Q. Please state your name, affiliation and title.

A. I am George D. Thurston, Sc.D. I am an Associate Professor at the New York University School of Medicine in the Department of Environmental Medicine. See Exhibit GT-1. My business address is: Three Catherine Ct., Chester, NY 10918. I offer this expert direct testimony on behalf of Orange Environment.

Q. What is the purpose of your testimony?

A. I am providing expert testimony that addresses the public health impacts of emissions of fine particulate matter ("$PM_{2.5}$") generally and, specifically, the expected public health impacts of $PM_{2.5}$ emissions from the proposed 540 MW Calpine Wawayanda Energy Center (Project). My testimony will address three relevant questions previously posed by the New York State Board on Electric Generation Siting and the Environment in its January 24, 2002 Order Concerning Interlocutory Appeals from Article X Issues Ruling regarding the NYPA Astoria plant in New York City, NY (CASE 99-F-1627). My testimony will also critique the $PM_{2.5}$ analysis performed by Calpine. I conclude that Calpine's analysis of the health impacts of $PM_{2.5}$ emissions from its proposed facility is inadequate, and that $PM_{2.5}$ emissions from this facility can be expected to increase adverse health risks in the surrounding community?].

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

A. I received 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. 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 in recent 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 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. Most recently, I was a Principal Investigator of a study that shows 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. 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. JAMA 2002; 287: 1132-1141. The publications reviewed or relied upon for this testimony are listed at the end of this report as "Literature Cited."

Q. 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. In the last 4 years, I have provided testimony in the case: U.S. v. LTV Steel Company, Inc., Case No. 1:98CV3012, N.D. Ohio, Judge O'Malley. I have also

provided verbal testimony on health impacts of $PM_{2.5}$ at the Department of Environmental Conservation ("DEC") issues conference in In the Matter of the Application of St. Lawrence Cement Company, Application Number 4-1040-00011/00001(July 20, 2001, Hudson, NY) and in the Article X issues conference in the Wayawanda Energy Center proceeding, Case 00-F-1256 (Application by Calpine Construction Finance Company, L.P.) (December 18, 2001, Middletown, NY). Most recently, I submitted direct and rebuttal expert testimony on $PM_{2.5}$ health impacts in the Con Edison East River Repowering Proceeding, Case 99-F-1314 and the NYPA power plant in Northern Queens, NY (CASE 99-F-1627).

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

A. Yes.

The following direct testimony is submitted in response to Question 1 of the Siting Board's January 24 Order: "What is known about the sources of PM2.5, the nature of PM2.5 and the chemical composition of PM2.5? Also, is there a significant regional transport component to PM2.5?"

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

A. The United States Environmental Protection Agency ("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 of each

regulated air pollutant's National Ambient Air Quality Standard ("NAAQS"). The EPA recognized the adverse health effects of small particulate matter (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 50 micrograms per cubic meter on an annual average basis, and to no more than 150 micrograms per cubic meter on an average 24-hour period. Prior to 1987, EPA's particulate NAAQS had only regulated total suspended particulate matter. Its focus in 1987 on smaller particles -- that is, 10 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 $PM_{2.5}$. These consisted of: 1) a long-term annual standard of 15 ug/m^3, annual arithmetic mean, averaged over three years

from single or multiple community-oriented monitors; and 2) a 24-hour standard that is met when the three-year average of the 98th percentile of 24-hour $PM_{2.5}$ concentrations at each population-oriented monitor within an area does not exceed 65 ug/m^3. 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 provide "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).

In addition, the U.S. EPA's most recent reanalysis of the issue of the potential for the impacts of fine particles on human health has reconfirmed that fine particles, such as those that result from the air emissions from power plants, are significantly adverse to human health (U.S. EPA, 2001).

Q. What are the relevant sections of the 1996 PM Criteria Document and the EPA PM Staff Paper that address the sources, nature and chemical composition of PM2.5?

A. Chapter 3 of Volume I of the 1996 PM Criteria Document (EPA/600/P-95/001aF), and Section IV of the 1996 OAQPS Staff Paper (EPA-452\R-96-013).

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

A. The EPA PM Staff Paper concludes 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, 1996b).

Q. Is control of PM_{10} sufficient to protect the public health with an adequate margin of safety?

A. No. PM_{10} is composed of both coarse and fine particles, while $PM_{2.5}$ focuses on the fine particles, which are thought to contain the most toxic constituents, and can efficiently bypass the lung's defenses and reach and damage the deepest recesses of the lung, unlike coarse particles. As pointed out in the EPA Staff paper: "In summary, the fine and course mode particles are distinct entities with

differing sources and formation processes, chemical composition, atmospheric lifetimes and behaviors and transport distances. The CD concludes that these profound differences alone justify consideration of fine and coarse fraction particles as separate pollutants for measurement and development of control strategies. The fundamental differences between fine and coarse particles are also important considerations in assessing the available health effects and exposure information." (U.S. EPA, 1996b).

Moreover, EPA's ongoing research on the health impacts continues to affirm the serious dangers to human health posed by PM and $PM_{2.5}$ specifically. In the preamble to a Final Rule issued on January 18, 2001 with respect to air pollution emissions from certain heavy diesel-vehicles, EPA found that:

> Particulate matter . . . has been linked to a range of serious respiratory health problems. . . . The key health effects categories associated with ambient particulate matter include premature mortality, aggravation of respiratory and cardiovascular disease (as indicated by increased hospital admissions and emergency room visits, school absences, work loss days, and restricted activity days), aggravated asthma, acute respiratory symptoms, including aggravated coughing and difficult or painful breathing, chronic bronchitis, and decrease lung function that can be experienced as shortness of breath.

Control of Air Pollution from New Motor Vehicles: Heavy-Duty Engine and Vehicle Standards and Highway Diesel Fuel Sulfur Control Requirements, 66 Fed. Reg. 5002, 5018 (Jan. 18, 2001).

Significantly, in the preamble to this Final Rule EPA also explicitly noted that "[t]he epidemiologic science points to fine PM as being more strongly associated with some health effects, such a premature mortality, than coarse PM." and that adverse health impacts from $PM_{2.5}$ emissions will even be found in cities which are in compliance with the NAAQS for PM_{10}:

> Many epidemiological studies have shown statistically significant associations of ambient PM levels with a variety of human health endpoints in sensitive populations, including mortality, hospital admissions and emergency room visits, respiratory illness and symptoms measured in community surveys, and physiologic changes in mechanical pulmonary function. <u>These effects have been observed in many areas with ambient PM levels at or below the current PM_{10} NAAQS. The epidemiological science points to fine PM as being more strongly associated with some health effects, such as premature mortality, than coarse PM.</u>

66 Fed. Reg. at 5020 (emphasis added)..

Finally, I note that the State of California is in the process of considering new state standards for PM, including stricter state standards for PM_{10} and new

state standards for $PM_{2.5}$. The proposed state annual $PM_{2.5}$ standard recommended by the California Air Resources Board ("CARB") is stricter than EPA's annual NAAQS for $PM_{2.5}$ (12 micrograms per cubic meter, annual arithmetic mean, compared to EPA's 15 micrograms per cubic meter annual standard). Just yesterday, CARB issued a draft proposal to establish a state 24-hour standard for $PM_{2.5}$, recommending that it be set at 25 ug/ m^3, a standard that is substantially lower than EPA's 65 ug/m^3 annual NAAQS for $PM_{2.5}$. CARB is expected to act on these recommendations later this year.

CARB estimates that its recommended state PM standards will result in a reduction of 6,500 cases of premature mortality per year and reduce annual hospitalizations by an estimated 600 for chronic obstructive pulmonary disease, 900 for pneumonia, 1,500 for cardiovascular disease, and 500 for asthma. Among children ages 7-14, CARB estimates that attainment of the recommended $PM_{2.5}$ standard will result in about 209,000 fewer days of lower respiratory symptoms per year. The CARB Public Review Draft Report and Draft Proposal to Establish a 24-hour standard for PM2.5 can be found at: http://www.arb.ca.gov/research/aaqs/std-rs/pm-draft/pm-draft.htm.

Q. 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 2001 PM Criteria documents. In addition, my research was cited by the U.S. EPA as a "key study" in promulgating both the most recent $PM_{2.5}$ and ozone air quality standards. 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.

Q. 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 1 below.

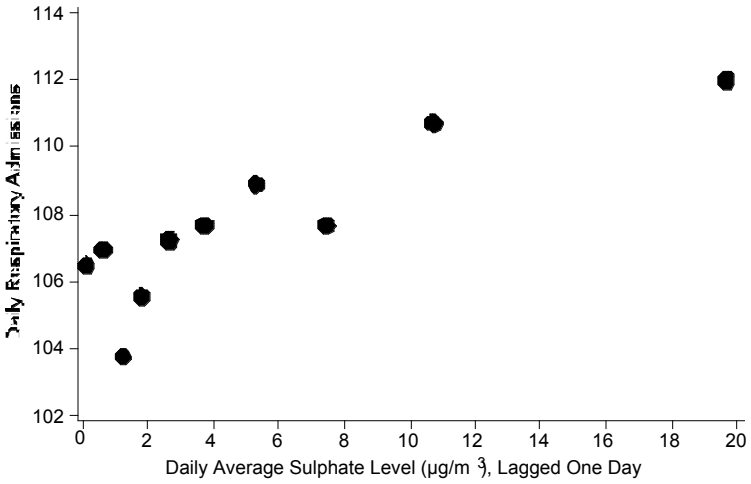


Figure 1. Average number of respiratory admissions among Ontario hospitals adjusted for other factors, by decile of the daily average sulfate fine particle concentration (ug/m^3). (Burnett et al, 1994).

Recent studies of fine PM associations with adverse health effects support the occurrence of significant adverse health effects at levels below the U.S. EPA $PM_{2.5}$ long-term mean concentration standard of 15 ug/m^3. For example, mean $PM_{2.5}$ levels in a study of asthma hospital visits by Seattle children less than 18 was estimated by the authors at $PM_{2.5}$ = 12 ug/m^3, and the $PM_{2.5}$ association with excess ER visits was still significant at these low levels, even after controlling for co-pollutants. Norris, Young-Pong; Koenig; et al. (1999). This indicates that $PM_{2.5}$ adverse health effects associations can be documented below the present long-term standard. and is consistent with the expectation that there is no threshold below which incremental effects of $PM_{2.5}$ will not cause an associated increase in the risk of severe adverse health effects, such as increased emergency room visits by children. I also note that, consistent with my conclusion, EPA has designated in its Air Quality Index that especially susceptible populations should be more careful to avoid outdoor pollution exposures (e.g. by minimizing exercise) when the daily $PM_{2.5}$ levels are expected to rise above 40 ug/m3.

Q. Is there a significant regional transport component to $PM_{2.5}$?

A. Yes, there can be. Since fine particles can be transported long distances in the atmosphere once emitted, there tends to me a greater transport component for fine $PM_{2.5}$ than for larger, coarse PM. Local $PM_{2.5}$ therefore tends to be primary in

nature (i.e., emitted directly from, or condensing into a particle immediately upon emission from, an exhaust stack), rather than secondary in nature (i.e., formed in the atmosphere over time).

Q. 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" $PM_{2.5.}$.

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

A. There is ever-growing scientific evidence indicating that particulate matter (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 1950's, 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.

Q. What is known about the toxic composition of PM 2.5 emitted by power plants that burn fossil fuels?

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. Therefore, these 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, and Lay et al, 1999). This may also be especially true in the case of power plant particles because power plant PM is composed of very small 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 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 (i.e., fossil fuel combustion products) are among the most toxic particles (e.g., Ozkaynak and Thurston,1987; Dockery et al.,1993; and Pope et al.,1995). In my own published research examining the associations of PM with human mortality, we have found that PM emitted from fossil-fuel combustion and from the metals industry are more strongly associated with mortality than particles from other sources, such as soil-derived and automobile emission-related particles (Ozkaynak, H. and Thurston, G.D., 1987, Associations between 1980 U.S. mortality rates and alternative measures of airborne particle concentration. *Risk Analysis* 7:44 s been found between sulfate fine pa 2.

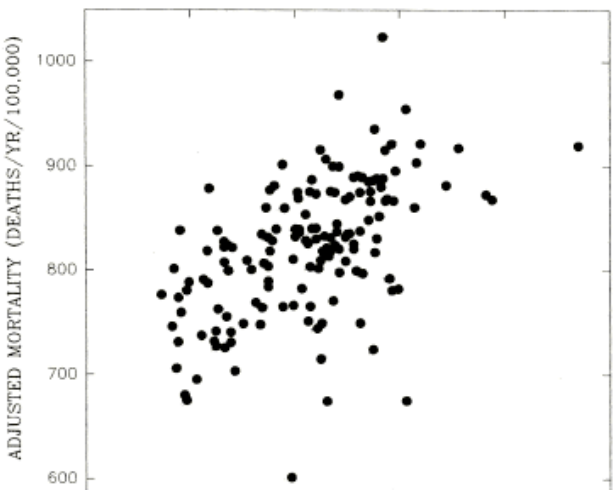


Figure 2. Age-, sex-, and race-adjusted population-based mortality rates for U.S. metropolitan areas in 1980 plotted versus mean sulfate fine particle air pollution levels. (From: Pope, et al 1995).

Lab studies also suggest that the presence of acidity in particles 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.

Q. Is there any evidence that primary $PM_{2.5}$ emitted from natural gas-burning facilities such as the proposed NYPA Astoria 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 before emission control technologies are applied, there is no evidence that, on a pound for pound basis, the particles from gas-fired power plant are any less toxic than $PM_{2.5}$ from other fossil fuels. Indeed, because gas-fired sources can emit a greater percentage of the particles as ultra-fine particles, which have a much

higher surface area per mass than larger particles, it is possible that there is a greater effect per pound for gas-fired $PM_{2.5}$ than for $PM_{2.5}$ emitted by sources burning other fossil fuels.

The following testimony is submitted in response to Question 2 of the Siting Board's January 24 Order: What is the state of the science regarding PM 2.5 and its health effects, and what is known about the general health impacts and health risks associated with PM 2.5?

Q. Please describe the general health impacts and health risks associated with $PM_{2.5}$ emissions.

A. Over the past few decades, medical researchers examining air pollution and public health, including myself, have shown that $PM_{2.5}$ pollution is associated with a host of serious adverse human health effects. See pp. 8-11 above. This documentation includes impacts demonstrated by controlled chamber exposures and by observational epidemiology showing consistent associations between $PM_{2.5}$ and adverse impacts across a wide range of human health outcomes. Amongst this evidence, observational epidemiology studies, such as the ones I have conducted, have provided the most compelling and consistent evidence of adverse effects from $PM_{2.5}$. Such epidemiological studies statistically evaluate changes in the incidence of adverse health effects in a single population as it undergoes varying real-life exposures to pollution over time, or across multiple populations experiencing different exposures from one place to another. These

studies have largely shown confirmatory associations between exposure to inhalable PM pollution and increased adverse health 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,
- increased numbers of daily deaths.

$PM_{2.5}$ can bypass the defensive mechanisms of the lung, and become lodged deep in the lung where they can cause a variety of health problems. Indeed, the latest evidence indicates that short-term exposures can cause not only respiratory damage, but also cardiac effects, including heart attacks. With respect to $PM_{2.5}$ from power plants, my recent study 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. Moreover, long-term exposure to fine particles increases the risk of death, and 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 15 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 10 successive five-year categories that make up the 25-75 year old age range. Life expectancy of a 25 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 25. 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 25. Thus, these calculations indicate that differences in long-term exposure to ambient $PM_{2.5}$ can have substantial effects on life expectancy.

Q. What, if any, evidence establishes a causal link between $PM_{2.5}$ and the health effects you have enumerated?

A. The fact that the effects of air pollution have both been shown so consistently for so many health endpoints and in so many locales indicates these associations to be causal. Furthermore, controlled human and animal exposures of air pollution have also shown consistent adverse effects. For example, when particulate matter stresses on the lung (e.g., by inducing edema), it places extra burden on the heart, which could induce fatal complications for persons with cardiac problems. Indeed, Peters et al (2001) found that elevated concentrations

of fine particles in the air can elevate the risk of Myocardial Infarctions (MI's) within a few hours, and extending 1 day after PM exposure. The Harvard University team found that a 48 percent increase in the risk of MI was associated with an increase of 25 ug/m^3 $PM_{2.5}$ 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 ug/m^3 $PM_{2.5}$ in the 24-hour average 1 day before the MI onset (Peters et al., 2001). Furthermore, recent animal experiments by Godleski and coworkers at Harvard indicate that controlled exposures to elevated concentrations of ambient particulate matter (PM) can result in cardiac related problems in animals. The most biologically and clinically significant finding was that in dogs with induced coronary occlusion, particles affected one of the major electro-cardiogram (ECG) signs of myocardial ischemia in humans, known as elevation of the ST segment. Cardiac effects at the biological level have also been found in human 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. Overall, such consistency between the epidemiological study associations and experimental studies' results support the conclusion that there is indeed a cause-effect relationship underlying the PM air pollution-health effect relationships that have been seen so consistently in epidemiological studies.

Q. 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; and see: U.S. EPA, 2001). Most recently, I was a Principal Investigator of a study published in the Journal of the American Medical Association (JAMA) that shows 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 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. (Pope et al, 2002).

Q. What conclusions were reached in the 1996 EPA PM Criteria Document and Staff Paper in regards to the health effects of $PM_{2.5}$?

A. The PM Criteria Document ("CD") concluded that "The evidence for PM-related effects from epidemiologic studies is fairly strong, with most studies showing increases in mortality, hospital admissions, respiratory symptoms, and pulmonary

function decrements associated with several PM indicators." The 1996 Staff Paper went on to conclude that: "While it is difficult to distinguish the effects of fine or coarse fraction particles from those of PM_{10}, consideration of comparisons between fine and coarse fraction particles suggests that fine particles are a better surrogate for those particle components linked to mortality and morbidity effects."

Q. Do you agree with those conclusions?

A: Yes, and the published evidence since the 1996 PM CD and Staff Paper were written also largely support those conclusions.

Q. Do the EPA's 1996 Criteria Document, 2001 Draft Criteria Document and the Staff Paper reflect the consensus in the scientific community in regards to $PM_{2.5}$?

A. Yes, I would say that the general scientific consensus supports the EPA conclusions regarding the health risks associated with fine $PM_{2.5}$ exposures.

Q. 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).

Q. 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. New York City has one of the highest burdens of asthma problems in the nation (see, e.g. Weiss and Wagner, 1990). Thus, there is a large population in New York City that, because of pre-existing asthma, is especially susceptible to these effects of increases in air pollution. Among the groups that have a high rate of asthma are Hispanics, especially those of Puerto Rican descent (see Carter-Polkras and Gergen, 1993). These ethnic groups are highly represented in New York. For example, Hispanics made up only about 12.5 percent of the U.S. population in 2000 (U.S. Bureau of the Census, 2002). Furthermore, in the three of the six census tracts (11, 14 and 15) surrounding the proposed plant location , between 27-28% of the population were Hispanics, well above the national average (U.S. Bureau of the Census, 2002). Thus, it is clear from these data, as compared with the U.S. in general, that there are a disproportionately larger number of people who are especially susceptible to asthma in the census tracts immediately surrounding the plant in question.

Moreover, recent 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). Overall, the percent of families living below the poverty line in the U.S. was some 13 percent in 1989 (U.S. Census). However, , some 20% of persons living in 2 of the 6 key census tracts (14 and 15 in

Middletown) within two miles of the proposed facility live below the poverty line. It is clear from these data that a disproportionately large number of persons living in poverty are affected by the pollution from this plant.

Q. Is there substantial evidence that some of these subpopulations, particularly those with pre-existing respiratory health conditions, live in the area around the proposed Calpine plant?

A. Overall, when compared to the general U.S. population, (from the U.S. Census Bureau) there is a higher percentage and/or number of especially affected persons in the areas immediately surrounding the plant.

Q. What are your conclusions in regard to the expected health impacts of increased $PM_{2.5}$ emissions from the proposed Calpine Facility?

A. The existing levels of particulate air pollution in Orange County (as collected and reported by the NY DEC) are already in the concentration range where incremental exposures have been shown by published epidemiology to produce incremental health risks in the general population. Thus, additional emissions from the proposed Calpine facility will add to the existing levels of $PM_{2.5}$ in the vicinity of the plant, In addition, because no threshold of air pollution effects has yet been found, any incremental $PM_{2.5}$ exposures, such as the $PM_{2.5}$ increments indicated by Calpine's "Assessment of Potential PM2.5 Facility Impacts" (April 2000), add an incremental adverse health risk to New Yorkers from power plant air pollution. Moreover, Census Tracts 11, 14, 15, 16 and 112 have higher concentration of some classes of persons who are likely to be especially susceptible to adverse

effects from particulate air pollution, relative to the general U.S. population (especially for the Hispanic and the poor populations). 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. Am J Respir Crit Care Med. 2000 Feb;161(2 Pt 1):665-73.) Therefore, any action that increases 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 Calpine facility will emit additional levels of $PM_{2.5}$, it will cause an increase in the risk of adverse health effects among residents of the surrounding communities who breathe that pollution.

Q. Have you reviewed Calpine's Analysis of $PM_{2.5}$ 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. While it is reasonable to assume that all PM_{10} emitted by the plant is $PM_{2.5}$, it is not sufficient to present the increments alone. It is necessary to do an

assessment of the potential health implications of the $PM_{2.5}$ impacts, which may well be larger than for PM_{10}, as $PM_{2.5}$ is thought to be more toxic on a per pound basis than PM_{10}, thus increasing the health risks associated with each increment of $PM_{2.5}$ versus if it were PM_{10}. Such an assessment would entail utilizing the published risk factors for $PM_{2.5}$ air pollution and applying it 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 (RIA) for the $PM_{2.5}$ standards.

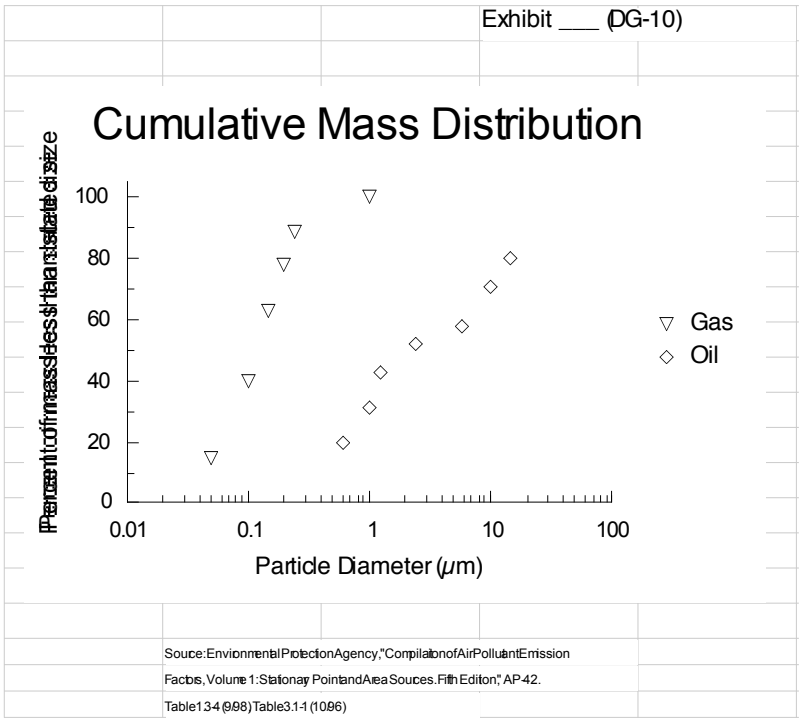
Q. Please discuss your critique of Calpine's conclusion with respect to the composition of $PM_{2.5}$ emissions from the proposed facility.

A.

Calpine's statement that "PM 2.5 that is rich in either biologically-active material or in various metals is likely to be more harmful than PM 2.5 that has little to no biologic or metallic content. Therefore it is important to note that since PM emitted from a gas-fired turbine has no biologic and very little metal content it is far less likely to have health impacts than even ordinary dust that may contain aeroallergens,"(refer to: Assessment of Potential PM2.5 Facility Impacts, pg. 9), is incorrect and without scientific basis.

There are at least two known characteristics of natural gas-fired power plant emissions that make it likely to have especially high health effects, on a per pound basis: 1) it has a higher percentage of ultrafine particles that have very high surface areas, relative to other fossil-fuel emissions, which may increase the health impacts of gas-fired PM considerably; 2) some metals are present in gas combustion particles that are as high a percentage, or higher percentage than for oil combustion; and 3) there are acidic sulfates associated with these emissions, especially as strongly acidic ammonium bisulfate Both of these factors would tend to increase the "bio-availability" of the toxins for gas-fired PM, which would therefore likely increase the toxicity of gas-fired power plant particles, relative to other ambient particles.

There are several points to make with respect to the metals content of $PM_{2.5}$ emissions from gas-fired power plants. As summarized in Exhibit DG-6 to the Testimony of Daniel Gutman in the Astoria plant case (CASE 99-F-1627), a comparison of the metal content of gas-and oil-fired particles shows that gas-fired particles have just as high or higher a percentage of a number of metals as do oil-fired particles, including barium, cadmium, chromium, molybdenum and zinc. Moreover, even if one assumes that metals content is generally lower in gas-fired particles, a higher percentage of gas-fired particles are ultrafine particles, compared to particles from other combustion sources (e.g. residual oil combustion particles). This can be expected to increase the toxicity of these metals, relative to other combustion sources with a smaller percentage of ultrafines (e.g., residual oil



combustion particles). The fact that natural gas-fired turbines have $PM_{2.5}$ emissions with a high content of ultrafine particles is demonstrated by the particle size distribution of the stack emissions from such turbines. The U.S. EPA AP-42 emission factors for large gas-fired turbines, such as are being proposed, indicates that some 40% of $PM_{2.5}$ from this source is as ultrafines (diameter <0.1 um) (U.S. EPA, 1996); NYPA Application, Table 5.34, page 5-94 (see below).. Thus, there is a high presence of ultrafine particles that may be of especially high toxicity, especially in the co-presence of metals and acidic aerosols.

Plot showing the higher percentage of ultrafine particles in gas combustion emissions vs. in oil combustion emissions (from Dan Guttman's NYPA testimony).

Source: Dan Guttman Testimony, Case Case 99-F-1627..

Furthermore, the expected co-presence of strongly acidic ammonium bisulfate will tend to further enhance the bio-availability, and hence the toxicity, of the metals that are present. In contrast, the data presented in the table below for $PM_{2.5}$ from a similar gas-fired plant indicate that there are insufficient ammonium ions present to neutralize the sulfates in the PM2.5 emitted by the proposed project. Moreover, if the plant sulfates were in the form of the more strongly acidic ammonium bisulfate, then for the 7.9 percent sulfate given in the table below, there would have to be some 1.6 percent ammonium, which is very close to what is observed. If the sulfate from the plant were present in the more neutralized form, there would be much more ammonium present than reported for these source PM2.5 in Table 1. Thus, it is clear from these data that the PM2.5 emitted by the proposed project will contain a higher percentage of particle acidity than NYC's usual ambient air. The potential toxicity of these gas-fired power plant metals cannot be dismissed.

Table 1

$PM_{2.5}$ Components	Composition of $PM_{2.5}$ Emitted by Project	Composition of $PM_{2.5}$ in NYC's Ambient Air
Sulfates	7.9%	32.1%
Nitrates	0%	11.7%
Ammonium	1.5%	14.0%
Metals	0.6%	4.9%
Elemental Carbon	83.4%	7.0%
Organic Carbon	6.6%	30.6%

Sources: Project data are from Exhibit RH-2. Data for New York City are from the DEC Report, Part II, Exhibit B, using the average of the values presented for the Bronx and Queens.

Table 1. Data for the composition of particles from a gas-fired turbine similar to Calpine's proposed facility vs. NYC ambient air.

Source: Robert S. Hall Testimony, Case 99-F-1314..

Indeed, although the mass of particles per Btu from gas combustion is lower than for oil and coal, it is of concern just how many characteristics that gas combustion particles share with oil and coal emission particles. For all these reasons, the $PM_{2.5}$ emissions from the new plant cannot be assumed to be less toxic than other particles and, because of their high ultrafine fraction, their acidity, and their metal content, they potentially could be even more toxic than other forms of particulate matter. Thus, Calpine's conclusion is unsupported.

Q. Does this conclude your testimony?

A. Yes.

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