BEFORE THE COUNCIL FOR THE CITY OF NEW ORLEANS

APPLICATION OF ENTERGY NEW ORLEANS, INC. FOR APPROVAL TO CONSTRUCT NEW ORLEANS POWER STATION AND REQUEST FOR COST RECOVERY AND TIMELY RELIEF	**)**	**DOCKET NO. UD-16-02**

PRE-FILED DIRECT TESTIMONY

OF

DR. GEORGE THURSTON, SC.D.

ON BEHALF OF

ALLIANCE FOR AFFORDABLE ENERGY,

DEEP SOUTH CENTER FOR ENVIRONMENTAL JUSTICE,

AND SIERRA CLUB

Filed on: JANUARY 6, 2017

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I. QUALIFICATIONS

Q1. Please state your name, affiliation, and title.

A. I am George D. Thurston, Sc.D. I am a Professor at the New York University School of Medicine in the Department of Environmental Medicine, where I am the Director of the Program in Human Exposures and Health Effects of Air Pollution. See Exhibit GT-1. My business address is: Three Catherine Ct., Chester, NY 10918. I offer this direct testimony on behalf of the Alliance for Affordable Energy, the Deep South Center for Environmental Justice, and Sierra Club.

Q2. What is the purpose of your testimony?

A. I am providing expert testimony that addresses the public health impacts of the emission of fine particulate matter ("$PM_{2.5}$") generally and, specifically, the expected public health impacts of $PM_{2.5}$ emissions from the proposed New Orleans Power Station ("NOPS"). My testimony will address the potential health effects of the plant, if approved. I conclude that the $PM_{2.5}$ emissions from this facility can be expected to increase adverse health risks in the surrounding community.

Q3. Briefly describe your qualifications to provide testimony in this matter.

A. I received my undergraduate degree in Engineering from Brown University (with a Concentration in Environmental Engineering) in 1974, and my doctorate in Environmental Health Sciences from the Harvard University School of Public Health in 1983. I was Chairman of the Health and Environment Panel of the Canadian Joint Industry/Government Study of Sulfur in Gasoline and Diesel Fuels in 1997. I also served on the National Academy of Science's Committee on the Health Effects of Incineration from January 1995 through November 1999,

and am presently serving as the Chair of the Environmental Health Policy Committee of the American Thoracic Society. I have published extensively regarding the health effects of inhaled air pollutants on humans, particularly as it relates to asthma attacks, hospital admissions, and mortality. I have been called upon by both the U.S. House of Representatives and the U.S. Senate on multiple occasions over the years to provide testimony before them regarding the human health effects of air pollution. I have also been a contributing author to both the 1996 and 2001 Environmental Protection Agency ("EPA") Particulate Matter ("PM") Criteria Documents, which the EPA uses as a scientific basis for its decisions regarding the setting of the nation's PM ambient air quality standards. More recently, I served on the U.S. EPA's Clean Air Science Advisory Committee ("CASAC") on the human health effects of Nitrogen Oxides and Sulfur Oxides. I was a Principal Investigator of a study that has shown that long-term exposure to combustion-related fine particulate air pollution is an important environmental risk factor for cardiopulmonary and lung cancer mortality in the U.S. See Pope, CA, 3rd; Burnett, RT; Thun, MJ; Calle, EE; Krewski, D; Ito, K; and; Thurston, GA. (2002). Lung Cancer, Cardiopulmonary Mortality, and Long-term Exposure to Fine Particulate Air Pollution. Journal of the American Medical Association 2002; 287: 1132-1141. The publications reviewed or relied upon for this testimony are listed at the end of this report as "Literature Cited."

Q4. Have you ever testified as an expert witness on air pollution issues generally, and $PM_{2.5}$ health impacts specifically, in a legal proceeding?

A. Yes, I have provided testimony about the human health impacts of $PM_{2.5}$ from electricity power plant combustion sources on numerous occasions, and on the health effects of natural gas plants specifically, including in the Issues Conference in Case 00-F-1256, in the Matter of the Application of Calpine Construction Finance Company, L.L.P. (2001) and on the application by TransGas Energy Systems LLC for a Certificate of Environmental Compatibility and Public Need to Construct and Operate a 1,100 Megawatt Combined Cycle Generating Facility (2003).

Q5. Does your resume, attached as Exhibit GT-1, fairly represent your education and experience?

A. Yes.

II. THE STATE OF THE SCIENCE REGARDING PARTICULATE MATTER ($PM_{2.5}$) AIR POLLUTION AND ITS HUMAN HEALTH EFFECTS

Q6. What are the human health effects of exposure to ambient $PM_{2.5}$ air pollution at levels experienced in the US today?

A. The adverse health consequences of breathing air pollution from sources such as utility power plants are well documented in the published medical and scientific literature. During the past decades, medical research examining air pollution and public health has shown that air pollution is associated with a host of serious adverse human health effects. This documentation includes impacts revealed by observational epidemiology, and confirmed by controlled chamber exposures, showing consistent associations between air pollution and adverse impacts across a wide range of human health outcomes.

Observational epidemiology studies provide the most compelling and consistent evidence of the adverse effects of air pollution. "Epidemiology" is literally "the study of epidemics," but includes all statistical investigations of human health and potentially causal factors of good or ill health. In the case of air pollution, such studies follow people as they undergo varying real-life exposures to pollution over time, or from one place to another, and then statistically inter-compare the health impacts that occur in these populations when higher (versus lower) exposures to pollution are experienced. In such studies, risks are often reported in terms of a Relative Risk ("RR") of illness, wherein an RR =1.0 is an indication of no change in risk after exposure, while an RR>1.0 indicates an increase in health problems after pollution exposure, and that air pollution is damaging to health.

These epidemiological investigations are of two types: 1) population-based studies, in which an entire city's population might be considered in the analysis; and 2) cohort studies, in which selected individuals, such as a group of asthmatics, are considered. Both of these types of epidemiologic studies have shown confirmatory associations between air pollution exposures and increasing numbers of adverse impacts, including:

- decreased lung function (a measure of our ability to breathe freely);
- more frequent asthma symptoms;
- increased numbers of asthma and heart attacks;
- more frequent emergency department visits;
- additional hospital admissions; and
- increased numbers of deaths.

The fact that the effects of air pollution have been shown so consistently for so many health endpoints and in so many locales indicates these associations to be causal.

In addition to lung damage, recent epidemiological and toxicological studies of $PM_{2.5}$ air pollution have shown adverse effects on the heart, including an increased risk of heart attacks. For example, when PM stresses the lung (*e.g.*, by inducing edema), it places extra burden on the heart, which can induce fatal complications for persons with cardiac problems. Indeed, for example, Peters *et al.* (2001) found that elevated concentrations of fine particles in the air can elevate the risk of myocardial infarctions ("MIs") within a few hours, and extending up to one day after PM exposure. The Harvard University team found that a 48% increase in the risk of MI was associated with an increase of 25 *u*g/m^3 $PM_{2.5}$ during a two-hour period before the onset of MI, and a 69% increase in risk to be related to an increase of 20 *u*g/m^3 $PM_{2.5}$ in the twenty-four-hour average one 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 an emergency department for MI (ICD-9 code, and Zanobetti et al. (2009) that examined the relationship between daily $PM_{2.5}$ concentrations and emergency hospital admissions for cardiovascular causes, MI, and congestive heart failure in twenty-six U.S. communities during 2000–2003.

Cardiac effects at the biological level have also been documented in both animal and human studies. Animal experiments at Harvard University by Godleski *et al.* (1996, 2000) indicate that exposures to elevated concentrations of ambient PM 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 ("HRV"). Such changes in heart rate variability may reflect changes in cardiac autonomic function and the risk of sudden cardiac death. In the Pope *et al.* study, repeated ambulatory ECG monitoring was conducted on seven subjects for a total of twenty-nine 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. This confirms, at the individual level, that 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.

Epidemiologic research conducted on U.S. residents has indicated that acute short-term exposures to PM air pollution are associated with increased risk of mortality. For example, a nationwide time-series statistical analysis of daily death counts by the Health Effects Institute (HEI, 2003) examined mortality and PM_{10} air pollution (a subset of particulate matter air pollution that is less than 10 μm in diameter, including $PM_{2.5}$) in ninety cities across the United States, finding that, for each increase of 10 $\mu g/m^3$ in daily PM_{10} air pollution concentration, there is an associated increase of approximately 0.3% in the *daily* risk of death by the public. Indeed, and I concur, the most recent U.S. EPA Particulate Matter Integrated Science Assessment ("ISA") (USEPA, 2009) unequivocally states that "[t]ogether, the collective evidence from epidemiologic, controlled human exposure, and toxicological studies is sufficient to conclude

that *a causal relationship exists between short term exposures to $PM_{2.5}$ and cardiovascular effects . . . and mortality.*"[1]

Q7. What about long-term exposures to $PM_{2.5}$? Are there cumulative effects of exposures, day after day, year after year, by people routinely exposed to such pollution, such as those who will reside near the proposed facility?

A. With respect to $PM_{2.5}$ from power plants, my recent studies have found that long-term exposure to combustion-related fine particulate air pollution is an important environmental risk factor for cardiopulmonary and lung cancer mortality. In addition to the acute health effects associated with daily PM pollution, long-term exposure to fine PM is also associated with increased lifetime risk of death and has been estimated to take years from the life expectancy of people living in the most polluted cities, relative to those living in cleaner cities. For example, in the Six-Cities Study (that was a key basis for the setting of the original $PM_{2.5}$ annual standard in 1997), Dockery *et al.* (1993) analyzed survival probabilities among 8,111 adults living in six cities in the central and eastern portions of the United States during the 1970s and 80s. The cities were: Portage, WI (P); Topeka, KS (T); a section of St. Louis, MO (L); Steubenville, OH (S); Watertown, MA (M); and Kingston-Harriman, TN (K). Air quality was averaged over the period of study in order to study long-term (chronic) effects. As shown in Figure 1, it was 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.

[1] U.S. Environmental Protection Agency (2009). (emphasis added).

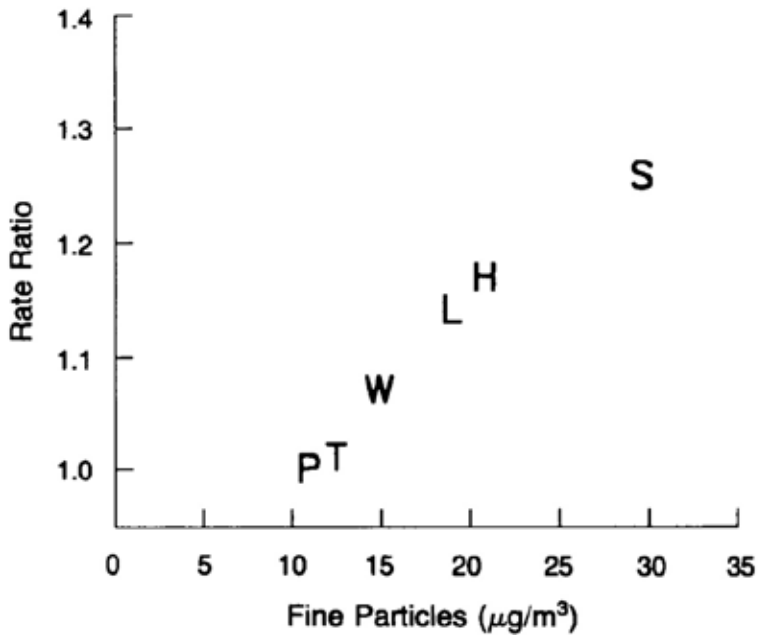

Figure 1. The Harvard Six-Cities Study showed that the lifetime risk of death increased across 6 U.S. cities as the average fine PM levels increased. (Source: Dockery et al., 1993.)

In addition, a study that I and co-authors published in the Journal of the American Medical Association ("JAMA"), shows that long-term exposure to combustion-related fine particulate air pollution is an important environmental risk factor for cardiopulmonary and lung cancer mortality. Indeed, as shown in Figure 2, this study indicates that the increase in risk of lung cancer from long-term exposure to $PM_{2.5}$ in a polluted city 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).

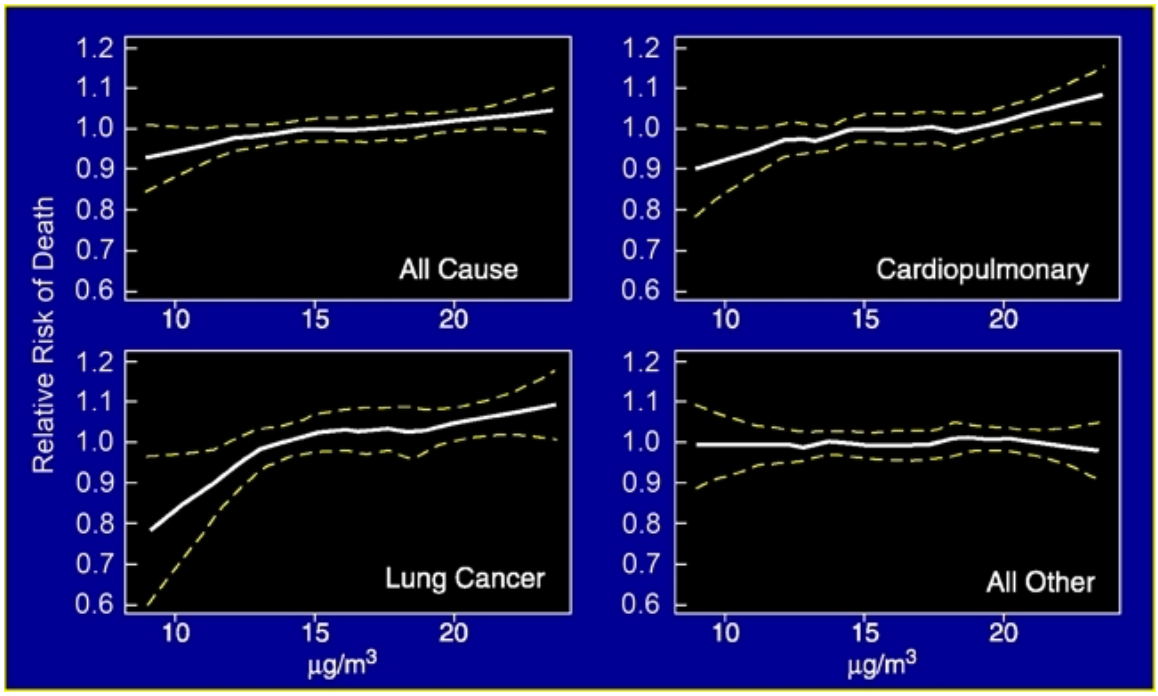

Figure 2. Cardiopulmonary and Lung Cancer Mortality Risks Increase Monotonically with Exposure to Long-Term Fine PM. (Adapted from: Pope, Burnett, Thun, Calle, Krewski, Ito, and Thurston, 2002.)

Moreover, long-term exposure to fine particles has been estimated to take more than a year from the life expectancy of people living in the most polluted cities, relative to those living in cleaner cities. For example, Brunekreef (1997) reviewed the available evidence of the mortality effects of long-term exposure to PM air pollution and, using life table methods, derived an estimate of the reduction in life expectancy implied by those effect estimates. Based on the results of Pope *et al.* (1995) and Dockery *et al.* (1993), a relative risk of 1.1 per 10 ug/m^3 exposure over fifteen years was assumed for the effect of fine PM air pollution on men 25–75 years of age. A 1992 life table for men in the Netherlands was developed for ten successive five-year categories that make up the 25–75 year old age range. Life expectancy of a twenty-five-year-old was then calculated for this base case and compared with the calculated life expectancy for the PM exposed case where the death rates were increased in each age group by a factor of

1.1. A difference of 1.11 years was found between the "exposed" and "clean air" cohorts' overall life expectancy at age twenty-five. A similar calculation by the authors for the 1969–71 life table for U.S. white males yielded an even larger reduction of 1.31 years for the entire population's life expectancy at age twenty-five. Thus, these calculations indicate that differences in long-term exposure to ambient $PM_{2.5}$ can have substantial effects on life expectancy.

The above-discussed increases in mortality documented by these studies represents only the "tip of the iceberg" of effects that would result. As shown in Figure 3 below, for every death associated with air pollution, there is a pyramid of much greater numbers of morbidity effects, including hospital admissions, emergency department visits, doctor visits, missed work days, missed school days, asthma symptoms days, etc. Clearly, when the whole scope of other adverse health effects associated with these air pollution deaths is considered, there is no doubt as to the significance of these adverse effects.

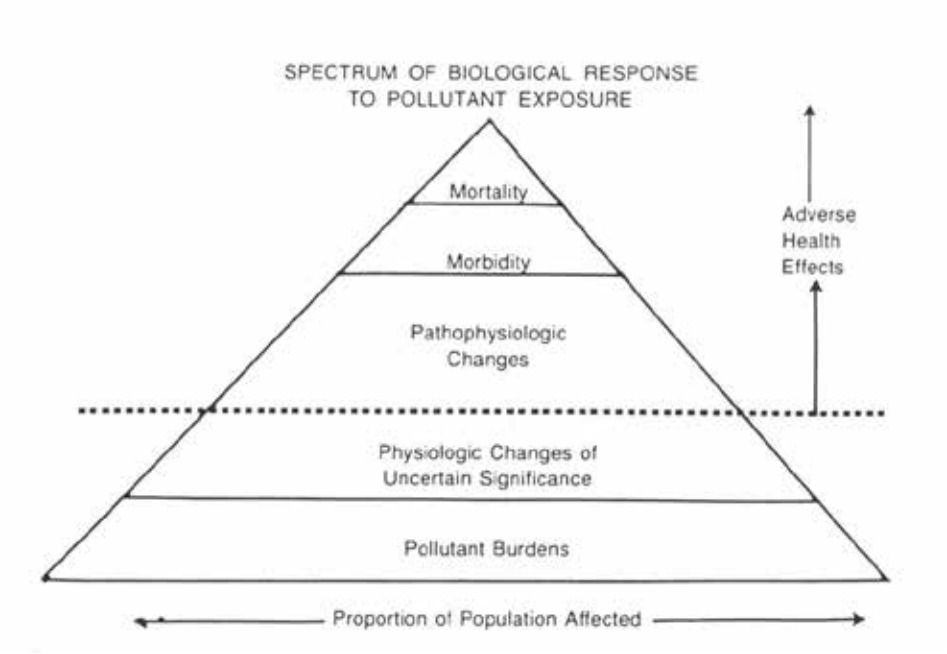

Figure 3. The Pyramid of Adverse Health Effects of Air Pollution on Health. (From: Guidelines as to what constitutes an adverse respiratory health effect, with special reference to epidemiologic studies of air pollution. Am. Rev. Respir. Dis. 1985 Apr; 131(4):666-8.)

Q8. Is there epidemiological evidence that power plant $PM_{2.5}$ emissions are more toxic per pound of pollution than other sources' $PM_{2.5}$?

A. With respect to $PM_{2.5}$ from power plants, my recent studies, and those by others, have also found that long-term exposure to combustion-related fine particulate air pollution is an important environmental risk factor for cardiopulmonary and lung cancer mortality. Air pollutants associated with fossil fuel combustion (e.g., from oil, coal, and natural-gas-fired power plants) have well-documented adverse human health effects. The health impact is particularly high for particulate matter from fossil-fuel-burning facilities, such as coal burning, which has been associated with an ischemic heart disease mortality risk that is roughly five times that of the average for $PM_{2.5}$ particles in general (Thurston *et al.*, 2016), and more damaging per $\mu g/m^3$ than $PM_{2.5}$ than other common sources (Figure 4).

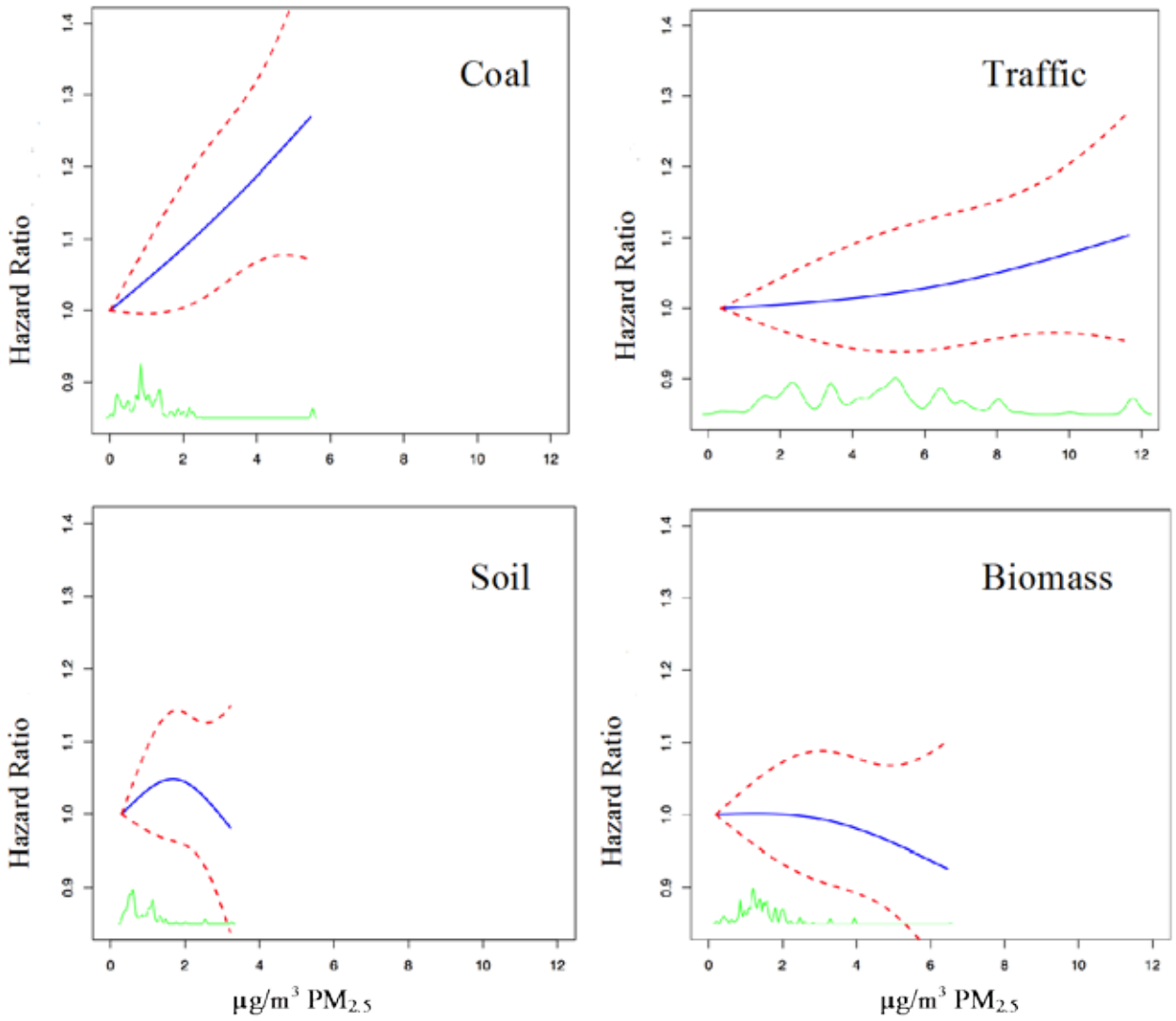

Figure 4. Concentration-response curve (solid lines) and 95% confidence intervals (dashed lines) for source-specific $PM_{2.5}$ mass in the US American Cancer Society (ACS) Cohort. (Thurston et al., 2016).

Q9. Why did the United States Environmental Protection Agency develop National Ambient Air Quality Standards ("NAAQS") for $PM_{2.5}$?

A. The EPA is required under Sections 108 and 109 of the Clean Air Act to periodically evaluate the air quality criteria that reflect the latest scientific information relevant to review each of the regulated air pollutant's NAAQS. The EPA recognized the adverse health effects of

small PM air pollution as early as 1987 when, pursuant to its authority under the Clean Air Act, it promulgated a NAAQS for particulate matter that is 10 micrometers in diameter or smaller ("PM_{10}"). The NAAQS promulgated by EPA are required for certain air pollutants "that may reasonably be anticipated to endanger public health and welfare." The NAAQS' air criteria must be "requisite to protect the public health" with an "adequate margin of safety." Under the particulate matter NAAQS, states must reduce PM_{10} concentrations in their ambient atmosphere to no more than fifty micrograms per cubic meter on an annual average basis, and to no more than 150 micrograms per cubic meter on an average twenty-four-hour period. Prior to 1987, EPA's particulate NAAQS had only regulated total suspended particulate matter. The focus in 1987 on smaller particles—that is, ten micrometers or less—resulted from increasing scientific evidence that human inhalation of smaller particles had more serious respiratory effects than larger particles.

In 1994, EPA began the process of again reviewing its particulate matter standards. In 1996, EPA proposed a new NAAQS for even smaller particles—those that are 2.5 micrometers in diameter or smaller ("$PM_{2.5}$"). In July 1997, upon determining that the PM_{10} NAAQS is no longer protective of human health, 62 Fed. Reg. 38652, 38665 (July 18, 1997), EPA issued a final rule revising the NAAQS for PM to include two new NAAQS for PM2.5. These consisted of: (1) a long-term annual standard of 15 ug/m3, annual arithmetic mean, averaged over three years from single or multiple community-oriented monitors; and (2) a twenty-four-hour standard that is met when the three-year average of the 98th percentile of twenty-four-hour $PM_{2.5}$ concentrations at each population-oriented monitor within an area does not exceed 65 ug/m3. 62 Fed. Reg. 38652, 38679 (July 18, 1997). These new $PM_{2.5}$ standards were based on an increasing scientific consensus that the current NAAQS for PM_{10} was not sufficiently protective

of human health. EPA's scientific review concluded that fine particles, in the 2.5 micrometer and smaller range, penetrate more deeply into the lungs, and may be more likely than coarse particles to contribute to the health effects (e.g., premature mortality and hospital admissions) found in a number of recently published community epidemiological studies at concentrations that extend well below those allowed by the current PM_{10} standards. As EPA stated in its rulemaking, a greatly expanded body of community epidemiological studies provides "evidence that serious health effects (mortality, exacerbation of chronic disease, increased hospital admissions, etc.) are associated with exposures to ambient levels of PM, even in concentrations below current U.S. PM standard." (Federal Register, 1997). Since that time, the U.S. EPA has lowered the allowable limits of ambient concentration of $PM_{2.5}$ to 35 $\mu g/m^3$ and 12 $\mu g/m^3$ for the daily and annual standards, respectively, in recognition of its effects at lower levels of exposure.

Q10. What were the findings and conclusions recorded in those studies and documents?

A. The EPA PM Staff Paper at the time of the setting of the $PM_{2.5}$ standards concluded that "fine and coarse particles can be differentiated by their sources and formation processes, chemical composition, solubility, acidity, atmospheric lifetime and behavior, and transport distances." EPA also concludes that: "Primary fine particles are formed from condensation of high temperature vapors during combustion"; and that: "Fine mode PM is mainly composed of varying proportions of several major components: sulfates, nitrates, acids, ammonium, elemental carbon, organic carbon compounds, trace elements such as metals, and water." U.S. EPA, 1996.

Q11. What involvement have you had in the establishment of EPA's NAAQS for $PM_{2.5}$?

A. I have served as a contributing author of the 1996 and the 2003 PM Criteria documents. In addition, my research was cited by the U.S. EPA as a "key study" in promulgating both the

$PM_{2.5}$ and ozone air quality standards in the past. I was also called upon by both the U.S. House and Senate to testify regarding the human health effects of air pollution when they were considering these new air quality standards.

Q12. Is there a known safe level of exposure to $PM_{2.5}$?

A. There is no evidence to date that there is any threshold below which the adverse effects of air pollution will not occur. For example, the incremental effects of sulfate containing fine particles, and the lack of a threshold of air pollution effects at ambient levels are indicated for sulfate and hospital admissions in Figure 5 below.

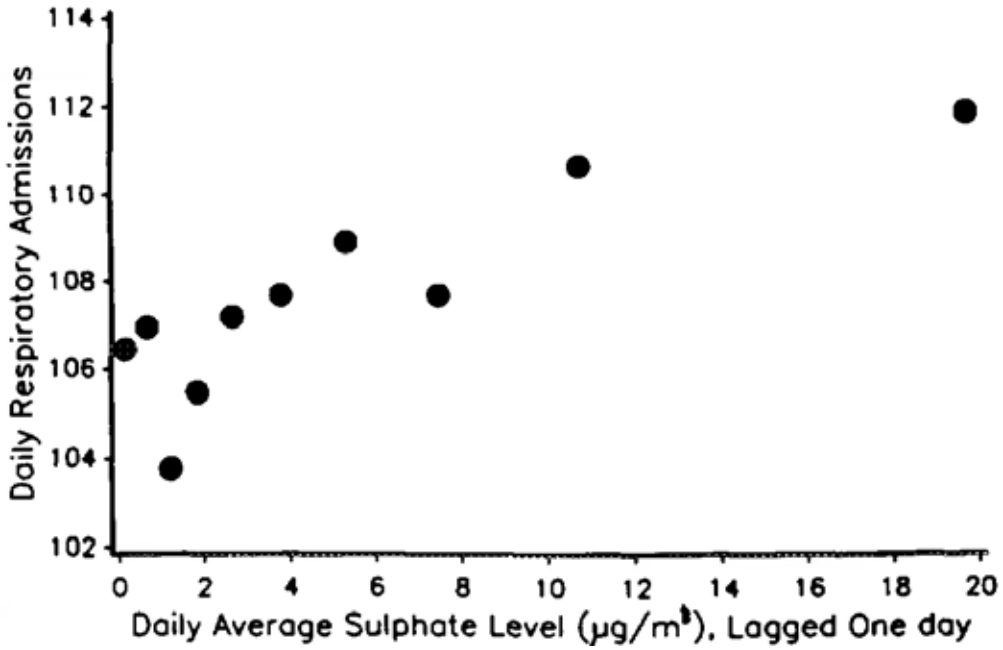

Figure 5. Average number of respiratory admissions among Ontario hospitals adjusted for other factors, by decile of the daily average sulfate fine particle concentration ($\mu g/m^3$). (Burnett *et al.*, 1994).

In addition, as displayed in Figure 6 below, my research has shown that increases in long-term exposure to $PM_{2.5}$ particulate matter air pollution are associated with increases in the risk of cardiovascular death among those exposed, even well below the present 12 µg/m^3 annual $PM_{2.5}$ air quality standard (Thurston *et al.*, 2016b). 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, even below the legal ambient pollution standards.

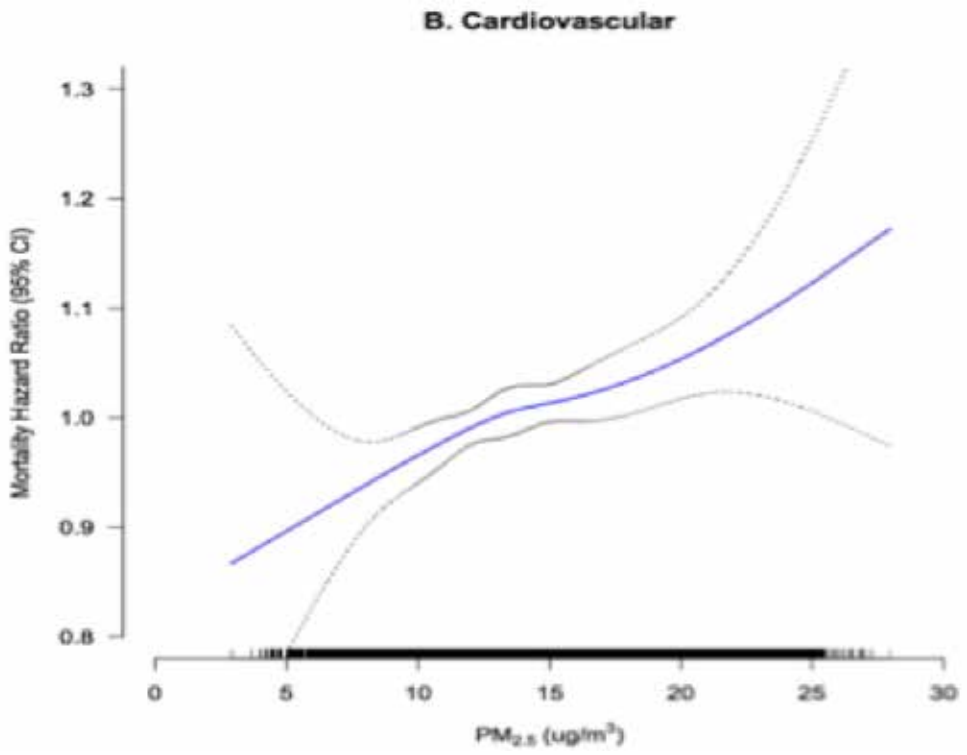

Figure 6. Mortality Risk from Cardiovascular Disease Increases with Rising PM2.5 Exposure, Even Well Below the Present US Ambient Air Quality Standard annual limit for PM2.5 (12 µg/m3). Thurston *et al*., 2016b.

Furthermore, in its calculations of the benefits of potentially reducing the $PM_{2.5}$ NAAQS, EPA has also implicitly acknowledged that there can be extant adverse health risks occurring below the NAAQS. For example, in a recent EPA Regulatory Impact Analysis for reducing the annual $PM_{2.5}$ standard from 15 $\mu g/m^3$ to 12 $\mu g/m^3$ (U.S. EPA, 2012), EPA included a figure summarizing the best, most current science regarding $PM_{2.5}$ health effects, which clearly illustrates that air pollution deaths occur below the existing $PM_{2.5}$ NAAQS (35 $\mu g/m^3$ for the daily standard, and 12 $\mu g/m^3$ for the annual standard). Figure 7 provides EPA's best estimate of the deaths that would be avoided by implementing the proposed more stringent standard, with roughly half of the avoided deaths occurring in places where the air would be cleaned to levels below (i.e., with air quality better than) the proposed air quality standard. While this particular EPA analysis is for the annual average concentrations, the same principle of effects occurring below the standard applies to the short-term $PM_{2.5}$ standard as well. Thus, just as cleaning the air below the standards would avoid more of those deaths, any increase in pollution will increase the risk of adverse effects at all levels of prevailing air pollution, even when the NAAQS standards are not violated.

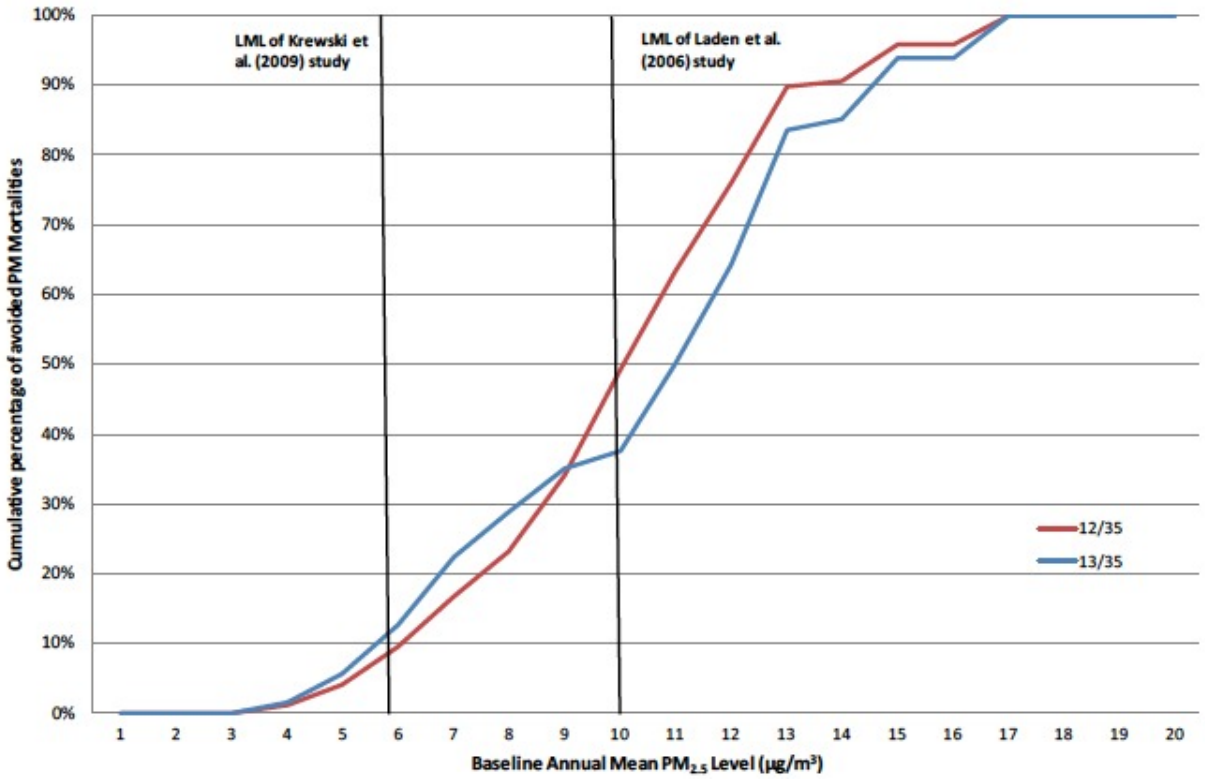

Figure 7. U.S. EPA Regulatory Impact Assessment of the Number of Premature PM2.5-Related Deaths Avoided for 12/35 vs. 13/35 Ambient PM2.5 Air Quality Standards. (LML = Lowest Measured Level of PM2.5 in the study population) (U.S. EPA 2012, Fig. 5-7).

It should be noted that the U.S. EPA agrees with me that meeting an air quality standard does not prevent significant adverse health effects from occurring in the exposed population. Indeed, in its 2013 rulemaking, adopting the revised annual particulate matter NAAQS standard, EPA explained that "evidence- and risk-based approaches using information from epidemiological studies to inform decisions on $PM_{2.5}$ standards are complicated by the recognition that *no population threshold, below which it can be concluded with confidence that $PM_{2.5}$-related effects do not occur, can be discerned from the available evidence*." (U.S. EPA, 2013) (emphasis added).

III. SOURCES, NATURE, CHEMICAL COMPOSITION, AND HUMAN HEALTH EFFECTS OF $PM_{2.5}$ AIR POLLUTION

Q13. Please describe the main sources of $PM_{2.5}$ emissions.

A. $PM_{2.5}$ is directly emitted by both stationary sources (e.g., power plants and other industrial sources) and mobile sources, such as diesel buses and trucks. $PM_{2.5}$ is also formed in the atmosphere from gaseous emissions, such as sulfur oxides from fossil fuel combustion in power plants, resulting in "secondary" $PM_{2.5.}$

Q14. Please explain the nature and chemical composition of $PM_{2.5}$ emitted from fossil-fuel burning power plants.

A. $PM_{2.5}$ air pollution has been carefully studied in the past few decades. PM 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 ("SOx") and nitrogen oxides ("NOx") also emitted by coal-fired power plants. After formation in the atmosphere, this secondary PM 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 ("µm")—a fraction of the diameter of a human hair. For example, after it is released from a smokestack, gaseous SOx is chemically converted in the atmosphere to become sulfate PM.

There is ever-growing scientific evidence indicating that PM air pollution emitted by fossil-fuel-burning electrical utility power plants is among the important contributors to the toxicity of PM. Evidence from historical pollution episodes, notably the London Fog episodes of the 1950s, indicate that extremely elevated daily particulate matter concentrations from fossil

fuel combustion may be associated with excess acute human mortality (Ministry of Health of Great Britain, 1954).

Recent epidemiological and toxicological evidence also suggests that the particles resulting from fossil-fuel utility power plant air emissions are among the most toxic in our air. Indeed, my own published analysis of U.S. mortality and PM by source category found that combustion-related particles were more strongly associated with variations in annual mortality rates across U.S. cities than were other components of PM (Ozkaynak and Thurston, 1987). More recently, an analysis by Laden and co-authors (2000) at Harvard University of PM sources and daily pollution confirms that power plant combustion particles, along with automobile pollution, were among the PM components that most affected daily variations in mortality. In addition, toxicological studies have indicated that particles resulting from fossil-fuel combustion that contain metals are very toxic to cells in the lung. Thus, both the toxicological and epidemiological evidence available indicate that pollution from fossil-fuel power plants are of great human health concern.

Q15. What is known about the toxic composition of $PM_{2.5}$ emitted by power plants that burn fossil fuels, such as natural gas?

A. The conclusion that power plant particle pollution is one of the more toxic types of particles that we breathe is supported by the facts that combustion particles have different sizes, physio-chemical characteristics, and deposit in different parts of the lung than other more "natural" particles, such as wind-blown soil.

In the past, I have testified that this is especially true of coal-fired power plant emissions, but since all fossil-fuel emissions particles share certain key characteristics, such as containing

transition metals, this is also true of oil-fired and natural-gas-fired emissions. Although the mass of particles emitted per kilowatt-hour ("kW/hr") is less for oil- and gas-fired units, there is no reason to believe that they are less toxic on a pound for pound basis, and every reason to expect they would be more toxic, since there are so many more ultrafine particles emitted by natural gas burning plants, per pound of emission, and ultrafine (e.g., nanoparticles) are thought to be far more toxic than large particles because they can reach deep into the lung, and even pass across the lung's membranes into the bloodstream to travel systemically throughout the body of a person who breathes them.

Such fossil-fuel combustion particles can defeat the body's natural defenses, and thereby have a far greater adverse effect on health. In particular, these power plant particles are enriched in toxic metals, such as arsenic and cadmium, as well as in transition metals, such as iron and vanadium, that can cause damaging oxidative stress in lung cells (*see, e.g.*, Costa *et al.*, 1997; Dreher *et al.*, 1997; Lay *et al.*, 1999). This may also be especially true in the case of natural-gas-fired power plant particles because that $PM_{2.5}$ is composed of very small ultrafine particles that bypass the natural defenses of the lung, and therefore can penetrate deep into the lung where they are not easily cleared, and can therefore reside there for long times, potentially causing significant damage to the lung and to the human body. Thus, PM air pollution from power plants**,** including natural-gas-fired units, is cause for special concern, and the health of persons in nearby populations can be adversely affected by this power-plant-related air pollution.

Recent epidemiological studies support the conclusion that sulfate containing particles, such as those that come from the proposed NOPS, are among the most toxic particles (e.g., Ozkaynak and Thurston,1987; Dockery *et al.*,1993; Pope *et al.*, 1995, and 2002). In my own published research examining the associations of PM with human mortality, we have found that

PM emitted from fossil-fuel combustion is more strongly associated with mortality than particles from many other sources, such as soil-derived and automobile-emission-related particles (Ozkaynak and Thurston, 1987). An example of the relationship that has been found between sulfate fine particle pollution and mortality is shown in Figure 8.

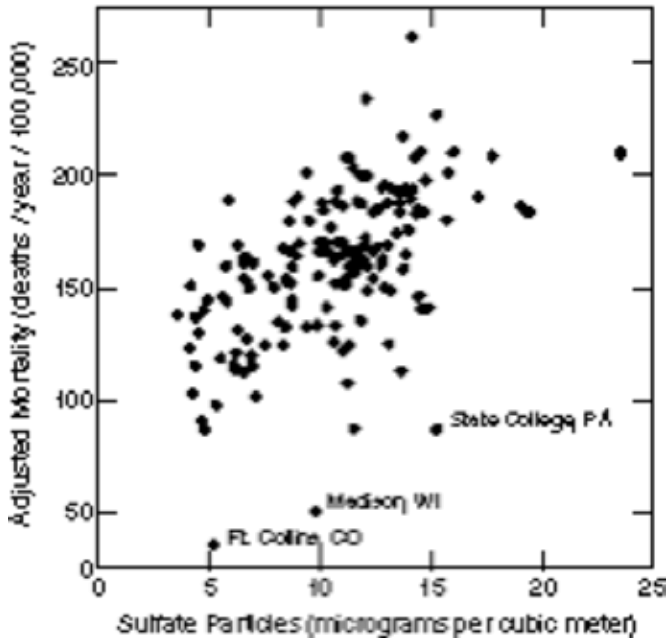

Figure 8. Age-, sex-, and race-adjusted population-based mortality rates for U.S. metropolitan areas in 1980 plotted versus mean sulfate fine particle air pollution levels. (Adapted from: Pope et al., 1995).

Lab studies also suggest that the presence of acidity in particles, which is usually the case for power-plant-combustion emissions, increases the toxicity of PM (e.g., Chen, *et al.*, 1990). This conclusion is supported by studies of human respiratory cells (e.g., Veronesi *et al.*, 1999). The presence of acidity increases the solubility of toxic metals, thereby making them more biologically available to damage the body. This may be an important pathway by which acidic particles, such as those resulting from fossil-fuel-burning power plants, can have heightened toxicity versus other ambient particles, and provides a plausible physiological mechanism for the

epidemiological associations found between acidic particle exposures and adverse human health effects.

Q16. What is the significance of "active sites" on the particles?

A: Freshly combusted particles will have sharp edges, and will be composed, in part, of unoxidized compounds that have not been neutralized. The sharp edges are the active sites at which these particles irritate and interact with the lining of the lung. Natural gas particles from combustion turbines, also include sulfuric acid mist ("SAM"), as acknowledged on page 1-4 and Table 1-1 of the Information Package 4, dated July 22, 2016.[2] Unoxidized and acidic compounds would be more reactive, and therefore, be more likely to irritate and interact with the lining of the lung, and, in combination with the metallic components of fine particles, cause more damage than aged and neutralized particles.

Q17. Is there any evidence that primary $PM_{2.5}$ emitted from natural-gas-burning air pollution sources, such as the proposed facility, is less toxic than $PM_{2.5}$ emitted from power plants that burn other forms of fossil fuel?

A. No. Although the quantities, in terms of mass per unit Btu are lower, there is no evidence that, on a pound for pound basis, the particles from gas-fired power plants are any less toxic than the $PM_{2.5}$ from other fossil fuels. Indeed, because natural-gas-fired sources can emit a much greater percentage of the particles as ultrafine particles, which have a much higher surface area per mass than larger particles, it is likely that there is a much *greater* effect per pound of $PM_{2.5}$ emitted by gas-fired sources than for $PM_{2.5}$ emitted by sources burning other fossil fuels. For

[2] *See* Exhibit GT-3.

this reason, the impacts of the proposed plant in terms of $PM_{2.5}$ mass concentration are an inadequate indication of the health risks associated with the proposed NOPS.

Q18. Can you summarize the results from your own research on the health effects of $PM_{2.5}$?

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, hospital admissions, and mortality. In particular, I have found that both ozone and 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; *see also* U.S. EPA, 2001). I was a Principal Investigator of a study published in the Journal of the American Medical Association (JAMA) in March of 2002, that shows that long-term exposure to combustion-related fine particulate air pollution is an important environmental risk factor for cardiopulmonary and lung cancer mortality. In fact, it was found that the increase in risk of lung cancer from long-term exposure to $PM_{2.5}$ in a polluted city 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. (Pope *et al.*, 2002).

Q19. Is there scientific evidence that certain subpopulations are particularly susceptible to adverse health impacts from an increase in $PM_{2.5}$ in the ambient air?

A. Among the groups of persons found in scientific research to be especially affected by environmental insults, including particulate matter air pollution, are: the very young, the poor, the very old, and persons with pre-existing health conditions, such as heart disease and asthma. (*see, e.g.*, U.S. EPA, 1996).

Q20. Do ethnicity, age, or pre-existing medical conditions play a role in determining whether adverse health impacts are the predictable result of exposure to increased $PM_{2.5}$ emissions?

A. Yes, they do. Analyses by me and by others in the field of air pollution health effects indicate that the poor are especially at risk from air pollution (*e.g.*, Gwynn and Thurston, 2001). Similarly, older adults are at greater risk of severe adverse outcomes from air pollution. Also, children, a population known to be especially susceptible to the effects of air pollution because their bodies are developing (and because they spend larger amounts of time exercising outside) are an especially affected sub-population that is well represented in the community surrounding the plant. This subpopulation of children can be expected to be among those most strongly affected by any increases in $PM_{2.5}$ concentrations in the vicinity of the plant.

IV. $PM_{2.5}$ ASSESSMENT OF THE PROJECT

Q21. Have you reviewed the analysis of $PM_{2.5}$ impacts from the proposed facility and, if so, do you believe that it is an adequate analysis of the $PM_{2.5}$-related health impacts from the proposed facility?

A. I have reviewed this analysis and find it to be inadequate because it makes no attempt to perform a health-risk analysis of $PM_{2.5}$ emissions from the proposed facility. It is not sufficient

to consider the pollution increments alone. It is necessary to do an assessment of the potential health implications of the $PM_{2.5}$ impacts. Such an assessment would entail utilizing the published risk factors for $PM_{2.5}$ air pollution and applying them to the appropriate populations in order to derive the numbers of added incidences of adverse health effects (e.g., deaths, hospital admissions, emergency room visits, asthma symptom days, etc.), such as has been done by the U.S. EPA in conducting its Regulatory Impact Analysis for the $PM_{2.5}$ standards. While the increases in risk from the addition of even small increments of $PM_{2.5}$ might be relatively small across the city, when multiplied by the hundreds of thousands of New Orleans residents, they would add up quickly. This is exemplified in a recent publication in the journal SCIENCE that I co-authored, which presented estimates of the potential health benefits in New York City and other cities that would be achievable by reductions in ozone and particulate matter air pollution associated with implementing measures to reduce "greenhouse gas" emissions from fossil fuel combustion, as shown below.

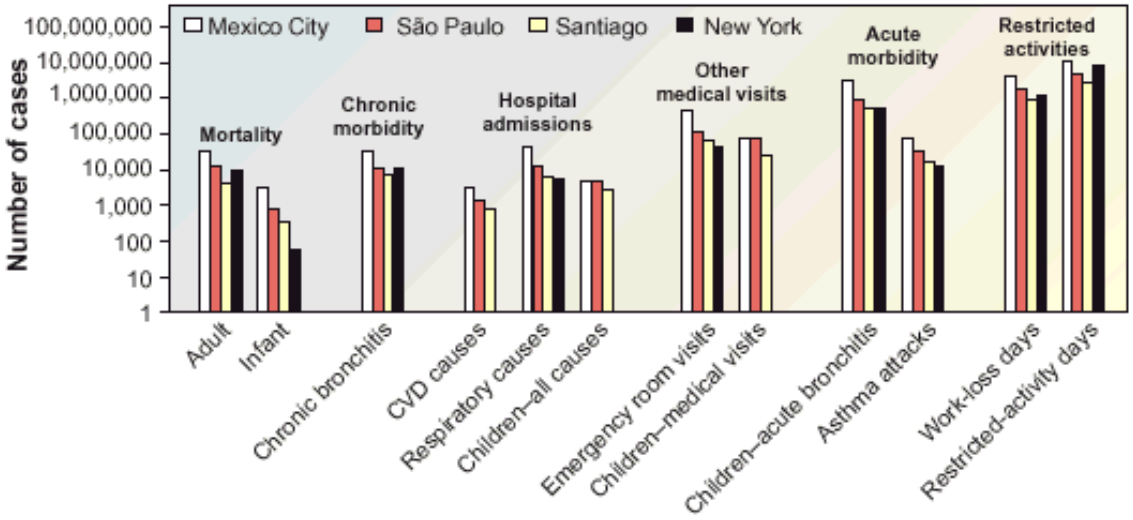

Figure 9. Potential human health benefits from reductions in ozone and particulate matter air pollution associated with implementing GHG mitigation measures (2001 –2020). (CVD= cardiovascular disease) (Cifuentes et al., 2001).

Q22. Do you find it reasonable to assume that nearby residents will be exposed to these emissions even while inside their homes?

A. Yes, outdoor air pollution, and especially fine particle pollution, is known to infiltrate into buildings with high efficiency as exchanges between outdoor and indoor air occur (via transfer through windows, doors, ventilation systems, etc.). As the levels of air pollution in the air outside a building increase, the exposures of residents inside the building to PM of outdoor origins will therefore also rise.

Q23. Wouldn't exposure to indoor sources of PM pollution be much greater than exposure to PM pollution coming indoors from outside sources?

A. While other $PM_{2.5}$ exposures, such as indoor air pollution, may have health effects, they are independent of the impacts of increases of exposures to $PM_{2.5}$ of outdoor origins in general and of the proposed plant in particular. If the levels of outdoor $PM_{2.5}$ impinging the living areas of residents increase, then it can be expected that their personal exposures to $PM_{2.5}$ of outdoor origins, and their associated health risks, will rise proportionately.

Q24. Please discuss in more detail your critique of the supplemental conclusion and analysis with respect to the $PM_{2.5}$ impacts from the proposed facility.

A. I disagree with the statement in the NOPS application Supplement that: "in no case, will the emissions cause air quality to exceed regulatory standards, which are protective of human health." This is the case because, as noted above, there are potentially significant adverse health effects experienced below the ambient standards, and because fossil fuel combustion particles to be exhausted into the atmosphere by this proposed plant are much more toxic than typical

particles. Thus, the present mass-based $PM_{2.5}$ standards are not effective in protecting the public health from such ultrafine particle emissions that will be caused by the proposed NOPS.

There are two known characteristics of natural gas-fired power plant particles that make them likely to have especially strong health effects, on a per-pound basis, than most ambient $PM_{2.5}$. First, they have a higher percentage of ultrafine particles, as compared with other fossil fuel options (see Figure 10). These ultrafine particles have very high surface areas, relative to other fossil fuel emissions, and penetrate deep into the lungs when breathed. Second, they, like other fossil fuel combustion particles, contain a high percentage of toxic transition metals. These characteristics tend to increase the dose and toxicity of gas-fired power plant particles, relative to most other ambient particles.

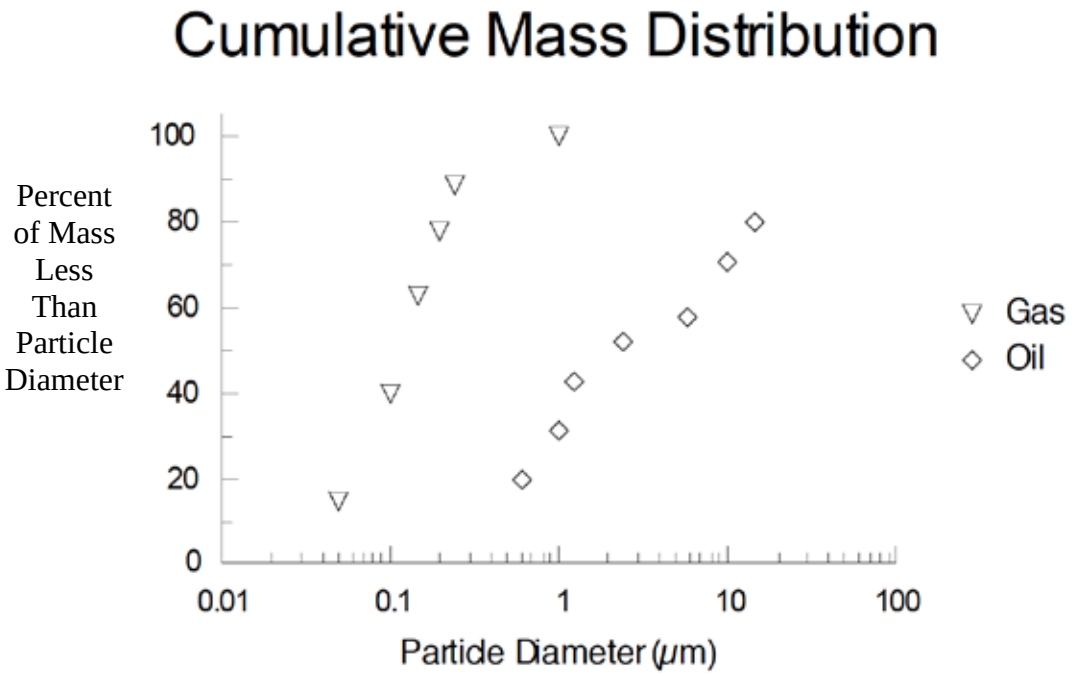

Figure 10. Comparison of Particle Mass Size Distribution for Natural Gas vs. Oil Combustion Emissions. Source: Environmental Protection Agency, "Compilation of Air Pollutant Emission Factors, Volume 1: Stationary Point and Area Sources. Fifth Edition," AP-42. Table 1.3-4 (9/98), Table 3.1-1 (10/96).

Furthermore, the likely co-presence of strongly acidic vapor in the NOPS emissions will tend to further enhance the bio-availability, and hence the toxicity, of the metals that are present. Sulfuric acid is the most strongly acidic form, with a pH of less than 1 at 50% relative humidity (RH), and ammonium bisulfate is also very strongly acidic, with a pH of 1-2 at 50% RH, while ammonium sulfate is only weakly acidic, with a pH of 5-6 (vs. a pH of 7.0 for completely neutral conditions) (NRC, 1978). Although the applicant has provided no data indicating the breakdown of ammonia sulfates in its proposed plant's emissions, the plant emissions can be expected to be in a strongly acidic, and therefore more toxic, form. The potential toxicity of exposure to these gas-fired power plant metals cannot be dismissed, even at very low $PM_{2.5}$ mass levels.

Thus, the potential health effects of $PM_{2.5}$ emissions from the new plant cannot be dismissed. Because of their high ultrafine fraction, their composition, and the likely co-presence of acidic vapors, they potentially could be more toxic than other forms of particulate matter.

Q25. Do studies using laboratory animals and humans support the conclusion that ambient or moderately elevated concentrations of soluble sulfates or nitrates harm health?

A. Yes. Some controlled animal exposures of air pollution have shown adverse effects at $PM_{2.5}$ at levels close to ambient levels. Recent animal experiments by Godleski and co-workers at Harvard indicate that exposures to elevated concentrations of ambient particulate matter can result in cardiac-related problems in animals (Godleski *et al.*, 1996; Godleski, 2000). The most biologically and clinically significant finding was that in dogs with induced coronary occlusion, particles affected one of the major ECG signs of myocardial ischemia in humans, known as elevation of the ST segment. Consistent cardiac effects at the biological level have also been found in human epidemiological studies, as well. For example, Pope *et al.* (1999) and Gold *et al.*

(2000) report that PM exposure is associated with changes in human heart rate variability, confirming that biological changes do occur in heart function as a result of PM exposure.

Q26. 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 consist of very tiny particles that have large surface areas available to interact with the lung. And the particles have toxic transition metals in them.

Second, because fossil fuel particles, especially those near a power plant, are freshly combusted, they have more active sites on them. Work by Oberdorster *et al.* (1995) has shown PM concentrations at ambient levels, 60 $\mu g/m^3$ and less, cause mortality in healthy rats. And then they found the aging of those fumes with aggregation of the ultrafine particles significantly decreased their toxicity. Thus, fresher, recently emitted, particles, such as those breathed by persons living near a power plant, are more toxic.

Q27. The applicant claims (Entergy Supplemental Testimony, Nov. 18, 2016) that "the emissions from the proposed NOPS will result from combustion of clean burning natural gas; in no case, will the emissions cause air quality to exceed regulatory standards, which are protective of human health and the environment." Do you agree?

A. No. As discussed above, even the U.S. EPA acknowledges that there are health effects from air pollution at levels below the prevailing ambient standards, so any addition to the local air pollution will have a proportionate addition to the risk of adverse health effects from that

exposure. Furthermore, the particles emitted from the proposed new facility will be dominated by ultrafine nanoparticles, which are not protected against by the $PM_{2.5}$ mass standard, which is affected primarily by larger particles with less surface area per pound of pollution, and less able to enter the bloodstream and affect those breathing this pollution than more usual $PM_{2.5}$ mass.

Q28. Do you agree with the designation, in Table 1-1 of the "Application for Modification of the Part 70 Operating Permit Acid Ran(sic) Michoud Electric Generating Plant" (March 2016),[3] of the decommissioned former power plant air quality emissions and impacts as the baseline for comparison to the impacts of the proposed new plant?

A. No. Since the old plant is no longer in operation, the logical and relevant baseline would be zero emissions, not the emissions of a now-decommissioned, and logically irrelevant, plant. The air quality impacts of the proposed plant would be in addition to the present prevailing ambient conditions and pollutant concentrations. Similarly, the deactivated unit should not be employed to compute net project emissions, as seen in Table 1-1 in the Information Package 4, dated July 22, 2016.[4]

Q29. Are the startup/shutdown operations an important factor in assessing the potential adverse health impacts of the proposed power plant?

A. Yes, because of the expected "peaking" use of this plant, ENOI (in the Information Package 4, dated July 22, 2016)[5] reports a potential/expected total of 500 startup/shutdowns in a year (TABLE D-2D: StartUp/Shutdown Emissions Summary Table), which is many more than would be associated with a base power generating plant in the same time period. Thus, this

[3] Attached herein as Exhibit GT-2.
[4] *See* Ex. GT-3.
[5] *See id*.

would be a very prevalent case, and the extreme increases in Volatile Organic Compounds and in carbon monoxide emissions predicted in Tables D-2B and D-2D (versus Normal Operations emissions) are of concern, due to their frequent occurrence throughout the year during the startups/shutdowns at this proposed plant.

Q30. Do you agree that with the application's Technical Report (November 16, 2016, pg. 16) that an impact comparison of this plant's emissions to those of a hypothetical residential outdoor gathering beside a 2-kW home generator facility is a useful or appropriate comparison?

A. No. These are very different and incomparable situations in that the power plant being proposed is being operated by a business for their profits, so the benefits of its operation all go to the business, while the impacts of the resulting air pollution fall on the nearby residents who do not share in the proceeds. In contrast, the residential generator's operation would be by a property owner for their own use, and therefore they would be taking the pollution risks for their own personal benefit. Thus, a much more stringent environmental impact standard must apply to the power plant operation, since the adversely affected parties get no direct benefit. In addition, if you ever heard one of these residential generators in operation, you would know that they are so loud that no one would ever gather beside one of them even briefly, while the power plant will operate year-round, so it is both an impractical and an inappropriate example, irrelevant to this situation.

Q31. What are your final conclusions in regard to the expected health impacts of increased $PM_{2.5}$ emissions from the proposed facility?

A. Additional emissions from the proposed facility will add to the existing levels of $PM_{2.5}$ in the vicinity of the plant, and, because no threshold of air pollution effects has yet been found, any incremental $PM_{2.5}$ exposures add an incremental adverse health risk to nearby residents from power plant air pollution. Also, the fact that an increased population risk of health effects constitutes an individual adverse health effect has been confirmed by the American Thoracic Society (American Thoracic Society, What constitutes an adverse health effect of air pollution? Official statement of the American Thoracic Society (2000). Therefore, any action that increases the ambient concentration of $PM_{2.5}$ in this area will have an adverse impact on human health in the exposed population. These incremental health effects risks would in no way be mitigated or negated by other respiratory health effects risks, such as indoor air pollution exposures, which would represent independent health risks of their own. I therefore conclude that, to the extent that the proposed facility will emit additional levels of $PM_{2.5}$, it will cause an increase in the risk of adverse health effects among those who breathe that pollution, and especially for those who live within the most affected areas immediately surrounding the plant.

Q32. Does this conclude your testimony?

A. Yes.

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