause respiratory effects in healthy and asthmatic individuals, there has been an overall downward trend in SO2 concentrations in the state.

The letter also references a 2010 study conducted by the National Research Council (NRC) that indicated that annual health related damages from particulate, SO2 and NO2 cost 62 billion dollars in 2005 alone. The concentration-response (C-R) relationship used in the NRC Health Impact Assessment (HIA) to estimate damages associated with SO2-related hospital admissions was from a study conducted by Sheppard et al. (1999). However, this study, like most SO2 epidemiology studies, failed to find an association between ambient SO2 concentrations and asthma-related hospital admissions, as was clearly acknowledged by the authors.

The C-R function is a key component of HIAs because it is this function that allows the effect of interest to be linked in a quantitative way to incremental changes in concentrations by assuming a response continuum. However, C-R relationships are calculated for all pollutants and health endpoints examined in a scientific study by the authors, even for those pollutant-health effect pairings that are determined through statistics not to be associated with the exposure of interest. Therefore, it is up to those conducting the HIA (individuals other than the scientific study authors) to choose appropriate C-R relationships for use in modeling. It defies logic that the NRC study would use a C-R function from a study in which the ambient SO2 concentrations and asthma-related hospital admissions were determined not to be correlated and casts doubt on the validity of the entire NRC report. This is a clear example of how findings published in the scientific literature are often misinterpreted and inadvertently or intentionally misused by activist groups.

Comments of Samuel Dorevitch, MD

Comments from Dr. Dorevitch were provided by Andrew Armstrong with the ELPC. Mr. Armstrong read excerpts from Dr. Dorevitch's written comments, which stated that research has demonstrated that even moderate levels of SO2 are associated with bronchospasm but provided no information on which research he was referring to or what he considers a moderate concentration of SO2. However, the doctor does cite the EPA's ISA (EPA, 2008) and notes that it states that "epidemiologic studies observed respiratory effects in areas where the SO2 concentration was below the regulatory level in place at the time." The evidence of respiratory effects below the 24-hour NAAQS referenced in the ISA comes entirely from epidemiological studies. To put this finding into context it is important to understand what epidemiological studies can and cannot do.

Observational epidemiological studies attempt to determine which factors are associated with diseases (risk factors), and which factors may protect people or animals against disease

(protective factors). However, epidemiological studies cannot prove that a specific risk factor actually causes the disease being studied. This is because epidemiological studies cannot control for, nor can they necessarily identify, all of the factors that may influence a health outcome. Therefore, they are plagued with issues of confounding. For example, if coffee drinkers were more likely to also be cigarette smokers, and a study was conducted to explore potential associations between coffee drinking and lung cancer, without taking the smoking habits of the coffee drinkers into account, smoking would be a confounder and the results may seem to show that coffee drinking increases the risk of lung cancer.

Epidemiological evidence can only show that a risk factor is or is not associated (correlated) with a higher incidence of disease in the population exposed to that risk factor. The higher the correlation, the more certain the association; but causation cannot be proven in these studies. Therefore, the fact that there may be studies showing that hospital admissions or emergency room visits were increased in an area where short-term SO2 concentrations were below the NAAQS does not necessarily mean that the SO2 concentrations caused the increase in hospital admissions.

Studies that use population level data, such as respiratory hospital admissions or emergency room visits obtained from databases without collecting any data on the individuals involved, are prone to what is known as "ecologic fallacy". This occurs when a correlation observed at the population level is assumed to apply at the individual level. Without information on whether the correlations were statistically significant and whether co-pollutants and other intervening variables were properly controlled for, it is not possible to know what the implications of the findings are or if the statement even accurately characterizes the findings.

Thus, while EPA's ISA may have noted that epidemiologic studies observed respiratory effects in areas where the SO2 concentration was below the regulatory level in place at the time, the respiratory effects were not necessarily due to SO2 exposure.

Prairie Rivers Network

A representative from the Prairie Rivers Network indicated that the air pollution that is released from the Newton facility kills about 25 people per year (pg. 186 of transcript). This value appears to be from the Power Plant Impact Estimator Software Tool located at http://www.catf.us/resources/publications/view/138 and another report entitled, "Toll from Coal: An Updated Assessment of Death and Disease from America's Dirtiest Energy Source". This study was commissioned by the Clean Air Task Force (CATF, 2010). However, even upon examination of the technical support document for the estimator tool and Toll from Coal report (Abt, 2010), the underlying assumptions used in deriving these numbers are not particularly transparent. However, one thing noted is that the C-R relationship for SO2 exposure and asthma-related hospital admissions used in the tool was from a study (Sheppard et al., 1999) that failed to find an association between ambient SO2 concentrations and asthma-related hospital admissions. Although these types of evaluations are becoming increasingly more common, use of these C-R relationships are subject to significant uncertainty related to their generalizability and accuracy. One thing is certain, C-R relationships from studies that fail to establish an association between the exposure and effect of concern should not be extrapolated to other situations in the hopes of providing

accurate predictions of adverse health outcomes. The other comments made above about C-R relationships and their use apply here as well.

Miscellaneous Comments

Many commenters made vague statements such as "Coal power plants kill people.", "Hundreds of people in Illinois die from what comes out of coal-fired power plant stacks." etc, but gave no support for these statements.

The public debate on air pollution coupled with the sensationalized air pollution health stories in the media have created the appearance that harm from air pollution is much greater and more certain than suggested by the underlying scientific evidence. According to Dr. Joel Schwartz (2006), whose work on the relationship between respiratory hospital admissions and ambient air pollutants is amongst some of the most highly cited and one of the premier authors of many epidemiological studies that have been relied upon by EPA in establishing and re-evaluating US NAAQS, "the incentives in air pollution health research encourage risk exaggeration...Through exaggeration, omission of contrary evidence, and lack of context, regulators, activists, and even many health scientists misrepresent the results of air pollution health studies and the overall weight of the evidence from the research literature" (Schwartz, 2006). Dr. Schwartz across multiple fields, including Clinical Medicine, Environment & Ecology, Biology & Biochemistry, and Pharmacology & Toxicology. http://www.esi-topics.com/airpoll/interviews/JoelSchwartz.html

It is not surprising that ordinary citizens perpetuate the misinformation that is rampant in the media in hearings such as the one on AER's request for variance from the SO2 MPS. We urge the board to consider the scientific evidence on the health effects of SO2 rather than statements from citizens that have been mislead by the news media and reports published by activist groups. In support of this perspective, we have attached a copy of "Air Pollution and Health: Do Popular Portrayals Reflect the Scientific Evidence?" by Joel Schwartz, which provides several case studies aimed at demonstrating that misinformation about air pollution is a pervasive problem. The Schwartz (2006) article demonstrates why it is so important for those involved in evaluating and setting health policy to look deeper than the summaries of studies provided by other governmental agencies, health scientists in press conferences, and activists in hearings. Likewise, because it is not uncommon for even those who should know better to inadvertently misrepresent the evidence, interpreting air pollution health effects information published in the media requires a level of vigilance that few citizens are capable of and as a result, while citizens may believe that their health effects can be attributed to AER's emissions, they are often misinformed or even manipulated by the local activists.

There is no question that very high levels of pollution *can* kill, as occurred during the "London Fog" of 1952, when soot and SO2 were at levels orders of magnitude higher than those experienced in developed countries today and visibility was less than 20 feet. However, claims that low levels of pollutants cause death is based purely on scientific evidence from observational studies that are incapable of controlling for co-pollutants, lifestyle variables, and usually do not even contemplate regional disease patterns that have nothing to do with air pollution. It is noteworthy that researchers have been unable to evoke adverse health outcomes in animals with SO2 concentrations anywhere near as low as those found in ambient air today.

Sincerely,

AECOM

Lucy Fraiser, PhD, DABT
Associate Vice President/Senior Toxicologist

Lisa JN Bradley, PhD, DABT
Vice President/Senior Toxicologist

Enclosures:

Attachment 1. References
Attachment 2. Schwartz, J. 2006. Air Pollution and Health: Do Popular Portrayals Reflect the Scientific Evidence? American Enterprises Institute for Public Policy Research. Environmental Policy Outlook, No. 2.
Attachment 3. Resume of Dr. Lucy Fraiser
Attachment 4. Resume of Dr. Lisa Bradley

Attachment 1: References

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Attachment 2: Schwartz, J. 2006. Air Pollution and Health: Do Popular Portrayals Reflect the Scientific Evidence? American Enterprises Institute for Public Policy Research. Environmental Policy Outlook, No. 2.

American Enterprise Institute for Public Policy Research

AEI

No. 2, 2006

Environmental Policy Outlook

Air Pollution and Health: Do Popular Portrayals Reflect the Scientific Evidence?

By Joel Schwartz

Environmentalists, regulators, health scientists, and journalists are the main purveyors of information on air pollution health risks. Unfortunately, these groups create the appearance that harm from air pollution is much greater and more certain than suggested by the underlying evidence. The incentives in air pollution health research encourage risk exaggeration, because information purveyors depend on public fear to maintain their funding and influence. Investigative reporters are in the best position to assess how the political economy of environmental health research affects the production and portrayal of the evidence. Public debate on air pollution will continue to proceed from false premises until journalists take up this challenge.

In a nationwide survey in 2004, 85 percent of Americans rated air pollution as a "very serious" or "somewhat serious" problem, with similar results for state surveys.[1] In a recent Gallup Poll, 78 percent of Americans said they worry about air pollution "a fair amount" or "a great deal."[2] Public fear of air pollution is understandable, because most popular information about air pollution is indeed alarming.

Activist groups regularly issue reports with scary titles such as *Danger in the Air; Death, Disease and Dirty Power; Highway Health Hazards; Plagued by Pollution; and Children at Risk*.[3] Health researchers often issue alarming summaries of their research as well. Recent press-release headlines from health research institutes include "Smog May Cause Lifelong Lung Deficits," "Link Strengthened between Lung Cancer, Heart Deaths and Tiny Particles of Soot," "USC Study Shows Air Pollution May Trigger Asthma in Young Athletes," and "Traffic Exhaust Poisons Home Air."[4]

Regulators declare "code orange" and "code red" alerts on days when air pollution is predicted to exceed federal health standards. And news stories on air pollution often feature menacing headlines such as "Air Pollution's Threat Proving Worse than Believed," "Don't Breathe Deeply," "Study Finds Smog Raises Death Rate," "State's Air Is among Nation's Most Toxic," and "Asthma Risk for Children Soars with High Ozone Levels."[5]

Headlines like these might be warranted if they accurately reflected the weight of the scientific evidence. But they do not. Through exaggeration, omission of contrary evidence, and lack of context, regulators, activists, and even many health scientists misrepresent the results of air pollution health studies and the overall weight of the evidence from the research literature. They create the appearance that harm from air pollution is much greater and more certain than suggested by the underlying evidence.

Journalists are the final line of defense between the public and the proponents of air pollution health scares. Unfortunately, the majority of media air pollution health stories are sensationalized exaggerations of air pollution's risks.

Through several case studies, this essay shows that misinformation on air pollution and health is a pervasive problem. As a result, public fear of air pollution is out of all proportion to the minor risks posed by current, historically low air pollution levels.

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False Alarm on Asthma and Air Pollution

Beginning in 1993, the California Air Resources Board (CARB) funded the Children's Health Study (CHS). Researchers from the University of Southern California (USC) tracked several thousand California children living in twelve communities with air pollution ranging from near-background to the worst in the nation.

At a joint press conference in 2002, the USC researchers and CARB managers reported that children who played three or more team sports were more than three times as likely to develop asthma if they lived in the six highest-ozone communities in the study, when compared with the six lowest-ozone communities.[6] They also claimed the study's results applied to cities across the United States.

Ironically, the CHS asthma study actually showed just the opposite. While higher ozone was associated with a greater risk of developing asthma for children who played three or more team sports (8 percent of children in the study), higher ozone was associated with a 30 percent *lower* risk of developing asthma in the full sample of children in the study.[7] While this fact was discussed in a journal article on the study, it was not mentioned at the press conference.[8]

Higher levels of other pollutants, including nitrogen dioxide and particulate matter (PM_{10}), were also associated with a lower asthma risk.[9] Also mentioned in the journal article, but not at the press conference, was that when the researchers divided the twelve communities in three groups of four (rather than two groups of six), the association of ozone with increased asthma prevalence in child athletes applied only to the four communities in the highest ozone group and not to the medium-ozone group.

The assertion that the study is relevant for other parts of the country was also false. The four high-ozone areas in the study averaged 89 days per year exceeding the federal eight-hour ozone standard and 59 days per year exceeding the one-hour standard during 1994–1997, the years used to assess pollution exposure in the study.[10] No area of the United States, outside of a few parts of California, has *ever* had ozone levels this high even for a single year, much less for several years running.

In fact, by the time of its release in February 2002, the study no longer applied even in the southern California areas where it was performed. Eight-hour ozone exceedances had declined 55 percent, and one-hour exceedances had declined 78 percent in the interim. By 2002, communities that were "high-ozone" areas during the study had become "medium-ozone" areas, for which ozone had no effect on asthma risk.

At the press conference releasing the CHS asthma results, the chairman of the Air Resources Board claimed: "This study illustrates the need not to retreat but to continue pushing forward in our efforts to strengthen air pollution regulations."[11] But if anything, the CHS asthma study showed that current standards already include a large safety margin. Ozone was not associated with a change in asthma risk in the medium-ozone areas of the study. Yet these areas exceeded federal ozone standards by large margins—an average of 41 eight-hour exceedance days per year and 17 one-hour exceedances.

> Through exaggeration, omission of contrary evidence, and lack of context, regulators, activists, and even many health scientists misrepresent the results of air pollution health studies and the overall weight of the evidence from the research literature.

False information on the CHS asthma results was not limited just to CARB officials or USC scientists. Health experts from around the country misinterpreted the study's results. For example, on the day the study was released, a professor at the State University of New York at Stony Brook, who has since become the American Lung Association's medical director, claimed: "This is not just a Southern California problem. There are communities across the nation that have high ozone."[12] According to the *Houston Chronicle*, Houston asthma specialists said the study showed that "Houston [should] step up its efforts to implement a state plan to reduce ozone."[13] The director of the pediatric asthma program at the University of California at Davis claimed "Sacramento is a very high ozone area, so this [the CHS asthma study] is going to be very relevant to us."[14]

Not only were all of these nominal experts wrong about whether the study is relevant to actual ozone levels in the United States, all of them completely missed the fact that ozone and other air pollutants were associated with an overall lower risk of developing asthma.

In a recent commentary on air pollution and asthma in the *Journal of the American Medical Association*, two prominent air pollution health researchers claimed:

"Some evidence suggests that air pollution may have contributed to the increasing prevalence of asthma."[15] The "evidence" they cite is the CHS asthma study.

Journalists also often act as cheerleaders for air pollution alarmists when reporting on air pollution and health. For example, a recent editorial headline in the *Sacramento Bee* declared "Smog and Asthma: The Link—and Threat—Are Real."[16] The *Bee*'s source for this claim? Once again, the CHS asthma study.

Much Ado about Very Little

The Children's Health Study also suggests that even the highest air pollution levels in the nation are having little or no effect on children's lung development. But once again, the scientists involved in the study obscured that fact.

After following more than 1,700 children from ages ten to eighteen (years 1993 to 2001), CHS scientists reported that there was no association between ozone and lung-function growth.[17] This is despite the fact that the twelve communities in the study ranged from zero to more than 120 eight-hour ozone exceedance days per year, and zero to more than 70 one-hour ozone exceedance days per year during the study period.[18] Once again, no area outside California has ever had anywhere near this frequency of elevated ozone, even for a single year, so we can conclude that ozone is not causing any reduction in children's lung capacity. This has not stopped environmental groups from claiming otherwise. For example, in *Impacts of Ozone on Our Health*, the Carolinas Clean Air Coalition claims: "Children have a 10 percent decrease in lung function growth when they grow up in more polluted air."[19]

The Children's Health Study also suggests that fine particular matter ($PM_{2.5}$) is causing little or no long-term harm to lung growth. Unlike ozone, $PM_{2.5}$ actually was associated with a small effect on lung development. Annual-average $PM_{2.5}$ levels ranged from about 6 to 32 micrograms per cubic meter ($\mu g/m^3$) in the twelve communities in the study.[20] Across this range, $PM_{2.5}$ was associated with about a 2 percent decrease in forced expiratory volume in one second (FEV_1) and a 1.3 percent decrease in force vital capacity (FVC), both measures of lung capacity.

But even this small effect drastically inflates the apparent importance of the results. First, no location outside of the CHS communities has $PM_{2.5}$ levels anywhere near 32 $\mu g/m^3$. In fact, outside California there is not a single area with $PM_{2.5}$ above 21 $\mu g/m^3$. And by the time the study was published in 2004, even the highest $PM_{2.5}$ area in California was at 25 $\mu g/m^3$.

It is also worth noting that the children in the CHS were already ten years old when they entered the study in 1993 and had therefore been breathing the even-higher air pollutant levels extant during the 1980s in southern California. For example, Riverside averaged about 48 $\mu g/m^3$ $PM_{2.5}$ during the 1980s, or about 50 percent greater than the highest $PM_{2.5}$ level measured during the CHS years.[21] If it were really these higher 1980s $PM_{2.5}$ levels that caused the lung-function declines, then the current worst $PM_{2.5}$ in the country would be causing about a 1 percent decrease in FEV_1 and a 0.5 percent decrease in FVC. Thus, taking the CHS results at face value, ozone is having no effect on children's lung development anywhere in the United States. $PM_{2.5}$ is having virtually no effect.

Nevertheless, the USC researchers' press release on the study created an unwarranted appearance of serious harm. Titled "Smog May Cause Lifelong Lung Deficits," the press release asserted: "By age 18, the lungs of many children who grow up in smoggy areas are underdeveloped and will likely never recover."[22] The National Institutes of Health (NIH) also misled the public about the study's findings and relevance. The director of the National Institute of Environmental Health Sciences claimed the study "shows that current levels of air pollution have adverse effects on lung development in children."[23]

Furthermore, although the study is relevant only to a few areas of California with uniquely high air pollution levels, by asserting that it applies to all "smoggy areas" and to "current levels of air pollution," NIH and USC created the false impression that the study applies to much of the United States.

The scientists were able to create these false impressions, because the journal article on the study, which was published in the prestigious *New England Journal of Medicine* (*NEJM*), does not explicitly reveal the magnitude of the percentage change in children's lung capacity. Instead, readers have to be vigilant enough to realize that the percentage change can be calculated by combining information found in three different places in the article.[24] It is odd that a study whose main outcome measure is changes in lung capacity never actually states the percentage change explicitly.

The researchers reported a different outcome measure in their *NEJM* paper: the percent of children in

each community with a lung capacity of less than 80 percent of the "predicted" value for their age.[25] Between the least and most polluted communities, $PM_{2.5}$ was associated with nearly a five-fold increase in this percentage, from about 1.6 percent of children in the lowest-$PM_{2.5}$ community, up to about 7.9 percent in the highest-$PM_{2.5}$ community.

This seems like a large effect, but it is not. What is going on is that the 2 percent average decline in lung function in the highest-$PM_{2.5}$ community relative to the lowest meant a shift of some children who were at, say, 80 or 81 percent of "predicted" lung capacity for their age, down to maybe 78 or 79 percent. Because lung-capacity scores have a bell-curve distribution, and few children have low lung capacity, there are many more children slightly above 80 percent than slightly below 80 percent. A small shift in average lung-capacity scores therefore results in a large change in the fraction of children scoring below a given cutoff level.[26]

Reporting that even the highest air pollution levels in the country were associated with only a 2 percent decrease in lung capacity would not have caused much alarm. This probably explains why that number is nowhere to be found in the *NEJM* report or the press releases on it.

NIH took advantage of this omission in its press release, which begins: "Children who live in polluted communities are five times more likely to have clinically low lung function—less than 80 percent of the lung function expected for their age."[27] Note how this statement creates the appearance of a decline of more than 20 percent in average lung function by leading readers to tacitly make the incorrect assumption that all children would be at 100 percent if there were no air pollution.

This is exactly the mistake environmentalists have made in promoting the study. For example, the American Lung Association's (ALA) *State of the Air 2005* report claims the "average drop in lung function was 20 percent below what was expected for the child's age."[28] The Carolinas Clean Air Coalition made a similar error.[29]

The ALA clearly did not understand the study's results. But NIH and the USC researchers created the confusion. The editors and peer reviewers at the *New England Journal of Medicine* also bear responsibility for not requiring that its article on the study explicitly state the percentage change in lung capacity associated with air pollution.

> Scientists, regulators, and environmentalists have ignored these weaknesses and continue to make believe these spurious statistical correlations are telling us something real about the effects of low-level air pollution.

Monkey Business

A University of California at Davis press release begins "Primate Research Shows Link between Ozone Pollution, Asthma."[30] The press release goes on to claim the ozone exposures in the study "mimic the effect of exposure to occasional ozone smog—for example as it occurs in the Sacramento area."

In fact, the ozone exposures in the study were far higher than the actual ozone levels in American air—including the air in Sacramento. The monkeys were exposed to 0.5 parts per million (ppm) ozone for eight hours a day for five days in a row, followed by nine days of clean air. This cycle was repeated eight times. To give you an idea of the magnitude of these ozone exposures, during the last thirty years only one site in the U.S. has ever exceeded 0.5 ppm ozone for even one hour, and that happened in 1976. Today, the worst site in the United States never reaches even 0.25 ppm for one hour, and the average site never reaches 0.11 ppm.

Despite the real-world irrelevance of this study, environmental activists cite it to support claims that ozone is causing permanent lung damage in people. For example, under the headline "Lung Development of Young Monkeys Drastically Changed when Exposed to Ozone Pollution," the American Lung Association concludes, "This study presents data suggesting that the changes caused by ozone pollution are long-lasting, and maybe even permanent."[31]

Some reporters also failed to compare ozone levels in the study to real-world ozone levels. For example, according to the *Modesto Bee*, "Monkeys were exposed to air contaminated with ozone, mimicking the smog in the [Central] valley."[32] But even more nuanced stories still took an alarmist tack. For example, the *Sacramento Bee* explicitly compared ozone levels in the Sacramento region with the far higher ozone levels used in the study.[33] But you have to go halfway into the 1,100-word story to find this information. The story's headline—"Study Suggests Asthma Culprit; Young

Lungs Exposed to Ozone Seem More Prone to Problems with Development"—leaves no doubt that readers are supposed to conclude that ozone is causing Americans to develop asthma.

Of Mice and Men

By far the most serious health claim about air pollution is that it kills tens of thousands of Americans each year, mainly due to exposure to $PM_{2.5}$. There is no question that high levels of air pollution can kill. About 4,000 Londoners died during the infamous five-day "London Fog" of December 1952, when soot and sulfur dioxide soared to levels tens of times greater than the highest levels experienced in developed countries today, and visibility dropped to less than 20 feet.[34]

However, current fears center on whether today's comparatively low levels of air pollution are also deadly. An embarrassment for proponents of low-level air pollution as a cause of death is that the evidence is almost solely circumstantial, being based on statistical studies reporting small correlations between long- or short-term air pollution levels and risk of dying. These "observational" studies are not based on randomized trials, but on non-random data that inherently suffer from confounding by non-pollution factors with much larger effects on health than the purported effects of air pollution.

Observational studies could be taken more seriously if they were supported by evidence from randomized, controlled studies that eliminate the possibility of confounding by non-pollution factors. Such studies cannot, of course, be done with people, but they can be done with animals. However, researchers have been unable to kill animals with air pollution at levels anywhere near as low as the levels found in ambient air. As a recent review of particulate matter toxicology concluded:

> It remains the case that no form of ambient PM—other than viruses, bacteria, and biochemical antigens—has been shown, experimentally or clinically, to cause disease or death at concentrations remotely close to U.S. ambient levels.[35]

This seemingly changed in December 2005 when the *Journal of the American Medical Association* (*JAMA*) published the results of a study that claimed $PM_{2.5}$ at current ambient levels is increasing Americans' risk of developing heart disease. The study exposed mice to 85 $\mu g/m^3$ of $PM_{2.5}$ concentrated from ambient air for six hours per day for six months, or about one-fourth of a typical mouse life span.[36]

Mice fed a high-fat diet and exposed to $PM_{2.5}$ had more than a 50 percent greater rate of atherosclerosis (as measured by arterial plaque area) and other signs of heart disease, when compared with a control group that was fed a high-fat diet, but not exposed to $PM_{2.5}$. $PM_{2.5}$ was associated with greater atherosclerosis in mice on a low-fat diet as well, but the effect was not statistically significant.

NIH highlighted the study with a press release that begins: "Test results with laboratory mice show a direct cause-and-effect link between exposure to fine particle air pollution and the development of atherosclerosis . . . [The study] may explain why people who live in highly polluted areas have a higher risk of heart disease."[37] The study caused a minor media sensation, with both journalists and health experts claiming the study provides strong evidence that $PM_{2.5}$ is causing serious harm to human beings.[38]

Despite the enthusiastic reception, there is much less here than meets the eye. The mice used in the study were genetically engineered in ways that make them unrepresentative of even real-world mice, much less of humans. The mice were designed to lack the gene for apolipoprotein E (ApoE), a key substance for fat and cholesterol metabolism. As a result, these ApoE "knockout" mice have blood cholesterol levels 5 to 6 times greater than normal mice when fed regular rat chow. ApoE knockout mice have 14 times the cholesterol of normal mice when both are fed a high-fat diet.[39]

These are stupendous cholesterol levels. For comparison, medical authorities define "high cholesterol" as a serum cholesterol level greater than 240 milligrams per deciliter (mg/dl), which is about 20 percent greater than the average cholesterol level in American men.[40] Only one in 50 American men exceeds 1.5 times the U.S. average, and only one in 500 exceeds twice the average.[41]

The very reason for using such grossly unrealistic mice to study $PM_{2.5}$ is that $PM_{2.5}$ does not kill regular mice or other animals at PM concentrations relevant to real-world human exposures. For that matter, $PM_{2.5}$ did not actually kill the high-cholesterol mice in the study either.

NIH downplayed the vast gulf between the genetically engineered mice and normal mice, stating only that they were "genetically programmed to develop atherosclerosis at a higher-than-normal rate." This is a bit

like doing a study on people who weigh 500 pounds and referring to them merely as "overweight."

If you build a house out of cards, you would expect even a gentle breeze to knock it down. But this does not tell you much about the ability of a real house to withstand a gentle breeze. Likewise, if you design an artificial mouse that cannot regulate its fat or cholesterol levels, it is not surprising that even a minor environmental insult can cause it some health problems. But this does not tell you much about the effects of low-level air pollution levels on regular mice or on people.

Unfortunately, news articles on the study failed to provide the context that would show that study has little real-world relevance. A Nexis search turned up ten news reports on the study. Seven did not even mention that the mice had been genetically engineered, leaving the impression that real-world $PM_{2.5}$ levels caused heart disease in normal mice.

Three other news outlets followed NIH's lead, creating the impression that the mice in the study were merely analogous to people with a higher-than-average risk of heart disease. For example, according to the *Los Angeles Times*, the mice were "bred to be susceptible to developing heart disease."[42]

NIH and the study authors also misled reporters about the relevance of the $PM_{2.5}$ doses to real-world $PM_{2.5}$ levels. According to NIH, "The fine particle [$PM_{2.5}$] concentrations used in the study were well within the range of concentrations found in the air around major metropolitan areas." The press release also quotes one of the study's authors saying that "the average exposure over the course of the study was 15 micrograms per cubic meter, which is typical of the particle concentrations that urban area residents would be exposed to, and well below the federal air quality standard of 65 $\mu g/m^3$ over a 24-hour period."[43]

In fact, the $PM_{2.5}$ levels in the study were nothing like real-world $PM_{2.5}$ levels. The mice were exposed to $PM_{2.5}$ at 85 $\mu g/m^3$ for six hours in a row during five days of each week, and filtered air the rest of the time. Over the six-month study period, this does indeed average out to about 15 $\mu g/m^3$, the level of the federal $PM_{2.5}$ annual standard. But in the real world, areas that average 15 $\mu g/m^3$ of $PM_{2.5}$ over a year rarely approach short-term $PM_{2.5}$ levels of 85 $\mu g/m^3$.

For example, in the mouse study, the mice spent the equivalent of 1,560 hours per year breathing 85 $\mu g/m^3$ $PM_{2.5}$ (30 hours per week times 52 weeks per year). In contrast, Modesto California averaged 16 $\mu g/m^3$ of $PM_{2.5}$ over the past year, but spent only 80 hours at 85 $\mu g/m^3$ or above.[44] Furthermore, 40 percent of those high-$PM_{2.5}$ hours occurred between 11 p.m. and 6 a.m., when most people are in bed. There were only 420 hours when Modesto exceeded even 50 $\mu g/m^3$ of $PM_{2.5}$.

Even areas with the highest $PM_{2.5}$ levels in the country have far fewer hours of high $PM_{2.5}$ than were used in the mouse study. For example, Riverside California averaged 27 $\mu g/m^3$ $PM_{2.5}$ over the past year, but had only 135 hours at or above 85 $\mu g/m^3$, and 1,055 hours above 50 $\mu g/m^3$.

Health effects depend not only on the average dose, but on the acute dose. For example, you could take 2 aspirins 4 times per day, or you could take 8 all at once each day. Either way, your average dose is 8 aspirins per day. But you are more likely to suffer ill effects if you take the aspirins all at once. The mice received an analogously unrealistic daily $PM_{2.5}$ exposure. NIH and the scientists involved in the study then created the false appearance that this unrealistic exposure schedule has some relevance to the real world.

There is nothing wrong with the *JAMA* mouse study in principle. It shows that when you take a mouse specially designed to have unrealistically stupendous cholesterol levels, feed it a high-fat diet, and repeatedly expose it to unrealistically high acute levels of $PM_{2.5}$, that $PM_{2.5}$ increases the extent of heart disease. The problem arose when the study's proponents claimed that this has something to do with $PM_{2.5}$ risks faced by human beings.

You can now find a summary of the study on NIH's website. Its title? "Particulate Air Pollution and a High Fat Diet: A Potentially Deadly Combination."[45]

Sins of Omission

At the March meeting of the California Air Resources Board, staff members gave a detailed presentation on Jerrett et al. (2005)—a new epidemiological study of the Los Angeles region that reported a stronger link between $PM_{2.5}$ and mortality than suggested in previous research regulators have used to support tougher $PM_{2.5}$ standards.[46] What CARB's staff did not tell its board is that right around the same time that Jerrett et al. was published, another study of $PM_{2.5}$ risks in California by Enstrom (2005) concluded that $PM_{2.5}$ was having no effect on mortality.[47] Several California papers, including the *Los Angeles Times*, covered the alarming findings

of Jerrett et al. But none covered the benign results reported by Enstrom.

This is a typical pattern. Studies that report harm from air pollution receive a great deal of attention from regulators, environmentalists, and journalists. Studies finding no harm from air pollution are ignored. As a result, claims of harm from air pollution appear more consistent and robust than suggested by the actual weight of the evidence.

The American Lung Association's website includes an area called Medical Journal Watch, which summarizes hundreds of air pollution health studies.[48] But the site omits studies that do not report any harm from air pollution. For example, the site does not include any studies by Fred Lipfert, Suresh Moolgavkar, Richard Smith, Gary Koop, William Keatinge, or James Enstrom—all of whom have provided evidence against a connection between low-level air pollution and risk of death.[49]

The ALA also excludes specific studies and portions of studies that fail to find any harm from air pollution. For example, Medical Journal Watch does not mention Gong et al. (2003) and Holgate et al. (2003), which found little or no adverse health effects in human volunteers who breathed high levels of $PM_{2.5}$ and diesel soot, respectively.[50] The ALA does summarize the CHS findings on children's lung capacity discussed earlier, but does not mention that the study found that even the highest ozone levels in the country had no effect on lung growth.

Three studies have used CHS data to assess whether ozone is associated with increases in school absences. One study reported an increase.[51] Two reported no effect.[52] The ALA mentions only the first study on Medical Journal Watch. CARB likewise cites only the first study in its review of California's ozone standard.[53]

Coal-fired power plants have been one of environmentalists' premier targets during the last several years. In reports such as *Danger in the Air; Death, Disease and Dirty Power; Power to Kill; Children at Risk*; and many more, environmental groups claim that particulate pollution from power plants is killing thousands of Americans each year.[54] The Bush administration, a constant target of environmental groups for supposedly "gutting" power plant pollution requirements, last year adopted the Clean Air Interstate Rule (CAIR).[55] CAIR requires that power plants reduce their sulfur dioxide emissions by more than 70 percent below current levels.[56] Some sulfur dioxide is converted to ammonium sulfate in the atmosphere, and this is the main form of $PM_{2.5}$ from power plants. EPA claims these $PM_{2.5}$ reductions will prevent 17,000 premature deaths each year.[57]

Environmental groups want to increase support for ever more stringent regulations, maintain and enhance their control over other people's lives, and bring in the donations that support their activism. Regulators want to show the success of their efforts to reduce air pollution, but they also want to justify the need to preserve or expand their powers and budgets.

There is just one problem: ammonium sulfate is not toxic, even at levels many times those ever found in ambient air.[58] In fact, ammonium sulfate is used as an inert control—that is, a compound not expected to have any health effects—in studies of the health effects of acidic aerosols.[59] If ammonium sulfate is not toxic, then the campaign against $PM_{2.5}$ from power plants is based on a false premise.

Last year CARB adopted a tougher ozone standard for California.[60] To justify the tougher standard, CARB prepared a detailed report summarizing ozone health effects research. The report analyzes hundreds of health studies in nearly 1,000 pages, but fails to mention a study reporting that *higher* ozone was associated with a *lower* rate of hospital visits in California's Central Valley.[61] CARB was certainly aware of the existence of this study, because CARB funded and published it. EPA also failed to mention the study in its latest review of the federal ozone standard.[62]

EPA based its annual $PM_{2.5}$ standard mainly on the American Cancer Society (ACS) study, which followed more than 500,000 Americans in fifty cities from 1982 to 1989 and looked for correlations between $PM_{2.5}$ levels and risk of death.[63] The most recent ACS report covered the period from 1982 to 1998 and reported that each 10 $\mu g/m^3$ increase in long-term $PM_{2.5}$ levels is associated with a 4 percent increase in risk of death.[64]

The validity of epidemiological studies, such as the ACS study, depends on the assumption that correlations between air pollution and health outcomes represent genuine causal relationships. The implicit assumption is that after researchers have controlled for non-pollution

health factors like income or smoking, any residual correlation between air pollution and health represents a genuine causal linkage. Experience has shown that this assumption is false.

For example, a reanalysis of the ACS data showed that the apparent $PM_{2.5}$-mortality link was spurious. According to sensitivity analyses of the ACS data, $PM_{2.5}$ apparently kills men, but not women; those with no more than a high school degree, but not those with at least some college; and those who said they were moderately active, but not the very active or the sedentary.[65] Results like these are biologically implausible and suggest a failure to adequately control for confounding by non-pollution factors.

When migration rates into and out of various cities over time were added to the statistical model relating $PM_{2.5}$ and risk of death, the apparent effect of $PM_{2.5}$ disappeared.[66] Cities that lost population during the 1980s—Midwest "rust belt" cities—also had higher $PM_{2.5}$ levels. People left these cities, which were in economic decline, in search of work in more economically dynamic parts of the country. But people who work and have the wherewithal to migrate also tend to be healthier than the average person. Hence, what appeared to be an effect of $PM_{2.5}$ was actually the result of differential migration. Migration was just one of several confounding factors that diminished or erased the apparent harm from $PM_{2.5}$, but that were not accounted for by the ACS researchers.

This problem of spurious air pollution risk estimates is not limited to the ACS study, but is endemic to air pollution epidemiology and to epidemiology in general.[67] Nevertheless, scientists, regulators, and environmentalists have ignored these weaknesses and continue to make believe these spurious statistical correlations are telling us something real about the effects of low-level air pollution.

The Politics of Air Pollution Health Science

Most public information on air pollution and health comes from environmental activists, regulators, and health researchers. As these case studies show, their claims of harm from current, historically low air pollution levels are at best exaggerations and at worst fabrications. The result is unwarranted public fear, and continued support for ever more costly regulatory requirements that deliver little or no benefit in exchange for their high costs.

Regulators, environmentalists, and scientists enjoy substantial credibility with the public and the press. But like other interest groups, their goals often do not coincide with the interests of the vast majority of Americans. Environmental groups want to increase support for ever more stringent regulations, maintain and enhance their control over other people's lives, and bring in the donations that support their activism. Regulators want to show the success of their efforts to reduce air pollution, but they also want to justify the need to preserve or expand their powers and budgets. Maintaining a climate of crisis and pessimism meets these institutional goals, but at the expense of encouraging people to exaggerate the risks they face.

While it is not surprising that activists and regulators exaggerate air pollution risks, they would not be taken as seriously without scientific authority to back them up. The credibility of science and scientists flows from the power of scientific methods to uncover truths about the world, and from the perceived objectivity of scientists themselves. As the case studies above show, trust in scientific authority is often misplaced.

Scientific and medical research does have checks and balances that are absent from more explicitly political endeavors. Environmental health research nevertheless suffers from its own set of pressures that militate against evenhanded inquiry and dispassionate analysis and presentation of evidence. Studies that report harm from air pollution are more likely to be published than studies that do not. Regulatory agencies, whose power and budgets depend on the perception that air pollution is a serious health problem, are also major funders of the research intended to demonstrate the severity of the problem. Scientists who believe air pollution is a serious health threat and who report larger health effects are more likely to attract research funding. It is not a big leap to conclude that there is a great deal of selection bias in who does environmental health research, what questions they ask, and how they report their results.

Journalists should be acting as a check on air pollution misinformation, but they are not. Media outlets face their own pressures to sensationalize stories. Good news does not sell newspapers or attract viewers. As a result, journalists and editors are more likely to cover studies claiming harm from air pollution, and to pass along these claims with little or no critical review.

True, few journalists have the expertise to evaluate the technical merits of specific studies. But continuing

to rely on scientific authority will only perpetuate the problem of risk exaggeration. Among the major providers of public information on environmental risks, investigative reporters are in the best position to assess how the political economy of environmental health research affects the production and portrayal of scientific evidence. It would be a breath of fresh air if journalists and editors took up this challenge.

AEI editor Scott R. Palmer worked with Mr. Schwartz to edit and produce this Environmental Policy Outlook.

Notes

1. *The Environmental Deficit: Survey on American Attitudes on the Environment* (New Haven, CT: Yale Center for Environmental Law and Policy, May 2004), available at www.yale.edu/forestry/downloads/yale_poll_globalwarming.pdf. For similar results in state surveys, see *Recent Texas Statewide Survey Findings Prepared for Public Citizen and the Seed Coalition* (Alexandria, VA: American Viewpoint, 2002); New York Conservation Education Fund, *Key Findings of a Statewide Survey of New York State Residents on Environmental Issues* (New York: New York League of Conservation Voters, 2001); *Sprawl: New Jerseyans Dislike the Problems, and the Solutions* (New Brunswick, NJ: Newark *Star-Ledger*/Eagleton-Rutgers, September 29, 2002), available at http://slerp.rutgers.edu/retrieve.php?id=138-6; and M. Baldassare, *PPIC Statewide Survey: Special Survey on Californians and the Environment* (San Francisco: Public Policy Institute of California, July 2004).

2. *Water Pollution Tops Americans' Environmental Concerns* (Washington, D.C.: Gallup Poll, April 21, 2006), available at http://poll.gallup.com/content/Default.aspx?ci=22492&VERSION=p.

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4. Johns Hopkins School of Public Health News Center, "Traffic Exhaust Poisons Home Air," news release, August 31, 1999, available at www.jhsph.edu/PublicHealthNews/Press_Releases/PR_1999/traffic_exhaust.html; A. Di Rado, "USC Study Shows Air Pollution May Trigger Asthma in Young Athletes," news release, February 1, 2002, available at www.usc.edu/hsc/info/pr/1vol8/803/air.html; A. Di Rado, "Smog May Cause Lifelong Lung Deficits," University of Southern California, September 8, 2004, available at www.usc.edu/uscnews/stories/10495.html; and National Institutes of Health (NIH), "Link Strengthened between Lung Cancer, Heart Deaths and Tiny Particles of Soot," March 5, 2002, available at www.niehs.nih.gov/oc/news/lchlink.htm.

5. T. Avril, "Air Pollution's Threat Proving Worse than Believed," *Philadelphia Inquirer*, November 17, 2004; M. Cone, "State's Air Is among Nation's Most Toxic," *Los Angeles Times*, March 22, 2006, available at www.latimes.com/news/printedition/la-me-cancer22mar22,1,7087336.story; M. Cone, "Study Finds Smog Raises Death Rate," *Los Angeles Times*, November 17, 2004; T. Freemantle, "Asthma Risk for Children Soars with High Ozone Levels—Study," *Houston Chronicle*, February 1, 2002; and T. Webber, "Don't Breathe Deeply," *Indianapolis Star*, June 23, 2005.

6. California Air Resources Board, "Study Links Air Pollution and Asthma," news release, January 31, 2002, available at www.arb.ca.gov/newsrel/nr013102.htm.

7. The risk of developing asthma was 30 percent lower based on one-hour ozone levels and was statistically significant. Asthma risk was 20 percent lower based on eight-hour ozone levels and was just a hair short of statistical significance. (The top of the 95 percent confidence interval for relative risk was 1.0. Anything less than that would have been statistically significant.)

8. The journal article is R. McConnell, K. T. Berhane, F. Gilliland et al., "Asthma in Exercising Children Exposed to Ozone: A Cohort Study," *Lancet* 359 (2002): 386–91.

9. Once again the risk was 20 percent lower and was just barely short of statistical significance.

10. Pollution monitoring data from the Children's Health Study were provided by CARB's staff.

11. California Air Resources Board, "Study Links Air Pollution and Asthma," news release, January 31, 2002, available at www.arb.ca.gov/newsrel/nr013102.htm.

12. Dr. Norman Edelman, quoted in S. Borenstein, "Air Pollution Is a Cause of Asthma, Study Contends," *Philadelphia Inquirer*, February 1, 2002.

13. T. Freemantle, "Asthma Risk for Children Soars with High Ozone Levels—Study."

14. In fact, even the worst areas of Sacramento never average more than a few days per year exceeding the one-hour ozone standard and 20 or so days per year exceeding the eight-hour standard—ozone levels typical of the "medium-ozone" CHS communities, in which there was no relationship between air pollution and asthma risk. Dr. Jesse Joad, quoted in C. Bowman, "Asthma's Toll: A New Study Links Children's Sports Activities in Smoggy Areas to the Illness," *Sacramento Bee*, February 1, 2002.

15. G. D. Thurston and D. V. Bates, "Air Pollution as an Underappreciated Cause of Asthma Symptoms," *Journal of the American Medical Association* 290 (2003): 1915–17.

16. "Smog and Asthma: The Link—and Threat—Are Real," *Sacramento Bee*, May 6, 2003.

17. W. J. Gauderman, E. Avol, F. Gilliland et al., "The Effect of Air Pollution on Lung Development from 10 to 18 Years of Age," *New England Journal of Medicine* 351 (2004): 1057–67.

18. The CHS study set up special-purpose monitors to measure pollution levels in the communities where the study was performed. CARB staff provided data from these monitors.

19. *Impacts of Ozone on Our Health* (Charlotte, NC: Carolinas Clean Air Coalition, undated), available at http://003af56.netsolhost.com/air_basics_ozone_impact.htm.

20. Based on research by CARB staff, these values have been adjusted upward by 13.6 percent to make them comparable with $PM_{2.5}$ levels determined by the Federal Reference Method, which has been used nationwide since 1999 for determining compliance with federal $PM_{2.5}$ standards. See N. Motallebi, J. Taylor, A. Clinton, B. E. Croes et al., "Particulate Matter in California: Part 1—Intercomparison of Several $PM_{2.5}$, $PM_{10\text{-}2.5}$, and PM_{10} Monitoring Networks," *Journal of the Air & Waste Management Association* 53 (2003): 1509–16.

21. Based on IPN data for Riverside collected in the early 1980s, and $PM_{2.5}$ data collected by CARB in 1988 and 1989 and retrieved from CARB's 2006 Air Pollution Data CD, www.arb.ca.gov/aqd/aqdcd/aqdcd.htm. Once again, I have corrected these values for the change in measurement methods.

22. Di Rado, "Smog May Cause Lifelong Lung Deficits."

23. Dr. Kenneth Olden, quoted in NIH, "New Research Shows Air Pollution Can Reduce Children's Lung Function," news release, September 9, 2004, www.nih.gov/news/pr/sep2004/niehs-08a.htm.

24. Here's how: First, note from table 3 of the *NEJM* article that $PM_{2.5}$ was associated with a 79.7 milliliter (ml) reduction in FEV_1 between the least and most polluted community. Then from table 2, note that at eighteen years of age average FEV_1 was 3,332 ml for girls and 4,464 ml for boys. Given that there were 876 girls and 883 boys in the study (p. 1,059, column 1), the weighted average FEV_1 for the study population was 3,900 ml. The percentage decline is then 79.7/3,900 = 0.02 or 2 percent. A similar calculation can be done to show that the average decline in FVC was 1.3 percent. Gauderman, Avol, Gilliland et al., "The Effect of Air Pollution on Lung Development from 10 to 18 Years of Age."

25. The researchers used a regression model to create this "predicted" value.

26. This is assuming the "predicted" lung capacity values are valid. The *NEJM* paper provides few details on the model or the underlying distribution of lung-function test scores by community. Thus, another problem with this outcome measure is that it depends on something that was not actually measured!

27. NIH, "New Research Shows Air Pollution Can Reduce Children's Lung Function."

28. American Lung Association, *State of the Air 2005* (Washington, D.C.: May 2005), 60.

29. N. Bryant, "What Air Quality Problem?" *Charlotte Observer*, September 1, 2005, available at www.charlotte.com/mld/charlotte/news/opinion/12530112.htm?BMIDS=13194.

30. A. Fell, "Primate Research Shows Link between Ozone Pollution, Asthma," U.C.-Davis news release, October 13, 2000, available at www.dateline.ucdavis.edu/101300/DL_asthma.html.

31. *Recent Scientific Findings on Health Effects of Air Pollution and Diesel Exhaust* (Oakland, CA: American Lung Association of California, 2003), available at www.californialung.org/spotlight/cleanair03_research.html.

32. Melanie Turner, "Kids Focus of Air Quality Study Researcher Looking for Link Between Ozone, Asthma in Youth," *Modesto Bee*, May 11, 2001.

33. Edie Lau, "Study Suggests Asthma Culprit; Young Lungs Exposed to Ozone Seem More Prone to Problems with Development," *Sacramento Bee*, April 15, 2001.

34. I. M. Goklany, *Clearing the Air: The Real Story of the War on Air Pollution* (Washington, D.C.: Cato Institute, 1999).

35. L. C. Green and S. R. Armstrong, "Particulate Matter in Ambient Air and Mortality: Toxicologic Perspectives," *Regulatory Toxicology and Pharmacology* 38 (2003): 326–35.

36. Q. Sun, A. Wang, X. Jin et al., "Long-Term Air Pollution Exposure and Acceleration of Atherosclerosis and Vascular Inflammation in an Animal Model," *Journal of the American Medical Association* 294 (2005): 3003–10.

37. NIH, "Air Pollution, High-Fat Diet Cause Atherosclerosis in Laboratory Mice," news release, December 22, 2005, available at www.nih.gov/news/pr/dec2005/niehs-22.htm.

38. Newspapers carrying articles on the study included the *Los Angeles Times*, *Houston Chronicle*, *Philadelphia Inquirer*, and several others.

39. A. S. Plump, J. D. Smith, T. Hayek et al., "Severe Hypercholesterolemia and Atherosclerosis in Apolipoprotein E-Deficient Mice Created by Homologous Recombination in Es Cells," *Cell* 71 (1992): 343–53; and S. H. Zhang, R. L. Reddick, J. A. Piedrahita et al., "Spontaneous Hypercholesterolemia and Arterial Lesions in Mice Lacking Apolipoprotein E," *Science* 258 (1992): 468–71.

40. See table 70 in National Center for Health Statistics, *Health, United States, 2005* (Hyattsville, MD: U.S. Department of Health and Human Services, 2005), available at www.cdc.gov/nchs/data/hus/hus05.pdf#070.

41. Based on National Health and Nutrition Examination Survey (NHANES) data on 4,090 adult men collected from 1999–2002. Data were downloaded from www.cdc.gov/nchs/nhanes.htm.

42. M. Bustillo and M. Cone, "EPA Issues New Plan to Limit Soot; Critics Say the Revised Standard Is Too Weak to Properly Protect the Public from Health Dangers Caused by Breathing Particulates," *Los Angeles Times*, December 21, 2005.

43. NIH, "Air Pollution, High-Fat Diet Cause Atherosclerosis in Laboratory Mice."

44. Hourly $PM_{2.5}$ data were downloaded from CARB at www.arb.ca.gov/aqmis2/paqdselect.php.

45. M. Lippmann, L. C. Chen, and S. Rajagopalan, "Particulate Air Pollution and a High Fat Diet: A Potentially Deadly Combination," NIH, available at www.niehs.nih.gov/dert/profiles/hilites/2005/pm-diet.htm.

46. *Stronger Relationship between Particulate Matter (PM) and Premature Death* (Sacramento: California Air Resources Board, March 23, 2006), available at ftp://ftp.arb.ca.gov/carbis/board/books/2006/032306/06-3-1pres.pdf. This presentation was based on the results of M. Jerrett, R. T. Burnett, R. Ma et al., "Spatial Analysis of Air Pollution and Mortality in Los Angeles," *Epidemiology* 16 (2005): 727–36.

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51. F. D. Gilliland, K. Berhane, E. B. Rappaport et al., "The Effects of Ambient Air Pollution on School Absenteeism Due to Respiratory Illnesses," *Epidemiology* 12 (2001): 43–54.

52. K. Berhane and D. C. Thomas, "A Two-Stage Model for Multiple Time Series Data of Counts," *Biostatistics* 3 (2002): 21–32; and V. Rondeau, K. Berhane, and D. C. Thomas, "A Three-Level Model for Binary Time-Series Data: The Effects of Air Pollution on School Absences in the Southern California Children's Health Study," *Statistics in Medicine* 24 (2005): 1103–15.

53. *Review of the California Ambient Air Quality Standard for Ozone* (Sacramento: California Air Resources Board, March 2005), available at www.arb.ca.gov/research/aaqs/ozone-rs/ozone-final/ozone-final.htm; J. Schwartz, "Rethinking the California Air Resources Board's Ozone Standards" (working paper, AEI, Washington, D.C., September 2005), www.aei.org/publication23145.

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Attachment 3: Resume of Dr. Lucy Fraiser, PhD, DABT

Lucy H. Fraiser, Ph.D., DABT
Senior Toxicologist/Associate Vice President

Professional History

AECOM Corporation
Signature Science, LLC
MFG Inc.
URS Corporation
Texas Natural Resource Conservation Commission
Radian Corporation

Education

Ph.D (Toxicology) University of Texas at Austin
B.A. (Psychology) University of Texas at Austin

Years of Experience 25

Technical Specialties

Toxicological Evaluations
Risk Assessments
Risk Communication
Litigation Support
Agency Negotiations
Development of Innovative Risk-Based Approaches
Exposure Modeling

Professional Affiliations

Diplomat of the American Board of Toxicology
National Member, Air & Waste Management Association
Society of Environmental Toxicology and Chemistry

Dr. Fraiser is a Senior Consulting Toxicologist, Operations Manager for AECOM Environment's Austin and Dallas offices, and Associate Vice President of AECOM Environment North America (Austin, Texas). She has 21 years of experience in the areas of human health risk assessment, development of quantitative toxicity criteria, health effects and toxicology research, risk communication, and litigation support. While she has extensive experience in all areas of risk assessment, she specialized in air quality risk assessment. She has acted as Health Risk Assessment Team Lead and/or Project Manager for numerous human health and ecological risk assessments of air pollutants emitted from chemical plants, waste management facilities, army depots, cement kilns and power plants. She is part of a successful proposal team for a study on the development of POP (persistent organic pollutant) inventory and monitoring program in the Philippines and will act as the Project Manager and Health Risk Assessment Team Leader on that project. Dr. Fraiser was the Health Risk Assessment Team Lead for a recently completed Phase 1 Engineering Investigation and Environmental Study conducted by AECOM for the siting of a Municipal Solid Waste Incinerator in Hong Kong. Because of her extensive experience with human health risk assessments for hazardous waste combustion units, Dr. Fraiser was invited, along with several others at AECOM, to develop U.S. Environmental Protection Agency (EPA) training materials on various aspects of the U.S. National Emission Standards for Hazardous Air Pollutants (NESHAPs) regulations for Hazardous Waste Combustors and to teach the site-specific risk assessment component of the training in 2008.

Representative project experience follows.

Experience

Toxicological Evaluations and Risk-Based Regulatory Criteria Development . As part of a federal litigation case on behalf of a major multi-national petrochemical company, developed a health-based effects screening level for hydrogen sulfide in air.

Toxicological Evaluations and Risk-Based Regulatory Criteria Development. Developed interim Effects Screening Levels (air action levels) for Toluene Diisocyanate and

Hexamethylene Diisocyanate on behalf of a major chemical company for use in permitting Maintenance, Startup, Shutdown activities at the facility. A report documenting the development of the Effects Screening Levels is currently being reviewed by the Texas Commission on Environmental Quality.

Toxicological Evaluations and Risk-Based Regulatory Criteria Development. Researched toxicity and risk-based criteria information on Perfluorooctanic Acid (PFOA) and Perfluorooctane Sulfonate (PFOS) to educate management and potential client on the toxicity and status of risk-based criteria development for perfluorinated compounds.

Toxicological Evaluations and Risk-Based Regulatory Criteria Development. Developed a risk-based soil cleanup objective for insoluble inorganic mercury based on a review of available toxicity, bioavailability, and plant uptake studies for insoluble mercury salts.

Toxicological Evaluations and Risk-Based Regulatory Criteria Development. Critically reviewed an Air Quality Health Consultation conducted by the Texas Department of State Health Services (DSHS) and the Agency for Toxic Substances and Disease Registry (ATSDR) on behalf of a commercial client. Responsibilities included working with the client's attorney to develop formal comments and representing the client in meetings with the DSHS and ATSDR. Health department data on birth defects were reviewed to evaluate whether community concerns about birth defects were likely related to emissions from the client's hazardous waste combustion unit. Constituents of concern included aldehydes, chlorinated and non-chlorinated volatile organic compounds, and metals.

Toxicological Evaluations and Risk-Based Regulatory Criteria Development. Evaluated potential health effects associated with methanol and ethanol emissions from tailpipe emissions under different test scenarios. The exhaust concentrations were derived from composite emission results from two different diesel engines. It was determined that ethanol and methanol concentrations in tailpipe exhaust are below acute and chronic health protective concentrations.

Toxicological Evaluations and Risk-Based Regulatory Criteria Development. Developed an alternate to EPA's acute inhalation toxicity benchmark for nickel and nickel compounds on behalf of a commercial client based on site-specific speciation data and information from the toxicological literature.

Toxicological Evaluations and Risk-Based Regulatory Criteria Development. Used the National Library of Medicine's ChemIDPlus database to search for compounds with similar chemical structures to dye constituents and information on chemical structure to identify appropriate surrogate health benchmarks for dyestuffs lacking health benchmarks.

Toxicological Evaluations and Risk-Based Regulatory Criteria Development. Developed an Emergency Response Planning Guideline (ERPG) for a reactant (thionyl chloride) used in organic synthesis, which involved estimating toxicological properties of the previously uncharacterized compound on the basis of knowledge of chemistry concepts (i.e., stoichiometry of hydrolysis).

Toxicological Evaluations and Risk-Based Regulatory Criteria Development. Developed water quality criteria for methyl isobutyl ketone and methyl isobutyl carbinol that are protective of human health and domestic animals utilizing criteria set forth in the Illinois Environmental Protection Agency Water Quality Standards. Reviewed available toxicology data and recommended a suitable chemical for use as a surrogate in calculating aquatic criteria in the absence of toxicological data for methyl isobutyl carbinol.

Toxicological Evaluations and Risk-Based Regulatory Criteria Development. Performed a human health exposure and risk assessment for Rodeo® herbicide based on its intended use patterns, including applicator exposure, consumer safety, and terrestrial wildlife. The product subsequently received registration approval in the state of Connecticut.

Toxicological Evaluations and Risk-Based Regulatory Criteria Development. Reviewed toxicity data and derived an inhalation reference concentration (RfC) for cesium oxide, a gasoline additive that is used in France, using EPA's "Interim Methods for Development of Inhalation Reference Concentrations" on behalf of a commercial client.

Toxicological Evaluations and Risk-Based Regulatory Criteria Development. Served as primary author on a successful delisting petition for di-n-octylphthalate in which similarly-structured phthalates were evaluated.

Toxicological Evaluations and Risk-Based Regulatory Criteria Development. Reviewed toxicity studies from the chemical inventory of a large chemical company and made

recommendations for submittal under TSCA 8E (Toxic Substances Control Act).

Air Quality Risk Assessment. Health Risk Assessment Advisor for a study recently completed on behalf of the Electric Power Research Institute (EPRI) that evaluated the potential health risk from emissions of coal fired power plants throughout the U.S. Responsibilities included consultation with project risk assessors on various technical issues, senior review of human health risk assessments and associated calculations, and presentation of information in a joint EPRI/Utility Air Research Group meeting in May, 2012.

Air Quality Risk Assessment. Recently provided comments on Boiler MACT Health Based Emissions Limitations on behalf of the American Forest and Paper Association.

Air Quality Risk Assessment. Health Risk Assessment Team Leader responsible for overseeing the conduct of a risk assessment as part of an engineering investigation (EI) and environmental impact assessment (EIA) study for two potential sites of an Integrated Waste Management Facility in Hong Kong. The project entailed using air dispersion and deposition modeling to evaluate exposure, and calculating potential risk associated with both stack and fugitive air emissions from the facilities.

Air Quality Risk Assessment. Recently acted as project manager for multi-pathway risk assessment updates for two chemical plants in the U.S. (Houston Texas and Baton Rouge Louisiana) to support permitting activity that reflected the installation of new sulfur dioxide (SO2) abatement equipment on sulfuric acid regeneration units.

Air Quality Risk Assessment. Conducted a Best Available Control Technology (BACT) analysis for carbon monoxide (CO), particulate matter, sulfur dioxide (SO2), nitrogen oxides (NOx), and volatile organic compounds (VOCs) on behalf of a meat processing and rendering plant in Texas.

Air Quality Risk Assessment. Health risk assessment lead for vapor intrusion evaluation, using crawl-space soil vapor and ambient air samples collected beneath and near a house in the vicinity of a crude oil release from a buried flow line.

Air Quality Risk Assessment. Health risk assessment lead for vapor intrusion evaluation, using indoor and ambient air samples at manufacturing facility.

Air Quality Risk Assessment. Served as task leader for over two dozen human health risk assessments conducted in support of RCRA Part B permit applications for hazardous waste combustion units at chemical plants, waste management facilities, army depots, and cement kilns. Responsibilities included oversight and coordination of staff conducting modeling, reporting for the project, representation of clients in Agency meetings, and complete responsibility for all financial and technical aspects of the risk assessments. Primary risk drivers routinely consisted of Polycyclic Aromatic Hydrocarbons (PAHs), mercury, chromium, phthalates, and dioxins.

Air Quality Risk Assessment. Evaluated the potential for health effects associated with particulates and metals present in air samples collected by neighbourhood volunteers when noxious odors were detected.

Air Quality Risk Assessment. Completed formal technical comments on behalf of a power plant client on the Strategic Toxic Air Reduction (STAR) program, a risk-based program intended to significantly reduce levels of toxic air contaminants in Louisville, KY.

Air Quality Risk Assessment. Conducted a risk evaluation of site operations that described potential impacts to on-site workers, the surrounding community, and ecological receptors. In combination with information on chemical hazards, local exposure patterns, and available local media concentrations, toxic release inventory (TRI) data were used to identify chemical release and material handling practices at the plant that may warrant further study or action.

Air Quality Risk Assessment. Served as project manager and technical lead for multipathway exposure and risk assessments conducted for three lignite-fired utility plants. Toxic Release Inventory (TRI) data were used as the basis for emission estimates.

Air Quality Risk Assessment. Served as project manager and technical lead for mercury exposure and risk assessments conducted for three lignite-fired utility plants in which the Electric Power Research Institute’s (EPRI’s) Dynamic Mercury Cycling model was used. TRI data were used as the basis for emission estimates.

Air Quality Risk Assessment. Developed technical comments on EPA Risk Assessment Protocols for Hazardous Waste

Combustion Facilities on behalf of the Louisiana Chemical Association (LCA) and the Cement Kiln Recycling Coalition (CKRC). Included evaluating chemical-specific approaches, including those for Polycyclic Aromatic Hydrocarbons (PAHs). In particular, the benefit of including a metabolism factor for PAHs in calculating PAH concentrations in beef, milk, pork, poultry, eggs, and fish was evaluated and commented upon.

Air Quality Risk Assessment. Had primary responsibility within the Toxicology & Risk Assessment Section for implementation of the Texas Natural Resource Conservation Commission's (TNRCC) Combustion Strategy. Was instrumental in recognizing issues associated with high Polycyclic Aromatic Hydrocarbon (PAH) detection limits typical in environmental media samples and spear-headed the development of guidance/approaches for ensuring that high PAH detection do not artificially inflate risk estimates.

Air Quality Risk Assessment. Represented the TNRCC in an EPA work group to evaluate Maximum Achievable Control Technology (MACT) standards developed under the Clean Air Act for hazardous waste incinerators, cement kilns, and lightweight aggregate kilns. Highly advanced technical support documentation was critically reviewed and concerns and ideas regarding each standard were conveyed to EPA during the pre-proposal stage.

Air Quality Risk Assessment. Served as an external peer reviewer for a draft exposure and risk assessment guidance document developed by EPA Region 6 for conducting exposure and risk assessments at facilities that burn hazardous waste ("Protocol for Screening Level Human Health Risk Assessment at Hazardous Waste Combustion Facilities" and "Screening Level Ecological Risk Assessment Protocol for Hazardous Waste Combustion Facilities") while employed by the TNRCC. Evaluated and commented upon various chemical-specific approaches, including those for Polycyclic Aromatic Hydrocarbons (PAHs).

Risk Based Corrective Action. Served as task leader for over 75 human health risk assessments and/or risk-based evaluations conducted in support of Resource Conservation Recovery Act (RCRA) closures or under the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA) for both commercial companies (petrochemical, agrochemical/pesticide, gas supply, aluminum, electric utility, cement kiln) and government clients (Army Corp of Engineers [USACE], Air Force

Center for Environmental Excellence [AFCEE], Air Force Bases [Cannon, Davis-Monthan, Holloman, Langley, Maxwell]). Responsibilities included oversight and coordination of staff conducting modeling and reporting, representation of clients in Agency meetings, and complete responsibility for all financial and technical aspects of the risk assessments.

Risk Based Corrective Action. Currently acting as Health Risk Assessment Lead for a multi-media health risk assessment for a high school at which incinerator ash was used as fill material beneath several sports fields. The primary constituents of concern are polychlorinated biphenyls (PCBs).

Risk Based Corrective Action. Currently acting as Health Risk Assessment Lead for a multi-media health risk assessment for a former refinery.

Risk Based Corrective Action. Recently completed a human health and screening ecological risk assessment for several areas within a former refinery in Southeast Asia in preparation for divestment.

Risk Based Corrective Action. Currently supporting an evaluation of a historical polychlorinated biphenyl (PCB) release under the risk-based provisions of 40 CFR 761.61(c) of the TSCA regulations for an Independent School District in the Northeast.

Risk Based Corrective Action. Currently providing risk assessment and toxicology support on a very large remediation project for a former battery manufacturing company. Contaminants of concern include mercury and lead.

Risk Based Corrective Action. Currently providing toxicology and risk-based corrective action support to a consortium of Potentially Responsible Parties on a remediation project for a river segment in the Northeast. Primary contaminants of concern include dioxins, polychlorinated biphenyls, polycyclic aromatic hydrocarbons (PAHs), arsenic, lead, mercury.

Risk Based Corrective Action. Currently involved in conducting a risk-based evaluation of a former production well site for submittal to the Texas Railroad Commission.

Risk Based Corrective Action. Currently involved in developing a risk-based strategy for achieving regulatory closure of numerous oil field leases at a very large Texas oil field. Responsibilities include developing site-specific standard

operating procedures, preparing reports on risk-based evaluations for submittal to the Texas Railroad Commission, and representing the client in Agency meetings.

Risk Based Corrective Action. Performed detailed human health and screening ecological risk assessments for a former lube oil refinery intended for divestiture in Southeast Asia. Responsibilities included interpretation of sampling data generated by another contractor for use in the risk assessment, coordination of groundwater modeling, exposure modeling, reporting, and complete responsibility for all financial and technical aspects of the risk assessment project. The risk assessment was conducted in a manner consistent with International guidance on the conduct of risk assessments. An extensive exposure pathway analysis was conducted for the site, including inhalation of vapors from in indoor and outdoor air, dermal contact with water and soil, ingestion of water and soil, and uptake through the food chain (i.e., vegetables).

Risk Based Corrective Action. Performed a post-remediation human health risk assessment for an active petroleum depot in Southeast Asia. Responsibilities included modeling and reporting for the project, representation of the client before a health panel of scientists convened to review the risk assessments, and complete responsibility for all financial and technical aspects of the risk assessment project. The risk assessment was conducted in a manner consistent with International guidance on the conduct of risk assessments.

Risk Based Corrective Action. Performed a human health risk assessment for an operational petroleum dispensing station in Southeast Asia. Responsibilities included coordination with both U.S. and Asian staff conducting sampling, modeling, and reporting for the project, representation of the client before a health panel of scientists convened to review the risk assessments, and complete responsibility for all financial and technical aspects of the risk assessment project. The risk assessment was conducted in a manner consistent with International guidance on the conduct of risk assessments. An extensive exposure pathway analysis was conducted for the site, including inhalation of vapors from in indoor and outdoor air, dermal contact with water, ingestion of water, and uptake through the food chain (i.e., vegetables, chicken, and eggs).

Risk Based Corrective Action. Served as task leader for three human health risk assessments and one ecological risk assessment for an active petroleum depot in Southeast Asia.

Responsibilities included oversight and coordination of both U.S. and Asian staff conducting sampling, modeling, and reporting for the project, representation of the client before a health panel of scientists convened to review the risk assessments, and complete responsibility for all financial and technical aspects of the risk assessment project. The risk assessment was conducted in a manner consistent with International guidance on the conduct of risk assessments. An extensive exposure pathway analysis was conducted for the site, including inhalation of vapors from in indoor and outdoor air, dermal contact with soil and water, ingestion of soil and water, and uptake through the food chain (i.e., vegetables, chicken, and eggs).

Risk Based Corrective Action. Developed numerous approaches to ensure internal consistency in risk assessments conducted by Texas Natural Resource Conservation Commission (TNRCC) personnel, the review of risk assessments submitted to the TNRCC, and data collection procedures while employed by the TNRCC. Dr. Fraiser also played a critical role in developing a risk-based corrective action rule package (Texas Risk Reduction Program) and technical support guidance documents for the proposed rule.

Risk Based Corrective Action. Significant involvement in a large-scale project in which risk-based tools for use by the Air Force were evaluated and recommended. Risk-based cleanup options at Installation Restoration Program (IRP) and Resource Conservation and Recovery Act (RCRA) sites located on Air Force Bases were reviewed to identify Records of Decision (RODs) amenable to modification.

Risk Based Corrective Action. Participated in the development of an approach for establishing cleanup criteria for Air Force Bases nationwide as part of the Rational National Standards Initiative (RNSI). During the course of the project, Dr. Fraiser was involved in negotiations with military personnel and regulators.

Testifying and Litigation Support. Currently providing litigation support in a federal case on behalf of a major multi-national petrochemical company. Constituents of concern include sulfur dioxide, hydrogen sulfide, nitrogen oxides, ozone and volatile organic compounds associated with emissions from a refinery, a chemical plant, and an olefins plant. The case also involves evaluating potential odor nuisances. Dr. Fraiser was deposed on June 20, 2012 and the hearing is expected to take place in the fall of 2012.

Testifying and Litigation Support. Provided testimony on potential risks associated with permitting of a rock crusher. Constituents of concern for the case are anticipated to be PM10, PM2.5, and crystalline silica. Hearing took place in 2011 and construction permit was granted.

Testifying and Litigation Support. Consulting toxicologist on behalf plaintiff in a lawsuit brought against a steel tank manufacturer by the State of Texas.

Testifying and Litigation Support. Provided toxicological support and testified in a contested case hearing involving a Texas rock crusher. Constituents of concern for the case are PM10, PM2.5, limestone and crystalline silica. The permit was granted in December 2010.

Testifying and Litigation Support. Criteria Pollutant, Air Toxics, and Odor Nuisance Litigation Support: Currently providing litigation support in a federal case on behalf of a major multi-national petrochemical company. Constituents of concern include sulfur dioxide, hydrogen sulphide, nitrogen oxides, ozone and volatile organic compounds associated with emissions from a refinery, a chemical plant, and an olefins plant. Dr. Fraiser will be deposed on June 20, 2012 and the hearing is expected to take place in the fall of 2012.

Testifying and Litigation Support. Developed an opinion regarding the likelihood that a level of exposure sufficient to cause known health effects could have occurred as a result of a hydrogen sulfide (H2S) and/or sulfur dioxide (SO2) release from a Sulfur Recovery Unit at a Refinery in Texas. Development of the opinion entailed review of medical records, air dispersion model results, depositions, and discovery responses of the defendant and plaintiff. The case settled in 2009.

Testifying and Litigation Support. Testified on potential risks associated with permitting of a concrete batch plant. Constituents of concern for the case were PM10, PM2.5, total suspended particulate and crystalline silica. Dr. Fraiser was deposed and prepared pre-filed testimony in this case. Permit was granted.

Testifying and Litigation Support. Testified on potential risks associated with permitting of a copper smelter on behalf of the City of El Paso. Constituents of concern for the case were metals, PM10, PM2.5, nitric oxide, sulfur dioxide, and sulfuric acid. Hearing took place in July 2005. Permit was denied.

Testifying and Litigation Support. Testified on potential risks associated with permitting of a rock crusher. Constituents of concern for the case are PM10, PM2.5, and crystalline silica. Dr. Fraiser was deposed and prepared pre-filed testimony in this case. Hearing took place July 2005.

Testifying and Litigation Support. Provided critical testimony on potential risks associated with emissions from a commercial hazardous waste incinerator. The Kentucky Department of Environmental Protection attempted to revoke the facility's RCRA Interim Status Part B permit for alleged violations of RCRA and the Clean Air Act. As a result of the combined testimony of Dr. Fraiser and one of her colleagues, the client's request for a restraining order was granted and 75 employees kept their jobs.

Testifying and Litigation Support. Developed expert opinion and provided testimony in a criminal case hearing regarding the potential for health effects associated with relatively short-term exposure to benzene concentrations above the Maximum Contaminant Level (MCL) in groundwater. Case was lost.

Testifying and Litigation Support. Prepared to provide expert testimony on potential risks associated with lead and total petroleum hydrocarbon (TPH) levels detected in street sweepings for a criminal case hearing. Issues considered included potential risk associated with contaminant levels that were detected and potential for matrix interferences associated with laboratory methods. The case settled before going to trial.

Testifying and Litigation Support. Served as an expert witness on toxicology and risk assessment issues in a contested case hearing involving the Texas Natural Resource Conservation Commission (TNRCC) and the first Boiler & Industrial Furnace (BIF) to be permitted under Subpart H of 40 CFR 266 in the state of Texas (only second BIF to be permitted in the nation).

Televised Press Conference. Peer reviewed of a paper entitled "Proximity to Point Sources of Environmental Mercury Release as a Predictor of Autism Prevalence" and conducted a televised press conference on the findings of the paper on behalf of the Clean Coal Technology Foundation of Texas.

Testimony Before Regulatory Agencies/Bodies. Provided technical support to the American Clean Coal Coalition for Electricity, a non-profit group dedicated to protecting the viability of coal-based electricity, in meetings with various state regulatory agencies (Texas Commission on Environmental Quality, North

Carolina Department of Environment and Natural Resources) and regulatory bodies (Environmental Regulations Committee of Texas House of Representatives and the Cultural, Recreation and Tourism Committee of Texas Senate) on mercury emission issues.

Publications, Presentations, and Training Courses

Fraiser, L.H. and Vosnakis, K.A.S. Evolution of PCB Regulations and Toxicity Assessment: Impact on Environmental Management. 27th Annual International Conference on Soils, Sediments, Water and Energy, Amherst, Massachusetts. October 17 – 19, 2011.

Fraiser, L.H. Toxicology & Risk Assessment in the News: Recent EPA Proposals with Broad Implications. Houston Air & Waste Management Association. June 2010.

Fraiser, L.H., Quintin, A. Durocher, K. Szembek, C. Heinold, D. EPRI Human Health and Environmental Risk Assessment Process. February 18, 2010.

Fraiser, L.H. Trends in International Risk-Based Screening Levels (RBSLs). Society of Toxicology and Chemistry, New Orleans, Louisiana. November 19 – 23, 2009.

Fraiser, L.H. Risk Assessment: How it Can Inform Site Closure Decisions. Invited Short Course presented to the Department of Environment Malaysia, Kuala Lumpur. March 4 – 5, 2009.

Fraiser, L.H. Incinerator Risk Assessment: Principles and Practices, Hong Kong. Regional Conference on Sustainable Waste Management in Carbon-Conscious Cities. December 2008.

Fraiser, L.H. and Quintin, A. International Screening Levels. White Paper prepared for ExxonMobil, Fairfax, VA. December, 2008.

Site-Specific Risk Assessments, RCRA Omnibus Provision and Combining Risk Burns and Comprehensive Performance Tests. MACT EEE EPA Training Workshop, Dallas, TX. November 3 – 8, 2008.

Fraiser, L.H., Quintin, A. and Ng, H.S. Comparison of International Risk-Based Screening Levels. Brownfield Asia 2008, Third International Conference on Remediation and Management of Contaminated Land: Focus on Asia October 21 –

23, 2008, Kuala Lumpur, Malaysia.

Fraiser, L.H. Involvement of Local Governments in Air Toxics Regulation. Texas Chemical Council/ Association of Chemical Industry of Texas's EH&S Seminar Moody Gardens Hotel, Galveston Texas. June 10, 2008.

Fraiser, L.H. Chemical and Microbial Risk Assessment: Establishing Environmental Endpoints for use in Regulatory Decision-Making. Presented at a meeting jointly organized by the Hong Kong Institute of Environmental Impact Assessment and Chartered Institution of Water and Environmental Management. May 9, 2008.

Coal-fired Power Plants: Assessing the Positive Health Benefits of Reliable and Low Cost Electricity. White Paper prepared for Center for Energy and Economic Development. March 2007. Introduction to Toxin Threat Agents, Confidential Government Client, Austin, TX. January 2006.

Toxins as Threat Agents, Confidential Government Client, Austin, TX. July 2003.

Environmental Risk and Impact: What Do the Numbers Really Mean? Society of Women Engineers, Atlanta, GA. October 11, 2003.

TRRP-13 - Review and Reporting of COC [chemical of concern] Concentration Data, Central Texas Chapter of the Air & Waste Management Association, 301 Congress Avenue, Room 360, Austin, TX. January 16, 2003.

Health Studies Conducted in Midlothian, TX, Tarrant County Medical Society, Renaissance Hotel, Austin, TX. January 18, 2002:

Fraiser, L.H., and Chaudhuri, I. Short-Term Toxicity Benchmark for Nickel Oxide. To be presented at the 42nd Annual Society of Toxicology Meeting, Salt Lake City, Utah. March 9 – 14, 2002.

Fraiser, L.H., and Ruffle, B. Chemical Regulations with Business Implications. Environmental Protection. June, 2002.

Fraiser, L.H., and Chaudhuri, I. Short-Term Toxicity Benchmark for Nickel Oxide. International Conference on Incineration & Thermal Treatment Technologies Proceedings, New Orleans, Louisiana. May 13 -17, 2002.

Fraiser, L.H., and Chaudhuri, I. Short-Term Toxicity Benchmark for Nickel Oxide. Proceedings of the Air & Waste Management Association, St. Louis, Missouri. April 16 – 19 2002.

Fraiser, L.H., Chaudhuri, I, and Smith, D. EPA's Dioxin Reassessment – Potential Impacts to the Regulated Community. Proceedings of the Air & Waste Management Association, Orlando, Florida. June 24 - 28, 2001.

Fraiser, L.H., Roeck, D., and Smith, D. New Developments in Dioxin Regulation – Potential Impacts on the Regulated Community. International Conference on Incineration & Thermal Treatment Technologies Proceedings, Philadelphia, Pennsylvania. May 14 -18, 2001.

Fraiser, L.H., Roeck, D., and Smith, D. Current Environment of Hazardous Waste Combustion. International Conference on Incineration & Thermal Treatment Technologies Proceedings, Philadelphia, Pennsylvania. May 14 -18, 2001.

Fraiser, L.H., and Pope, P.G. 'Hazardous Waste Combustion Risk Assessment — Artifact or True Risk?' International Conference on Incineration & Thermal Treatment Technologies Proceedings., Portland, Oregon. May 8-12, 2000.

Fraiser, L.H., and Lewis, D. 'Detection Limits: Practical Implications for Risk Assessments Conducted on Hazardous Waste Combustion Units.' Presented before the Louisiana Chemical Association. Baton Rouge, Louisiana. September 9, 1999.

Fraiser, L.H., Tachovsky, J.A., King, M.L., McCoy, J.T., and Haws, L.C. 'Hazardous Waste Combustion Risk Assessment Experience in the State of Texas.' International Conference on Incineration & Thermal Treatment Technologies Proceedings. pp. 189-196, Salt Lake City, Utah. May 11-15, 1998.

Fraiser, L., McCoy, J.T., Perry, C., King, M., and Haws, L.C. Screening Risk Analysis for the Bayer Corporation Facility in Baytown, Texas. TNRCC publication number AS-120, AS-120A, and AS-120B. November 1996.

Fraiser, L., Lund, L., Tyndall, K., King, M., Schultz, D., and Haws, L. 'Case Studies in Risk Assessment for Hazardous Waste Burning Cement Kilns in Waste Combustion' in Boilers and Industrial Furnaces Proceedings. pp.208-225. Kansas City, Missouri. March 26-27, 1996.

Fraiser, L., Lund, L., Hueske, K., and Haws, L.C. 'Indirect Risk Assessment: Case Studies of Hazardous Waste Combustors'. Toxicologist 30:6, 1996.

Fraiser, L., Lund, L., Hueske, K., King, M., and Haws, L.C. Screening Risk Analysis for the North Texas Cement Company (NTCC) Facility in Midlothian, Texas. TNRCC publication number AS-71, AS-71A, and AS-71B. January 31, 1996.

Fraiser, L., Lund, L., Hueske, K., King, M., and Haws, L.C. Screening Risk Analysis for the Texas Industries (TXI) Facility in Midlothian, Texas. TNRCC publication number AS-72, AS-72A, and AS-72B. November 2, 1995.

Ramu, K., Fraiser, L., Mamiya, B., Ahmed, T., and Kehrer, J.P. 'Acrolein Mercapturates: Synthesis, Characterization, and Assessment of Their Role in the Bladder Toxicity of Cyclophosphamide.' Chem. Res. Toxicol. 8:515-524, 1995.

Fraiser, L., and Kehrer, J.P. 'Effect of Indomethacin, Aspirin, Nordihydroguairetic Acid, and Piperonly Butoxide on Cyclophosphamide-Induced Bladder Damage.' Drug Chem. Toxicol. 16(2):117-133, 1993.

Fraiser, L., Barnett, J.W., and Hixson, E.J. 'Toxicity Equivalents for Chlorinated Hydrocarbon Pesticides Lacking EPA-Verified Toxicity Values.' Toxicologist 14: 1540, 1994.

Kanekal, S., Fraiser, L., and Kehrer, J.P. 'Pharmacokinetics, Metabolism, and Lung Toxicity of Cyclophosphamide in C57/Bl6 and ICR Mice.' Toxicol. Appl. Pharmacol. 114:1-8, 1992.

Fraiser, L., and Kehrer, J.P. 'Murine Strain Differences in Bladder Toxicity of Cyclophosphamide.' Toxicol. 75:257-272, 1992.
Fraiser, L., Kanekal, S., and Kehrer, J.P. 'Cyclophospharnide Toxicity: Characterizing and Avoiding the Problem.' Drugs. 42(5):781 -795, 1991.

Attachment 4: Resume of Dr. Lisa Bradley, PhD, DABT

Lisa J. N. Bradley, Ph.D., DABT
Vice President and Senior Toxicologist

Professional History

AECOM (formerly ENSR)
Massachusetts Institute of Technology
University of Idaho

Education

PhD (Toxicology) Massachusetts Institute of Technology, 1991
BS (Zoology) University of Idaho, 1983
BS (Chemistry) University of Idaho, 1983

Years of Experience 25

Technical Specialties

Toxicology
Risk Assessment
Environmental Communication
Regulatory Negotiation
Site Strategy Development

Professional Affiliations

Diplomate, American Board of Toxicology, 1994
Society of Toxicology
Phi Beta Kappa

Dr. Lisa Bradley is a Senior Toxicologist/Risk Assessor and Vice President with AECOM. She has a Ph.D. in toxicology from the Massachusetts Institute of Technology. She has 25 years of experience in risk assessment and toxicology, and is certified by the American Board of Toxicology. She has managed risk assessments for hazardous waste sites in many EPA Regions, and under many state programs. Dr. Bradley has also served as an advisor on strategic risk assessment issues for clients in the natural gas, utility, and railroad industries. She has developed the risk assessment approach for a large multi-site program for a railroad client, for a national steel client, and developed and managed the risk evaluation component of a large multi-site, multi-state federal program for a natural gas client. Dr. Bradley is experienced in public speaking and environmental communications, and she has published articles in peer reviewed scientific journals based on both her laboratory and risk assessment work. Dr. Bradley is the global risk practice technical lead for AECOM. She is the manager and technical lead for AECOM's coal combustion product (CCP) initiative and was recently elected to the Executive Committee of the American Coal Ash Association.

Experience

A. Representative Superfund Experience

Pines Area of Investigation, Indiana, USEPA Region 5.
Serving as project manager for a multi-disciplinary team conducting the Remedial Investigation/Feasibility Study for the Respondents of an Administrative Order on Consent (AOC) being administered under the Superfund Alternative program in USEPA Region 5. The AOC addresses the placement of coal combustion by-products (CCBs) within a local permitted landfill and allegedly used as fill in other locations within the Area of investigation. Activities to date include agency negotiations on the AOC and scope of work; submission of a Site Management Strategy document, and subsequent approval by the Agency; submittal of the RI/FS Work Plan (including a Field Sampling Plan, Human Health and Ecological Risk Assessment Work Plans, HASP,

QAPP, and a Quality Management Plan), and subsequent approval by the agency; submission of additional Sampling and Analysis Plans; and communications activities (including a website – www.pinesupdate.com - and regular mailings of information updates to the community). Regular communications with the agency is also a cornerstone of the project. As the site covers not a facility, but a town and surrounding area, executing access agreements with the land owners for sampling and well installation was a critical task. Four rounds of sampling and analysis have been successfully completed. The Final RI Report has been approved, and the Draft Human Health Risk Assessment Report and the Draft Ecological Risk Assessment Report have been submitted to the agency. Approved project documents to date are available on USEPA's website: http://www.epa.gov/region5/sites/pines/index.htm.

Delaware Sand & Gravel Remedial Trust, Delaware, USEPA Region 3. A human health risk assessment (HHRA) focusing on evaluation of the vapor intrusion exposure pathway was performed for the PRPs at a former drum disposal area to evaluate the effectiveness of a Bioremediation System installed as a result of an EPA Superfund Record of Decision Amendment. A tiered vapor intrusion assessment was performed consistent with USEPA guidance using groundwater and then soil gas data. It was successfully concluded, with acceptance from EPA Region 3, that no unacceptable risk to human health was posed to occupants of on-site buildings via the vapor intrusion inhalation pathway.

Solutia, Inc., Human Health Risk Assessment, Sauget Area 1, Illinois, USEPA Region 5. Prepared a human health risk assessment work plan to follow Superfund guidelines for several abandoned landfill areas and areas downgradient of the landfills. The work plan was accepted by U.S. EPA Region V. A comprehensive human health risk assessment was prepared that evaluated the former land fill areas as well as local residential areas, a creek, and a borrow pit lake. A total of 64 receptor and area scenarios were quantitatively evaluated. Supporting risk modeling included indoor and outdoor air from subsurface soil and groundwater. Activities included site visits, meetings with personnel from USEPA Region 5 and their contractors, and preparations of responses to comments and document revisions. The human health risk assessment has been accepted by the agency, and the results have been used to guide the feasibility study and remedy selection. Constituents of interest included PCBs in ditch sediments. The final report is available on EPA's website: http://www.epa.gov/region5/cleanup/saugetarea1/pdfs/

sauget1_deadcreek_final_remedy_200604.pdf

Sauget Area 2 Sites Group, Human Health Risk Assessment, Illinois, USEPA Region 5. Serving as the senior human health risk assessment manager for a multi-party PRP group. Prepared a human health risk assessment work plan to follow Superfund guidelines for a set of sites that include abandoned landfill areas. Conducted the multi-receptor, multi-pathway human health risk assessment, including vapor intrusion modeling for both indoor and outdoor air for the multiple multi-acre sites within the project area. Activities included a site visit, meetings and negotiations with USEPA Region 5 and their contractors, and preparation of responses to comments.

Columbia Gas Transmission, Strategic Risk Assessment Advisor, West Virginia, USEPA Region 3. Served as strategic risk assessment advisor to a multi-site, ten-state AOC with U.S. EPA Region III to assess environmental conditions along their pipeline system in the Mid-Atlantic States. Provided strategic risk assessment advice and technical support on the design and implementation of the program, and developed a programmatic approach to the evaluation of risk across the program. Was responsible for: review of other contractor reports, development of a common strategy for TPH and mercury to be used across the program, review and summary of risk assessment regulations and guidance for each of the states (Ohio, Pennsylvania, West Virginia, Virginia, Kentucky, North Carolina, Delaware, New Jersey, Maryland, New York, and Louisiana), conducted risk assessments, provided critical review of individual site characterization reports prepared by other contractors, and provided support in negotiations and meetings with regulators. Additional constituents of interest include PCBs, arsenic, and PAHs.

Tippecanoe Landfill, Human Health Risk Assessment, Indiana, USEPA Region 5. Conducted agency negotiations (U.S. EPA Region V) concerning the human health risk assessment for a Superfund site. Because arsenic concentrations in groundwater were of concern to the agency, researched and reviewed the toxicological information available for arsenic, and prepared a literature review and evaluation of the dose-response values developed by the U.S. EPA for arsenic.

Industri-Plex CERCLA Site, Risk Assessment Review and Strategy for PRP Group, Massachusetts, USEPA Region 1. Provided risk assessment review and strategy for PRP group, and developed risk assessment work plan to address surface water

and groundwater exposure pathways.

Tennessee Valley Authority, Human Health Risk Assessment, Tennessee, USEPA Region 4. Prepared human health risk assessment and developed target cleanup levels for an abandoned battery manufacturing site. Primary constituent was lead and both child and adult lead models were used in the evaluation.

Confidential Client, Human Health Risk Assessment, New Jersey, USEPA Region 5. Conducted a human health risk assessment for a school district's baseball fields located adjacent to a potential Superfund site. Report was prepared for community distribution, and results presented at a public meeting.

Motco Superfund Site, Review of AIC for Volatile Organics, Texas, USEPA Region 6. Reviewed U.S. EPA-developed acute inhalation criteria (AIC) for volatile organics. Developed a consistent and scientifically-defensible methodology for AIC development, and applied this methodology to provide alternative AICs for use at the site.

Brio Site Task Force, Texas, USEPA Region 6. Developed acute inhalation criteria for use in a remedial program for benzene, 1,1-dichloroethane, 1,2-dichloroethane, ethyl benzene, methylene chloride, styrene, toluene, 1,1,1-trichloroethane, 1,1,2-trichloroethane, and vinyl chloride.

B. Representative RCRA Experience

Solutia, Inc., Human Health Risk Assessment Oversight for the J.F. Queeny Facility, St. Louis, Missouri. Provided oversight for the human health risk assessment prepared for the facility under an order with USEPA Region 6. The risk assessment is designed to meet the requirements of both USEPA and the State of Missouri Risk-Based Corrective Action Program.

Solutia, Inc., Human Health Risk Assessment for the W.G. Krummrich Facility, Sauget, Illinois, USEPA Region 5. Developed the human health risk assessment workplan and report for the RCRA Sampling Plan for Solutia's W.G. Krummrich Facility. The workplan was designed to permit evaluation of the "Human Exposures Environmental Indicator" as well as human health risk. Used risk assessment and data visualization to identify extent of areas for remediation such that total site risk would not exceed target risk levels once remediation is complete. Also used the risk assessment to identify remedial treatment

objectives for soils and groundwater. Target chemicals included PCBs and chlorinated compounds.

U.S. Steel, Human Health Risk Assessment, Gary, Indiana, USEPA Region 5. Developed the RCRA RFI Human Health Risk Assessment Workplan for the U.S. Steel Gary Works. Activities included response to regulatory comments on previous reports, site visits, review of reports generated both by USS and by local groups about the facility and its environs, development of the risk-related portions of the facility-wide RCRA RFI workplan, in addition to the HHRA workplan, and agency negotiation. Participated in strategy development for and preparation of the human health sections of the Sampling and Analysis Plans for each of the Solid Waste Management Areas being addressed at Gary Works under RCRA (13 in total). Managed and prepared the human health risk evaluation of perimeter groundwater data. Work included conducting a two tiered well-by-well screening (55 wells total). The first tier comparison was to generic and readily available standards, and the second tier took into account background and dilution into receiving water bodies, and evaluated construction worker and indoor air scenarios.

U.S. Steel, Human Health Risk Assessment, Fairless Hills, Pennsylvania, USEPA Region 3. Prepared the human health risk evaluation under RCRA Corrective Action for a parcel of property to be leased by U.S. Steel at Fairless Works. The work was conducted to satisfy Pennsylvania Department of Environmental Protection (PADEP) requirements under the Pennsylvania Act 2 program, as well as USEPA Region 3 requirements. Activities included site visit, meetings and presentations to both agencies, as well as preparation of memoranda and reports. Included in the evaluation was a sensitivity analysis of the parameters used to evaluate a construction worker scenario; site-specific parameters, parameters from the scientific literature, and parameters provided by the agency were evaluated.

U.S. Steel, Human Health Risk Assessment, Fairfield, Alabama, USEPA Region 4. Developed the RCRA RFI Human Health Risk Assessment Workplan for the U.S. Steel Fairfield Works under USEPA Region 4 and Alabama Department of Environmental Management (ADEM) requirements. Activities included site visits, preparation of strategy, review of the full RFI workplan to ensure consistency with risk objectives, and preparation of responses to agency comments. Work included a detailed evaluation of USEPA's current and proposed adult soil ingestion rates.

Hartford Working Group, Hartford Hydrocarbon Plume Site, Hartford, Illinois, USEPA Region 5. Provided toxicology and risk assessment services to the PRP group for the Hartford Hydrocarbon Plume site in Hartford, IL. Provided review of indoor air screening levels developed by the Agencies for benzene, butane, isopentane, trimethylbenzene and other petroleum-related constituents used in vapor intrusion evaluations.

C. Representative Risk Assessment Experience Under Other Programs

NiSource, Risk Assessment Issues, Columbus, Ohio. Serving as the human health risk assessment expert for NiSource's environmental programs. Have addressed issues related to PCBs (including conducting employee informational meetings), MGP-related constituents (benzene, PAHs), radon, and mercury.

Confidential Utility. Have provided PCB expert support for issues related to PCBs in natural gas pipeline systems and potential residential and commercial exposures.

Bureau of Land Management, Environmental Impact Statement, Western States. Developed human health risk assessment to evaluate five pesticides proposed for use in BLM vegetation treatment programs. Risk assessment uses standard USEPA Office of Pesticide Policy risk assessment methods and includes use of the AgDRIFT model to evaluate off-site spray drift and deposition, and transport models to evaluate surface water impacts. Worker, public and Native American subsistence receptors were evaluated. Work has included interagency scoping meetings. Report available at: http://www.blm.gov/wo/st/en/prog/more/veg_eis.html. 2007.

Bureau of Land Management, Environmental Impact Statement, Western States. Conducting human health risk assessment for additional pesticides for the BLM vegetation treatment programs following the protocol developed for the 2007 BLM Vegetation EIS.

Confidential Client, Indiana. Evaluated groundwater and soil gas data for vapor intrusive to indoor air using the USEPA version of the Johnson and Ettinger model. Used the Johnson (2002) sensitivity analysis method to ensure that critical model parameters were within acceptable/realistic ranges. Provided deposition testimony and testimony in a court hearing on both the vapor intrusion pathway risk assessment and the toxicology of benzene.

U.S. Steel, Development of a Standardized Risk Evaluation Guidance Manual, Pennsylvania. Worked in conjunction with another firm and USS personnel to develop a standardized Risk Evaluation Guidance Manual for USS. The manual addresses important issues in human health and ecological risk assessment, provides background for the issues, USS strategy to address the issues, and examples of standard language and references to be used in future USS reports. The manual will allow for more cost-effective and consistent risk evaluations to be conducted for USS facilities and sites.

U.S. Steel, Review and Comment on Indiana's RISC Program, Indiana. Reviewed several draft versions of Indiana's "Risk Integrated System for Closure" guidance, and submitted comments to the agency. Detailed comments were provided on the following topics: construction worker soil ingestion rate, soil saturation limit, arbitrary caps for metals concentrations in soil. Have also prepared comments on Indiana's draft groundwater policy and The User's Guide that details how the RISC program will be applied to RCRA sites under state authority.

U.S. Steel, Human Health Risk Assessment, Fairfield, Alabama. Conducted a human health risk evaluation for a parcel of property to be leased by U.S. Steel at Fairfield Works. Activities included evaluation of a construction worker scenario, and use of the Johnson & Ettinger and ASTM models to evaluate indoor and outdoor air.

West Virginia Manufacturer's Association, West Virginia. Worked with the WVMA on a committee to review and provide language to the West Virginia Department of Environmental Protection in development of their tiered site closure guidance.

Indiana Department of Environmental Management, Indiana. Served on an IDEM committee to review and provide language in the development of revisions to the "Risk Integrated System for Closure" guidance.

D. Representative Toxicology Experience

Utility Solid Waste Activities Group (USWAG), Washington, DC. Reviewed and developed comments on the risk assessment aspects of USEPA's June 2010 proposed rulemaking for the disposal of coal combustion residuals (CCRs). Comments focused on a critique of the USEPA's updated human health and ecological risk assessment, a critique of the USEPA's fugitive dust model report, and a critique of USEPA's proposed listing of CCRs as a hazardous waste under RCRA Subtitle C.

Utility Solid Waste Activities Group (USWAG), Washington, DC. Reviewed and developed comments on the USEPA's risk assessment for coal combustion wastes. The risk assessment was released in 2007, and comments were submitted under USWAG cover in January 2008. AECOM addressed all aspects of the risk assessment including human health, ecological risk and fate and transport. Provided oral comments during a national teleconference.

Utility Solid Waste Activities Group (USWAG), Washington, DC. Developed information sheet on "What is Coal Ash" for use by the USWAG membership for community relations.

Electric Power Research Institute, Palo Alto, CA. Developed the report "Comparison of Risks for Leachate from Coal Combustion Product Landfills and Impoundments with Risks for Leachate from Municipal Solid Waste Landfill Facilities," EPRI Report Number 1020555, available at www.epri.com.

Prairie State Energy Campus, Washington County, IL. Provided presentation to county board on coal ash composition and health risk issues.

We Energies, Milwaukee, WI. Reviewed the basis of the state and USEPA screening levels and toxicity values for molybdenum, and demonstrated the over-conservatism used in their derivation. Provided the review to the state agency, and developed a fact sheet on molybdenum in groundwater for communications with a local community.

We Energies, Milwaukee, WI. Reviewed the basis of the state screening levels and toxicity values for aluminum as part of review of the Wisconsin Department of Natural Resources proposed groundwater standards under NR 140. Provided testimony for a board hearing, and met with the state regulators, and demonstrated the over-conservatism used in their derivation.

Ameren UE, St. Louis, MO. Developed a human health and ecological risk assessment to support the regulatory closure under the state agency of a former ash impoundment located along a major river at the Hutsonville, IL Power Station. Boron and molybdenum were constituents of interest. Pathways evaluated in the risk assessment included use of groundwater for irrigation purposes and the migration of groundwater to the river and potential impact on the benthic community. Work included negotiation meeting with the local agency.

Ameren UE, St. Louis, MO. Serving as an expert for a landfill siting project in Missouri, for issues related to exposure, toxicity and risk assessment. Provided public testimony at a county board meeting as well as written comments that have been submitted into the record.

AES, New York. Provided expert testimony on the lack of human health effects of ammonia in groundwater associated with coal ash landfills. Developed expert opinion, reviewed and critiqued opposing opinions, and testified at hearing.

AES, Puerto Rico. Provided review and synthesis of data associated with a beneficial use product, AGREMAX™ manufactured by AES Puerto Rico using bottom ash and fly ash from the coal-fired power plant. Specifically, evaluation of data on metals content, leaching of metals, and radionuclides were shown not to pose a human health or environmental risk based on the beneficial uses of AGREMAX™. Testified on AES behalf at a Puerto Rican Senate subcommittee hearing on coal ash issues.

South Carolina Electric & Gas, Columbia, SC. Provided presentation materials for use in a landfill siting and zoning process. Materials addressed the comparison of arsenic and other metals and radionuclides in coal ash and in our natural environment, and background levels of arsenic in foods and background levels of exposure to radioactivity in our natural environment.

Utility Solid Waste Activities Group (USWAG), Washington, DC. Provided oversight of comments developed on the proposed listing of naphthalene as a carcinogen by the National Toxicology Program, and on the USEPA's childhood cancer document.

Electric Power Research Institute, California. Worked with another ENSR toxicologist to develop a critique of the benzo(a)pyrene toxicity value developed by the United Kingdom for their Contaminated Lands program.

Confidential Natural Gas Client, Toxicity Assessment, Ohio. Provided toxicity assessment of cleaning compounds proposed for use in the decommissioning of a natural gas pipeline laid on the bed of a reservoir that serves as the primary drinking water source for a community. Demonstrated that even should a catastrophic release of cleaning fluid and/or PCBs occur, human and ecological health would not be adversely affected and that concentrations at the drinking water intake would be much lower than health-based values or detection limits.

Confidential Client, Toxicology Review, Indiana. Provided a review of the toxicology and potential carcinogenicity of two structurally similar proprietary industrial chemicals. Used recent data on the nongenotoxic/cytotoxic mechanism of action of a class of potential carcinogens to demonstrate that a safe level for worker exposure exists.

U.S. Environmental Protection Agency, Literature Review. Developed a strategy for evaluating absorption data in the literature and applied it to the development of absorption adjustment factors for oral and dermal exposures to soil and water for 5 metals of concern at hazardous waste sites (arsenic, cadmium, chromium III, chromium VI, inorganic mercury, organic mercury, and nickel) based on a thorough review of the literature.

Georgia Pacific, Literature Review, Georgia. Reviewed literature and summarized the current scientific knowledge of the endogenous synthesis of halogenated compounds in humans.

E. Representative MGP Experience

Natural Gas Company, Risk Assessment Advisor, Ohio. Serving as strategic risk assessment advisor to the manager of MGP sites. Work includes conducting risk assessments for MGP sites under various state programs, evaluation of program-wide vapor intrusion data, regulatory negotiations, environmental communications, and employee meetings.

Natural Gas Company, Former MGP Site Advisor, Wisconsin. Have reviewed remediation plans and fenceline monitoring plans, gave presentation at public meetings discussing the air monitoring plan, and have reviewed fenceline monitoring data for a remediation project.

Energy Company, Former MGP Site Review, Rhode Island. Provided senior review of an air monitoring program and identified where flexibility can be used in the development of fenceline air monitoring standards.

Village of Oak Park, Former MGP Site Advisor, Illinois. Provided senior review of remediation plans, and fenceline monitoring plans, and provided air monitoring data evaluation. Was involved in regulatory meetings, negotiations, and presentations to the Village council, including public meetings concerning air monitoring aspects of the project.

Committees

Leader of AECOM's Risk Assessment Technical Practice Group including practitioners internationally within AECOM with specialties in human health and ecological risk assessment and other supporting disciplines.

Leader of AECOM's Coal Combustion Product (CCP) Initiative; responsible for following regulatory developments, and keeping AECOM staff and clients updated on the issues.
Elected member of the American Coal Ash Association (ACAA) Executive Committee, and member of the Government Relations Committee, and the Women's Leadership Forum.
Task group leader for AECOM's Women's Leadership Collaborative program.

Publications and Presentations

"Hexavalent Chromium in Perspective" Presentation and invited Chair – Human Health Risk Panel, MGP 2012, Chicago, IL, March 29, 2012.

"Health Risk of CCPs." Presented at the EUCI conference on CCR Management: Impacts of Regulations and Technological Advances. , Nashville, TN, February 28-29, 2012.

"Risk Assessment: How the EPA Looks at Coal Combustion Products." Presented at the ACAA Fall meeting, Indianapolis, IN, September 27, 2011.

"Risk assessment: An overview of how the U.S. Environmental Protection Agency looks at coal combustion residuals." Presented at the American Chemical Society meeting in Denver, CO, August 28, 2011.

"Is Coal Ash Toxic?" Keynote Presentation at the World of Coal Ash May 10-12, 2011, and invited presentation at The Coal Institute/NCCI meeting July 11, 2011.

"Potential Effect of Proposed Coal Combustion Residuals Regulation and Alternative Leach Testing on Beneficial Reuse." World of Coal Ash May 10-12, 2011.

"Comparison of Risks for Leachate from Coal Combustion Product Landfills and Impoundments with Risks for Leachate from Municipal Solid Waste Landfill Facilities." World of Coal Ash May

10-12, 2011, and poster at Society of Toxicology March 6-10, 2011.

“Overview of Coal Ash Regulatory Issues.” NCASI Northern Regional Meeting May 18-19, 2011.

“Perspectives on Health Risks Associated with Beneficial Re-Use of Byproducts of Coal Combustion.” McIlvaine Hot Topic Hour. April 28, 2011.

”Risk Assessment: How the EPA Looks at Coal Combustion Products.” EUCI March 13-14, 2011.

“Risk Assessment: How the EPA Looks at Coal Combustion Products.” Presented at the EUCI conference on Future of Coal Combustion Products (CCPs): Regulatory, Legal, Technical, and New Markets, March 2011, Denver, CO.

“Development of a Realistic Risk Assessment Conceptual Site Model for an Urban River Sediment Site.” B. Ruffle, L. Bradley, K. Durocher, and L. Fraiser. Battelle Sediment Conference February 7-10, 2011.

Press Conference with ACAA (American Coal Ash Association) , October 27, 2010, Knoxville, TN.

“USEPA’s Proposed rule for Coal Combustion Residual (CCRs): Beneficial Use Aspects.” Keynote address given at the June 2010 meeting of the American Coal Ash Association, Baltimore, MD. Bradley, L.J.N, and A. Ellis.

“Overview of a CCP Site Investigation Conducted Under the Superfund Alternative Program.” Presented at the ACAA spring meeting, March 2010, Nashville, TN.

“Coal Ash Business Planning and Management: Addressing Risks and Liabilities in a Changing Regulatory Environment.” Workshop presented at the EUCI Conference on the Future of Coal Combustion Products, March 2010, Houston, TX. L.J.N.

Bradley, J. Trast, J. Matus,, and A. Kier.
“PAHs and Dioxins Not Present in Fly Ash at Levels of Concern.” World of Coal Ash, May 2009 and Society of Toxicology, March 2009.

Bradley, L.J.N., G.M. Fent, and S.W. Casteel. “In Vivo Bioavailability of Arsenic in Coal Combustion By-Products.”

Poster presented at the Society of Toxicology 2008 annual meeting in Seattle, WA.

Bradley, L.J.N., K. Sullivan, and M. Garcia. “Background Levels of Benzene in Indoor and Outdoor Air.” Paper presented at the Gas Technology Institute’s Natural Gas Technologies II Conference, Phoenix, Arizona. February, 2004

Bradley, L.J.N., and K.A. Sullivan. "Risk-Based Action Levels for Remediation Project Fence-Line Air Monitoring Programs." The Toxicologist. 72(S-1): 395. March, 2003

Bradley, L.J.N., and M. Gerath. "Generic Risk and Fate Analysis for Mercury at Natural Gas Meters." Paper presented at the December 1998 Society for Risk Analysis Annual Meeting, Phoenix, AZ.

Bradley, L.J.N., K.B. Lemieux, M.C. Garcia, A.H. Parsons, and D.E. Rabbe. "Comparison of Concentrations of Selected Metals and Organics in Fish Tissue and Sediment in the Grand River, Ohio, and the Southern Lake Erie Drainage Basin." Human and Ecological Risk Assessment 4(1):57-74 (1998).

Bradley, L.J.N. "TPH Analyses Provide Means of Direct Assessment of Diesel Releases." Paper presented at the October, 1997, Contaminated Soils Conference, Amherst, MA.

Bradley, L.J.N. "Risk Assessment of Hazardous Air Pollutants in Arizona." Paper presented at the December, 1996 Society for Risk Analysis Annual Meeting, New Orleans, LA.

Bradley, L.J.N. "Cost-Effective Use of Tiered Approaches in Risk Assessment." Paper presented at the October, 1996 Annual Conference on Contaminated Soils, Amherst, MA.

Bradley, L.J.N. "Role of Risk Assessment in Environmental Management." Invited paper presented at the West Virginia Manufacturers Association Environmental Compliance Conference, May, 1996, Charleston, WV.

Bradley, L.J.N. "New Toxicology Data for Chloroform: Implications for the Pulp and Paper Industry." Proceedings of the 1996 Environmental Conference of the Technical Association of the Pulp and Paper Industry. Vol 1, pp. 13-16 (1996).

Bradley, L.J.N. "Ingested Arsenic - Are the Taiwanese Data Appropriate for Risk Assessment in the U.S." Paper presented at

the December, 1994, Society of Risk Analysis Conference, Baltimore, MD.

Magee, B.H., and L.J.N. Bradley. "Absorption Adjustment Factors for Use in Risk Assessment." Proceedings of the International Congress on the Health Effects of Hazardous Waste. (1994).

Bradley, L.J.N., B.H. Magee, and S.L. Allen. "Background Levels of Polycyclic Aromatic Hydrocarbons and Selected Metals in New England Urban Soils." J. Soil Contam. 3(4):349-361. (1994).

Magee, B.H., L.J.N. Bradley, E.L. Butler, A. Dasinger, J. Grabowski. "Risk-Based Target Clean-Up Levels for TPH in Soils." In: Hydrocarbon Contaminated Soils. Vol. 3. pp. 303-319. Edited by P.T. Kostecki and E.J. Calabrese. 1993.

Bradley, L.J.N. "Human Health Risk Assessment Workshop." Presented at the September, 1992, Hydrocarbon Contaminated Soils Conference, Amherst, MA.

EXHIBIT 4

Ameren Energy Resources Alternative SO2 Limit Comparison to the Current MPS

Year	Baseline Heat Input MMBtu	MPS SO2 Rate lb/MMBtu	MPS Baseline SO2 Tons	Variance SO2 Rate lb/MMBtu	Variance SO2 Tons	SO2 Reduced Tons*	Net Variance SO2 Tons	Cumulative Reductions in SO2 Variance Tons
2010	340,446,252	0.50	85,112	0.50	85,112	14,552	70,560	14,552
2011	340,446,252	0.50	85,112	0.50	85,112	12,573	72,539	27,125
2012	340,446,252	0.50	85,112	0.38	64,685	7,699	56,986	55,251
2013	340,446,252	0.50	85,112	0.35	59,578	7,699	51,879	88,483
2014	340,446,252	0.43	73,196	0.35	59,578	7,699	51,879	109,800
2015	340,446,252	0.25	42,556	0.35	59,578	7,699	51,879	100,477
2016	340,446,252	0.25	42,556	0.35	59,578	7,699	51,879	91,153
2017	340,446,252	0.23	39,151	0.35	59,578	7,699	51,879	78,426
2018	340,446,252	0.23	39,151	0.35	59,578	7,699	51,879	65,698
2019	340,446,252	0.23	39,151	0.35	59,578	7,699	51,879	52,970
2020	340,446,252	0.23	39,151	0.23	39,151	7,699	31,452	60,669
Total			655,359		691,108	96,416	594,690	60,669

Note for the "Cumulative SO2 Variance Reduced Tons" column, a positive number indicates an emission decrease (benefit).

* Tons shown for 2010 and 2011 are based on actual SO2 emissions. Reduced tons shown for 2012-2020 are based on the not operating Hutsonville and Meredosia less potential SO2 emissions from FutureGen 2.