



August 21, 2019

Ms. Barbara Kunkle  
Acting Executive Secretary  
Michigan Public Service Commission  
7109 W. Saginaw Hwy.  
P. O. Box 30221  
Lansing, MI 48909

***Via E-Filing***

RE: MPSC Case No. U-20471

Dear Ms. Kunkle:

The following is attached for paperless electronic filing:

Direct Testimony of George Thurston on behalf of Michigan Environmental  
Council, Natural Resources Defense Council, and Sierra Club

Exhibits MEC-85 through MEC-87

Proof of Service

Sincerely,

Tracy Jane Andrews  
[tjandrews@envlaw.com](mailto:tjandrews@envlaw.com)

xc: Parties to Case No. U-20471

DIRECT TESTIMONY OF GEORGE THURSTON  
CASE NO. U-20471

STATE OF MICHIGAN

BEFORE THE MICHIGAN PUBLIC SERVICE COMMISSION

MPSC Case No. U-20471

In the matter of the Application of )  
DTE ELECTRIC COMPANY for )  
approval of its Integrated Resource Plan )  
pursuant to MCL 460.6t, and for other relief. )  
\_\_\_\_\_ )

**DIRECT TESTIMONY OF GEORGE D. THURSTON**

**ON BEHALF OF  
THE MICHIGAN ENVIRONMENTAL COUNCIL, NATURAL RESOURCES DEFENSE  
COUNCIL AND THE SIERRA CLUB**

August 21, 2019

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1    **INTRODUCTION AND QUALIFICATIONS**

2    **Q:     Please state for the record your name, position, and business address.**

3    A:     My name is George D. Thurston. I am Professor of Environmental Medicine at the New  
4    York University (NYU) School of Medicine. My business address is 3 Catherine Ct., Chester, New  
5    York.

6    **Q:     On whose behalf is this testimony offered?**

7    A:     I am testifying on behalf of Michigan Environmental Council, Natural Resources Defense  
8    Council, and Sierra Club.

9    **Q.     Please summarize your experience in the field of the health impacts of air pollution.**

10   A:     I have a Bachelor of Science degree in Engineering from Brown University, and a Masters  
11   and Doctorate of Environmental Health Sciences from the Harvard University School of Public  
12   Health. I have over 30 years of subsequent experience in the evaluation of the human health effects  
13   of air pollution. I have served on the U.S. Environmental Protection Agency's Clean Air Scientific  
14   Committee (CASAC) that advised the EPA on the promulgation of ambient air quality standards  
15   from 2007 through 2010, and I have served on the National Academy of Science's Committee on  
16   the Health Effects of Incineration from 1995 through 1999. I have published extensively regarding  
17   the health effects of inhaled air pollutants on humans, particularly as it relates to asthma attacks,  
18   hospital admissions, and mortality, in prominent scientific journals, such as Science, Lancet,  
19   Thorax, the New England Journal of Medicine, and the Journal of the American Medical  
20   Association (JAMA). I have also been called upon by both the U.S. House of Representatives and  
21   the U.S. Senate on multiple occasions in recent decades to provide testimony before them  
22   regarding the human health effects of air pollution, and have served on an US EPA Clean Air  
23   Science Advisory Committee (CASAC). A detailed statement of my qualifications is attached as  
24   Exhibit MEC-85.

25   **Q.     What is the purpose of your testimony?**

26   A.     The purpose of this testimony is:

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- To document the health impacts resulting from exposure to air pollutants (particularly fine particulates, ozone, sulfur dioxide, nitrogen oxides, and toxic pollutants) from fossil-fueled electric generating units (EGUs), including DTE's fleet of fossil-fueled EGUs;
- To document ways to model and assess the health impacts on an affected community resulting from exposure to air pollution, including the results of two analyses of DTE's EGUs; and
- To provide recommendations for the Commission to recognize these impacts in resource planning and incorporate them into decision-making.

**Q. Are you sponsoring exhibits?**

A. Yes, I am sponsoring the following exhibits:

Ex MEC-85: Curriculum Vitae

Ex MEC-86: Nicholas Bakalar, *Cleaner Air Brings Drop in Death Rate*, New York Times (Mar. 21, 2006), page F7.

Ex MEC-87: A joint ERS/ATS policy statement: What constitutes an adverse health effect of air pollution? An analytical framework

**Q. How is your testimony organized?**

A. The first section below begins by introducing the scientific research on the adverse health consequences of air pollution exposure, particularly related to emissions from fossil-fueled generating facilities. It continues with an overview of the scientific research on the health impacts of the four pollutants of particular concern. The second section introduces the methodology for evaluating the health effects of emissions from a particular source of air pollution. It also presents the results of two studies that have evaluated and quantified the health impacts of some of DTE's fossil-fueled generating units. The final section recommends that the Commission recognize these health impacts and consider ways to integrate them into future decision-making.

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1    **A.    THERE IS SCIENTIFIC CONSENSUS THAT EXPOSURE TO AIR POLLUTION**  
2    **FROM FOSSIL GENERATING PLANTS CAUSES ADVERSE HEALTH EFFECTS.**

3    **Q.    Please summarize your findings on the health impacts of air pollution.**

4    A.    The adverse health consequences of breathing air pollutants are well documented in the  
5    published medical and scientific literature. During the past few decades, medical research examining  
6    air pollution and public health has definitively shown that air pollution exposure is associated with a  
7    host of serious adverse human health effects. This documentation includes impacts revealed by  
8    observational epidemiology, and confirmed by controlled chamber exposures, showing consistent  
9    associations between air pollution and adverse impacts across a wide range of human health outcomes.

10   **Q.    What kinds of studies provide evidence of the adverse effects of air pollution?**

11   A.    Observational epidemiology studies provide the most compelling and consistent evidence of  
12   the adverse effects of air pollution. “Epidemiology” is literally “the study of epidemics,” but includes  
13   all statistical investigations of human health and potentially causal factors of good or ill health. In the  
14   case of air pollution, such studies follow people as they undergo varying real-life exposures to  
15   pollution over time, or from one place to another, and then statistically inter-compare the health  
16   impacts that occur in these populations when higher (versus lower) exposures to pollution are  
17   experienced. In such studies, risks are often reported in terms of a Relative Risk (RR) of illness,  
18   wherein a  $RR = 1.0$  is an indication of no change in risk after exposure, while a  $RR > 1.0$  indicates an  
19   increase in health problems after pollution exposure. An RR significantly above 1 demonstrates that  
20   the air pollutant studied is damaging to the community’s health.

21   **Q.    What types of epidemiological investigations of environmental health effects are there?**

22   A.    There are two types of epidemiological investigations: (a) population-based studies, in which  
23   an entire city’s population might be considered in the analysis; and (b) cohort studies, in which  
24   selected individuals, such as a group of asthmatics, are considered.

25   **Q.    What have these studies been able to show?**

26   A.    Both of these types of epidemiologic studies have shown confirmatory associations between  
27   air pollution exposures and increasing numbers of adverse impacts, including:

- 28       • decreased lung function (a measure of our ability to breathe freely);

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- more frequent asthma symptoms;
- increased numbers of asthma and heart attacks;
- more frequent emergency department visits;
- increased incidence of new onset childhood asthma;
- additional hospital admissions; and
- increased numbers of deaths.

**Q. What do the consistent results of these studies tell us about the association between air pollution and health impacts?**

A. The fact that the adverse health effects of air pollution have been shown so consistently for so many health endpoints, in so many locales, and by so many different investigators using a variety of approaches, indicates these associations to be causal.

**Q. Which are the particular pollutants that you are concerned about, related to fossil fuel powered electric generation?**

A. The United States 1970 Clean Air Act and its amendments require the US Environmental Protection Agency (EPA) to set National Ambient Air Quality Standards (NAAQS) for six common air pollutants (also known as “criteria air pollutants”) in outdoor air with the aim of reducing the health risks associated with those pollutants in our air. Fossil fuel powered EGUs are major sources of emissions of criteria air pollutants or their precursors, including fine particulate matter (PM<sub>2.5</sub>), nitrogen dioxide (NO<sub>2</sub>), sulfur dioxide (SO<sub>2</sub>), and ground-level ozone (O<sub>3</sub>). In addition, EPA identifies and regulates Hazardous Air Pollutants or toxic air pollutants, as addressed below. My testimony focuses in particular on the health impacts from these four pollutants (fine particulate matter, nitrogen dioxide, sulfur dioxide, and ozone).

**Q. Please summarize the following sections.**

A. The operation of fossil fueled EGUs will have consequences to public health, as has been well-documented in the studies discussed below. Each subsection that follows discusses in detail the nature of public health impacts resulting from air pollutants emitted from EGU operations.

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**1. Human Health Effects of Exposure to Fine Particulate Matter (PM<sub>2.5</sub>)**

**Q. What is Fine Particulate Matter?**

A. Fine Particulate Matter (PM<sub>2.5</sub>) is among the key air pollutants emitted from fossil-fuel power plants that have been revealed by research to adversely affect human health. These research studies have been conducted for a wide array of geographic areas, including eastern North America. PM<sub>2.5</sub> air pollution has been carefully studied in recent decades. PM<sub>2.5</sub> is composed of two major components: “primary” particles, or soot, emitted directly into the atmosphere by pollution sources; and “secondary” particulate matter, formed in the atmosphere from gaseous pollutants, such as the sulfur oxides (SO<sub>x</sub>) and nitrogen oxides (NO<sub>x</sub>) also emitted by fossil fuel-fired power plants. After formation in the atmosphere, this secondary PM<sub>2.5</sub> largely condenses upon the smallest existing primary particles that, collectively, represent the greatest surface area for the secondary PM to condense upon. These particles are very small, commonly having an aerodynamic diameter of less than 1.0 micrometer ( $\mu\text{m}$ ) – a fraction of the diameter of a human hair. For example, after it is released from a smokestack, gaseous SO<sub>x</sub> is chemically converted in the atmosphere to become sulfate PM<sub>2.5</sub>.

**Q. Please summarize the human health effects of exposure to fine particulate matter.**

A. As elaborated upon below, the health effects from fine particulate air pollution include:

- Increased risk of heart attacks
- Increased risk of death
- Lung damage
- Increased risk of cardiopulmonary and lung cancer mortality

**Q. Please explain further about the human health effects related to the heart from exposure to Fine Particulate Matter (PM<sub>2.5</sub>).**

A. Consistent epidemiological and toxicological studies of PM air pollution have shown adverse effects on the heart from exposures, including an increased risk of Myocardial Infarctions (MI's) (*i.e.*, heart attacks). Peters et al. (2001) found that elevated concentrations of fine particles in the air can elevate the risk of a heart attack within a few hours, and extending 1 day after PM<sub>2.5</sub> exposure. When PM stresses the lung (*e.g.*, by inducing edema), it places extra burden on the heart, which can induce fatal complications for people with cardiac problems. The Harvard University



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team found that a 48 percent increase in the risk of MI was associated with an increase of 25  $\mu\text{g}/\text{m}^3$   $\text{PM}_{2.5}$  during a 2-hour period before the onset of MI, and a 69 percent increase in risk to be related to an increase of 20  $\mu\text{g}/\text{m}^3$   $\text{PM}_{2.5}$  in the 24-hour average 1 day before the MI onset (Peters et al., 2001). Numerous other U.S. studies have also shown qualitatively consistent acute cardiac effects, such as the Zanobetti and Schwartz (2006) study of hospital admissions through emergency departments for myocardial infarction (ICD-9 code 410), and the Zanobetti et al. (2009) study that examined the relationship between daily  $\text{PM}_{2.5}$  concentrations and emergency hospital admissions for cardiovascular causes, myocardial infarction, and congestive heart failure in 26 U.S. communities during 2000-2003.

Animal and human studies have confirmed that at the individual level, biological changes do occur in heart function as a result of PM exposure, supporting the biological plausibility of the epidemiological associations between PM exposure and cardiac illnesses. Cardiac effects at the biological level have been documented in both animal and human studies. Animal experiments at Harvard University by Godleski et al. (1996, 2000) indicated that exposures to elevated concentrations of ambient particulate matter can result in cardiac related problems in dogs that had been pre-treated (in order to try to simulate sensitive individuals) to induce coronary occlusion (*i.e.*, narrowed arteries in the heart) before exposing them to air pollution. The most biologically and clinically significant finding was that, in these dogs, the PM affected one of the major electrocardiogram (ECG) markers of heart attacks (myocardial ischemia) in humans, known as elevation of the ST segment. Cardiac effects at the biological level have been found in human studies, as well. For example, Pope et al. (1999) and Gold et al. (2000) found that PM exposure is associated with changes in human heart rate variability. Such changes in heart rate variability (HRV) may reflect changes in cardiac autonomic function and risk of sudden cardiac death. In the Pope et al. study, repeated ambulatory ECG monitoring was conducted on 7 subjects for a total of 29 person-days before, during, and after episodes of elevated pollution. After controlling for differences across patients, elevated particulate levels were found to be associated with (1) increased mean heart rate, (2) decreased SDNN, a measure of overall HRV, (3) decreased SDANN, a measure that corresponds to ultra-low frequency variability, and (4) increased r-MSSD, a measure that corresponds to high-frequency variability.

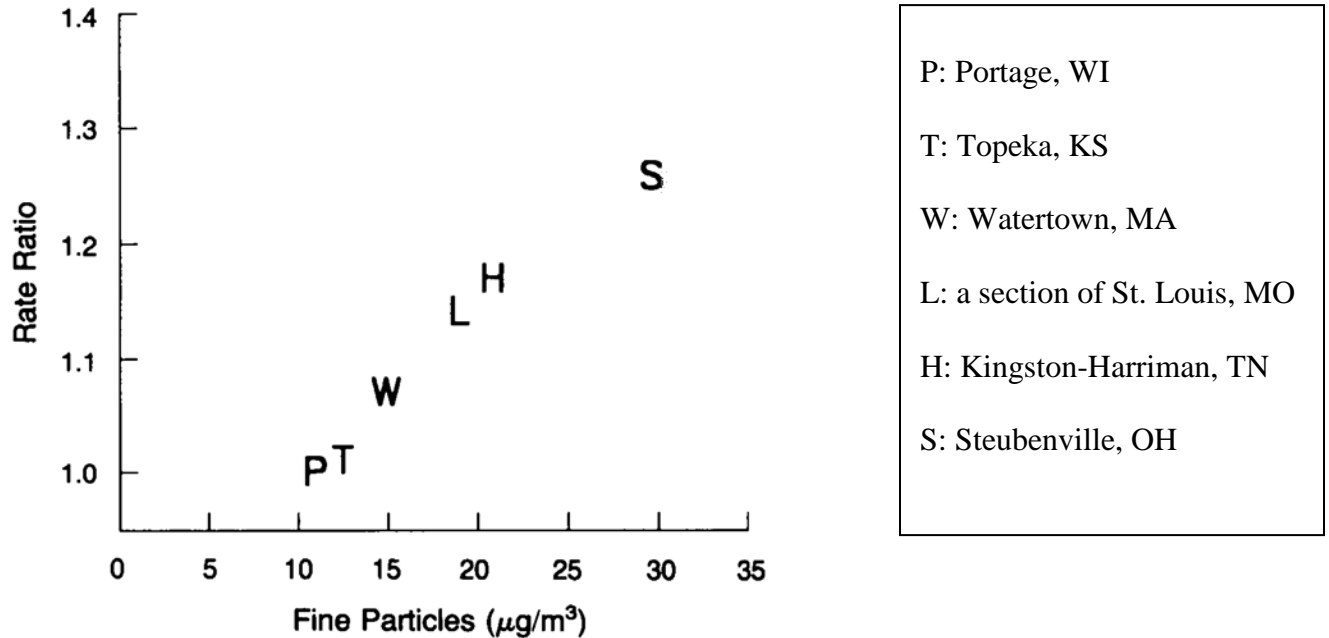
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1   **Q.    Is there evidence linking exposure to fine particulate matter air pollution with**  
2   **increased risk of mortality?**

3    A.    Yes. I concur with the most recent U.S. EPA Particulate Matter Integrated Science  
4    Assessment (ISA) (USEPA, 2009), which unequivocally states that “Together, the collective  
5    evidence from epidemiologic, controlled human exposure, and toxicological studies is sufficient  
6    to conclude that a causal relationship exists between short term exposures to PM<sub>2.5</sub> and  
7    cardiovascular effects . . . and mortality.” Epidemiologic research conducted on U.S. residents  
8    has indicated that acute exposure to particulate air pollution is associated with increased risk of  
9    mortality. A nationwide time-series statistical analysis by the Health Effects Institute (HEI, 2003)  
10   of mortality and PM<sub>10</sub> (i.e., the mass of particles less than 10 µm in diameter) air pollution in 90  
11   cities across the US indicated that, for each increase of 10 µg/m<sup>3</sup> in daily PM<sub>10</sub> air pollution  
12   concentration, there is an associated increase of approximately 0.3% in the *daily* risk of death.  
13   While a 0.3 % change in the daily death risk may seem small, it is important to realize that such  
14   added risks apply to the entire population, and accumulate day after day, week after week, and  
15   year after year, until they account for thousands of needless daily deaths from air pollution in the  
16   U.S. each year.

17        In addition to the acute health effects associated with daily PM pollution, the long-term  
18   exposure to fine PM is also associated with increased lifetime risk of death and has been estimated  
19   to take years from the life expectancy of people living in the most polluted cities, relative to those  
20   living in cleaner cities. For example, in the Six-Cities Study (which was one key basis for the  
21   setting of the original PM<sub>2.5</sub> annual standard in 1997), Dockery et al. (1993) analyzed survival  
22   probabilities among 8,111 adults living in six cities in the central and eastern portions of the United  
23   States during the 1970’s and 1980’s.

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**Figure 1.** The Harvard Six-Cities Study showed that the lifetime risk of death increased across 6 U.S. cities as the average fine PM levels increased. (Source: Dockery et al., 1993).

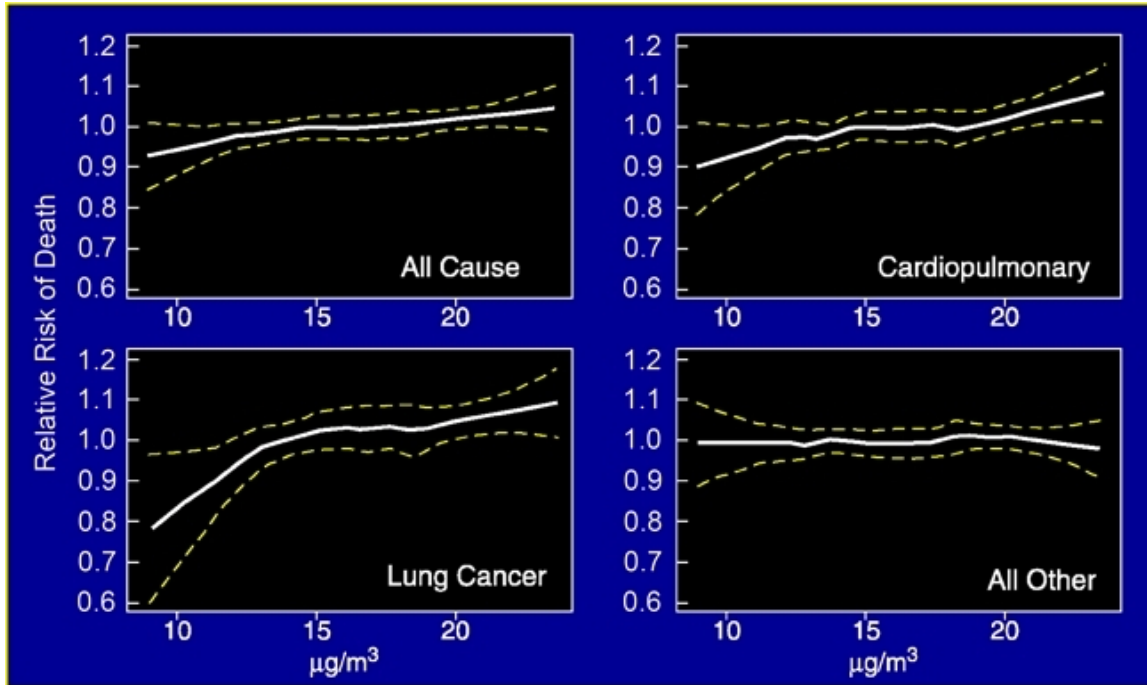
As shown in Figure 1, the Six-Cities Study found that the long-term risk of death, relative to the cleanest city, increased with fine particle exposure, even after correcting for potentially confounding factors such as age, sex, race, smoking, etc. Air quality was averaged over the period of study in order to study long-term (chronic) effects.

**Q. Please explain the human health effects related to the lungs from exposure to fine particulate matter.**

A. In my own research, I have found that acute (short-term) increases in PM air pollution are associated with increases in the number of daily asthma attacks, respiratory hospital admissions, and mortality. In particular, I have found that particulate matter air pollution is associated with increased numbers of respiratory hospital admissions in New York City; Buffalo, NY; and Toronto, Ontario, as well as with mortality in cities such as Chicago, IL; and Los Angeles, CA (see, e.g., Thurston *et al.*, 1992). My results have been confirmed by other researchers considering locales elsewhere in the U.S. and throughout the world (see, e.g., Schwartz, J., 1997). I co-authored a study published in the Journal of the American Medical Association (JAMA), showing

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that long-term exposure to combustion-related fine particulate air pollution is an important environmental risk factor for cardiopulmonary and lung cancer mortality.



**Figure 2.** Cardiopulmonary and lung cancer mortality risks increase monotonically with exposure to long-term fine PM (Adapted from: Pope, Burnett, Thun, Calle, Krewski, Ito, and Thurston, 2002)

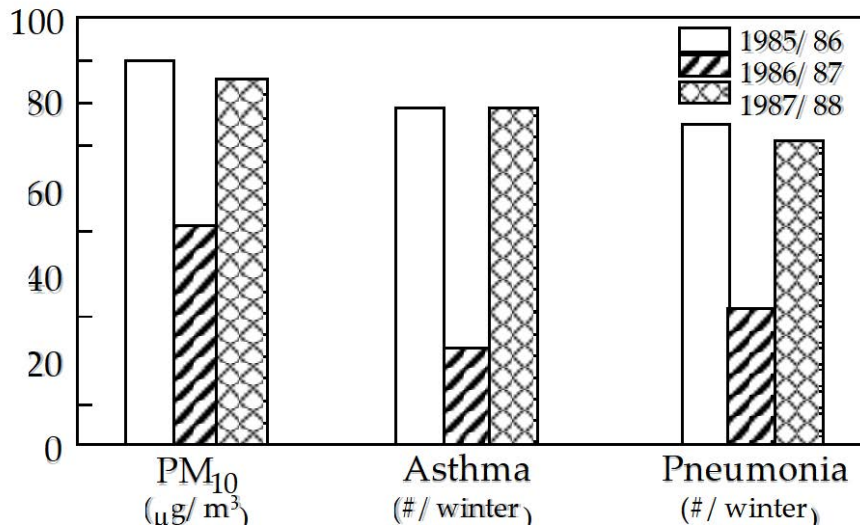
Indeed, as shown in Figure 2, this study indicates that the increase in risk of lung cancer from long-term exposure to  $\text{PM}_{2.5}$  in a city like New York was of roughly the same size as the increase in lung cancer risk of a non-smoker who breathes passive smoke while living with a smoker, or about a 20% increase in lung cancer risk. *See Pope, CA, et al, 2002.*

**Q. Are there studies showing the health benefits of avoiding or reducing fine particulate matter levels?**

A. Yes. While many studies evaluate whether rising air pollution levels worsen health, others have shown that reducing pollution in the air can result in health benefits to the public. For example, Pope (1989) conducted a compelling study clearly showing that, when pollution levels diminish, the health of the general public improves. He investigated a period during the winter of 1986-87 when the Geneva Steel mill in the Utah Valley shut down during a strike. The PM

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levels dropped dramatically in that strike-year winter, as opposed to the winters preceding and following when the steel mill was in operation.



**Figure 3.** Decreasing PM pollution lowered the number of children's hospital admissions (Source: Pope, 1989).

As shown in Figure 3, hospital admissions in the Utah Valley showed the same pattern as the PM air pollution, decreasing dramatically during the 1986-87 strike. As a control, Pope also examined the pollution and hospital admissions records in nearby Cache Valley, where the mill's pollution was not a factor, and no such drop in respiratory admissions was seen, showing that the drop in admissions in the Utah Valley was not due to some cause other than the reduction in the air pollution levels.

These four studies of the health improvements associated with decreases in PM<sub>2.5</sub> pollution show that any reduction can be expected to result in commensurate health benefits to the public at ambient levels, irrespective as to whether the National Ambient Air Quality Standards (NAAQS) are already met. A follow-up analysis of the Harvard Six-Cities Study cohort discussed earlier (Dockery *et al*, 1993), published in the March 15, 2006 issue of The American Journal of Respiratory and Critical Care Medicine (Laden *et al*, 2006), shows that mortality is decreased by lowering PM pollution. This study was carried out in the same six metropolitan areas evaluated in the earlier study, study participants' ages ranged from 25 to 74 at enrollment in 1974, and the scientists tracked both PM air pollution and mortality through 1998 in these populations. The Laden study found that improved overall mortality (*i.e.*, a risk

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ratio significantly below 1.0) was associated with decreased mean PM<sub>2.5</sub> over the study follow-up time (RR = 0.73; 95% per 10 µg/m<sup>3</sup>, CI = 0.57-0.95). In other words, for each decrease of 1 µg/m<sup>3</sup> of PM<sub>2.5</sub>, the overall death rate from causes such as cardiovascular disease (CVD), respiratory illness and lung cancer decreased by nearly 3% (i.e., 10 µg/m<sup>3</sup> x 2.7% = 27% decrease, or RR=0.73). The study also found that people who are exposed to lower pollution live longer than they would if they were exposed to higher pollution. Francine Laden, the study's lead author, explained its key findings in the March 21, 2006 issue of the New York Times: "For the most part, pollution levels are lower in this country than they were in the 70's and 80's," and "the message here is that if you continue to decrease them, you will save more lives."<sup>1</sup> "Consistently," Dr. Laden said, "in the cities where there was the most cleanup, there was also the greatest decrease in risk of death."

**Q. Do health effects occur at low levels of Fine Particulate Matter?**

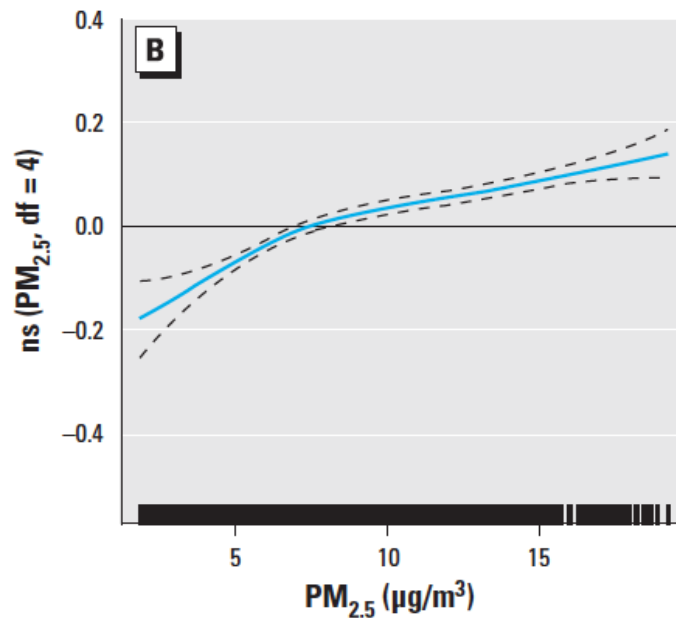
A. Yes. Although the Laden study took place in urbanized areas, the same principle can be applied in more rural areas where the air is more pristine: higher concentrations of PM<sub>2.5</sub>, even at very low overall levels, are associated with greater health risks. In addition, the three studies discussed below confirm that, even in places where background air is relatively clean, small changes in air pollution concentration can have population health impacts. As these studies show, there is no convincing evidence to date showing that there is any threshold below which such adverse effects of PM air pollution will not occur. This lack of a threshold of effects indicates that any reduction in air pollution can be expected to result in commensurate health benefits to the public at ambient levels.

A more recent Canadian national-level cohort study, Crouse *et al* (2012), has shown that the adverse effects of air pollution extend down to very low levels of PM<sub>2.5</sub>.

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<sup>1</sup> Ex MEC-86, Nicholas Bakalar, *Cleaner Air Brings Drop in Death Rate*, New York Times (Mar. 21, 2006), pg F7.

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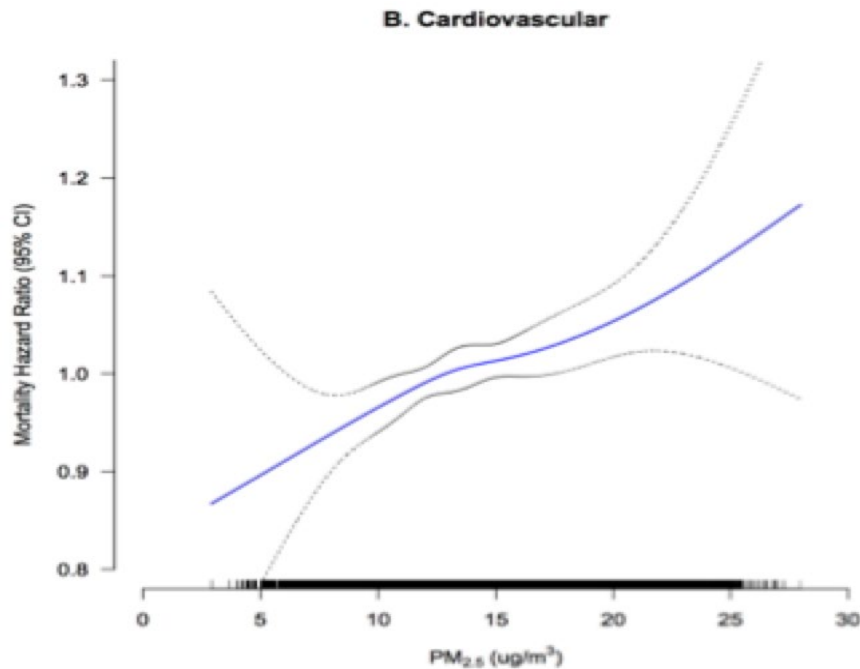
**Figure 4.** Cardiovascular Mortality Risk vs. PM<sub>2.5</sub> exposure (solid line) and 95% CIs (dashed lines), showing increasing risk of death with increasing PM<sub>2.5</sub>, even at very low ambient levels of PM<sub>2.5</sub> air pollution (Source: Crouse *et al*, 2012).

Figure 4, taken from the Crouse study, illustrates the finding that mortality risk decreases with decreasing levels of PM<sub>2.5</sub>, even at ambient PM<sub>2.5</sub> levels down to 1 µg/m<sup>3</sup>. These investigators calculated hazard ratios (*i.e.*, risk ratios) and 95% confidence intervals (CIs), adjusted for available individual-level and contextual covariates, using a Cox proportional hazards survival models with spatial random-effects. They found a relative risk (or hazard ratio) of 1.30 (95% CI: 1.18, 1.43) for cardiovascular mortality from a 10 µg/m<sup>3</sup> increase in PM<sub>2.5</sub>.

Similarly, my own research has verified (as shown in Figure 5) that the association between PM<sub>2.5</sub> air pollution and cardiovascular mortality extends down to very low PM<sub>2.5</sub> concentration levels in the US as well (Thurston *et al*, 2016).

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1



**Figure 5.** Mortality Risk from Cardiovascular Disease Increases with Rising PM<sub>2.5</sub> Exposure, Even Well Below the Present US Ambient Air Quality Standard annual limit for PM<sub>2.5</sub> (12 µg/m<sup>3</sup>). (Source: Thurston *et al*, 2016a).

2 Importantly, this study is highly regarded, as it was conducted in a well characterized and large  
3 US population: the National Institutes of Health – American Association of Retired Persons (NIH-  
4 AARP) Diet and Health Study cohort. The NIH-AARP Study was initiated when members of the  
5 AARP, aged 50 to 71 years from 6 US states (California, Florida, Louisiana, New Jersey, North  
6 Carolina, and Pennsylvania) and 2 metropolitan areas (Atlanta, Georgia, and Detroit, Michigan),  
7 responded to a mailed questionnaire in 1995 and 1996. The NIH-AARP cohort questionnaires  
8 elicited information on demographic and anthropometric characteristics, dietary intake, and  
9 numerous health-related variables (*e.g.*, marital status, body mass index, education, race, smoking  
10 status, physical activity, and alcohol consumption), that was used to control for these factors in the  
11 air pollution mortality impact assessment. An extended analysis of the PM<sub>2.5</sub> - cardiovascular  
12 mortality association in the NIH-AARP Cohort has shown statistically significantly increased  
13 CVD mortality effects in subjects exposed to 8 to 12 µg/m<sup>3</sup> of long-term average PM<sub>2.5</sub> vs. those  
14 participants who resided in areas with concentrations less than 8 µg/m<sup>3</sup> (Hayes et al, 2019). This  
15 even more definitively confirms that the effects of PM<sub>2.5</sub> occur at levels below the prevailing



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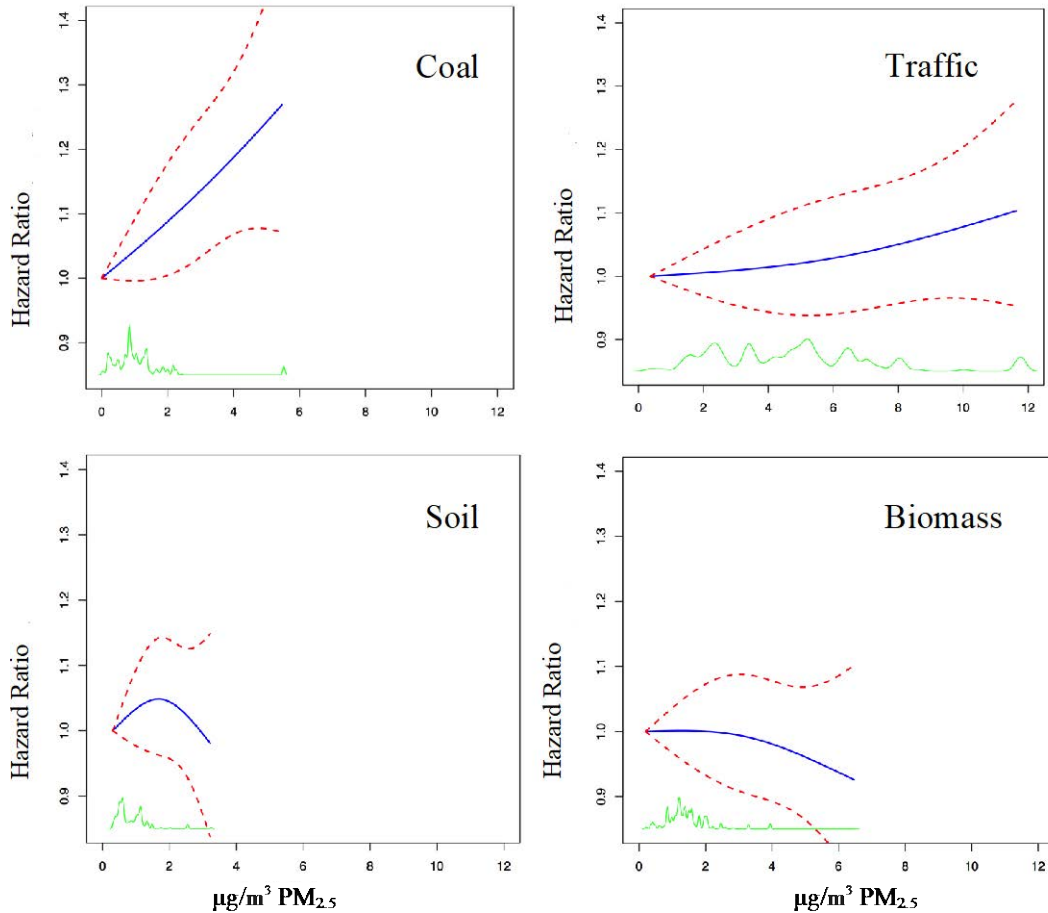
1 ambient air quality standard ( $12 \mu\text{g}/\text{m}^3$ ), and is consistent with the conclusion that  $\text{PM}_{2.5}$  is a non-  
2 threshold air pollutant, meaning it can have health impacts at any level of exposure.

3 Although published too late to be considered by the U.S. EPA in their 2013 standard setting  
4 process, the Crouse *et al* (2012), Thurston *et al* (2016a), and Hayes *et al* (2019) results indicate  
5 that the mortality effects of  $\text{PM}_{2.5}$  air pollution can occur at even lower ambient air pollution levels  
6 than shown by Pope *et al.* 2002, and even lower long-term mean concentrations than that level at  
7 which the U.S. EPA assumed the effects of  $\text{PM}_{2.5}$  to exist in its 2012 Regulatory Impact  
8 Assessment for the revised annual PM NAAQS (US EPA, 2012). The results of these three studies  
9 are consistent with the assumption that any reduction in  $\text{PM}_{2.5}$  air pollution can be expected to  
10 result in a commensurate health benefits to the public at ambient levels.

11 **Q. Are there studies that show that the health effects of  $\text{PM}_{2.5}$  from fossil fuel**  
12 **combustion are higher than from other sources?**

13 A. Yes. With respect to  $\text{PM}_{2.5}$  from power plants, specifically, my recent studies, and those by  
14 others, have also found that long-term exposure to fossil fuel combustion-related fine particulate  
15 air pollution is a particularly toxic environmental risk factor for cardiopulmonary and lung cancer  
16 mortality. The health impact is particularly high for particulate matter from fossil-fuel-burning  
17 facilities, such as coal burning, which has been associated with an ischemic heart disease mortality  
18 risk that is roughly five times that of the average for  $\text{PM}_{2.5}$  particles in general (Thurston *et al.*,  
19 2016b), and more damaging per  $\mu\text{g}/\text{m}^3$  than  $\text{PM}_{2.5}$  from other common sources (Figure 6). This  
20 new study, combined with past studies of US mortality and source-specific  $\text{PM}_{2.5}$  (*e.g.*, Ozkaynak  
21 and Thurston, 1987) indicate that the particles resulting from fossil-fuel burning (and especially  
22 from coal-combustion) at fossil-fueled EGUs are far more toxic to human health than the average  
23  $\text{PM}_{2.5}$  mass, when considered on per  $\mu\text{g}/\text{m}^3$  mass basis.

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**Figure 6.** Concentration-response curve (solid lines) and 95% confidence intervals (dashed lines) for source-specific PM<sub>2.5</sub> mass in the US American Cancer Society (ACS) Cohort. (Source: Thurston *et al*, 2016b).

**Q. Is particulate matter emitted by power plants toxic, regardless of which fossil fuel is burned?**

**A.** Particles from the combustion of different fossil fuels generally have shared characteristics. Fossil fuels have all undergone a similar process—they have a similar derivation, they have been underground and compressed, and they are combusted in relatively similar ways. Also, fossil fuel emissions all consist of very tiny particles that have large surface areas available to interact with the lung. And all of the various fossil-fuel combustion particles contain toxic transition metals,

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1 which cause can cause damaging oxidative stress in the body, once breathed into the lung and  
2 transported to the bloodstream along with oxygen.

3 **Q. How does toxicity of Particulate Matter from Fossil EGUs change over time, once**  
4 **emitted into the air?**

5 A. Recently emitted particles, such as those breathed by persons living near a power plant, are  
6 more toxic because they have more active sites on them to readily interact with the lung. Work by  
7 Oberdorster *et al* (1995) has shown PM concentrations at ambient levels, 60 µg/m<sup>3</sup> and less, cause  
8 mortality in healthy rats. And then they found the aging of those fumes with aggregation of the  
9 emitted ultrafine particles significantly decreased their toxicity over time, so proximity to such a  
10 combustion source of fine particles is of greatest health concern.

11 **Q. Is there scientific evidence that certain subpopulations are particularly susceptible**  
12 **to adverse health impacts from an increase in PM<sub>2.5</sub> in the ambient air?**

13 A. Among the groups of persons found in scientific research to be especially affected by  
14 environmental insults, including particulate matter air pollution, are: the very young, the poor, the  
15 very old, and persons with pre-existing health conditions, such as heart disease and asthma. (*see*,  
16 *e.g.*, US EPA, 2009a). Analyses by me and by others in the field of air pollution health effects  
17 indicate that the poor are especially at risk from air pollution (*e.g.*, Gwynn and Thurston, 2001).  
18 Similarly, older adults are at greater risk of severe adverse outcomes from air pollution. Also,  
19 children, a population known to be especially susceptible to the effects of air pollution because  
20 their bodies are developing (and because they spend larger amounts of time exercising outside) are  
21 an especially affected sub-population that is well represented in the community surrounding  
22 sources. These subpopulations can be expected to be among those most strongly affected by any  
23 exposures to PM<sub>2.5</sub> concentrations in the vicinity of these plants.

24 **Q. What is your conclusion about the relationship between PM<sub>2.5</sub> and health based on**  
25 **these studies?**

26 A. Any PM<sub>2.5</sub> exposure, such as the PM<sub>2.5</sub> air pollution resulting from the DTE EGU's  
27 emissions, has an adverse human health effect on those breathing that air pollution at any  
28 prevailing PM<sub>2.5</sub> air pollution level.

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**2. Human Health Effects of Exposure to Ozone (O<sub>3</sub>)**

**Q. Please summarize the human health effects of exposures to ozone.**

A. Health effects from ozone include:

- Aggravation of asthma; triggering of more asthma attacks
- Aggravation of lung diseases such as emphysema and bronchitis
- Damaged and diminished lung function
- Diminished immune system effects
- Increased risk of mortality

**Q. What is Ozone?**

A. Ozone (O<sub>3</sub>) is a gaseous air pollutant that is not emitted directly by the EGUs under consideration here, but is instead a secondary air pollutant formed in the atmosphere from the pollutants emitted by these units, especially nitrogen oxides and hydrocarbons, both products of fossil fuel combustion. This is especially the case in the summer months, when there is more sunlight and warmer weather, which enhances the formation of ozone in the atmosphere.

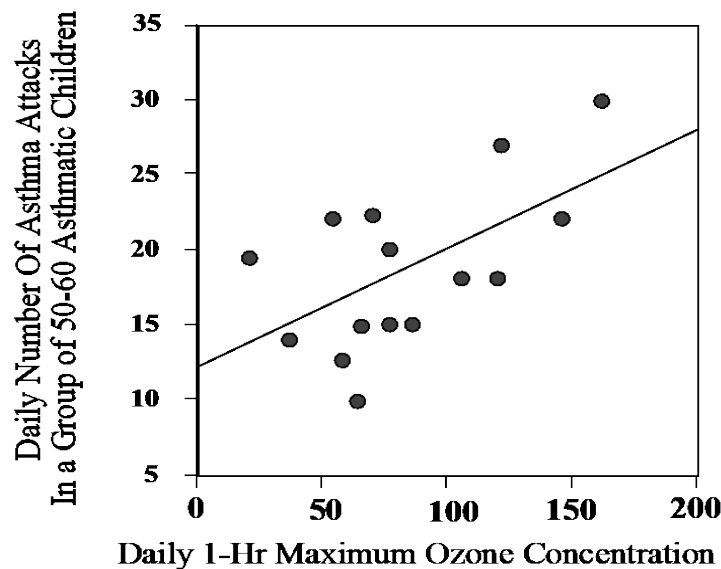
**Q. What are the human health effects associated with exposure to Ozone.**

A. Ozone can irritate the human respiratory system when breathed, causing exposed people to cough, feel an irritation in the throat, and/or experience an uncomfortable sensation in the chest area. Ozone has also been shown to reduce the lung's ability to inhale and exhale, thereby making it more difficult for people to breathe as deeply and vigorously as they normally would (*See e.g.*, Bates, 1995). Research shows that ozone can also acutely aggravate asthma, and new evidence suggests that it may cause more children to get asthma (Garcia *et al*, 2019). When ozone levels are high, people with asthma have more attacks that require a doctor's attention or the use of additional medication. One reason this happens is that ozone makes people more sensitive to allergens, which are the most common triggers for asthma attacks. Ozone can inflame and damage cells that line the human lung, and ozone has been compared by some to "getting a sunburn on your lungs." Ozone may also aggravate chronic lung diseases, such as emphysema and bronchitis, and can reduce the immune system's ability to fight off bacterial infections in the respiratory system.

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**Q. Please explain the connection between ozone exposure and asthma attacks on children.**

A. Among the important adverse effects associated with ozone exposure to asthmatics is the triggering of asthma attacks. The effects of ozone air pollution on children with asthma have been demonstrated in my own research following a group of children at an asthma summer camp located in Connecticut. This study of a group of about 55 moderate to severely asthmatic children showed that these children experienced statistically significant reductions in lung function, increases in asthma symptoms, and increases in the use of unscheduled asthma medications as ozone pollution levels rose.



**Figure 7.** The number of asthma attacks among children at an “Asthma Camp” in Connecticut increase as the ozone levels rise (Source: Thurston *et al.*, 1997)

As shown in Figure 7, the risk of a child having an asthma attack was found to be approximately 40 percent higher on the highest ozone days than on an average study day (Thurston *et al.*, 1997). Consistent with other research in this area, there is no indication in this plot of a threshold concentration below which children with asthma are safe from the effects of ozone increases. These asthma camp results have been confirmed by a larger study published in the Journal of the American Medical Association (JAMA). Gent *et al* (2003) presented a cohort study of asthmatic children from the New Haven, CT area, including 130 children who used maintenance medications for asthma and 141 children who did not. The more severe asthmatics were identified as those using maintenance medication. For these severe asthmatics, the study found that the level of ozone exposure was

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1 significantly associated with worsening of symptoms and an increase in the use of rescue medication.  
2 Each 50 parts per billion (ppb) increase in 1-hour average O<sub>3</sub> was associated with an increased  
3 likelihood of wheezing (by 35%) and chest tightness (by 47%). The findings indicate that asthmatic  
4 children are particularly vulnerable to ozone, even at pollution levels below EPA air quality  
5 standards.

6 **Q. Please explain the adverse impacts of ozone exposure beyond asthmatic children.**

7 A. My own research has also shown ozone air pollution to be associated with diminished lung  
8 function in non-asthmatic healthy children at a YMCA summer camp in a pristine area in the  
9 Kittatinny Ridge, in the northwestern part of the state (Spektor *et al*, 1988a). Similarly, in the  
10 summer of 1988, Berry *et al* (1991) conducted a field health study at two summer day camps in  
11 suburban-central New Jersey. Thirty-four campers and counselors had daily lung function tests,  
12 and it was found that the campers had a statistically significant decrease in peak expiratory flow  
13 rate associated with increasing ozone concentrations, indicating an acute loss in the children's  
14 ability to inhale and exhale after ozone exposure.

15 The adverse effects of exposure to ozone in ambient air on the lungs of individuals has  
16 been demonstrated in studies that I have conducted in the State of New York, as well. For example,  
17 respiratory function damage was demonstrated in a study I co-authored of 30 healthy adult non-  
18 smokers engaged in a regular daily program of outdoor exercise in Tuxedo, New York, during the  
19 summer of 1985 (Spektor *et al*, 1988b). All measured health indices showed statistically  
20 significant O<sub>3</sub>-associated decreases in the lung function of the runners as ozone levels increased.  
21 More recently, using lung bronchoscopy (which allows a visualization of the main tubes of the  
22 lungs, by means of a flexible lighted instrument introduced through the vocal cords and windpipe)  
23 and broncho-alveolar lavage (BAL, or a washing of the lining of the lung), Kinney *et al* (1996)  
24 examined some 19 normal volunteer joggers from Governors Island, New York. The joggers  
25 exercised in the afternoon during the 1992 summer season. These results indicate a significant  
26 inflammatory response in the lungs of recreational joggers in New York City exposed to regional  
27 ozone and associated co-pollutants during the summer months.

28 Airway inflammation in the lung is among the serious effects that have also been  
29 demonstrated by controlled human studies of ozone at levels typically experienced by most

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Americans. Airway inflammation is especially problematic for children and adults with asthma, as it makes them more susceptible to having asthma attacks, consistent with the asthma camp results discussed above. For example, controlled human studies have shown that prior exposure to ozone enhances the reactivity of asthmatics to aeroallergens, such as pollens, which can trigger asthma attacks (*see e.g., Molfino et al, 1991*).

The increased inflammation of the lung, and diminished immune system effects associated with ozone air pollution can also make the elderly more susceptible to pneumonia, a major cause of illness and death in this age group. Both *in vivo* and *in vitro* experimental studies have demonstrated that O<sub>3</sub> can affect the ability of the immune system to defend against infection. Increased susceptibility to bacterial infection has been reported in mice at below 80ppb ozone for a single 3-hr exposure (Ehrlich *et al, 1977*). Related alterations of the pulmonary defenses caused by short-term exposures to O<sub>3</sub> include impaired ability to inactivate bacteria in rabbits and mice (Coffin and Gardner, 1972; Ehrlich *et al, 1979*) and impaired macrophage defense mechanisms in the lung (Dowell *et al, 1970*; Goldstein *et al, 1971*; McAllen *et al, 1981*; Amoruso *et al, 1981*). Thus, the biological plausibility of the adverse air pollution health effects associations found by epidemiological studies is supported by a body of controlled exposure animal studies.

The O<sub>3</sub> - morbidity associations indicated by the above-presented epidemiological studies are also supported by a large body of data from controlled human exposure studies that give consistent and/or supportive results, and that have demonstrated pathways by which ozone can damage the human body when breathed. Clinical studies have demonstrated decreases in lung function, increased frequencies of respiratory symptoms, heightened airway hyper-responsiveness, and cellular and biochemical evidence of lung inflammation in healthy exercising adults. For example, in controlled exposure studies, McDonnell *et al* (1991) and Devlin *et al* (1991) found that prolonged controlled exposures of exercising men to levels of ozone common in present-day U.S. (only 80 ppb) resulted in significant decrements in lung function, induction of respiratory symptoms, increases in nonspecific airway reactivity, and cellular and biochemical changes in the lung.

Ozone exposure has also been shown to have adverse effects on athletic performance. Epidemiological evidence compiled more than three decades ago suggested that the percentage of high school track team members failing to improve performance increased with increasing oxidant concentrations the hour before a race (Wayne *et al, 1967*). Controlled exposure studies of heavily

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exercising competitive runners have demonstrated decreased function at 200 to 300 ppb (Savin and Adams, 1979; Adams and Schelegle, 1983). More recent studies have shown reduced athletic performance at even lower O<sub>3</sub> concentrations. Schlegle and Adams (1986) exposed 10 young male adult endurance athletes to 120, 180, and 240 ppb O<sub>3</sub> while they exercised for 60 minutes. Although all 10 completed the protocol for filtered (clean) air exposure, 1, 5, and 7 of them could not complete it for the 120, 180 and 240 ppb O<sub>3</sub> exposures, respectively, indicating that higher O<sub>3</sub> concentrations made exercising more difficult.

Ozone may also cause permanent lung damage. For example, repeated short-term ozone damage to children's developing lungs may lead to reduced lung function in adulthood (*e.g.*, see Kunzli *et al*, 1997). In adults, ozone exposure may accelerate the natural decline in lung function that occurs as part of the normal aging process (*e.g.*, see Detels, *et al*, 1987). One important study suggests that long-term ozone exposure can increase the chances that children will develop asthma disease (McConnell *et al*, 2002).

Ozone has also been shown to have long-term cumulative health effects in the State of New Jersey in a study that included cadets from the U.S. Military Academy at West Point who attended special summer training in Fort Dix, New Jersey. There was a statistically significant drop in forced expiratory volume in 1 sec of 44 ml ( $p = .035$ ), and there were also significant increases in reports of cough, chest tightness, and sore throat at the follow-up clinic visit: a larger decline in long-term mean Forced Expiratory Volume lung function was observed in cadets at Fort Dix, where ozone exposures were the highest (Kinney and Lippmann, 2000).

**Q. Is there a connection between ozone pollution and increased hospital admissions?**

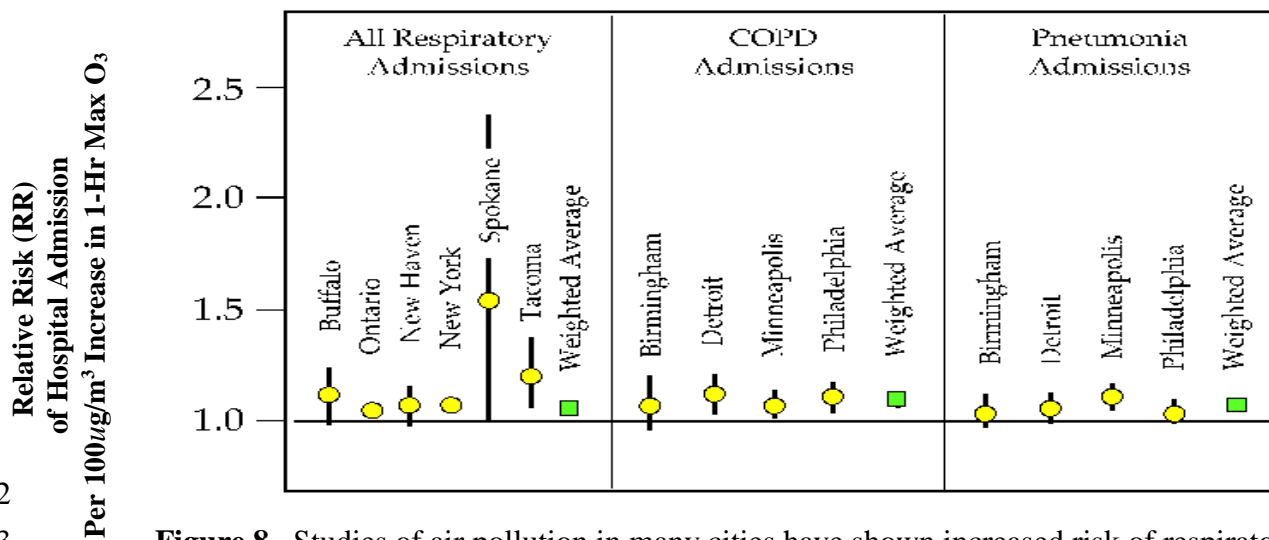
A. Yes. Emergency Room Visits and Hospital Admissions are increased by O<sub>3</sub> air pollution. Cody *et al* (1992) analyzed data on New Jersey hospital emergency department (ED) visits for asthma, bronchitis, and finger wounds (a non-respiratory control) for the period May through August for 1988 and 1989, finding that, when temperature was controlled for in a multiple regression analysis, a highly significant relationship between asthma visits and ozone concentration was identified. In addition, a 5-year retrospective study by Weisel *et al* (1995) of the association between ED visits for asthma with mean ambient ozone levels was conducted for



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hospitals located in central New Jersey. An association was identified in each of the years (1986-1990), and ED visits occurred 28% more frequently when the mean ozone levels were greater than 60 ppb O<sub>3</sub>, as compared to when they were less than 60 ppb O<sub>3</sub>.

Epidemiological evidence has accumulated over the years indicating a role of O<sub>3</sub> in daily hospital admissions. As displayed in Figure 8, time-series studies conducted in the U.S. have shown increased risk of hospital admissions (Relative Risk>1.0) at higher O<sub>3</sub> levels, even after accounting for the effects of PM (Schwartz, J. in Health at the Crossroads, 1997). This work has now been expanded to consider 36 cities across the U.S., finding that, during the warm season of the year, the 2-day cumulative effect of a 5-ppb increase in O<sub>3</sub> was an estimated 0.3% increase in the risk of chronic obstructive pulmonary disease admissions, and a 0.4% increase in the risk of pneumonia admissions (Medina-Ramon *et al*, 2006).

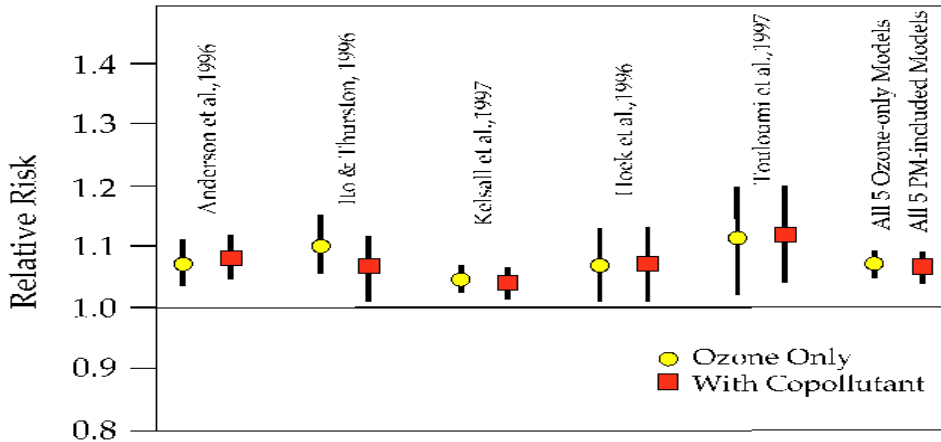


**Figure 8.** Studies of air pollution in many cities have shown increased risk of respiratory hospital admission (RR >1.0) on days of high ozone air pollution (Source: Schwartz, J. in Health at the Crossroads, 1997).

**Q. Is there evidence that ozone pollution has an impact on human mortality?**

A. Yes. Epidemiological evidence has also accumulated over recent years indicating a role by ozone in daily human mortality. As shown in Figure 12, time-series studies conducted in cities around the world have shown increased mortality (Relative Risk>1.0) at higher ozone concentrations, even after accounting for the mortality effects of PM (Thurston and Ito, 2001).

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**Figure 9.** Studies indicate an increased risk of mortality ( $RR > 1.0$ ) at higher ozone concentrations, even after considering the effects of PM. (Source: Thurston and Ito, 2001).

Multi-city analyses have confirmed the ozone-mortality relationship. These include meta-analyses of multiple past ozone studies that show consistent associations between ozone and increases in mortality (Levy *et al*, 2005; Ito *et al*, 2005; Bell *et al*, 2005). In one analysis of some 95 U.S. cities over two decades published in *JAMA*, Bell *et al* (2004) showed that, even after controlling for PM and weather, an increase of 10 parts-per-billion in daily ozone pollution was associated with approximately a 0.5% increase in daily risk of death. As discussed earlier, this size percent increase in daily admissions, though small, affects a huge portion of the population and accumulates day after day, week after week, and month after month, so that it accumulates to account for thousands of deaths each year in the U.S.

More recently, mortality effects from long-term exposure to ozone air pollution has now been confirmed in major cohort studies (Jerrett *et al*, 2009; Turner *et al*, 2016, Lim *et al*, 2019). In Jerrett *et al*, data from the study cohort of the American Cancer Society Cancer Prevention Study II were correlated with air-pollution data from 96 metropolitan statistical areas in the United States. 448,850 subjects, with 118,777 deaths in an 18-year follow-up period were considered. Data on daily maximum ozone concentrations were obtained from April 1 to September 30 for the years 1977 through 2000. Data on concentrations of fine particulate matter ( $PM_{2.5}$ ) were obtained for the years 1999 and 2000. Associations between ozone concentrations and the risk of death were evaluated with the use of standard and multilevel Cox regression models. In single-pollutant models, ozone was associated with the risk of death from respiratory causes. The estimated relative

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1 risk of death from respiratory causes that was associated with an increment in ozone concentration  
2 of 10 ppb was 1.040 (95% confidence interval, 1.010 to 1.067). The association of ozone with the  
3 risk of death from respiratory causes was insensitive to adjustment for confounders and to the type  
4 of statistical model used. In a follow-up analysis of this same database, Turner *et al* (2016)  
5 improved ozone exposure estimates by employing estimates of O<sub>3</sub> concentrations at the  
6 participant's residence, as derived from a hierarchical Bayesian space-time model. In two-  
7 pollutant models, adjusted for PM<sub>2.5</sub>, significant positive associations remained between O<sub>3</sub> and  
8 all-cause (hazard ratio [HR] per 10 ppb, 1.02; 95% confidence interval [CI], 1.01–1.04),  
9 circulatory (HR, 1.03; 95% CI, 1.01–1.05), and respiratory mortality (HR, 1.12; 95% CI, 1.08–  
10 1.16) that were unchanged with further adjustment for NO<sub>2</sub>. Lim *et al* (2019) confirmed these  
11 findings in another large cohort study conducted across the US. We investigated associations of  
12 long-term (annual or warm season average) O<sub>3</sub> exposure with all-cause and cause-specific  
13 mortality in the NIH-AARP Diet and Health Study, a large prospective cohort of U.S. adults with  
14 17 years of follow-up from 1995 to 2011. Long-term annual average exposure to O<sub>3</sub> was  
15 significantly associated with deaths due to cardiovascular disease (per 10 ppb, HR=1.03; 95% CI:  
16 1.01-1.06), ischemic heart disease (HR=1.06; 95% CI: 1.02-1.09), respiratory disease (HR=1.04;  
17 95% CI: 1.00-1.09), and chronic obstructive pulmonary disease (HR=1.09; 95% CI: 1.03-1.15) in  
18 single-pollutant models.

19 **Q. Are there studies showing the health benefits of avoiding or reducing Ozone levels?**

20 A. Yes. Another study considers a broadly relevant case showing the benefits of cleaner air.  
21 During the Atlanta Summer Olympics of 1996, traffic-related ozone and PM declined significantly  
22 as a result of the alternative mass transportation strategy implemented to reduce road traffic during  
23 the Games (Friedman *et al*, 2001). These improvements were correlated with changes in the rate  
24 of children's hospital admissions. Compared to a baseline period, traffic related ozone and PM<sub>10</sub>  
25 levels declined by 28% and 16%, respectively. Concentrations of both PM and ozone also rose  
26 noticeably after the end of the Olympics. The study showed a significant reduction in asthma  
27 events associated with these pollution improvements. This study supports the hypothesis that  
28 improvements in acute air pollution can provide immediate health benefits.

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**3. Human Health Effects of Exposures to Sulfur Dioxide (SO<sub>2</sub>)**

**Q. Please summarize the human health effects of exposures to Sulfur Dioxide.**

A. EGUs are the leading source of SO<sub>2</sub> in the United States. Coal-fired EGUs remain the dominant sources by nearly fivefold above the next highest source (industrial fuel combustion), emitting 3.2 million tons of SO<sub>2</sub> annually (US EPA, 2017). Exposures to SO<sub>2</sub> have also been associated with adverse health effects, in addition to leading to the secondary formation of acidic PM<sub>2.5</sub> in the atmosphere. As concluded in the most recent U.S. EPA Integrated Science Assessment for SO<sub>2</sub> (EPA-600/R-17-451), research studies have provided scientific evidence that is sufficient to infer a similar causal relationship to also exist between short-term (e.g., daily) SO<sub>2</sub> exposure and adverse effects on the respiratory system, such as asthma. This finding of a causal relationship between SO<sub>2</sub> exposure and increased respiratory morbidity is supported by a large body of recent epidemiologic evidence, as well as by findings from human and animal experimental studies. These epidemiologic and experimental studies encompass a number of endpoints, including ED visits and hospitalizations, respiratory symptoms, airway hyperresponsiveness, and lung function (US EPA, 2009).

**4. Human Health Effects of Exposures to Nitrogen Dioxide (NO<sub>2</sub>)**

**Q. Please summarize the human health effects of exposures to Nitrogen Dioxide.**

A. Exposures to nitrogen oxides themselves have also been associated with adverse human health effects, in addition to leading to the formation of PM<sub>2.5</sub> and ozone. As concluded in a U.S. EPA Risk and Exposure Assessment Report for NO<sub>x</sub> (EPA-452/R-08-008a), research studies have provided scientific evidence that is sufficient to infer a similar relationship to also exist between short-term (e.g., daily) NO<sub>2</sub> exposure and adverse effects on the respiratory system. This finding is supported by the large body of recent epidemiologic evidence as well as findings from human and animal experimental studies. These epidemiologic and experimental studies encompass a number of endpoints including ED visits and hospitalizations, respiratory symptoms, airway hyperresponsiveness, airway inflammation, and lung function (US EPA, 2008).

**5. Human Health Effects of Exposure to Toxic Pollutants**

**Q. Please summarize the human health effects of exposures to toxic pollutants.**

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A. Air toxics, also known as hazardous air pollutants (HAPs), are pollutants that are known or suspected as causing cancer or other serious health effects, such as birth defects or reproductive effects. An EPA assessment of air toxics from utility power plants identified twelve HAPs as priority pollutants of potential health concern (arsenic, beryllium, cadmium, chromium, manganese, nickel, hydrogen chloride [HCl], hydrogen fluoride [HF], acrolein, dioxins, formaldehyde, and radionuclides) (US EPA, 1998). Of these, the EPA report assessed the impact on public health due to non-inhalation exposures for radionuclides, mercury, arsenic, and dioxins, finding that, of these four, mercury from coal-fired utilities is the HAP of greatest potential concern to public health. The primary health impacts for toxicity of mercury and mercury compounds are to the nervous system, kidney, and developing fetus. Other biological systems that may be impacted include the respiratory, cardiovascular, gastrointestinal, hematologic, immune, and reproductive systems (US EPA, 1998).

**Q. Would you agree that emissions from EGUs have an adverse impact on the community of impact?**

A. Overall, there is a consistency between the epidemiologic study associations and experimental study results, supporting the conclusion that (a) there is indeed a cause-effect relationship between air pollution and negative health effects; and, (b) there is no known threshold below which no effects are experienced. Thus, air pollution from the EGUs, including DTE's EGUs, result in commensurate public health risks from those air pollutants, the continued operation of these units continues those adverse health effects to accumulate, and decreasing their emissions would reduce those health effects in future years.

**B. EVALUATING THE IMPACTS OF AIR POLLUTION EXPOSURES FROM FOSSIL FUEL EGUs**

**Q. What are DTE Electric Company's key fossil-fuel powered EGUs?**

A. DTE Electric has numerous fossil-fuel powered EGUs, including several coal units, gas fired peakers, oil fueled units, and others. In addition, DTE Electric is proposing to extend the life of its currently coal-fired River Rouge 3 plant by two years by fueling it with gas rather than coal, and is building a new 1100 MW CCGT, which is currently under construction and expected to be in-service in 2022. The following table summarizes DTE's coal-fueled fleet, as reported by DTE in its IRP:

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**Table 1: DTE Coal Units and Present Operational Plans:**

<b>Fossil-Fueled Generation Units</b>	<b>Year Operational</b>	<b>Starting Point Retirement Date<sup>2</sup></b>	<b>IRP Retirement Treatment<sup>3</sup></b>	<b>Location</b>
River Rouge Unit 3	1958	2020	Coal combustion ends 2020; burn recycled industrial gases until 2022	Wayne County
St. Clair Unit 1	1953	2022	2019	St. Clair County
St. Clair Unit 2	1953	2022	2022	St. Clair County
St. Clair Unit 6	1961	2022	2022	St. Clair County
St. Clair Unit 7	1969	2023	2022	St. Clair County
Trenton Channel Unit 9	1968	2023	2022	Wayne County
Belle River Unit 1	1984	2029	Evaluated 2025 retirement; maintains 2029 retirement	St. Clair County
Belle River Unit 2	1985	2030	Evaluated 2026 retirement; maintains 2030 retirement	St. Clair County
Monroe Unit 1	1971	2040	Consider in future IRP	Monroe County
Monroe Unit 2	1973	2040	Consider in future IRP	Monroe County
Monroe Unit 3	1973	2040	Consider in future IRP	Monroe County
Monroe Unit 4	1974	2040	Consider in future IRP	Monroe County

<sup>2</sup> IRP, p. 53; Mikulan, p. 37.

<sup>3</sup> IRP, pp. 129, 142, 153; Mikulan pp. 51-61, 87-89.

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DTE is proposing to chart a course towards continued operation of some coal-fired EGUs, imminent retirement of other coal-fired EGUs, and the addition of a new gas-fired EGU in the near term and the potential addition of another gas-fired EGU in the longer term. Other witnesses discuss those plans. The purpose of my testimony, as noted above, is to document the nature of the health impacts associated with the continued operation of DTE's fossil-fuel EGUs.

**Q. Is it feasible for an electric utility to consider health impacts as part of resource decision-making?**

A. Yes, the utility has first-hand emissions data and facility details (*e.g.*, stack heights, production rates) to provide for an economic and health impact analysis.

**Q. Are there tools available to assist with the evaluation of economic and human health effects resulting from air pollution?**

A. Yes. One way to estimate such health and economic impacts from such pollution sources is the widely applied US EPA approved Environmental Benefits Mapping and Analysis Program (BenMAP) (Sacks *et al*, 2018). BenMAP is a Windows-based computer program that uses a Geographic Information System (GIS)-based method to estimate the health impacts and economic impacts occurring when populations experience changes in air quality (Abt Associates, 2010; USEPA, 2015). Analysts have relied upon BenMAP to quantify the health impacts from air quality changes at the city and regional scale, both within and beyond the U.S.

BenMAP is primarily intended as a tool for estimating the health impacts, and their associated economic values, associated with changes in ambient air pollution. It accomplishes this by running health impact functions, which relate a change in the concentration of a pollutant with a change in the incidence of a health endpoint. Inputs to health impact functions include:

- the change in ambient air pollution level;
- pollutant health effect estimates (based upon the scientific literature);
- the exposed population, as provided in the BenMAP model; and
- the baseline incidence rate of the health endpoint, from the BenMAP model.

By way of example, in the case of a premature mortality health impact function, the

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BenMAP calculation can be represented, in a simplified form, as:

$$\text{Mortality Change} = (\text{Air Pollution Change}) * (\text{Air Pollution Mortality Effect Estimate}) * (\text{Mortality Incidence}) * (\text{Exposed Population})$$

- **Air Pollution Change.** The air quality change is calculated as the difference between the starting air pollution level, also called the baseline, and the air pollution level after some change, such as that caused by a regulation. In the case of ozone, this is typically estimated in parts per million (ppm).
- **Mortality Effect Estimate.** The mortality effect estimate is an estimate of the percentage change in mortality due to a one-unit change in ambient air pollution. Epidemiological studies provide a good source for effects, as discussed above.
- **Mortality Incidence.** The mortality incidence rate is an estimate of the average number of people that die in a given population over a given period of time, as provided in BenMAP. For example, the mortality incidence rate might be the probability that a person will die in a given year.
- **Exposed Population.** The exposed population is the number of people affected by the air pollution increases caused by upwind emissions controls not being applied.

**Q. Are there examples where health impact modeling tools like BenMAP have been used to develop a health impact assessment?**

A. Yes, this is an air quality evaluation approach widely used by governments around the world. For example, the Canadian government uses the very similar Air Quality Benefits Assessment Tool (AQBAT)<sup>4</sup> (). AQBAT is a computer application developed by Health Canada which is designed to estimate the human health impacts of changes in Canada's ambient air quality. It is used to estimate the benefits (positive impacts) or damages (negative impacts) of proposed regulatory initiatives related to outdoor air quality. A tool that quantifies the economic and dollar valuation of health effects from air pollution resulting from power plant emissions, using the same health effects and valuation approach as the EPA BenMAP, is demonstrated in studies issued by the Clean Air Task Force ("CATF") based on work by Abt Associates, US EPA's health benefits consultant relying on peer-reviewed, published methodology. Using recent available emissions data (2016), MSB Energy Associates on behalf of CATF used the Powerplant Impact Estimator

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<sup>4</sup> [http://www.science.gc.ca/eic/site/063.nsf/eng/h\\_97170.html](http://www.science.gc.ca/eic/site/063.nsf/eng/h_97170.html)



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(PIE) tool developed by Abt Associates to estimate the death and disease due to coal plant fine particulate matter (PM<sub>2.5</sub>) in that year. This latest report finds that over 3,000 deaths each year are attributable to fine particle pollution from U.S. coal-fired power plants (Clean Air Task Force, 2010). One part of this study, referred to as the *Toll from Coal Report*, provides plant by plant estimates of the annual health impacts of individual coal-fired power plants.<sup>5</sup> In addition, I am familiar with a health impacts modeling analysis that was performed for the Community Action to Promote Healthy Environments (CAPHE), with results compiled in the report, *Air Pollutant Sources, Exposures & Health Impacts* (2016). (Milando, Huang and Batterman, 2016;).<sup>6</sup>

**Q. What do you conclude based on the health impacts modeling tools that you have reviewed?**

A. Based on the modeling tools I have reviewed, in particular the *Toll from Coal* tool, I conclude that the human health effects being visited on the public living in the surrounding and downwind populations are many, and of significant monetary valuation. As such, any consideration of the costs of lowering emissions from the DTE EGUs should also consider these costs being visited on the public.

I further note that experts have been using models like BenMAP for decades to quantify the health and economic impacts from air pollution. These are industry accepted models that follow a thorough and rigorous process. However, to generate dependable and reliable outputs, these tools require accurate data. Because current, reliable data sets (*e.g.*, actual emissions) may not be available, data may be provided by the EGU-operator so that the valuations of the health benefits from cleaner air can be considered in the regulatory process.

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<sup>5</sup> [www.TollFromCoal.org](http://www.TollFromCoal.org)

<sup>6</sup> See also [www.caphedetoirt.sph.umich.edu](http://www.caphedetoirt.sph.umich.edu).

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1     **C.           CONCLUSIONS**

2     **Q.           Please summarize your findings on air pollution from DTE EGUs and health**  
3     **impacts.**

4     A.           My findings are as follows:

- 5         **1. Emissions from the DTE EGUs add to the existing levels of PM<sub>2.5</sub>, sulfur oxides, and**  
6         **nitrogen oxides in the vicinity of these DTE EGU units, and to ozone downwind of the**  
7         **plants.** Because no threshold of air pollution health effects has yet been found, these air  
8         pollution exposures add an incremental adverse health risk to residents downwind of these  
9         sources of fossil fuel combustion air pollution. Such an increased population risk of health  
10        effects constitutes an individual adverse health effect has been confirmed by a joint  
11        Statement of the European Respiratory Society and the American Thoracic Society.<sup>7</sup>
- 12        **2. Any reduction in air pollution emissions from these plants (including earlier EGU**  
13        **retirements) would lead to reductions in these adverse health effects, as well as a**  
14        **reduction in the economic impacts of these exposures on the public.** The operation of  
15        the fossil-fuel burning DTE EGUs have both local and downwind adverse human health  
16        consequences. To the extent that these facilities continue to emit PM<sub>2.5</sub> and other air  
17        pollutants, they cause an associated risk of adverse health effects among those who breathe  
18        that pollution, and especially for the socio-economically disadvantaged populations living  
19        immediately surrounding these EGUs. Furthermore, in addition to the effects of PM<sub>2.5</sub>, the  
20        proposed facility's emissions of sulfur dioxide and nitrogen oxides will also contribute to  
21        health risks from added local air pollution, as well as to the downwind formation of, and  
22        exposures to, ozone air pollution, and to associated downwind adverse human health  
23        effects caused by O<sub>3</sub> exposures.

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<sup>7</sup> Ex MEC-87 (Thurston *et al*, A Joint ERS/ATS Policy Statement: What Constitutes An Adverse Health Effect Of Air Pollution? An Analytical Framework. *European Respiratory Journal* (2017).

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1    **Q.     Why should the Commission recognize these adverse human health impacts?**

2    A.     There are multiple factors that impact the decision to operate fossil-fueled generating units  
3    – economic factors, reliability factors, demand requirements, alternative resources to meet  
4    demand, public policies, and others. There is a clear, direct, well-documented link between the  
5    emissions from fossil-fueled electric generating units and human health in the impacted downwind  
6    communities. A comprehensive, long-term electric generation resource planning process would be  
7    incomplete if it did not recognize the community health impacts associated with the resource  
8    planning decisions, and their economic disbenefits to the public. The affected communities are  
9    currently bearing the costs of those decisions. Decisions made by the Commission to approve a  
10   proposed course of action should consider the health and economic costs to the community of  
11   those decisions as part of the decision-making process.

12   **Q.     How do you recommend the Commission proceed?**

13   A.     In future proceedings, the Commission should require the utility to provide an analysis of  
14   the community health impacts (and their economic valuations) resulting from its various resource  
15   planning decisions, particularly as it relates to the operation of fossil-fired generation. As discussed  
16   above, there are acceptable modeling tools (*e.g.*, BenMAP) to assess and monetize health impacts  
17   of resource decisions. In order to effectively integrate the analysis of health impacts into resource  
18   planning and decision-making, the Commission may set the appropriate parameters (*e.g.*, scope,  
19   time frame, resources, data sets) for such an analysis, to ensure consistency as well as useful  
20   analysis.

21   **Q.     Does this conclude your testimony?**

22   A.     Yes.

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## Curriculum Vitae

### GEORGE D. THURSTON

3 Catherine Court  
Chester, NY 10918  
Hm: (845) 783-1978  
Wk: (845) 731-3564  
Fax: (845) 351-5472

Email: [george.thurston@nyu.edu](mailto:george.thurston@nyu.edu)  
<http://www.med.nyu.edu/biosketch/gdt1>

### Education

Degree	Field	Institution
Diploma	Academic	Barrington High School, RI
Sc.B. (Honors)	Environmental Engineering	Brown University
A.B.	Environmental Studies	Brown University
S.M.	Environmental Health Sciences	Harvard Univ. Schl. of Public Health
Sc.D.	Environmental Health Sciences	Harvard Univ. Schl. of Public Health

### Postdoctoral Training

<u>Specialty</u>	<u>Mentor</u>	<u>Place of Training</u>
Environ. Epidemiology	Dr. H. Ozkaynak	Harvard Univ., Kennedy Schl. of Gov., Camb., MA

### Internships and Residencies N/A

### Clinical and Research Fellowships N/A

**Licensure and Certification:** Environmental Benefits Mapping and Analysis Program - Community Edition (BenMAP-CE) Training Certification (August 2014).

### Academic Appointments

1987-1993	Assistant Professor, Dept. of Environmental Medicine, New York University School of Medicine, New York City, NY.
1993-2006	Associate Professor (Tenured), Dept. of Environmental Medicine, New York University School of Medicine, New York City, NY.
2007-present	Professor (Tenured), Dept. of Environmental Medicine, New York University School of Medicine, New York City, NY.
2007-present	Affiliated Faculty, Environmental Studies Program, College of Arts and Sciences, New York University, New York City, NY.
2012-present	Affiliated Faculty, Marron Institute on Cities and the Urban Environment, New York University, New York City, NY
2012-present	Faculty Mentoring Champion, Dept. of Environmental Medicine, New York University School of Medicine, New York City, NY.

### Hospital Appointments: N/A

### Other Professional Positions and Visiting Appointments:

Oak Ridge Institute for Science and Education (ORISE) Fellow (2008-2010)

## Major Administrative Responsibilities

<i>Year</i>	<i>Title, Place of Responsibility</i>
1995-2004	Director, Community Outreach and Environmental Education Program, NYU-NIEHS Center of Excellence, Nelson Inst. of Environ. Med., NYU School of Medicine, Tuxedo, NY
2002-2012	Deputy Director, NYU Particulate Matter Research Center, Nelson Inst. of Environmental Medicine, NYU School of Medicine, Tuxedo, NY
2007-2008	Director, Environmental Epidemiology Core, NYU-NIEHS Center of Excellence, Department of Environmental Medicine, Tuxedo, NY
2010-2015	Co-Leader, Metals Research Focus Group, NYU-NIEHS Center of Excellence, Department of Environmental Medicine, Tuxedo, NY.
2012-2016	Chair, Appointments and Promotions Committee, Department of Environmental Medicine, NYU School of Medicine.
2014-2016	Co-Chair, Environmental Health Research Affinity Group, NYU Global Institute of Public Health (GIPH), New York University, Washington Square.
2012-present	Director, Program in Exposure Assessment and Human Health Effects, Department of Environmental Medicine, NYU School of Medicine.

## Teaching Experience

<i>Year</i>	<i>Name of course</i>	<i>Type of Teaching</i>
1984-1994	Air Poll. Transport Modeling (G48.2048)	Course Director
2006-present	Climate, Air Pollution, & Health (G48.1010)	Course Director
1986-present	Aerosol Science (G48.2033)	Course Director
1984-2010	Environmental Contamination (G48.2305)	Lecturer
1984-present	Environ. Hygiene Measurements (G48.2035)	Lecturer/Lab
1990-1998	Environmental Toxicology (G48.1006)	Lecturer
1993-1995	Environmental Epidemiology I (G48.2039)	Lecturer
2001-2003	NYU Summer Institute, Wagner School	Lecturer
2006-present	Environmental Epidemiology I (G48.2039)	Lecturer
2006-present	Science, Health & Envir. Journalism (G54.1017.0)	Lecturer
2009-2011	Global Environmental Health (U10.2153.1)	Course Director
2009-2012	Global Issues in Environ. Health (G48.1011)	Course Director
2009-present	Earth Systems Science (undergrad) (V36.0200)	Lecturer
2011-present	Principles of Environmental Health (G48.1004)	Course Director
2013-present	Environ. Hygiene Measurements (G48.2035)	Course Co-Director

## Awards and Honors

November 1999	Orange Environment Citizens Action Group, OE Award for Excellence in Translating Science to the Public
December 2000	NYU School of Medicine Dean's Research Incentive Award
October 2012	Recipient of the "Haagen Smit Prize" for Best Paper, <u>Atmospheric Environment</u> . <a href="http://geo.arc.nasa.gov/sgg/singh/winners12.html">http://geo.arc.nasa.gov/sgg/singh/winners12.html</a>
March 2013	Recipient of the "Best Paper of the Year – Science" Award from <u>ES&amp;T</u> <a href="http://pubs.acs.org/doi/full/10.1021/es400924t">http://pubs.acs.org/doi/full/10.1021/es400924t</a>
May 2018	Recipient of the "Public Service Award" from the American Thoracic Society <a href="https://conference.thoracic.org/about/conference-history/public-service.php">https://conference.thoracic.org/about/conference-history/public-service.php</a>

## Major Committee Assignments

### New York University Committees

2007-present:	University Sustainability Task Force
2010-2012:	University Faculty Senate Alternate

2012-2016: University Faculty Senator

NYU School of Medicine Departmental Committees

1992-1998: Sterling Forest Library Committee, Member, NYU SOM Dept of Environ. Medicine  
1991-1994 Health & Safety Committee, Member, NYU SOM Dept. of Environ.. Medicine  
1992-2004 Community Outreach and Education Comm., Chairman, NYSOM Dept. of Environ. Med.  
1999-2004 Dept. Chairman's Internal Advisory Comm., Member, NYUSOM Dept. of Environ. Med.  
2005-present Dept. Academic Steering Committee, Member, NYUSOM Dept. of Environ. Medicine  
2007-present Dept. Appointments & Promotions Comm., Member, NYUSOM, Dept. of Environ. Medicine  
2012-2016 Dept. Appointments & Promotions Comm., Chair, NYUSOM, Dept. of Environ. Medicine

Advisory Committees

Regional

1983-1984 Massachusetts Acid Rain Advisory Board, Member, Mass. Dept. of Env. Protection  
1984-1986 Committee on Environ. And Occup. Health. , NY State American Lung Association  
1991-1996 Air Management Advisory Comm., Member of Health Effects Subcom., NY State DEC  
1995-1999 Engineering Advisory Board, Member, Tuxedo, NY  
1997-1998 Advisory Committee to the Mayor on the Port of Newburgh, Member, Newburgh, NY  
1996-1999 CUES Asthma Working Group, Member, New York Academy of Medicine  
2008-2010 New York City Community Air Study (NYCCAS) Advisory Panel

National

1995-1999 Comm. on Health Effects of Waste Incineration, Member, National Academy of Sciences  
1995-1999 National Air Conservation Commission, Member, American Lung Association  
2000-2004 National Action Panel on Environment, Member, American Lung Association  
2005-present National Clean Air Committee, Member, American Lung Association  
2007-2010 U.S. EPA Clean Air Science Advisory Committee (CASAC) for SO<sub>x</sub> and NO<sub>x</sub>  
Mar. 2012 EPA Panelist for "Kickoff Workshop to Inform EPA's Review of the Primary NO<sub>2</sub> NAAQS"

International

1996-1997 Sulfur in Gasoline Health and Environment Panel, Chairperson, Health Canada  
Sept. 2007 Illness Cost of Air Pollution Expert Committee, Canadian Medical Association  
2008-2012 Global Burden of Disease (GBD), Committee on the Human Health Effects of Outdoor Air Pollution, World Health Organization (WHO)

Grant Review Committees (National)

March 1989 EPA Air Chemistry and Physics Extramural Grants Review Panel (*ad hoc member*)  
Oct. 1989 NIEHS P30 Center Special Review Panel (*ad hoc member*)  
July 1992 NIH R01 Epidemiology & Disease Control Study Section (*ad hoc member*)  
Nov. 1992 NIEHS P20 Center Development Grant Special Study Section, (*ad hoc member*)  
June 1996 EPA Special Review Panel of the Health Effects Institute (HEI) (*ad hoc member*)  
March 1997 EPA Office of Res. and Development External Grant Review Panel (*ad hoc member*)  
April 1997 NIEHS Community-Based Participatory Res. R01 Special Study Sect. (*ad hoc member*)  
July 1997 EPA National Environ. Research Lab Intramural Research Review Panel (*ad hoc member*)  
June 1998 EPA Office of Res. and Development External Grant Review Panel (*ad hoc member*)  
July 1998 EPA Climate Policy and Programs Division Grant Application Review (*ad hoc member*)  
Oct. 1998 Mickey Leland Center for Air Toxics Grant Review Panel (*ad hoc member*)  
April 2000 NIEHS P30 Center Special Review Panel (*ad hoc member*)  
July 2001 NIEHS Community-Based Participatory Res. R01 Special Study Sect. (*ad hoc member*)  
Dec. 2001 NIEHS Program Project P01 Site Visit Review Panel (*ad hoc member*)  
April 2003 NIH R21 Fogarty Health, Env. and Economic Development Study Sect. (*ad hoc member*)  
Nov. 2003 U.S. EPA STAR Grant Panel (Epidemiologic Research on Health Effects of Long-Term Exposure to Ambient Particulate Matter and Other Air Pollutants) (*member*)  
October 2004 NIEHS Program Project P01 Review Panel (*ad hoc member*)  
June 2005 NIH Special Emphasis Panel (ZRG1 HOP Q 90 S) (*ad hoc member*)

Nov. 2005	NIH Infectious Disease, Reproductive Health, Asthma/Allergy, and Pulmonary (IRAP) Conditions Study Section Review Panel ( <i>ad hoc member</i> )
Feb. 2006	NIH Infectious Disease, Reproductive Health, Asthma/Allergy, and Pulmonary (IRAP) Conditions Study Section Review Panel ( <i>ad hoc member</i> )
June 2006	NIH Infectious Disease, Reproductive Health, Asthma/Allergy, and Pulmonary (IRAP) Conditions Study Section Review Panel ( <i>ad hoc member</i> )
Dec. 2006	NIEHS Special Emphasis Panel on Genetics, Air Pollution, and Respiratory Effects (ZES1 TN-E FG P) ( <i>member</i> )
Nov. 2007	NIH Special Emphasis Panel on Community Participation in Research (ZRG1 HOP-S) ( <i>member</i> )
June 2009	NIH Study Section Review Panel on Challenge Grants in Health & Science Research
March 2011	U.S. EPA Science to Achieve Results (STAR) Graduate Fellowship Review Panel – Clean Air Panel ( <i>chair</i> )
Sept. 2011	NIH Special Epidemiology Study Section (ZRG1 PSE K 02 M) ( <i>member</i> )
Oct. 2012	NIH Cardiac and Sleep Epidemiology (CASE) Study Section ( <i>ad hoc member</i> )
June 2013	NIH Special NHLBI Dataset Study Section (ZRG1 PSEQ 56) ( <i>member</i> )
July 2013	NIH “Career Awards” Study Section (ZES1 LWJ-D, K9) ( <i>member</i> )
Sept. 2013-15	Permanent Member, NIH Cardiac and Sleep Epidemiology Study Section (CASE) Study Section
Sept. 2015-17	Permanent Member, NIH Cancer, Heart, and Sleep Epidemiology Study Section (CHSE) Study Section
Nov. 2016	NIEHS R13 Study Section ( <i>member</i> )
Nov, 2018	NHLBI U01 New Epidemiology Cohort Studies in Heart, Lung, Blood and Sleep Diseases and Disorders Study Section

#### Memberships, Offices, And Committee Assignments in Professional Societies

<i>Year</i>	<i>Society/Committees</i>
1980-1996	Air and Waste Management Association (Comm. on Health Effects and Exposure,)
1992-Present	American Thoracic Society (ATS): Environmental and Occup. Health (EOH) Assembly, 1995-1999, 2012-2013: ATS EOH Long Range Planning Committee; 1993-1994, 2002-2004: ATS Program Committee 2006-2007 Chairman of the ATS-EOH Nominating Committee 2010-2018: ATS Environmental Health Policy Committee, member 2012-2014: ATS Environmental Health Policy Committee, Vice-Chairman 2015-2018: ATS Environmental Health Policy Committee, Chairman
1990-present	International Society of Exposure Science
1992-present	International Society for Environmental Epidemiology (Annual Meeting Program Committee: 1998, 2000, 2003, 2004, 2006, 2016) (ISEE Conference Planning Committee: 2006-present)
2007-2009	New York Academy of Sciences (membership given in appreciation for a 1/23/07 NYAS forum presentation)
2017-present	American Public Health Association (APHA)

#### **Editorial Positions**

##### Journal Board Membership

<i>Year</i>	<i>Name of Board</i>
1993-2008	International Society of Exposure Analysis (J. of Exp. Anal. and Environ. Epid.)
2017-present	Environmental Health Perspectives (EHP) Editorial Review Board

Ad Hoc Manuscript Reviewer

<i>Years</i>	<i>Journal</i>
1996-1998	American Journal of Epidemiology
1994	Archives of Environmental Health
1995-present	Atmospheric Environment
1995-present	Environmental Health Perspectives
1994-present	Environmental Research
2004-present	Environmental Science and Technology
2011-present	Epidemiology
1993-present	Journal of Exposure Analysis and Environmental Epidemiology
1994-present	Journal of the Air and Waste Management Association
1996-present	Journal of the American Medical Association
1997-present	Journal of Occupational and Environmental Medicine
1997-present	Journal of Respiratory and Critical Care Medicine
2013-present	Nature: Climate Change
2006-present	Thorax

Scientific Report Reviewer

August, 1986	Reviewer for the National Academy of Sciences, Board on Environmental Studies and Toxicology report “The Airliner Cabin Environment: Air Quality and Safety”
October, 2002	Reviewer for the NAS, Board on Environmental Studies and Toxicology report “Estimating the Public Health Benefits of Proposed Air Pollution Regulations”

**Mentoring of Graduate Students, Residents, Post-Doctoral Fellows in Research**

Under direct supervision:

<i>Student Name</i>	<i>Type of Position</i>	<i>Time Period</i>	<i>Present Position</i>
Mark Ostapczuk	Masters	1984-1986	Industrial Hyg., Barr Labs, Pomona, NJ
Kazuhiko Ito	Masters/Doctoral	1984-1990	Scientist, NYC Dept. of Health, NYC, NY
Peter Jaques	Masters/Doctoral	1988-1998	Assoc. Prof., Clarkson Univ., Potsdam, NY
R. Charon Gwynn	Masters/Doctoral	1992-1999	Epidemiologist, Columbia Univ., NY
Ramona Lall	Masters/Doctoral	2000-2007	Research Sci. IV, NYC Dept. of Health, NY
Ariel Spira-Cohen	Masters/Doctoral	2003-2009	Research Sci. III, NYC Dept. of Health, NY
Kevin Cromar	Masters/Doctoral	2008-2012	Assistant Professor, NYU School Of Medicine
Lital Yinon	Doctoral	2011-2015	Self-Employed
Chris Lim	Doctoral	2012-2018	Post-Doc, Yale University
Mostafijur Rahman	Doctoral	2016-present	Doctoral Candidate, NYU School of Medicine

In advisory function (thesis committee):

<i>Student Name</i>	<i>Advisory Role</i>	<i>Time Period</i>	<i>Student's Supervisor</i>
Shao-Keng Liang	Doctoral Committee member	1990-1994	Dr. J. Waldman, UMDNJ, Rutgers
Jerry Formisano	Doctoral Committee member	1997-2000	Dr. M. Lippmann, NYU SOM
Yair Hazi	Doctoral Committee member	1993-2001	Dr. B. Cohen, NYU SOM
Samantha Deleon	Doctoral Committee member	1997-2003	Dr. K Ito, NYU SOM
Chun Yi Wu	Doctoral Committee member	2000-2004	Dr. L.C. Chen, NYU SOM
Carlos Restrepo	Doctoral Committee member	2002-2004	Dr. R. Zimmerman, Wagner, NYU
Shaou-I Hsu	Doctoral Committee member	2000-2009	Dr. M. Lippmann, NYU-SOM
Steven Schauer	Doctoral Committee member	2007-2009	Dr. B. Cohen, NYU-SOM
Christine Ekenga	Doctoral Committee Chair	2009-2011	Dr. G. Friedman-Jimenez, NYU-SOM

Rebecca Gluskin	Doctoral Committee Chair	2009-2012	Dr. Kazuhiko Ito, NYU SOM
Jiang Zhou	Doctoral Committee Chair	2008-2012	Dr. Kazuhiko Ito, NYU SOM
Eric Saunders	Doctoral Committee Chair	2012-2016	Dr. Terry Gordon, NYU SOM
Ruzmyn Vilcassim	Doctoral Committee Chair	2012-2018	Dr. Terry Gordon, NYU SOM

### **Teaching Awards Received**

N/A

### **Major Research Interests**

- 1) Air Pollution Epidemiology: Real-world air pollution exposures and human health effects in the general population and study cohorts of suspected susceptible individuals (e.g., children).
- 2) Aerosol Science: Ambient particulate matter aerosol exposures, including designing and implementing air monitoring equipment to collect human exposures to air pollution.
- 3) Environmental Exposure Assessment: Methods to assess human exposures and health effects from air pollution, especially the development of source apportionment models to separate human effects on the basis of pollution source. Design of epidemiological models/methods that better incorporate potential air pollution confounders/effect modifiers (e.g. weather and genetic influences).

### **Patents**

None

### **Boards and Community Organizations**

1990-1995	St. Mary's Episcopal Church, Tuxedo, NY, Vestry member
1992-2008	Monroe-Woodbury Soccer Club, Coach (Board Member: 1999-2000)
1994-1999	Orange County Citizen's Foundation, Member
1999-2009	Y2CARE Monroe-Woodbury, NY School District Residents Action Group, Founder
2005-present	St. Mary's Episcopal Church, Tuxedo, NY, Community Outreach Committee, Member
2006-present	EPISCOBUILD-Newburgh, NY Habitat for Humanity Advisory Board, Member
2012-2018	St. Mary's Episcopal Church, Tuxedo, NY, Vestry member

### **Military Service**

None

### **International Scientific Meetings Organized**

May 28-30, 2003	"Workshop on the Source Apportionment of PM Health Effects." U.S. EPA PM Centers, Harriman, NY.
Aug. 1-4, 2004	"Sixteenth Conference of the International Society for Environmental Epidemiology," Kimmel Conference Center, Washington Square, New York University, New York City, NY.

### **Scientific Forums for the Public Organized**

June 2001	"Science and Community Interaction Forum on the Environment." Held at Hostos Community College, Bronx, , New York City, NY.
October 2001	"Forum on Environmental Health Issues Related to the World Trade Center Disaster." Held at NYU Law School, Washington Square, New York City, NY.
October 2002	"2 <sup>nd</sup> Annual Forum on the Environmental Health Issues Related to the World Trade Center Disaster." Held at Manhattan Borough Community College, New York City, NY.
October 2003	"3 <sup>rd</sup> Annual Forum on the Environmental Health Issues Related to the World Trade Center Disaster." Held at NYU Lower Manhattan Campus, New York City, NY.



Sept. 2006 "Let's Clear the Air", South Bronx High School, New York City, NY

**Invited U.S. House and Senate Congressional Testimony**

- Feb. 5, 1997 "Human Health Effects of Ambient Ozone Exposures" Statement before the Committee on Environment and Public Works, Subcommittee On Clean Air, Wetlands, Private Property, And Nuclear Safety, U.S. Senate, Washington, DC.  
<http://epw.senate.gov/105th/thurston.htm>
- April 16, 1997 "Human Health Effects of Ambient Ozone and Particulate Matter Exposures." Statement before the Government Reform and Oversight Committee of the U.S. House of Representatives, Washington, D.C.
- May 8, 1997 "Human Health Effects of Ambient Ozone and Particulate Matter Exposures." Statement before the Subcommittee on Health and Environment, Committee on Commerce of U.S. House of Representatives, Washington, D.C.
- July 29, 1997, "The Human Health Effects of Ambient Ozone and Particulate Matter Air Pollution." Statement before the Subcommittee on Commercial and Administrative Law of the Judiciary Committee of the U.S. House of Representatives, Washington, D.C.  
<http://judiciary.house.gov/legacy/commercial.htm>
- October 22, 1997 "Ozone and Particulate Matter Air Pollution Health Effects." Statement before the U.S. Senate Committee on Environment and Public Works Subcommittee on Clean Air, Wetlands, Private Property, and Nuclear Safety. Washington, DC.  
<http://epw.senate.gov/105th/thursto2.htm>
- July 15, 1999: "The Mandated Release of Government-Funded Research Data." Statement before the Committee On Government Reform, Subcommittee on Government Management, Information And Technology, U.S. House of Representatives
- July 26, 2001 "The Human Health Effects Of Air Pollution From Utility Power Plants." Statement before the Committee on Environment and Public Works, U.S. Senate, Washington, D.C.  
<http://www.c-spanvideo.org/program/PlantE>
- Feb 11, 2002: "The Air Pollution Effects of The World Trade Center Disaster." Statement before the Committee on Environment And Public Works, Subcommittee On Clean Air, Wetlands, And Climate Change. United States Senate, New York, NY.  
<http://www.c-spanvideo.org/program/Qualitya>
- March 5, 2002 "The Use of the Nationwide Registries to Assess Environmental Health Effects." Statement before the Committee On Health, Education, Labor, And Pensions, Subcommittee On Public Health, U.S. Senate, Washington, DC.
- Sept. 3, 2002 "The Clean Air Act and The Human Health Effects of Air Pollution from Utility Power Plants." Statement before the U.S. Senate Committee on Health, Education, Labor, and Pensions, Subcommittee on Public Health, Washington, D.C. <http://www.c-spanvideo.org/program/AirStand>
- April 1, 2004 "The Human Health Benefits Of Meeting the Ambient Ozone And Particulate Matter Air Quality Standards." Statement before the Committee on Environment and Public Works, Subcommittee on Clean Air, Climate Change, and Nuclear Safety, U.S. Senate, Washington, D.C.  
<http://epw.senate.gov/epwmultimedia/epw040104.ram>
- July 19, 2006 "The Science And Risk Assessment Of Particulate Matter (PM) Air Pollution Health Effects." Statement before the Committee on Environment and Public Works, U.S. Senate, Washington, D.C.  
<http://epw.senate.gov/hearingstatements.cfm?id=258766>



May 7, 2008 “Science And Environmental Regulatory Decisions.” Statement before the Committee On Environment And Public Works of The U.S. Senate, Subcommittee on Public Sector Solutions to Global Warming, Oversight, and Children’s Health Protection, U.S. Senate, Washington, D.C.

<http://www.c-spanvideo.org/program/RegulatoryD>

<http://epw.senate.gov/public/index.cfm?FuseAction=Hearings.Hearing&HearingID=a1954f70-802a-23ad-4192-fc2995dda7f4>

October 4, 2011 “The Science of Air Pollution Health Effects and the Role of CASAC in EPA Standard Setting” Statement before the Subcommittee on Energy and the Environment, Committee on Science, Space and Technology, U.S. House Of Representatives, Washington, DC.

<http://science.house.gov/hearing/energy-and-environment-subcommittee---hearing-quality-science-quality-air>

## **Other Invited Presentations**

### Regional Presentations

April 21, 1993 “Summertime Smog and Hospital Admissions for Respiratory Illness”, Environmental and Occupational Health Sciences Institute Seminar Series Lecture, UMDNJ-Robert Wood Johnson Medical School, Piscataway, NJ.

Dec .14, 1995 “Health Effects of Acidic Aerosols”, NY State Dept. of Health, Wadsworth Center Seminar, Albany, NY

Jan. 18, 1996 “Outdoor Air Pollution and Asthma in Children “ American Lung Association Press Briefing, New York, NY.

June 1, 1996 “Asthma and Urban Air Pollution”, WHEACT, Harlem Hospital, New York, NY.

July17, 1996 “Asthma and Outdoor Air Pollution”, Making the Connection: Urban Air Toxics & Public Health. Northeast States for Coordinated Air Use Management (NESCAUM), Roxbury, MA

Feb. 11, 1997 “Outdoor Air Pollution and Asthma”,\_Bellevue Hospital Asthma Clinic *Grand Rounds*. New York City, NY.

Feb. 26, 1998 “Scientific Research for Ozone and Fine Particulate Standards “, Pace University School of Law, White Plains, NY

Nov. 30, 1998 “Outdoor Air Pollution and Asthma”, Center for Urban and Environmental Studies (CUES), NY Academy of Medicine,, New York, NY

Feb. 22, 1999 “Asthma and Air Pollution”, Cornell University, Ithaca, NY

April 28, 2001 “Asthma and Air Pollution in New York City”, NYC Council Environmental Candidate School, NY League of Conservation Voters, New York, NY.

Nov. 1, 2001 “Air Quality and Environmental Impacts Due to the World Trade Center Disaster”, Testimony before the Comm. on Environ. Protection, NYC Council, New York, NY.

Nov. 13, 2001 “WTC Pollution Impacts in Lower Manhattan”, Stuyvesant High School Parents Association General Meeting, Stuyvesant High School, New York, NY

Feb. 28, 2002 “Lung Cancer Effects of Long-Term Exposure to Ambient Fine Particulate Matter”, Mailman School of Public Health, Columbia University, New York, NY.

April 5, 2002 “Air Pollution Impacts of the WTC Disaster”, 23rd Annual Scientific Conference of the NY/NJ Education and Research Center: "Worker Health and Safety: Lessons Learned in the Aftermath of Sept. 11, 2001," Mt. Sinai School of Medicine, NYC, NY

April 21, 2002 “Adverse Health Effects of Power Plant Air Pollution on Children” Earth Day 2002, 14<sup>th</sup> Street Y, New York City, NY.

May 23, 2002 “Human Health Effects of Power Plant Pollution”, Rockland County Conservation Association, Suffern, NY

- May 31, 2002 “Environmental Health Impacts of the World Trade Center Disaster”, University of Rochester Medical School, Rochester, NY.
- Sept. 19, 2002 “Community Air Pollution Related to the World Trade Center Disaster”. NYC Council Forum: The Environmental Health Consequences of 9/11: Where Do We Stand One Year Later? Borough of Manhattan Community College, New York City, NY.
- Oct. 3, 2002 “Community Exposures to Particulate Matter Air Pollution from the World Trade Center Disaster”, Mount Sinai School of Medicine *Grand Rounds*, New York City, NY.
- April 11, 2003 “Environmental Impacts of the World Trade Center Disaster”, NIEHS Public Interest Liaison Group, New York City, NY.
- April 21, 2003 “Asthma and Air Pollution”, Airborne Threats to Human Health, NIEHS Town Hall Meeting, Syracuse, NY.
- May 7, 2003 “Asthma and Air Pollution in NY City” Environmental Candidate School for New York City Council Candidates, Wagner School, NYU, New York City, NY.
- July 21, 2003 “Health Effects of Particulate Matter Air Pollution”, Ozone Transport Commission, Philadelphia, PA.
- Nov. 18, 2004 “Ambient Air Pollution Particulate Matter (PM): Sources and Health Impacts”. U.S. Environmental Protection Agency, Region 2, New York City, NY.
- Feb. 17, 2005 “Community Air Pollution Aspects Of The Demolition Of 9-11 Contaminated Buildings”. Testimony before the Committee on Lower Manhattan Redevelopment, New York City Council, New York City, NY.
- Oct. 19, 2005 Air Pollution Health Effects: Consideration of Mixtures. Fall Meeting of the Mid-Atlantic Chapter of the Society of Toxicology (MASOT), East Brunswick, NJ.
- Dec.7, 2006 Asthma and Air Pollution Effects in the South Bronx. New York City Child Health Forum, The Children’s Health Fund, Harlem, NYC, NY.
- Jan. 18, 2007 Air Pollution Effects in New York City. NYU Environmental Sciences Seminar Lecture, Washington Square, NYC, NY.
- Jan. 23, 2007 The South Bronx Backpack Study: Asthma and Air Pollution in NYC. Presented at the forum "High Asthma Rates in the Bronx: What Science Now Knows and Needs to Learn." New York Academy of Sciences, 7 World Trade Center, NYC, NY.
- Oct. 2, 2009 “Diesel Air Pollution and Asthma in New York City”. Brown Superfund Research Program, Brown University, Providence, RI.
- June 19, 2012 “The Backpack Study of Asthma and Diesel Air Pollution in the South Bronx”. Region 1 U.S. EPA, Citizen Science Workshop, New York City, NY.

#### National Presentations

- Oct. 20, 1987. NIEHS Symposium on the Health Effects of Acid Aerosols: “Re-examination of London, England, Mortality in Relation to Exposure to Acidic Aerosols During 1963-1972 Winters” RTP, NC.
- Aug. 13, 1991 “Kuwait Mortality Risks from SO<sub>2</sub> and Particles: Insights from the London Fogs” The Kuwait Oil Fires Conf., American Academy of Arts and Sciences, Cambridge, MA.
- Jan. 24, 1994 “Air Pollution Epidemiology: Is the Model the Message?” The First Colloquium on Particulate Air Pollution and Human Morbidity and Mortality”. Beckman Center of the NAS, Irvine, CA.
- May 23, 1994 “Ozone Epidemiological and Field Studies”. American Thoracic Society Annual Meeting, Boston, MA.
- May 25, 1994 “Epidemiological Evidence Linking Outdoor Air Pollution and Increased Hospital Admissions for Respiratory Ailments” American Thoracic Society Annual Meeting, Boston, MA.

May 6, 1996	<u>"Associations Between PM<sub>10</sub> &amp; Mortality in Multiple US Cities"</u> . Second Colloquium on Particulate Air Pollution and Health. Park City, Utah.
Sept. 5, 1996	<u>"Particulate Matter Exposure Issues for Epidemiology"</u> U.S. EPA Particulate Matter Workshop, RTP, NC
April 3, 1997	<u>"Health Effects of Ambient Ozone &amp; Particulate Matter"</u> Air and Waste Assoc. Regional Conference On Impacts of EPA's Proposed Changes to Ozone and PM Standards, Oak Brook, IL
April 22, 1998	<u>"The New EPA Standards for Ambient PM and Ozone"</u> American Lung Association Annual Meeting, Chicago, IL.
Dec. 21, 1999	<u>"Global Overview of Human Death and Illness due to Air Pollution"</u> . California Air Resources, Sacramento, CA.
March 24, 2000	<u>"Estimating Ancillary Impacts, Benefits and Costs Of Proposed GHG Mitigation Policies For Public Health"</u> Resources for the Future, Wash., DC.
June 24, 2002	<u>"Investigations Into the Environmental Health Impacts Related to the WTC Disaster"</u> Air And Waste Management Annual Meeting, Baltimore, MD.
July 15, 2002	<u>"Air Pollution and Human Health"</u> NIEHS Built Environment Conference, RTP, NC
July 26, 2002	<u>"The Human Health Effects of Power Plant Emissions and Associated Air Pollution"</u> , The Environment & Health Forum, Physicians for Social Responsibility, Washington, DC.
October 7, 2002	<u>"Community Exposures to Particulate Matter Air Pollution from the World Trade Center Disaster"</u> Plenary Speaker at the American Association for Aerosol Research, Charlottesville, North Carolina.
Nov. 11, 2002	<u>"Characterization of Community Exposures to World Trade Center Disaster Airborne and Settled Dust Particulate Matter Air Pollution"</u> , American Public Health Association Annual Meeting, Philadelphia, PA.
Dec. 5, 2002	<u>"Susceptibility of Older Adults to Air Pollution"</u> , EPA Workshop on Differential Susceptibility of Older People to Environmental Hazards. National Academy of Sciences, Washington, DC.
Feb. 3, 2003	<u>"Health Effects of Particulate Matter Air Pollution"</u> , National Air Quality Conference, U.S. EPA, San Antonio, Texas
May 17, 2003	<u>"Assessing the Influence of Particle Sources and Characteristics on Adverse Health Effects of PM"</u> , PG18 - New Tools to Evaluate the Health Effects of Air Pollution in Epidemiologic Studies. American Thoracic Society Annual Meeting, Seattle, WA.
Sep. 10, 2003	<u>"Nature and impact of World Trade Center Disaster fine particulate matter air pollution at a site in Lower Manhattan after September 11."</u> Annual Meeting of the American Chemical Society, New York, NY.
October 20, 2003	<u>"Translating Air Pollution Risks to the Community"</u> Annual Meeting of the NIEHS Center Directors, Baltimore, MD.
May 18, 2004	<u>"The Health Imperative for Implementation of the Clean Air Act"</u> State and Territorial Air Pollution Program Administrators/ Association of Local Air Pollution Control Officials (STAPPA/ALAPCO) National Conference, Point Clear, Alabama.
Oct. 18, 2004	<u>"NIEHS Centers' Investigations of the World Trade Center Collapse Pollution Exposures and Effects: A Public Health Collaboration"</u> National Institute of Environmental Health Sciences Center Directors' Meeting, Research Triangle Park, NC.
May 25, 2005	<u>"Human Health Effects Associated with Sulfate Aerosols"</u> , American Thoracic Society Annual Meeting, San Diego, CA
Oct. 24, 2005	<u>"The Science Behind the Particulate Matter (PM) Standards"</u> State and Territorial Air Pollution Program Administrators/ Association of Local Air Pollution Control Officials (STAPPA/ALAPCO) National Conference, Alexandria, Virginia.

- Oct. 14, 2008 “Diesel Air Pollution and Asthma Exacerbations in a Group of Children with Asthma” Annual Meeting of the International Society for Environmental Epidemiology (ISEE). Pasadena, California.
- Feb. 26, 2010 “What studies are appropriate to use to estimate health impacts from specific sources such as diesel PM?” CARB Symposium: “*Estimating Premature Deaths from Long-term Exposure to PM<sub>2.5</sub>*”. Sacramento, CA.
- May 6, 2011 “Lung Cancer Risks from Exposure to Fine Particle Air Pollution” NYU Cancer Institute Symposium: “Cancer and the Environment”, NYC, NY.
- May 16, 2012 “The Human Health Effects of Air Pollution” The Air We Breathe: Regional Summit on Asthma and Environment at Allegheny General Hospital, Pittsburgh, PA.
- June 20, 2013 “Particles in our Air: A Global Health Risk”, Northeastern University, Research Seminar. Boston, MA.
- Mar 5, 2015 “Air Pollution, Climate Change and Health”. Stegner Institute Air Quality Symposium, Salt Lake City, Utah.
- Apr 22, 2017 “The Clean Air Health Benefits of Climate Mitigation Action”, Yale University, Global Health & Innovation Conference, New Haven, CT.

International Presentations

- May 1, 1987 “Acid Aerosols: Their Origins, Occurrence, and Possible Health Effects”, Canadian Environmental Health Directorate Seminar, Health and Welfare Canada, Ottawa, Canada
- July 2, 1987 “Health Effects of Air Pollution in the US”, University of Sao Paulo, Sao Paulo, Brasil
- Feb. 5, 1991 “Results from the Analysis of Toronto Summer Sulfate and Aerosol and Acidity Data”, Workshop on Current Use and Future Directions of Hospital-Based Data in the Assessment of the Effects of Ambient Air Pollution on Human Health. Health and Welfare Canada, Ottawa, Canada.
- April 23, 1997 “An Evaluation of the Role of Acid Aerosols in Particulate Matter Health Effects”, Conference on the Health Effects of Particulate Matter in Ambient Air. Air & Waste Management Association, Prague, Czech Republic.
- May 12, 1998 “The Health Effects of PM and Ozone Air Pollution”, Air Pollution: Effects on Ontario’s Health and Environment. Ontario Medical Association, Toronto, Canada
- Nov. 1, 1999 “Climate Change and the Health Impacts of Air Pollution”. The Public Health Opportunities and Hazards of Global Warming Workshop at the U.N. Framework Convention on Climate Change, Conference of Parties (COP5), Bonn, Germany.
- August 31, 2000 “Particulate Matter Air Pollution and Health in three Northeastern Cities”, World Congress on Lung Health, Florence, Italy
- January 29, 2001 “PM Exposure Assessment and Epidemiology”, NERAM International Colloquia: Health and Air Quality: Interpreting Science for Decision Makers. Ottawa, Canada.
- Feb. 4-5, 2002: “Air Pollution Exposure Assessment Approaches in U.S. Long-Term Health Studies”, Workshop on Exposure Assessment in Studies on the Chronic Effects of Long-term Exposure to Air Pollution, World Health Organization, Bonn, Germany
- May 2, 2002 “Health Effects of Sulfate Air Pollution” Air Pollution as a Climate Forcing Workshop, East-West Center, Honolulu, Hawaii
- Sept. 24, 2003 “Identification and Characterization of World Trade Center Disaster Fine Particulate Matter Air Pollution at a Site in Lower Manhattan Following September 11.” Annual Meeting of the International Society for Environmental Epidemiology (ISEE). Perth, Australia.
- Dec. 1, 2003 “Terrorism and the Pulmonary Effects of the World Trade Center Disaster Particulate Matter Air Pollution”, British Thoracic Society, London, England.
- Aug. 3, 2004 “A Study of Traffic-Related Pm Exposures And Health Effects Among South Bronx Children With Asthma”. Annual Meeting of the International Society for Environmental Epidemiology (ISEE). New York, NY.

- Sept 14, 2005 “Results And Implications of The Workshop on the Source Apportionment of PM Health Effects”, Annual Meeting of the International Society for Environmental Epidemiology (ISEE). Johannesburg, South Africa.
- Sept. 4, 2006 “A Source Apportionment of U.S. Fine Particulate Matter Pollution for Health Effects Analysis”, Annual Meeting of the International Society for Environmental Epidemiology (ISEE). Paris, France.
- Sept. 4, 2007 “Applying Attributable Risk Methods to Identify Susceptible Subpopulations”, Annual Meeting of the International Society for Environmental Epidemiology (ISEE). Mexico City, Mexico.
- Aug. 27, 2009 “Ischemic Heart Disease Mortality Associations with Long-Term Exposure to PM<sub>2.5</sub> Components”, Annual Meeting of the International Society for Environmental Epidemiology (ISEE). Dublin, Ireland.
- Dec. 1, 2010 "The Hidden Air Quality Health Benefits of Climate Change Mitigation". The Energy and Resources Institute (TERI), Lodhi Road, New Delhi, India.
- July 17, 2012 “Recent Findings on the Mechanisms and Health Risks of Particulate Matter Air Pollution”, European Centre for Environment & Human Health, Truro, England.
- Aug. 29, 2012 “Health Effects of PM Components: NYU NPACT Epidemiology Results and their Integration with Toxicology Results”, Annual Meeting of the International Society for Environmental Epidemiology (ISEE). Columbia, SC.
- May 20, 2013 “Long-term PM<sub>2.5</sub> Exposure and Mortality in the NIH-AARP Cohort”, Annual Meeting of the American Thoracic Society (ATS). Philadelphia, PA.
- Oct. 27, 2013 “Human Health Effects and Global Implications of Particle Air Pollution”, Center of Excellence in Exposure Science and Environ. Health, Technion University, Haifa, Israel.
- May 17, 2015 “Human Health Co-Benefits of Climate Change Mitigation Measures” in the Environment, Global Climate Change And Cardiopulmonary Health session of the American Thoracic Society (ATS) Annual Meeting in Denver, CO, USA.
- Jan. 21, 2016 “Particle Air Pollution: Its Adverse Human Health Effects and Potential Climate Mitigation Health Co-Benefits”. Imperial College. London, England.
- Sep. 1, 2016 “Air Quality Health Co-benefits from Climate Change Mitigation Measures”. 2016 Annual Meeting of the International Society for Environmental Epidemiology (ISEE). Rome, Italy.
- Feb. 12, 2017 “Human Health Effects and Global Implications of Particle Air Pollution”. MASDAR Institute. Abu Dhabi, United Arab Republic.
- Apr. 22, 2017 “Clean Air Health Benefits from Climate Change Mitigation Action”. Global Health & Innovation Conference. Yale University, New Haven, CT.
- May 22, 2017 “Air Pollution Health Effects of Energy Sources: Which Are the Most Toxic?” American Thoracic Society (ATS) Annual Meeting in Washington, DC. USA.
- Oct 29, 2018` “Health effects of dust, species and components of PM”, Workshop on Evaluating the short-term health effects of desert and anthropogenic dust. At the First WHO Global Conference on Air Pollution and Health / Improving Air Quality, Combatting Climate Change – Saving Lives. World Health Organization, Geneva, Switzerland.
- March 8, 2019 “Human Health Effects of Particulate Matter in Bangladesh, and Implications Regarding Biomass Combustion” Kings College, London, England
- May 21, 2019 “Policies That Protect Vulnerable Populations The Role of the EPA in the Current Climate” American Thoracic Society (ATS) Annual Meeting in Dallas, TX. USA.

#### **Scientific Meeting Sessions Chaired**

- May 1, 1996 “Epidemiological Findings”, 2<sup>nd</sup> Colloquium on Particulate Air Pollution & Health. Park City, UT.
- May 14, 1996 “Particulate Toxicity”, American Thoracic Society Annual Meeting, New Orleans, LA.

Jan. 30, 1998 “Evaluation of PM Measurement Methods”. PM<sub>2.5</sub>: A Fine Particulate Standard Specialty Conference. Los Angeles, CA.

August 18, 1998 “Communities and Airports: How to Co-Exist?”, Annual Meeting of the International Society for Environmental Epidemiology (ISEE). Boston, MA.

April 28, 1998 “Clean Air Act Update”, American Thoracic Society Annual Meeting, Chicago, IL.

Oct. 21, 1998 “Health Effects and Regulatory Issues in PM”, Particulate Methodology Workshop,. U.S. EPA Center, for Statistics and the Env., Univ. of Washington, Seattle, WA.

April 26, 1999 “Pulmonary Smoking and Air Pollution Epidemiology.” American Thoracic Society Annual Meeting, San Diego, CA

Sept. 6, 1999 “Personal exposures to Gases and Particles”, Annual Conference of the International Society for Environmental Epidemiology (ISEE), Athens, Greece.

March 31, 2000 “Epidemiology: Particles, Co-pollutants & Morbidity and Mortality”, Workshop on Inhaled Environmental/Occupational Irritants and Allergens: Mechanisms of Cardiovascular Responses, American Thoracic Society, Scottsdale, AZ

Jan. 26, 2000 “Epidemiology of Particulate Matter Air Pollution”, PM2000 Specialty Conference, Air & Waste Management Assoc., Charleston, SC

May 8, 2000 “Outdoor Air Pollution: Epidemiologic Studies”, American Thoracic Society Annual Meeting, Toronto, Canada

Sept. 5, 2001 “Mortality Epidemiology Studies”, Annual Meeting of the International Society for Environmental Epidemiology (ISEE). Garmisch, Germany.

May 20, 2002 “After September 11: Bio-terrorism and The Environmental Health Aftermath of The World Trade Center Disaster”, Plenary Session. American Thoracic Society Annual Meeting, Atlanta, GA.

April 1, 2003 “Epidemiology: Short-Term and Long-Term Health Effects”, Conference on Particulate Matter: Atmospheric Sciences, Exposure, and the Fourth Colloquium on PM and Human Health, Pittsburgh, PA

May 19, 2003 “Particulate Air Pollution and Diseases in Adults”, American Thoracic Society Annual Meeting, Seattle, WA.

May 21, 2003 “Air Pollution as a Cause of Childhood Asthma and Chronic Airway Disease”, American Thoracic Society Annual Meeting, Seattle, WA.

Sept. 2003 “Unexplained Medical Symptoms”, Annual Meeting of the International Society for Environmental Epidemiology (ISEE). Perth, Australia.

Sept. 25, 2005 “Technology and Health”, Annual Meeting of the International Society for Environmental Epidemiology (ISEE). Johannesburg, South Africa.

June 22, 2006 “Characteristics of PM and Related Considerations”, Annual Meeting of the Air and Waste Management Association, New Orleans, LA.

Sept. 3, 2006 “Air Pollution Mechanisms”, Annual Meeting of the International Society for Environmental Epidemiology (ISEE). Paris, France.

Sept. 20, 2006 “Linkage and Analysis of Air Quality and Health Data”, EPA & CDC Symposium on Air Pollution Exposure and Health, RTP, NC

Sept. 5, 2007 “Radiation Exposures and Health Risks”, 2007 Annual Meeting of the International Society for Environmental Epidemiology (ISEE). Mexico City, Mexico

Aug. 26, 2009 “Exploring the Range of Methodological Approaches Available for Environmental Epidemiology.” 2009 Annual Meeting of the International Society for Environmental Epidemiology (ISEE). Dublin, Ireland

March 23, 2010 “Exposure to and Health Effects of Traffic Pollution”, 2010 American Association for Aerosol Research Conference on Air Pollution and Health, San Diego, CA.

Sept. 16, 2011 “Susceptibility to Air Pollution”, 2011 Annual Meeting of the International Society for Environmental Epidemiology (ISEE). Barcelona, Spain.

- Aug. 27, 2012 “Source Apportionment Of Outdoor Air Pollution: Searching For Culprits”. 2012 Annual Meeting of the International Society for Environmental Epidemiology (ISEE). Columbia, SC.
- Aug. 21, 2013 “Source-specific health effects of air pollution”. 2013 Annual Meeting of the International Society for Environmental Epidemiology (ISEE). Basel, Switzerland.
- May 19, 2015 “Indoor and outdoor pollution: epidemiology and mechanisms”. 2015 Annual Meeting of the American Thoracic Society (ATS). Denver, CO, USA.
- Sep. 1, 2016 “Climate Change, Mitigation Measures and Co-Benefits”. 2016 Annual Meeting of the International Society for Environmental Epidemiology (ISEE). Rome, Italy.
- May 22, 2017 “Realizing the Paris Climate Agreement To Improve Cardio-Pulmonary Health: Where Science Meets Policy” American Thoracic Society (ATS) Annual Meeting in Washington, DC. USA.

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### *Book Chapters*

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***The New York Times***

## ***Cleaner Air Brings Drop in Death Rate***

**By NICHOLAS BAKALAR MARCH 21, 2006**

When air pollution in a city declines, the city benefits with a directly proportional drop in death rates, a new study has found.

For each decrease of 1 microgram of soot per cubic meter of air, death rates from cardiovascular disease, respiratory illness and lung cancer decrease by 3 percent -- extending the lives of 75,000 people a year in the United States. The association held even after controlling for smoking and body mass index.

The work, described in a paper in the March 15 issue of *The American Journal of Respiratory and Critical Care Medicine*, was carried out in six metropolitan areas: Watertown, Mass.; Kingston and Harriman, Tenn.; St. Louis; Steubenville, Ohio; Portage, Wyocena and Pardeeville, Wis.; and Topeka, Kan. The participants, ages 25 to 74 at enrollment, were followed from 1974 through 1998.

The scientists periodically measured concentrations of soot, or particulate air pollution, in each city. At the same time, they tracked disease and mortality among 8,096 residents. Particulate air pollution consists of a mixture of liquid and solid particles, mostly a result of fossil fuel combustion and high-temperature industrial processes. By definition, the particles have a diameter less than 2.5 microns, or about one ten-thousandth of an inch.

"For the most part, pollution levels are lower in this country than they were in the 70's and 80's," said Francine Laden, the study's lead author, "and the message here is that if you continue to decrease them, you will save more lives."

Further declines in air pollution are within reach, said Dr. Laden, an assistant professor of environmental epidemiology at Harvard. "The technology is out there," she said. "The cities that we've covered have cleaned up considerably over the course of the study."

In Steubenville, for example, soot declined to 22 micrograms per cubic meter from 27 over the course of the study, and the city had a corresponding 25 percent decrease in mortality risk. "Consistently," Dr. Laden said, "in the cities where there was the most cleanup, there was also the greatest decrease in risk of death."

Dr. Laden said the study supported what the federal scientific advisers had advocated: lowering the air quality standard below the present 15 micrograms per cubic meter. "There was discussion about lowering it to 12," she said, "and this study supports that."

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# American Thoracic Society

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## What Constitutes an Adverse Health Effect of Air Pollution?

THIS OFFICIAL STATEMENT OF THE AMERICAN THORACIC SOCIETY WAS ADOPTED BY THE ATS BOARD OF DIRECTORS, JULY 1999

### PURPOSE OF THE STATEMENT

As the twentieth century ends, the health effects of outdoor air pollution remain a public health concern in developing and developed countries alike. In the United States, the principal pollutants monitored for regulatory purposes (carbon monoxide, nitrogen dioxide, sulfur dioxide, particles, ozone, and lead; *see* Table 1) show general trends of declining concentrations, although ozone pollution now affects many regions of the country besides southern California (1). Yet, even at levels of air pollution now measured in many cities of the United States, associations between air pollution levels and health indicators are being demonstrated at concentrations around those set by standards of the U.S. Environmental Protection Agency (2, 3). In many countries of the developing world, concentrations of air pollutants are rising with industrialization and the increasing numbers of motor vehicles (4, 5). Extremely large and densely populated urban areas, often referred to as "megacities," have the potential to generate unprecedented air quality problems.

There are common principles to air quality management throughout the world. Public health protection unifies all approaches, whether based on voluntary guidelines, mandated standards for concentrations, or source control. The intent is to limit or to avoid any impact of air pollution on the public's health. Air quality management is thus based on a scientific foundation built from the epidemiologic, toxicologic, and clinical evidence on health effects of air pollution. In interpreting this evidence for public health protection, there is a need to identify those effects that are considered "adverse" and to separate them from those effects not considered adverse.

The American Thoracic Society has previously provided guidance on the distinction between adverse and nonadverse health effects of air pollution in its 1985 statement, "Guidelines as to What Constitutes an Adverse Respiratory Health Effect" (6). Definitions of adverse effects have also been offered by the World Health Organization (7–10), but the guidance of the American Thoracic Society has received particular emphasis in the United States. Preparation of the original statement was intended to coincide with consideration of the passage of an amended Clean Air Act and to provide a framework for interpreting scientific evidence relevant to the mandate of the act. In particular, the Clean Air Act requires that the Administrator of the Environmental Protection Agency promulgate, for certain pollutants, standards that will be sufficient to protect against adverse effects of the air pollutants on health. The act is silent on the definition of "adverse effect" and, at the time of the 1985 statement, there was considerable controversy around the interpretation of this language as revi-

sion of the act was being considered. Recognizing the need of policy makers for expert guidance, the American Thoracic Society released the 1985 statement, which to date constitutes the sole set of recommendations on this issue from an expert panel convened by a health organization.

The American Thoracic Society has revised the 1985 statement because new scientific findings, published since the original statement, have again raised questions as to the boundary between adverse and nonadverse in considering health effects of air pollution. These new findings reflect improved sensitivity of research approaches and the application of biomarkers that can detect even subtle perturbations of biologic systems by air pollutants. Epidemiologic research designs have been refined and large sample sizes and increasingly accurate methods for exposure assessment have increased the sensitivity of epidemiologic data for detecting evidence of effects. New statistical approaches and advances in software and hardware have facilitated analyses of large databases of mortality and morbidity information. The design of clinical studies—including controlled exposures of volunteers—has also advanced and biologic specimens may be obtained after exposure, for example, by fiberoptic bronchoscopy, to identify changes in levels of markers of injury. Toxicologic studies have also gained in sophistication through incorporation of more sensitive indicators of effect and the careful tracing of the relationship between exposure and biologically relevant doses to target sites, which may now be considered at a molecular level.

New dimensions have been added to the array of outcome measures. Medical outcomes research now recognizes that patient well-being should be broadly conceptualized and measured rigorously, in addition to considering the biological process of the disease itself. As a result, health-related quality of life, the perception of well-being, is now considered a necessary component of outcomes research. Validated instruments have been developed to assess the impact of health-related symptoms and impairment on functional status and quality of life (11–14). The formalization of the concept of environmental justice acknowledges that the effects of specific pollutants cannot be evaluated in isolation without giving consideration to the overlapping exposures of populations, often minority group members of low socioeconomic status, who live in neighborhoods that are heavily exposed to multiple environmental contaminants (15).

This new statement, like the 1985 statement, is intended to provide guidance to policy makers and others who interpret the scientific evidence on the health effects of air pollution for the purpose of risk management. The statement does not offer strict rules or numerical criteria, but rather proposes principles to be used in weighing the evidence and setting boundaries between adverse and nonadverse health effects. Even if the technical tools were available for scaling the consequences of air pollution on the multiple relevant axes, the placement of dividing lines should be a societal judgment and consequently

TABLE 1  
U.S. NATIONAL AMBIENT AIR QUALITY STANDARDS\*

Pollutant	NAAQS Concentration		Standard Type
	(ppm)	( $\mu\text{g}/\text{m}^3$ )	
Particulate matter $\geq 10 \mu\text{m}$ ( $\text{PM}_{10}$ )			
24-h average		150	Primary and secondary
Annual arithmetic mean		50	Primary and secondary
Particulate matter $\geq 2.5 \mu\text{m}$ ( $\text{PM}_{2.5}$ )			
24-h average		65	Primary and secondary
Annual arithmetic mean		15	Primary and secondary
Ozone ( $\text{O}_3$ )			
24-h average	0.12	235	Primary and secondary
Annual arithmetic mean	0.08	157	Primary and secondary
Sulfur dioxide ( $\text{SO}_2$ )			
24-h average	0.14	365	Primary
Annual arithmetic mean	0.03	80	Primary
3-h average	0.50	1,300	Secondary
Nitrogen dioxide ( $\text{NO}_2$ )			
Annual arithmetic mean	0.053	100	Primary and secondary
Carbon monoxide (CO)			
1-h average	35	40	Primary
8-h average	9	10	Primary
Lead (Pb)			
Quarterly average		1.5	Primary and secondary

\* For detailed information on scientific bases and policy considerations underlying decisions establishing the NAAQS listed here, see the AQCDs, staff papers, and NAAQS Promulgation notices cited in text. Such information can also be obtained from several internet websites (e.g., <http://www.epa.gov/airs/criteria.html>; <http://www.epa.gov/oar/oaqps/publicat.html>; and <http://www.epa.gov/ncea/biblio.htm>).

this committee does not propose specific boundaries for separating adverse from nonadverse effects.

## OVERVIEW OF THE 1985 STATEMENT

The 1985 statement of the American Thoracic Society was directed at respiratory health effects of air pollution and emphasized the interpretation of the epidemiologic evidence. The statement recognized the spectrum of responses to air pollution, which begins with exposure and evidence of exposure and ends at death. This spectrum has been characterized as a pyramid, based in the most common consequence—exposure—and having mortality, the least common and most severe consequence, at its tip. The statement included a table that lists adverse respiratory health effects, seemingly in order of declining severity (Table 2). The 1985 statement hinged the distinction between adverse and nonadverse effects on medical considerations. The committee recognized that the boundary is further influenced by societal considerations: "Where one draws the line to categorize it as an adverse health effect or an action level between pathophysiologic or physiologic change is probably best left to the individual or the community."

The committee's definition of adverse respiratory health effects was "... medically significant physiologic or pathologic changes generally evidenced by one or more of the following: (1) interference with the normal activity of the affected person or persons, (2) episodic respiratory illness, (3) incapacitating illness, (4) permanent respiratory injury, and/or (5) progressive respiratory dysfunction." The committee noted that all changes are not adverse, citing the example of carboxyhemoglobin. The level of carboxyhemoglobin, beyond that from endogenous production, is indicative of exposure but it is not predictive of adverse effects until reaching threshold levels, depending on the effect and the susceptibility of the exposed person. The statement recognized that a distinction should be

TABLE 2  
ADVERSE RESPIRATORY HEALTH EFFECTS

- Increased mortality (*Increased* as used here and subsequently means significantly [ $p < 0.05$ ] increased above that recorded in some standard, comparable population. In selected situation,  $p < 0.1$  may be appropriate)
- Increased incidence of cancer
- Increased frequency of symptomatic asthmatic attacks
- Increased incidence of lower respiratory tract infections
- Increased exacerbations of disease in persons with chronic cardiopulmonary or other disease that could be reflected in a variety of ways
  - Less able to cope with daily activities (i.e., shortness of breath or increased anginal episodes)
  - Increased hospitalization, both frequency and duration
  - Increased emergency ward or physician visits
  - Increased pulmonary medication
  - Decreased pulmonary function
- Reduction in  $\text{FEV}_1$  or FVC associated with clinical symptoms
  - Chronic reduction in  $\text{FEV}_1$  or FVC associated with clinical symptoms
  - A significant increase in number of persons with  $\text{FEV}_1$  below normal limits: chronically reduced  $\text{FEV}_1$  is a predictor of increased risk of mortality. Transient or reversible reductions that are not associated with an asthmatic attack appear to be less important. It should be emphasized that a small but significant reduction in a population mean  $\text{FEV}_1$  or  $\text{FEV}_{0.75}$  is probably medically significant, as such a difference may indicate an increase in the number of persons with respiratory impairment in the population. In other words, a small part of the population may manifest a marked change that is medically significant to them, but when diluted with the rest of the population the change appears to be small
  - An increased rate of decline in pulmonary function ( $\text{FEV}_1$ ) relative to the predicted value in adults with increasing age or failure of children to maintain their predicted  $\text{FEV}_1$  growth curve. Such data must be standardized for sex, race, height, and other demographic and anthropometric factors
- Increased prevalence of wheezing in the chest apart from colds, or of wheezing most days or nights. (The significance of wheezing with colds needs more study and evaluation.)
- Increased prevalence or incidence of chest tightness
- Increased prevalence or incidence of cough/phlegm production requiring medical attention
- Increased incidence of acute upper respiratory infections that interfere with normal activity
- Acute upper respiratory tract infections that do not interfere with normal activity
- Eye, nose, and throat irritation that may interfere with normal activity (i.e., driving a car) if severe
- Odors

drawn between effects to individuals and effects to populations and that populations are heterogeneous in their susceptibility. The comment was offered that a change in a population could be "medically significant" for that group. The statement also provides guidance on interpretation of reversible effects and on interpreting irreversible effects. In acknowledging that research would continue to address uncertainties, the committee recommended that the guidelines should be periodically reviewed and updated.

## METHODOLOGY FOR DEVELOPING THIS STATEMENT

Following the recommendation of the committee that authored the 1985 statement, the Environmental and Occupational Health Assembly of the American Thoracic Society recognized a need to reconvene a group to review and revise the prior statement. The statement had been used for more than a decade and new investigative approaches were being used to identify effects of air pollution that were not considered by the first committee. In addition, societal perspectives had shifted since the early 1980s and a formal concern for the impact of air

pollution on specific groups had been expressed through the environmental justice movement.

To revise the statement, a multidisciplinary committee was convened in 1997 that included expertise in pulmonary medicine, public health, epidemiology, both clinical and animal toxicology, biochemistry, and cellular and molecular biology. This committee conducted several planning meetings and consulted experts in environmental economics and in ethics. In addition, a multidisciplinary workshop was convened to gain input from the range of groups potentially interested in the statement and its application. The committee's approach was discussed at a symposium held at the 1999 Annual Meeting of the American Thoracic Society. After further revisions, the statement was reviewed and submitted to the Board of the American Thoracic Society.

## BACKGROUND ON THE CLEAN AIR ACT

The preparation of the original statement was largely motivated by potential ambiguity in interpreting the language of the Clean Air Act, which addresses adverse effects of air pollution without providing clear guidance as to the distinction between adverse and nonadverse effects. In addition, questions regarding this distinction arise repeatedly in interpreting the findings of research studies, whether observational or experimental. Consequently, the 1985 statement has had broader application than just the interpretation of evidence on air pollution and health for the purpose of promulgating air quality regulations. Nonetheless, the committee found the legislative history of the Clean Air Act to be relevant to its charge.

The first national legislation on air pollution, the Air Pollution Control Act, was passed in the mid-1950s; the original Clean Air Act was passed in 1963 and last revised in 1990. The act is lengthy and complex in its provisions; most relevant to considerations in defining an adverse health effect are Sections 108 (Air Quality Criteria and Control Techniques), 109 (National Ambient Air Quality Standards), and 112 (Hazardous Air Pollutants). National Ambient Air Quality Standards (NAAQS) are set individually for six prevalent pollutants (Table 1), often referred to as "criteria pollutants." They are so designated because of the requirement for comprehensively reviewing relevant information in a criteria document. The primary NAAQS are to be set at a level that protects the public health with an adequate margin of safety, regardless of economic or technical feasibility of attainment. The secondary standards are concerned with welfare and environmental consequences.

The hazardous air pollutants, as defined in Section 112, are not covered under Sections 108 and 109 as criteria pollutants. In 1990, the Congress offered a list of 189 such pollutants and a process for listing and delisting substances. The 1990 Clean Air Act states: "The Administrator shall periodically review [and revise] the list [of 189 hazardous air pollutants] by... adding pollutants which present, or may present, through inhalation or other routes of exposure, a threat of adverse human health effects (including, but not limited to substances which are known to be, or may reasonably be anticipated to be, carcinogenic, mutagenic, teratogenic, neurotoxic, which cause reproductive dysfunction, or which are acutely or chronically toxic)..." Section 112(f)(2) further directs the Environmental Protection Agency to assess whether the emissions standards for the listed hazardous air pollutants required under other subsections "provide an ample margin of safety to protect public health" and if not, then the agency is to develop standards that will address the "remaining risk."

The historical record provides an indication of the intent of the Congress in framing the language of the Clean Air Act with regard to protection of the public's health. Research now shows that the most highly susceptible individuals may respond to common exposures that are often at or close to natural background pollutant levels.

With regard to sensitivity, the 1970 Clean Air Act recognized that some persons were so ill as to need controlled environments, e.g., persons in intensive care units or newborn infants in nurseries; the act stated that the standards might not necessarily protect such individuals. It further stated, however, that the standards should protect "particularly sensitive citizens such as bronchial asthmatics and emphysematics who in the normal course of daily activity are exposed to the ambient environment." The act further suggested that the adequacy of any standard could be tested in a statistically representative sample of sensitive individuals. The hearing record on the 1970 act is informative. Dr. Hon T. Middleton (Commissioner, National Air Pollution Control Administration, Department of Health, Education, and Welfare) addressed the Senate Subcommittee on Air and Water Pollution of the Committee on Public Works on May 27, 1970. He testified that the intent of any national air quality standard is to be "protective of health in all places" and set at a level below which effects have not been observed. Dr. Middleton recognized the difficulty of finding a demarcation point of exposure below which there is no effect and he noted that there may be subtle effects and evolving scientific understanding.

Further difficulties in the language of the Clean Air Act were later noted in *A Legislative History of the Clean Air Act Amendments of 1977: A Continuation of the Clean Air Act Amendments of 1970*. This document noted the difficulty of applying the margin of safety and the erosion of margins of safety by advancing scientific knowledge. The document also commented on the implicit assumption of a safe threshold in the language of the act and the implausibility of this assumption. The report questioned whether the NAAQS (1) protect against genetic mutations, birth defects, and cancer, (2) take sufficient account of the consequences of long-term low-level exposures or short-term peaks, and (3) sufficiently consider synergism among pollutants and the formation of secondary pollutants, e.g., sulfates, with their own toxicity. These considerations remain relevant more than 20 years later.

This selective review of the historical record indicates that Congress intended that the NAAQS would afford health protection not only to the general population but to subgroups with enhanced susceptibility to air pollution, including people with asthma and people with chronic obstructive lung disease. Nevertheless, it is also clear that some exquisitely susceptible individuals might remain outside the ambit of protection of the NAAQS. A margin of safety was to be provided but quantitative specification was not offered. The evolutionary nature of the supporting scientific evidence was repetitively acknowledged and the need to distinguish adverse from nonadverse effects was at least implicitly recognized. The current language of Section 112 explicitly acknowledges the possibility of shifting understanding of risks of specific hazardous air pollutants.

## GENERAL CONSIDERATIONS

In preparing the statement, the committee identified several general considerations that are relevant to interpreting evidence on the health effects of air pollution. Each of these considerations and the committee's judgment as to their proper weighting are detailed below.

### Population Health versus Individual Risk

The effects of air pollution can be viewed in the complementary contexts of the increment of an individual's risk for disease—the clinician's measure of impact—and of the additional risk incurred by a population, which is the public health perspective (16). Both perspectives are relevant to interpreting research findings on air pollution and to regulations that are protective of the public's health. Any risk incurred by an exposed individual beyond some boundary, determined by the individual or on a societal basis, could be deemed unacceptable. For example, prolonged exposure to a respiratory carcinogen could result in an individual-level incremental risk of exposure of  $10^{-4}$ , more than two orders of magnitude lower than the baseline lifetime individual risk in the United States. Nevertheless, among an exposed population of  $10^7$ , the estimated number of cancer cases that might result from such an exposure would number  $10^3$ , illustrating that minute individual risks may be significant from the standpoint of population exposures.

Exposure could also enhance risk for a population to an unacceptable degree, perhaps without shifting the risks of any particular individuals to an unacceptable level. Figure 1 illustrates the distinction. In Figure 1A, the population's distribution of exposure shifts toward a higher level and some members of the population cross the boundary to an unacceptable risk. In Figure 1B, the shift affects the position of the population distribution, but no individuals move to an unacceptable level of risk. Effects on persons with asthma are illustrative. A population of children with asthma could have a distribution of lung function such that no individual child has a level associated with significant impairment. Exposure to air pollution could shift the distribution toward lower levels without bringing any individual child to a level that is associated with clinically relevant consequences. Individuals within the population would, however, have diminished reserve function and are at potentially increased risk if affected by another agent, e.g., a viral infection. Assuming that the relationship between the risk factor and the disease is causal, the committee considered that such a shift in the risk factor distribution, and hence the risk profile of the exposed population, should be considered adverse, even in the absence of the immediate occurrence of frank illness.

### Ethics and Equity

The past decade has brought increasing concern over the ethics of heterogeneous, inequitable distributions of environmental and occupational exposures (15). Within the United States, some groups receive disproportionate exposures to environmental agents that are injurious to health; the environmental justice movement seeks to redress these inequities. The exposures of concern originate in breathing polluted outdoor air, living in substandard housing with indoor air pollution problems, including exposures to certain bioaerosols and combustion products, and working in jobs with occupational respiratory risks. Groups encompassed by this movement in the United States include various racial and ethnic minority populations, particularly those living within urban areas, and the socioeconomically disadvantaged. In the developing world, such exposures can occur at substantially higher levels and may, in some instances, extend to a majority of a given nation's population. Limited access to care and medications may enhance susceptibility to pollution.

The concept of environmental equity had not been formally voiced when the 1985 statement was written. The present committee viewed inequities of exposure as potentially repre-

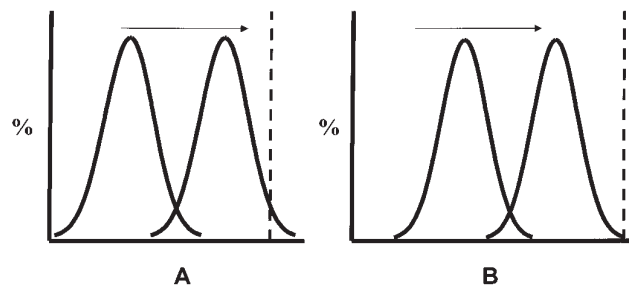


Figure 1. Hypothetical distributions of exposure for two populations, A and B. (See text for explanation.)

senting a form of susceptibility to air pollution. In other words, individuals within the target groups may be at increased risk of experiencing adverse effects from a given level of ambient air pollution because their baseline risk level may have been raised by other exposures. Moreover, in some instances there may be genetic and nutritional factors enhancing susceptibility as well. It should be noted, however, that there are other exposure scenarios and other subpopulations with increased baseline risks that are not formally included within the environmental justice movement. The heterogeneity of populations needs full acknowledgment, whether it reflects disproportionate noxious exposures or other factors. Observing that there have been few investigations of the effects of other exposures, genetics, or nutrition on susceptibility to air pollution-related effects, either in the United States or internationally, the committee issued a call for additional research in these areas.

### Economic Costs

Adverse health effects of air pollution incur costs, including direct costs of providing treatment for illness and indirect costs of lost work time and productivity. Cost-benefit analysis provides an estimate of the balancing of the costs of controls against the benefits; cost effectiveness analysis provides an indication of the level of control accomplished in relation to costs. Cost-benefit and cost-effectiveness analysis are assumption-laden tools now being used for policy-making purposes. Cost estimates depend on the valuation given to illness, lost work time and productivity, and even to lost life. It has been proposed that cost-benefit analysis may facilitate the process of deciding whether a given air pollution-related health impact should be considered adverse. The legislative history of the Clean Air Act explicitly excludes consideration of economic factors in setting ambient air quality standards or in developing emissions standards for hazardous air pollutants. In the context of air quality regulation, cost-benefit analysis is a multistep process involving the articulation of value judgments regarding potential costs (expenditures of public and private resources to reduce pollutant emissions and exposures) versus benefits (avoidance of specified adverse health impacts in a designated population). Benefits, in theory, should be quantified as the willingness of beneficiaries to pay to avoid the adverse impact. In practice, quantification of such health impacts from exposure to air pollution is often based on direct costs related to medical treatment and indirect costs such as school absenteeism, lost work time, decreased productivity, and, at the extreme, person-years of life lost. Valuations of a given effect may vary internationally, as differences in population age distributions, comorbidity, nutritional status, and other circumstances can affect this process. Ideally, cost-bene-

fit analysis should make explicit the value judgments underlying these assessments, highlighting distinctions among alternative pollution control strategies to achieve specific air quality standards. Willingness of individuals to pay to avoid adverse health effects is also estimated from responses to contingent valuation surveys and from market data concerning choices about employment that carries health risks.

Nevertheless, the committee concurred that the specification of which health effects should be considered adverse must precede the application of cost-benefit analysis for evaluation of air pollution control strategies. That is, once a given outcome is designated as adverse, this information can be used as input to cost-benefit analysis. Estimates of costs associated with a given health outcome, while useful from a public policy perspective, cannot be translated into any clinical or biological framework to distinguish adverse from nonadverse effects. Therefore, the committee concluded that however valuable this economists' tool may be for regulatory decision-making, cost-benefit analysis lay outside the scope of this position paper and, indeed, the expertise of the American Thoracic Society.

#### Susceptibility

The issue of susceptibility has been recognized throughout the history of our initiatives to regulate outdoor air pollution. Susceptibility, broadly defined, may include extrinsic factors, including the profile of exposures to other pollutants, for example, in the workplace or at home, and intrinsic factors, for example, genotype. The size of the population of individuals susceptible to indoor air pollution is large, potentially including infants and the elderly, persons with chronic heart and lung diseases, and the immunocompromised. Persons with multiple deleterious exposures may also be considered as having heightened susceptibility, particularly if the combined effects of the agents are synergistic. Even with the populations considered as susceptible there is a distribution of the degree of susceptibility. For example, levels of nonspecific airway responsiveness in persons with asthma span several orders of magnitude.

The current explosive growth in knowledge of the genetic basis of lung disease, including responses to environmental agents, will provide increasing insights into the mechanistic basis of susceptibility and provide markers of risk status. We already have evidence of between-person variation in the pulmonary function response to ozone and interstrain variation in the pulmonary effects of environmental exposures, including criteria pollutants, in rodent species. As we develop the capacity to more precisely identify those at risk, we may find it increasingly challenging to assure protection for all individuals against adverse health effects.

The present committee agreed with the principle espoused in the Clean Air Act: that regulations should extend protection to include those with enhanced susceptibility to air pollution, recognizing that some highly susceptible individuals may still respond to low-level exposures. Research now shows that some highly susceptible individuals may respond to common exposures that are often unavoidable. Furthermore, by definition, susceptible individuals cannot have the same margin of safety as the nonsusceptible groups within the population.

#### Heterogeneity of Perspectives

In society there is an extraordinary range of views on environmental issues and tolerance of risk. Looking more globally to other developed countries and to the developing countries, the range of perspectives is even broader. The committee acknowledges that any defined boundaries for distinguishing ad-

verse health effects may not be embraced by all groups. This heterogeneity and the possibility that some may reject the committee's proposal challenged the committee to recommend in principle that control measures should maximize public health benefits while assuring equity.

#### DIMENSIONS OF ADVERSE EFFECTS

##### Biomarkers

Biomarkers are indicators of exposure, effect, or susceptibility that are measured in biologic materials, such as blood or bronchoalveolar lavage fluid. The concept of biomarkers has been formalized since the 1985 statement (17) and since then, a continuously increasing number of candidate indicators of exposure, effect, and susceptibility have been developed and applied in laboratory studies of humans and animals and in both occupational and environmental population studies. The progressive refinement of techniques in the field of cellular and molecular biology, and the burgeoning understanding of the complex chemical intracellular and cell-to-cell signaling pathways collectively termed "cytokines" (18), have rapidly expanded the spectrum of candidate markers of effects. It is now possible to detect very early, or initiating phases of responses at the molecular level, such as the production of mRNA for cytokines. Similarly, the progressive development of genetic assays and understanding of the human genome have provided numerous candidate markers of both effects and susceptibility (19).

Biomarkers relevant to air pollution have been measured in blood, exhaled air, urine, sputum, and in bronchoalveolar lavage fluids and tissue specimens collected by bronchoscopy. Bronchoalveolar lavage fluids, for example, are now frequently analyzed for cell numbers and types, cytokines (e.g., several interleukins and tumor necrosis factor  $\alpha$ ), enzymes (e.g., lactate dehydrogenase and  $\beta$ -glucuronidase), fibronectin, protein, arachidonic acid metabolites, and reactive oxygen species. Because many of the epithelial cell types of the nasopharyngeal region are similar to epithelia and responses in the trachea, bronchi, and bronchioles, responses of nasal cells have been examined as potential biomarkers for their ability to predict parallel responses in lung airways, which are more difficult to sample.

Biomarkers have been extensively applied in toxicologic studies of air pollution, both in animals and in clinical studies involving exposures of human volunteers. The biomarkers are examined for their ability to provide evidence of "biologically effective" doses, including the earliest phases of homeostatic responses, the occurrence of injury, outcomes that are intermediate between injury and disease, and the presence of established disease processes. Genetic markers of susceptibility have begun to be applied to the respiratory system, and this application will undoubtedly expand rapidly. A frequent goal of biomarker development is the ability to readily measure changes that precede and predict continued or progressive events leading to clinical effects and disease (Figure 2).

To date, although biomarkers have proved informative about homeostatic adjustments to exposure and the mechanisms of injury and disease, lack of validation against previously established measures of effect, such as clinical status or even physiologic impairment, remains an important weakness. We do not know if elevations of biomarkers during short-term experimental exposures signal risk for ongoing injury and clinical effects or simply indicate transient responses that can provide insights into mechanisms of injury. The utility of some older biomarkers is well established, such as the relationships among carboxyhemoglobin, exposure to carbon monoxide,



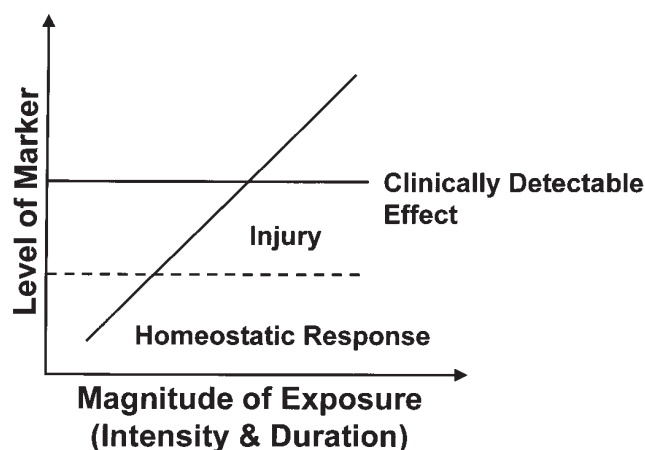


Figure 2. Schema for considering biomarkers of response.

impairment of oxygen-carrying capacity, and the risk for angina in the presence of ischemic heart disease. However, the interpretative value for the majority of the many promising new cytokine and genetic biomarkers remains to be established. Not only is it difficult to assess the value of many biomarkers for distinguishing between physiological, homeostatic responses and injury, but it is also difficult to judge the value of changes during short-term exposures for predicting ongoing injury or risk for longer-term clinical effects.

The committee concluded that the continued development of biomarkers is an important need because of their considerable potential not only for detecting the adverse effects of air pollution exposure, but also for aiding the determination of the types and levels of response that should be considered adverse. We often do not know in a parallel, iterative manner, whether the exploration and validation of biomarkers will unquestionably advance our understanding of the mechanisms of homeostatic and injury responses. At this time, however, few of the rapidly growing list of candidate biomarkers have been validated to such an extent that their responses can be used with confidence to define the point at which a response should be equated to an adverse effect warranting preventive measures. Thus, we presently have only a very modest ability to translate evidence from biomarkers directly into a taxonomy of adverse health effects. Consequently, the committee cautions that not all changes in biomarkers related to air pollution should be considered as indicative of injury that represents an adverse effect.

### Quality of Life

Health, in its broadest definition, includes not only the absence of disease but the attainment of well-being. Since the preparation of the 1985 statement, the National Institutes of Health, the Centers of Disease Control, the Food and Drug Administration, and the World Health Organization have broadened their perspective of health to incorporate the concept of health-related quality of life as a valid and important health outcome. Health-related quality of life (HRQL) refers to the individual's perception of well-being, and includes such factors as self-care functioning, mental health, pain, and sense of overall well-being. Decreased health-related quality of life is widely accepted to be an adverse health effect. For this reason, measurable negative effects of air pollution on quality of life, whether for persons with chronic respiratory conditions or the population in general, were consequently considered by

this committee to be adverse health effects. Air pollution exposure can adversely affect several domains of quality of life including physical functioning (particularly for persons with respiratory or cardiovascular conditions) and general well-being. Stinging, watery eyes resulting from air pollution not only reflect a chronic physical symptom but may decrease overall quality of life. Outdoor air pollution and odors have been associated with psychiatric symptoms, including anxiety and depression. Increased levels of some air pollutants have been reported to be associated with an increase in psychiatric admissions. The potential effects of air pollution and respiratory symptoms on different domains of quality of life are illustrated in Figure 3.

Measurement of the impact of air pollution on health-related quality of life can be accomplished either by measuring specific domains that may be influenced by air quality (e.g., anxiety, functional status), or by using specific quality of life instruments designed to measure multiple health-related domains (e.g., MOS-SF-36, St. George's Respiratory Questionnaire). The cost-benefits of improved air quality on health-related quality of life could also be measured by the use of quality of life measures that employ utility rating scales. The effects of air pollution of a magnitude considered to be clinically significant with these instruments should be regarded as adverse in interpreting evidence on the health effects of air pollution, regardless of the affected dimension. Additional research is needed to develop an information base for interpreting data from new and more sensitive instruments directed specifically at air pollution.

### Physiological Impact

The 1985 statement acknowledged a distinction between reversible and irreversible effects. Healthy persons may sustain transient reductions in pulmonary function associated with air pollution exposure, e.g., reduction of the forced vital capacity (FVC) with exercise at times of higher levels of ozone pollution. However, the committee recommends that a small, transient loss of lung function, by itself, should not automatically be designated as adverse. In drawing the distinction between adverse and nonadverse reversible effects, this committee recommended that reversible loss of lung function in combination with the presence of symptoms should be considered as adverse. This recommendation is consistent with the 1985 statement. The Environmental Protection Agency has also needed to address the interpretation of such data. The Envi-

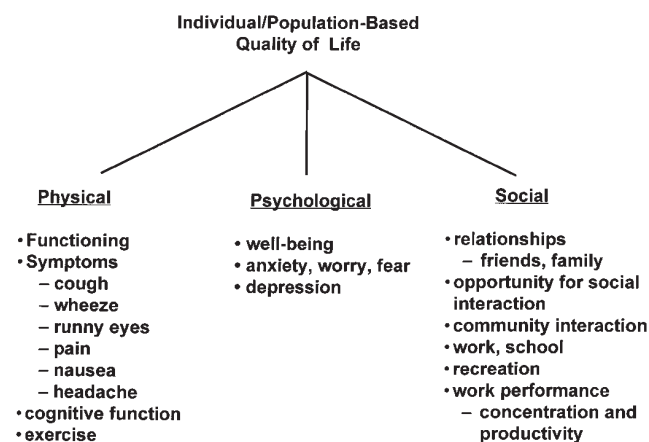


Figure 3. Quality of life domains vulnerable to the adverse health/respiratory effects of air pollution.



Environmental Protection Agency, in its 1989 review of ozone (20), offered a graded classification of lung function changes in persons with asthma. Reduction of the forced expiratory volume in 1 s (FEV<sub>1</sub>) was graded as mild, moderate, or severe for reductions of less than 10%, 10–20%, and more than 20%, respectively. This classification has not been validated for acceptability or against other measures.

There is also epidemiologic evidence that air pollution may adversely affect lung growth or accelerate the age-related decline of lung function. Epidemiologic studies are limited in their power to detect such permanent effects and any evidence of association between air pollution exposure and permanent loss of function is indicative of an adverse effect at the population level. Some individuals may sustain clinically relevant, permanent losses of lung function. This committee considered that any detectable level of permanent lung function loss attributable to air pollution exposure should be considered as adverse.

### Symptoms

Air pollution exposure can evoke symptoms in persons without underlying chronic heart or lung conditions and also provoke or increase symptom rates in persons with asthma and chronic obstructive lung disease. The Environmental Protection Agency also offered a scale for cough and pain on taking a deep breath in its 1989 ozone review (20). "Infrequent cough" was classified as "None/Normal."

Do all levels of increased symptom occurrence constitute an adverse health effect? The committee judged that air pollution-related symptoms associated with diminished quality of life or with a change in clinical status should be considered as adverse at the individual level. Characterizing the degree of symptomatology associated with diminished quality of life is an appropriate focus for research and a topic that could be investigated using new approaches for assessing quality of life. A change in clinical status can be appropriately set in a medical framework as one requiring medical care or a change in medication. At the population level, any detectable increment in symptom frequency should be considered as constituting an adverse health effect.

### Clinical Outcomes

A wide range of clinical outcome measures has been considered in relation to air pollution, including population-level effects, such as increases in numbers of emergency room visits for asthma or hospitalizations for pneumonia, and individual-level effects, such as increased need for bronchodilator therapy. The present committee shared the view of the previous group: detectable effects of air pollution on clinical measures should be considered adverse.

At the population level, the magnitude of the detectable air pollution effect will depend on the extent of the data available for evaluation and methodological aspects of the data, including the degree of error affecting exposure and outcome variables. With large databases, seemingly modest effects may be detectable. However, the committee recommends that no level of effect of air pollution on population-level clinical indicators can be considered acceptable.

### Mortality

Following the development of new approaches for the analysis of time-series data, extensive analyses have now been reported on the relationship between daily mortality counts and levels of air pollution on the same or prior days. Several prospective cohort studies have also addressed the effect of longer-term indicators of air pollution exposure on mortality,

controlling for relevant individual factors, including age, sex, cigarette smoking, and occupational exposures, among others. Cross-sectional studies—comparing mortality across locations having different levels of air pollution while controlling for a variety of potential confounding factors—have also been conducted. The air pollution-associated mortality findings figured prominently in the recent revision of the U.S. NAAQS for particulate matter.

Associations between air pollution levels and daily mortality counts have been interpreted by some as reflecting the impact of air pollution on a pool of frail individuals with severe underlying heart or lung disease. One explanation for the day-to-day associations attributes them to a brief advancement of the time of death for extremely frail individuals who would have been expected to die soon even in the absence of an air pollution-related insult (21). Work has shown, however, that while this phenomenon of advancement, referred to as mortality displacement, may occur, it cannot provide a full explanation of the associations repeatedly found between daily fluctuations of air pollution and mortality (22, J. Schwartz, "Harvesting and long term exposure effects in the relationship between air pollution and mortality" [1999, unpublished manuscript]). In addition, some mortality time-series studies have found effects across all age strata, not just among the elderly or the very young, suggesting potentially substantial effects on person-years of life lost. Finally, studies of long-term exposures have shown a gradient of mortality risk from cardiopulmonary disease as well as differences in life expectancy across cities with different long-term pollution levels. Thus, although we still have little insight into the extent to which mortality displacement occurs, the evidentiary ensemble from several types of study designs consistently shows that air pollution can shorten the life span to an unacceptable degree.

### Risk Assessment

Since the publication of the 1985 statement, quantitative risk assessment has emerged as a key tool for summarizing information on risks to health from environmental agents. Quantitative risk assessment offers a framework for organizing information on risks within its four elements: hazard identification, exposure assessment, dose–response assessment, and risk characterization. The findings of a risk assessment, encompassed in the risk characterization component, may include an overall assessment of impact, a description of the distribution of risk in the population, and an evaluation of risk for susceptible persons within the population. Quantitative risk assessment has been a cornerstone in evaluating risks of environmental carcinogens and we anticipate increasing application to non-carcinogenic health effects of environmental agents, including air pollution.

In interpreting the findings of risk assessments, guidance can be found in precedents offered by key interpretations of regulatory requirements, including the Supreme Court's decision on the benzene standard proposed by the Occupational Safety and Health Administration, and in pollutant-specific regulatory actions. Risks may be couched as the numbers of attributable events in the population and also as the level of risk incurred by individual members of the population.

The committee recognized the rising use and potential utility of quantitative risk assessment in characterizing the health effects of air pollution. However, the committee noted that the results of quantitative risk assessment can often be sensitive to assumptions regarding the distribution and magnitude of exposure, the choice of an appropriate dose–response relationship, and other input decisions. Judgments on acceptability of risk are societal and made through complex regulatory

processes involving extensive public input. The committee did not consider that its mandate extended to offering specific guidance on acceptable risk levels for populations or individuals, nor is risk assessment an appropriate basis for determining what constitutes an adverse effect.

## CONCLUSIONS

Since the preparation of the 1985 statement of the American Thoracic Society, there have been tremendous advances in the scientific methods used to investigate the health effects of air pollution. These advances range from the molecular to the behavioral levels of inquiry. As a result, this statement covers topics that are new since the 1985 statement. Yet, this committee, like the 1985 group, was confronted by a lack of formal research or investigation on the very topic of this statement: the boundary between adverse and nonadverse effects. Consequently, the committee needed to exercise its collective judgment on matters that should be based in some broader, societal decision-making process. Its recommendations are summarized below.

- **Biomarkers.** Few of the rapidly growing list of candidate biomarkers have been validated sufficiently that their responses can be used with confidence to define the point at which a response should be equated to an adverse effect warranting preventive measures. The committee cautions that not all changes in biomarkers related to air pollution should be considered as indicative of injury that represents an adverse effect.
- **Quality of life.** Decreased health-related quality of life is widely accepted as an adverse health effect. For this reason, measurable negative effects of air pollution on quality of life, whether for persons with chronic respiratory conditions or for the population in general, were consequently considered to be adverse by this committee.
- **Physiological impact.** The committee recommends that a small, transient loss of lung function, by itself, should not automatically be designated as adverse. In drawing the distinction between adverse and nonadverse reversible effects, this committee recommended that reversible loss of lung function in combination with the presence of symptoms should be considered adverse. This committee considered that any detectable level of permanent lung function loss attributable to air pollution exposure should be considered adverse.
- **Symptoms.** The committee judged that air pollution-related symptoms associated with diminished quality of life or with a change in clinical status should be considered adverse at the individual level.
- **Clinical outcomes.** The present committee shared the view of the previous group: detectable effects of air pollution on clinical measures should be considered as adverse.
- **Mortality.** This committee agreed with the conclusion articulated by the 1985 group that any effect on mortality should be judged as adverse. In addition, we are now faced with the challenge of interpreting the findings of time-series studies of effects on short time frames. In interpreting this type of evidence, consideration needs to be given to the extent of life-shortening underlying the association.
- **Population health versus individual risk.** Assuming that the relationship between the risk factor and the disease is causal, the committee considered that such a shift in the risk factor distribution, and hence the risk profile of the exposed population, should be considered adverse,

even in the absence of the immediate occurrence of frank illness.

This statement was prepared by an ad-hoc committee of the Assembly on Environmental and Occupational Health. Members of the committee are:

JON SAMET, M.D., *Co-chair*  
SONIA BUIST, M.D., *Co-chair*  
REBECCA BASCOM, M.D.  
JOE GARCIA, M.D.  
MICHAEL LIPSETT, M.D.  
JOE MAUDERLY, D.V.M.  
DAVID MANNINO, M.D.  
CYNTHIA RAND, Ph.D.  
ISABELLE ROMIEU, M.D.  
MARK UTELL, M.D.  
GREGORY WAGNER, M.D.

### *Workshop Participants*

DAVID V. BATES, M.D.  
MELVIN L. BILLINGSLEY, Ph.D.  
MICHAEL GELOBTER, M.D.  
BENJAMI F. HOBBS, Ph.D.  
STEPHEN KLEENBERGER, Ph.D.  
NANCY KLINE LEIDY, Ph.D., R.N.  
STEPHANIE LONDON, M.D., Dr.P.H.  
WILLIAM F. McDONNELL, M.D., Ph.D.  
DIETER SCHWELA, M.D.  
JAMES C. WILEY, M.D.

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STATE OF MICHIGAN

BEFORE THE MICHIGAN PUBLIC SERVICE COMMISSION

In the matter of the Application of **DTE Electric Company** for approval of its integrated resource plan pursuant to MCL 460.6t, and for other relief.

Case No. U-20471

ALJ Sally L. Wallace

**PROOF OF SERVICE**

On the date below, an electronic copy of **Direct Testimony of George Thurston on behalf of Michigan Environmental Council, Natural Resources Defense Council and Sierra Club along with Exhibits MEC-85 through MEC-87** was served on the following:

Name/Party	E-mail Address
<b>Administrative Law Judge</b> Hon. Sally Wallace	<a href="mailto:wallaces2@michigan.gov">wallaces2@michigan.gov</a>
<b>Counsel for DTE Electric Co.</b> Lauren DuVal Donofrio Martin Heiser	<a href="mailto:mpscfilings@dteenergy.com">mpscfilings@dteenergy.com</a> <a href="mailto:donofriol@dteenergy.com">donofriol@dteenergy.com</a> <a href="mailto:martin.heiser@dteenergy.com">martin.heiser@dteenergy.com</a>
<b>Counsel for MPSC Staff</b> Amit T. Singh Heather M.S. Durian Daniel E. Sonneveldt Benjamin J. Holwerda Sarah Mullkoff	<a href="mailto:singha9@michigan.gov">singha9@michigan.gov</a> <a href="mailto:durianh@michigan.gov">durianh@michigan.gov</a> <a href="mailto:sonneveltdt@michigan.gov">sonneveltdt@michigan.gov</a> <a href="mailto:holwerdab@michigan.gov">holwerdab@michigan.gov</a> <a href="mailto:MullkoffS1@michigan.gov">MullkoffS1@michigan.gov</a>
<b>Counsel for Department of Attorney General</b> Joel B. King	<a href="mailto:ag-enra-spec-lit@michigan.gov">ag-enra-spec-lit@michigan.gov</a> <a href="mailto:kingJ38@michigan.gov">kingJ38@michigan.gov</a>
<b>Counsel for ABATE</b> Bryan A. Bradenburg Michael J. Pattwell	<a href="mailto:bbradenburg@clarkhill.com">bbradenburg@clarkhill.com</a> <a href="mailto:mpattwell@clarkhill.com">mpattwell@clarkhill.com</a>
<b>Counsel for Great Lakes Renewable Energy Association</b> Don L. Keskey Brian W. Coyer	<a href="mailto:donkesey@publiclawresourcecenter.com">donkesey@publiclawresourcecenter.com</a> <a href="mailto:bwcover@publiclawresroucecenter.com">bwcover@publiclawresroucecenter.com</a>
<b>Counsel for Energy Michigan Inc.</b> Justin K. Ooms Laura Chappelle Timothy Lundgren	<a href="mailto:jkooms@varnumlaw.com">jkooms@varnumlaw.com</a> <a href="mailto:lachappelle@varnumlaw.com">lachappelle@varnumlaw.com</a> <a href="mailto:tjlundgren@varnumlaw.com">tjlundgren@varnumlaw.com</a>
<b>Counsel for Environmental Law &amp; Policy Center, the Ecology Center, the Solar Energy Industries Association, the Union of Concerned Scientists and Vote Solar</b> Margrethe Kearney Jean-Luc Kreitner	<a href="mailto:mkearney@elpc.org">mkearney@elpc.org</a> <a href="mailto:jkreitner@elpc.org">jkreitner@elpc.org</a>
<b>Counsel for International Transmission Company</b> Richard J. Aaron Courtney Kissel	<a href="mailto:raaron@dykema.com">raaron@dykema.com</a> <a href="mailto:ckissel@dykema.com">ckissel@dykema.com</a>
<b>Counsel for Geronimo Energy</b> Timothy Lundgren Justin K. Ooms	<a href="mailto:tjlundgren@varnumlaw.com">tjlundgren@varnumlaw.com</a> <a href="mailto:jkooms@varnumlaw.com">jkooms@varnumlaw.com</a>

<b>Counsel for Cypress Creek Renewables, LLC</b> Jennifer Utter Heston	<a href="mailto:jheston@fraserlawfirm.com">jheston@fraserlawfirm.com</a>
<b>Counsel for Convergen Energy, LLC</b> Timothy Lundgren	<a href="mailto:tjlundgren@varnumlaw.com">tjlundgren@varnumlaw.com</a>
<b>Counsel for Association of Businesses Advocating Tariff Equity</b> Bryan A. Brandenburg	<a href="mailto:BBrandenburg@clarkhill.com">BBrandenburg@clarkhill.com</a>
<b>Counsel for City of Ann Arbor</b> Timothy Lundgren Justin Ooms	<a href="mailto:tjlundgren@varnumlaw.com">tjlundgren@varnumlaw.com</a> <a href="mailto:jkooms@varnumlaw.com">jkooms@varnumlaw.com</a>
<b>Counsel for Heelstone Development, LLC</b> <b>Midland Cogeneration Venture LP</b> Jason Hanselman John A. Janiszewski	<a href="mailto:jhanselman@dykema.com">jhanselman@dykema.com</a> <a href="mailto:jjaniszewski@dykema.com">jjaniszewski@dykema.com</a>
<b>Counsel for Soulardarity</b> Christopher M. Bzdok Lydia Barbash-Riley	<a href="mailto:chris@envlaw.com">chris@envlaw.com</a> <a href="mailto:lydia@envlaw.com">lydia@envlaw.com</a>
<b>Counsel for Michigan Public Power Agency</b> Nolan Moody Peter H. Ellsworth	<a href="mailto:nmoody@dickinsonwright.com">nmoody@dickinsonwright.com</a> <a href="mailto:pellsworth@dickinsonwright.com">pellsworth@dickinsonwright.com</a>
<b>Co-Counsel for Soulardarity</b> Rebecca J. Boyd Mark H. Templeton Robert A. Weinstock	<a href="mailto:rebecca.j.boyd@gmail.com">rebecca.j.boyd@gmail.com</a> <a href="mailto:templeton@uchicago.edu">templeton@uchicago.edu</a> <a href="mailto:rweinstock@uchicago.edu">rweinstock@uchicago.edu</a>

The statements above are true to the best of my knowledge, information and belief.

OLSON, BZDOK & HOWARD, P.C.  
Counsel for MEC-NRDC-SC

Date: August 21, 2019

By: \_\_\_\_\_

Kimberly Flynn, Legal Assistant  
Karla Gerds, Legal Assistant  
Breanna Thomas, Legal Assistant  
420 E. Front St.  
Traverse City, MI 49686  
Phone: 231/946-0044  
Email: [kimberly@envlaw.com](mailto:kimberly@envlaw.com)  
[karla@envlaw.com](mailto:karla@envlaw.com) and  
[breanna@envlaw.com](mailto:breanna@envlaw.com)