Exhibit E

IN THE MATTER

Of the Proposed Buckingham Compressor Station

Report of
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Chester, NY 10918

September 9, 2018

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Report of George Thurston, Sc.D.

A. Qualifications

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. My business address is: Three Catherine Ct., Chester, NY 10918. I am providing expert testimony that addresses the public health impacts of emissions of fine particulate matter (PM2.5) generally and, specifically, the expected public health impacts of PM2.5 emissions from the proposed Buckingham Compressor Station (BCS). My testimony will address the potential health effects of the facility, if approved. I conclude that the that the air pollution emissions from this facility can be expected to increase adverse health risks in the surrounding community.

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 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 (CASC) 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 US. (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."

In the past, I have provided testimony about the human health impacts of air pollution from fossil fuel combustion sources on numerous occasions, and on the health effects of natural gas combustion-related pollution 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).

B. The State of the Science Regarding Particulate Matter (PM) Air Pollution and its Human Health Effects

The adverse health consequences of breathing air pollution that results from sources such as fossil fuel combustion facilities 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 a RR =1.0 is an indication of no change in risk after exposure, while a 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 PM2.5 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 (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 *u*g/m3 PM2.5 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 *u*g/m3 PM2.5 in the 24-hour average 1 day before the MI onset (Peters *et al.*, 2001). Numerous other U.S. studies have also show qualitatively consistent acute cardiac effects, such as the Zanobetti and Schwartz (2006) study of hospital admissions through emergency department for myocardial infarction (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, myocardial infarction, and congestive heart failure in 26 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 particulate matter can result in cardiac related problems in dogs that had been pre-treated (in order to try to simulate sensitive individuals) to induce coronary occlusion (i.e., narrowed arteries in the heart) before exposing them to air pollution. The most biologically and clinically significant finding was that, in these dogs, the particulate affected one of the major electrocardiogram (ECG) markers of heart attacks (myocardial ischemia) in humans, known as elevation of the ST segment. Cardiac effects at the biological level have been found in human studies, as well. For example, Pope *et al.* (1999) and Gold *et al.* (2000) found that PM exposure is associated with changes in human heart rate variability. Such changes in heart rate variability (HRV) may reflect changes in cardiac autonomic function and risk of sudden cardiac death. In the Pope *et al.* study, repeated ambulatory ECG monitoring was conducted on 7 subjects for a total of 29 person-days before, during, and after episodes of elevated pollution. After controlling for differences across patients, elevated particulate levels were found to be associated with (1) increased mean heart rate, (2) decreased SDNN, a measure of overall HRV, (3) decreased SDANN, a measure that corresponds to ultra-low frequency variability, and (4) increased r-MSSD, a measure that corresponds to high-frequency variability. 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 PM10 air pollution (a subset of particulate matter air pollution that is less than 10 μm in diameter, including PM2.5) in 90 cities across the United States, finding that, for each increase of 10 μg/m3 in daily PM10 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, 2009a) unequivocally states that "Together, 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 PM2.5 and cardiovascular effects . . . and mortality.*"1

With respect to PM2.5 from fossil fuel combustion, 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/m3 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 PM2.5 can have substantial effects on life expectancy.

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 PM2.5 annual standard in 1997), Dockery *et al.* (1993) analyzed survival probabilities among 8,111 adults living in six cities in the central and eastern

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

portions of the United States during the 1970's and 80's. 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.

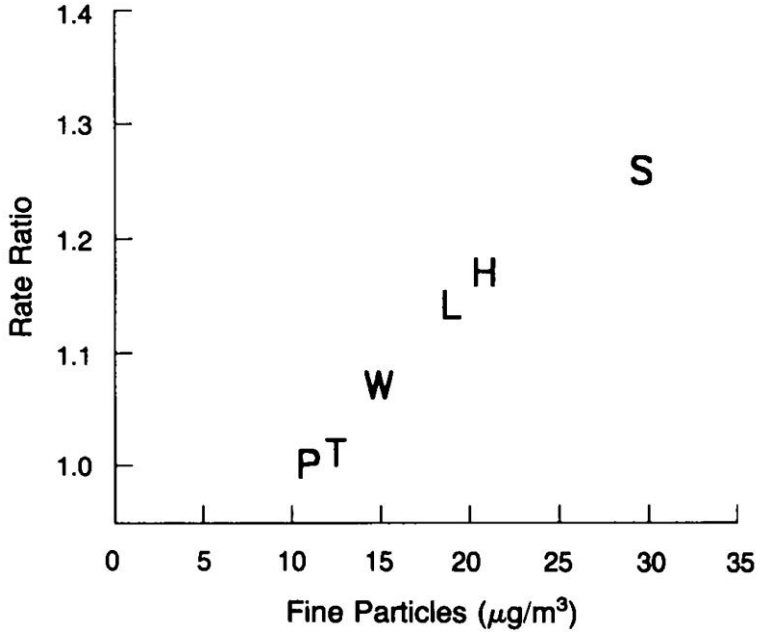

Figure 1. The Harvard Six-Cities Study showed that the lifetime risk of death increased across 6 U.S. cities as the average fine PM levels increased. (Source: Dockery *et al.*, 1993).

Moreover, 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 PM2.5 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).

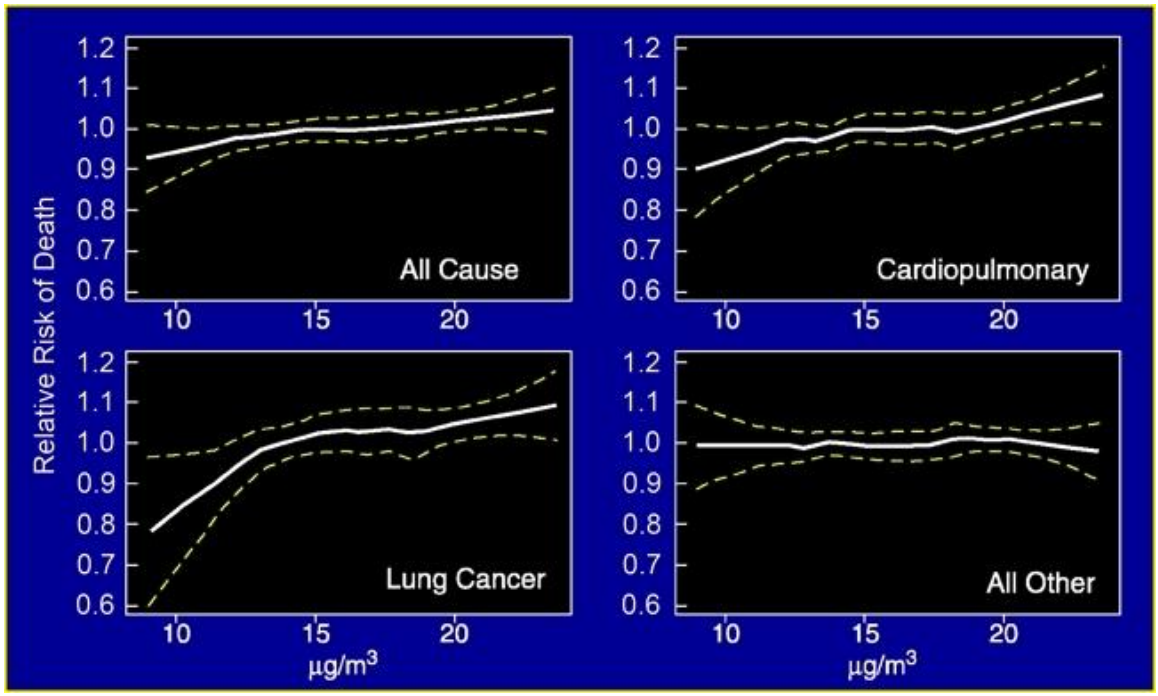

Figure 2. Cardiopulmonary and Lung Cancer Mortality Risks Increase Monotonically with Exposure to Long-Term Fine PM
(Adapted from: Pope, Burnett, Thun, Calle, Krewski, Ito, and Thurston, 2002)

Long-term exposure to fine particles has also 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/m3 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 PM2.5 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 are considered, there is no doubt as to the significance of these adverse effects.

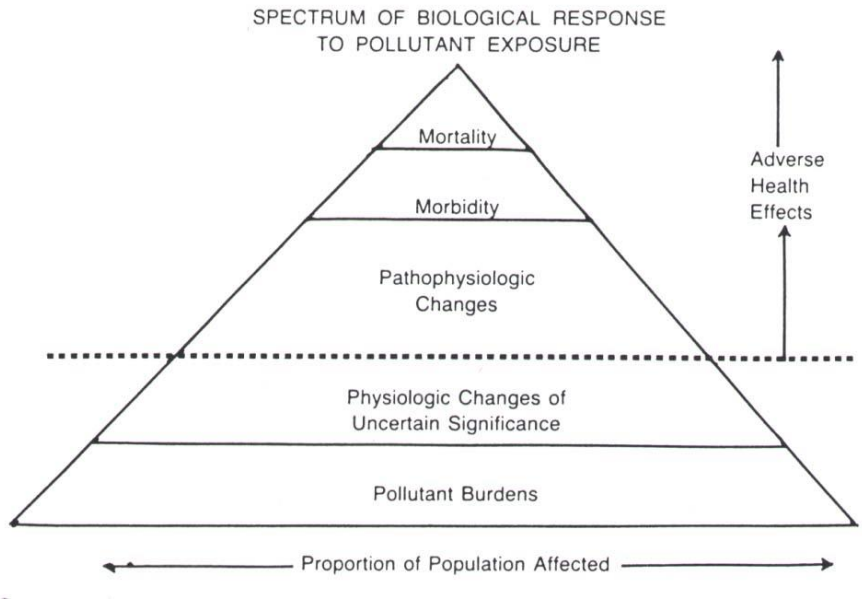

Figure 3. The Pyramid of Adverse Health Effects of Air Pollution on Health

(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.)

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 fossil fuel combustion sources) 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 PM2.5 particles in general (Thurston et al., 2016), and more damaging per µg/m3 than PM2.5 from other common sources (Figure 4).

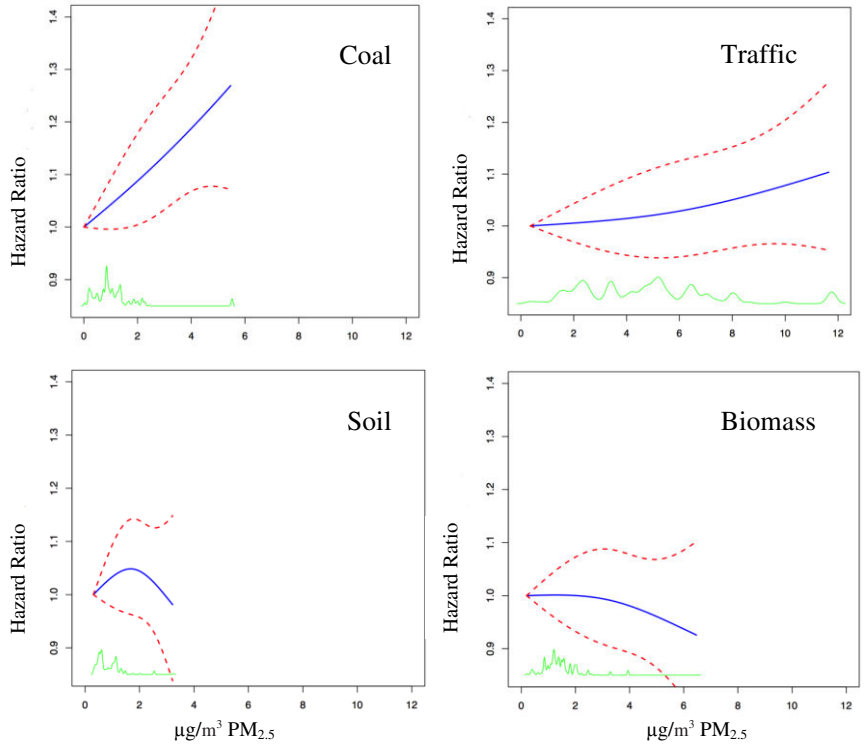

Figure 4. Concentration-response curve (solid lines) and 95% confidence intervals (dashed lines) for Source-Specific PM2.5 mass in the US ACS Cohort. (Thurston et al., 2016).

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 each of the 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 (PM10). 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 PM10 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. The 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 ("PM2.5"). In July 1997, upon determining that the PM10 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 24-hour standard that is met when the three-year average of the 98th percentile of 24-hour PM2.5 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 PM2.5 standards were based on an increasing scientific consensus that the current NAAQS for PM10 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 PM10 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). Since that time, the U.S. EPA has lowered the allowable limits of ambient concentration of PM2.5 to 35 µg/m3 and 12 µg/m3 for the daily and annual standards, respectively, in recognition of its effects at lower levels of exposure.

The EPA PM Staff Paper at the time of the setting of the PM2.5 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, 1996b).

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.

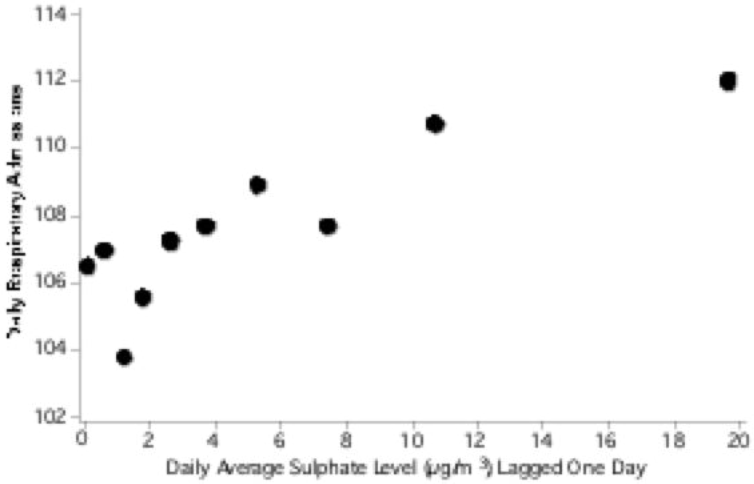

Figure 5. Average number of respiratory admissions among Ontario hospitals adjusted for other factors, by decile of the daily average sulfate fine particle concentration (ug/m3). (Burnett et al, 1994).

In addition, as displayed in the Figure 6 below, my research has shown that increases in long-term exposure to PM2.5 particulate matter air pollution are associated with increases in the risk of cardiovascular death among those exposed, even well below the present 12 µg/m3 annual PM2.5 air quality standard (Thurston et al., 2016). 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. 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 PM2.5 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.

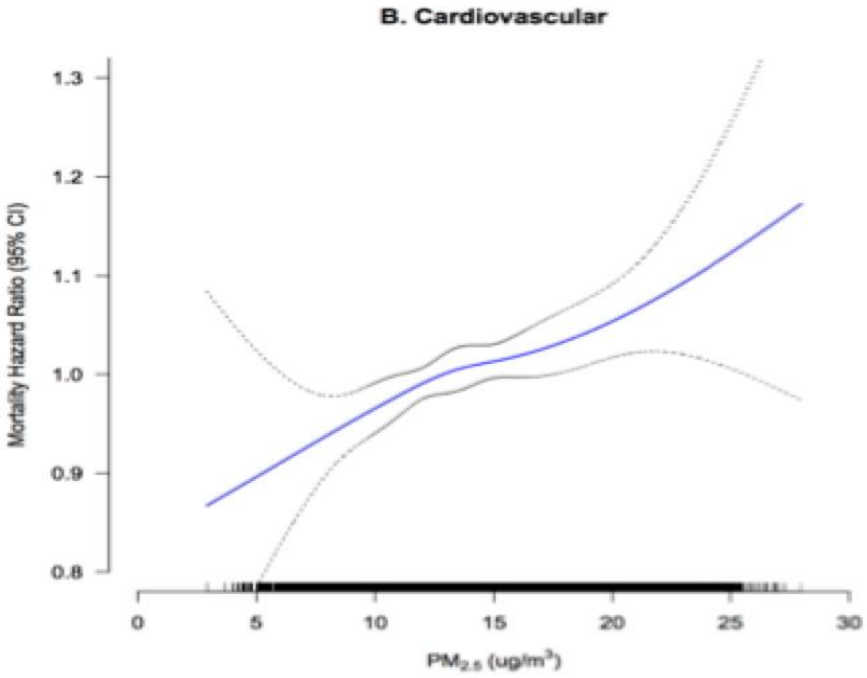

Figure 6. Mortality Risk from Cardiovascular Disease Increases with Rising PM2.5 Exposure, Even Well Below the Present US Ambient Air Quality Standard annual limit for PM2.5 (12 µg/m3). Thurston et al, 2016.

Furthermore, in its calculations of the benefits of potentially reducing the PM2.5 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 PM2.5 standard from 15 µg/m3 to 12 µg/m3 (U.S. EPA, 2012), EPA included a figure summarizing the best, most current science regarding PM2.5 health effects, which clearly illustrates that air pollution deaths occur below the existing PM2.5 NAAQS (35 µg/m3 for the daily standard, and 12 µg/m3 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 PM2.5

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.

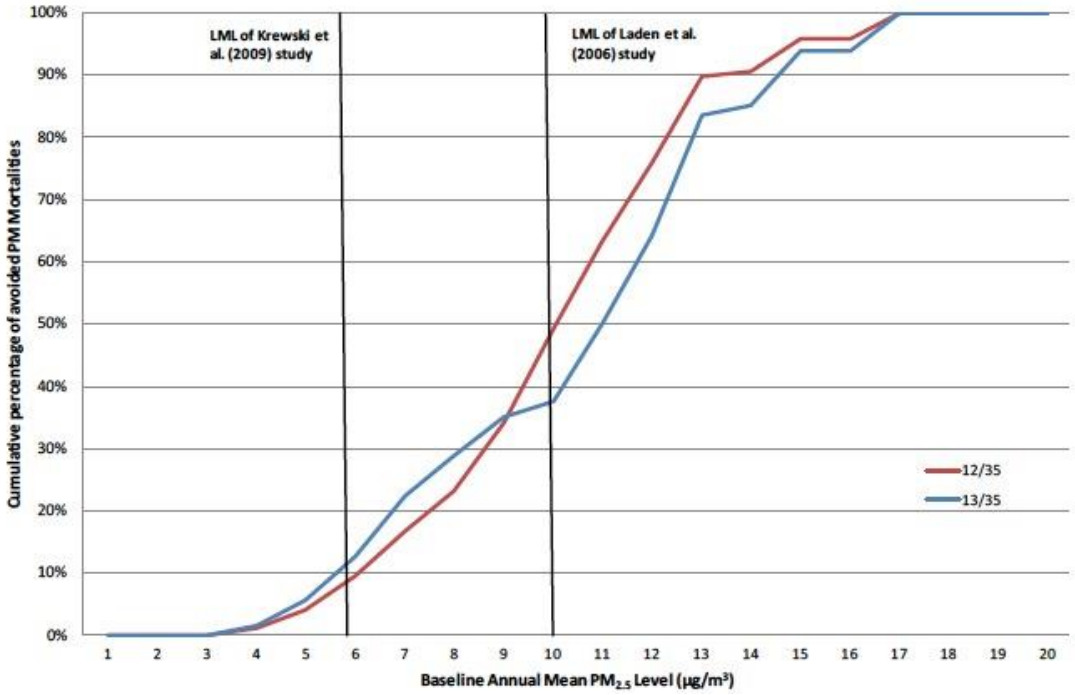

Figure 7. U.S. EPA Regulatory Impact Assessment of the Number of Premature $PM_{2.5}$-Related Deaths Avoided for 12/35 vs. 13/35 Ambient PM2.5 Air Quality Standards. (LML = Lowest Measured Level of PM2.5 in the study population) (U.S. EPA 2012, Fig. 5-7)

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 PM2.5 standards are complicated by the recognition that *no population threshold, below which it can be concluded with confidence that PM2.5-related effects do not occur, can be discerned from the available evidence*." (emphasis added). (U.S. EPA, 2013).

PM2.5 is directly emitted by both stationary sources (e.g. fossil fuel combustion sources and other industrial sources) and mobile sources, such as diesel buses and trucks. PM2.5 is also

formed in the atmosphere from gaseous emissions, such as sulfur oxides from fossil fuel combustion, resulting in “secondary” PM2.5.

PM2.5 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 fossil fuel combustion sources. 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 that 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 particulate matter (PM) air pollution emitted by fossil fuel combustion is among the important contributors to the toxicity of PM.2 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 combustion 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 fossil fuel combustion particles 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 combustion are of great human health concern.

The conclusion that fossil fuel combustion 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 unit energy 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 facilities, per pound of emission, and ultrafine (e.g., nanoparticles) are thought to be far more toxic per unit mass 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 are very small, and can defeat the body's natural defenses, thereby having a far greater adverse effect on health. In particular, these fossil fuel combustion 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 fossil fuel combustion particles because such 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 the combustion of fossil fuels**,** including natural gas-fired units, is cause for special concern, and the health of persons in nearby populations can be adversely affected by this fossil fuel combustion related air pollution.

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, 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 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* 7:449-460). An example of the relationship that has been found between sulfate fine particle pollution and mortality is shown in Figure 8.

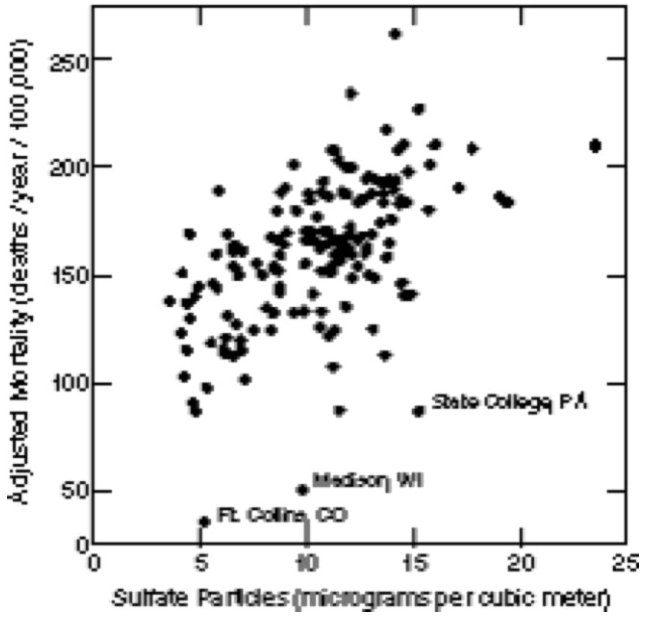

Figure 8. Age-, sex-, and race-adjusted population-based mortality rates for U.S. metropolitan areas in 1980 plotted versus mean sulfate fine particle air pollution levels. (Adapted from Pope, et al 1995).

Lab studies also suggest that the presence of acidity in particles, which is usually the case for fossil fuel 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 combustion, 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.

Particulate matter 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 fine particles, tiny particles, that have large surface areas available to interact with the lung. And the particles have transition metals in them. For example, the percentages of transition metals are similar for natural gas and residual oil.

Second because fossil fuel particles, especially those near a fossil fuel burning facility, are freshly combusted, they have more active sites on them by which to damage health. The work by Oberdorster has shown PM concentrations at ambient levels, 60 $\mu g/m^3$ and less, cause mortality in healthy rats. And then he found the aging of those fumes with aggregation of the ultrafine particles significantly decreased their toxicity. So "fresher" (more recently generated) particles are more toxic. Thus, living near a major fossil fuel combustion facility is more impactful because both the concentrations breathed are higher than downwind, but also because they are more recently emitted, and likely more reactive than more aged particles downwind.

Since fossil fuel particles are all fresh aerosols when they are coming out of the facility, and they are combustion aerosols, they share many characteristics. Hence, even though we haven't directly studied natural gas particles, since they share many of the same characteristics as particles from oil and coal combustion, it is very likely that they would share the toxicity of their "sister" fuels, and potentially at a higher effect per pound of pollution breathed.

Freshly combusted particles will have sharp edges, and will be composed, in part, of unoxidized compounds that haven't 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 that use ammonia as part of an SCR system, also include ammonium bisulfate, which is strongly acidic. 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.

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 facility are any less toxic than

PM2.5 from other fossil fuels. Indeed, because gas-fired sources can emit a much greater percentage of the particles as ultra-fine 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 PM2.5 emitted by gas-fired sources than for PM2.5 emitted by sources burning other fossil fuels. For this reason, the impacts of the proposed facility in terms of PM2.5 mass concentration are an inadequate indication of the health risks associated with the proposed BCS.

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). 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 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 PM2.5 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).

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). Ethnicity, age and pre-existing medical conditions play a role in determining whether adverse health impacts are the predictable result of exposure to increased PM2.5 emissions. 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 facility. This subpopulation of children can be expected to be among those most strongly affected by any increases in PM2.5 concentrations in the vicinity of the facility.

It is reasonable to assume that nearby residents will be exposed to these emissions even while inside their apartments? 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.

While other PM2.5 exposures, such as indoor air pollution, may have health effects, they are independent of the impacts of increases of exposures to PM2.5 of outdoor origins in general and of the proposed facility in particular. If the levels of outdoor PM2.5 impinging the living areas of residents increase, then it can be expected that their personal exposures to PM2.5 of outdoor origins, and their associated health risks,

There are two known characteristics of natural gas combustion particles that make them likely to have especially high health effects, on a per pound basis, than usual PM2.5: 1) they have a higher percentage of ultrafine particles, as compared with other fossil-fuel options (see Figure 9). These ultrafine particles have very high surface areas, relative to other fossil-fuel emissions, which likely increase the health impacts of gas-fired PM considerably; and 2) 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 combustion particles, relative to other ambient particles.

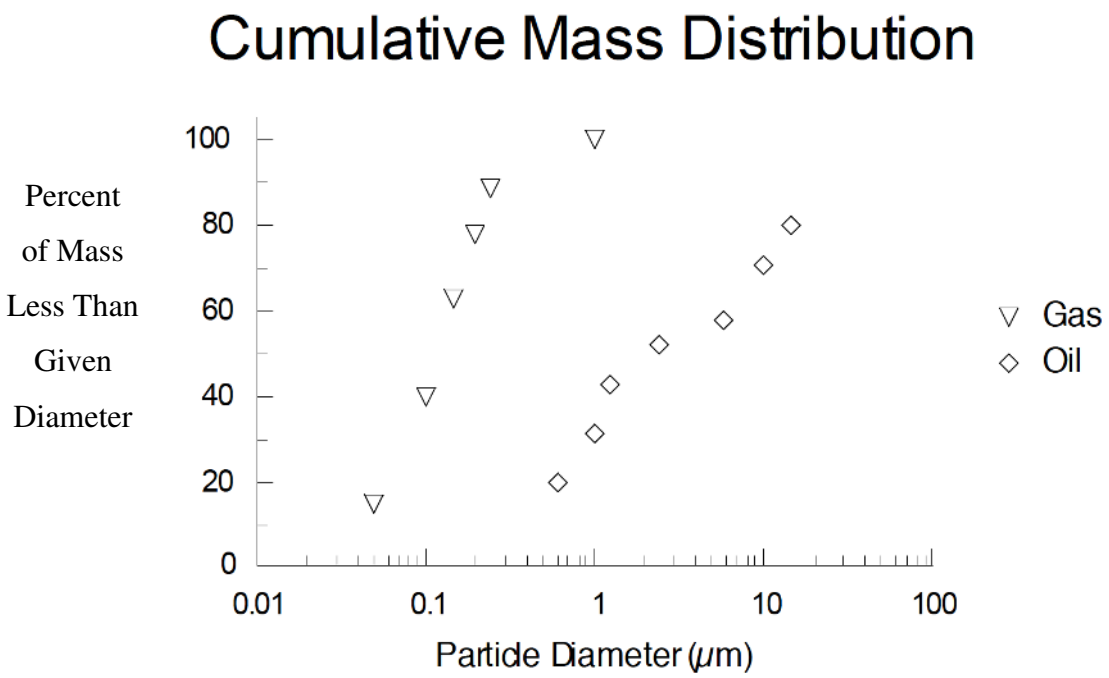

Figure 9. Comparison of Particle Mass Size Distribution for Natural Gas vs. Oil Combustion Emissions. Source: Environmental Protection Agency, "Compilation of Air Pollutant Emission Factors, Volume 1: Stationary Point and Area Sources. Fifth Edition," AP-42. Table 1.3-4 (9/98), Table 3.1-1 (10/96)

The composition of gas combustion particles also makes them to likely be more toxic than usual PM2.5. As summarized in Table 1, 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 oil-fired particles, including barium, cadmium, chromium, molybdenum and zinc. Moreover, even if one assumes that total 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). Thus, there is a high presence of ultrafine particles that are high in transition metal content, making them of especially high toxicity.

Table 1. Metal Content of Natural Gas vs. Residual Oil PM Emissions

Pollutant	PM Emissions (lb./MMBtu)		PM Emissions as % of PM Mass	
	Natural Gas	Residual Oil	Natural Gas	Residual Oil
Antimony		3.5E-05		0.070%
Arsenic	2.0E-07	<8.8E-06	0.003%	0.018%
Barium	4.3E-06	1.7E-05	0.058%	0.034%
Beryllium	<1.2E-08	<1.9E-07	0.000%	0.000%
Cadmium	1.1E-06	2.7E-06	0.014%	0.005%
Chloride		2.3E-03		4.641%
Chromium	1.4E-06	5.6E-06	0.018%	0.011%
Cobalt	8.2E-08	4.0E-05	0.001%	0.081%
Copper	8.3E-07	1.2E-05	0.011%	0.024%
Fluoride		2.5E-04		0.499%
Lead	4.9E-07	1.0E-05	0.007%	0.020%
Manganese	3.7E-07	2.0E-05	0.005%	0.040%
Mercury	2.5E-07	7.5E-07	0.003%	0.002%
Molybdenum	1.1E-06	5.2E-06	0.014%	0.011%
Nickel	2.1E-06	<5.6E-04	0.028%	1.130%
Phosphorus		6.3E-05		0.127%
Selenium	<2.4E-08	<4.6E-06	0.000%	0.009%
Vanadium	2.3E-06	2.1E-04	0.030%	0.425%
Zinc	2.8E-05	1.9E-04	0.382%	0.389%
Total PM	7.5E-03	5.0E-02		

Source: EPA, Compilation of Air Pollutant Emission Factors, Volume 1: Stationary Point and Area Sources. AP-42, Tables 1.3-1 (with S = 0.3), 1.3-2, 1.3-11, 1.4-2, 1.4-4. https://www.epa.gov/air-emissions-factors-and-quantification

Furthermore, the likely co-presence of strongly acidic vapor in the BCS 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% 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 facility's emissions, the facility emissions can be expected to be in a strongly acidic, and therefore more toxic, form. The potential toxicity of exposure to these natural gas combustion metals cannot be dismissed, even at very low PM2.5 mass levels.

For all these reasons, the PM2.5 emissions from the new facility 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. Thus, I

disagree with the Supplemental Report's conclusion that "The emissions from the proposed BCS 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."

Studies using laboratory animals and humans support the notion that ambient or moderately elevated concentrations of relatively non-acidic, soluble sulfates or nitrates in particular harm health. Some controlled animal exposures of air pollution have shown adverse effects at PM2.5 at levels close to ambient levels. Recent animal experiments by Godleski and coworkers at Harvard indicate that exposures to elevated concentrations of ambient particulate matter (PM) 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.

C. The Human Health Effects of Ozone (O3) and Nitrogen Oxides (NOx) Air Pollution

Ozone (O3) is an air pollutant, resulting from nitrogen oxide and hydrocarbon emissions from fossil fuel combustion, that adversely affects human health. Ozone is a highly irritating gas that is formed in the atmosphere in the presence of sunlight from other "precursor" air pollutants, including NOx and hydrocarbons that are emitted by combustion sources such as fossil fuel burning facilities. The adverse health consequences of breathing ozone are serious and well documented. This documentation includes impacts demonstrated in controlled chamber exposures of humans and animals, and observational epidemiology showing consistent associations between ozone and adverse impacts across a wide range of human health outcomes.

The noxious nature of ozone is also evidenced by the way it visibly "eats away" at materials such as rubber, an elastic substance, sharing characteristics with human lungs. Indeed, in the early years of air pollution monitoring, the number of cracks in a stretched rubber band left outdoors for weeks was used as an index of the ozone concentration in the

air. Similarly, ozone has been known to cause fading of certain colors in fabrics because they oxidize the dye, causing "O-fading." As a result, automobile manufacturers today utilize ozone-resistant rubbers, while carpet and drape manufacturers use ozone-resistant dyes (NRC, 1976). In addition, Cass *et al.* (1991) have discussed the importance of protecting works of art from damage due to O3. Given this evidence of ozone's devastating effects on solid materials, it comes as no surprise that ozone can also have serious adverse health effects on the more vulnerable human lung.

Ozone can irritate the human respiratory system, causing exposed people to cough, feel an irritation in the throat, and/or experience an uncomfortable sensation in the chest area. Ozone has also been shown to reduce the lung's ability to inhale and exhale, thereby making it more difficult for people to breathe as deeply and vigorously as they normally would (*e.g.*, *see* Bates, 1995). Research shows that ozone can also acutely aggravate asthma, and new evidence suggests that it may cause more children to get asthma. When ozone levels are high, people with asthma have more attacks that require a doctor's attention or the use of additional medication. One reason this happens is that ozone makes people more sensitive to allergens, which are the most common triggers for asthma attacks. Ozone can inflame and damage cells that line the human lung, and O3 has been compared by some to "getting a sunburn on your lungs." Ozone may also aggravate chronic lung diseases, such as emphysema and bronchitis, and can reduce the immune system's ability to fight off bacterial infections in the respiratory system.

Among the important adverse effects associated with ozone exposure to asthmatics is the triggering of asthma attacks. The effects of ozone air pollution on children with asthma have been demonstrated in my own research following a group of children at an asthma summer camp located in Connecticut. This study of a group of about 55 moderate to severely asthmatic children showed that these children experienced statistically significant reductions in lung function, increases in asthma symptoms, and increases in the use of unscheduled asthma medications as ozone pollution levels rose. As shown in Figure 10, the risk of a child having an asthma attack was found to be approximately 40 percent higher on the highest ozone days than on an average study day (Thurston *et al.*, 1997). Consistent with other research in this area, there is no indication in this plot of a threshold concentration below which children with asthma are safe from the effects of ozone increases.

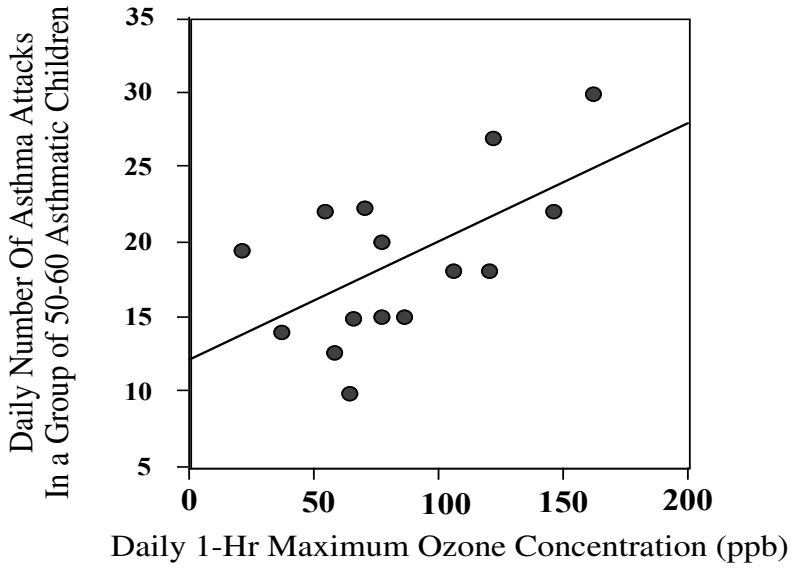

Figure 10. The number of asthma attacks among children at an "Asthma Camp" in Connecticut increase as the ozone levels rise (Source: Thurston *et al.*, 1997)

These asthma camp results have been confirmed by a larger study published in the Journal of the American Medical Association (JAMA). Gent *et al.* (2003) presented a cohort study of asthmatic children from the New Haven, CT area, including 130 children who used maintenance medications for asthma and 141 children who did not. The more severe asthmatics were identified as those using maintenance medication. For these severe asthmatics, the study found that the level of O3 exposure was significantly associated with worsening of symptoms and an increase in the use of rescue medication. Each 50 parts per billion (ppb) increase in 1-hour average O3 was associated with an increased likelihood of wheezing (by 35%) and chest tightness (by 47%). The findings indicate that asthmatic children are particularly vulnerable to ozone, even at pollution levels below the U.S. EPA air quality standards.

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

The adverse effects of exposure to ozone in ambient air on the lungs of individuals has been demonstrated in studies that I have conducted in the State of New York, as well. For example, respiratory function damage was demonstrated in a study I co-authored of 30 healthy adult non-smokers engaged in a regular daily program of outdoor exercise in Tuxedo, NY during the summer of 1985 (Spektor *et al.*, 1988b). All measured health indices showed statistically significant O3-associated decreases in the lung function of the runners as ozone levels increased. More recently, using lung bronchoscopy (which allows a visualization of the main tubes of the lungs, by means of a flexible lighted instrument introduced through the vocal cords and windpipe) and broncho-alveolar lavage (BAL, or a washing of the lining of the lung), Kinney *et al.* (1996) examined some 19 normal volunteer joggers from Governors Island, NY. The joggers exercised in the afternoon during the 1992 summer season. These results indicate a significant inflammatory response in the lungs of recreational joggers in New York City exposed to regional ozone and associated co-pollutants during the summer months.

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

The increased inflammation of the lung, and diminished immune system effects associated with ozone air pollution can also make the elderly more susceptible to pneumonia, a major cause of illness and death in this age group. Both *in vivo* and *in vitro* experimental studies have demonstrated that O_3 can affect the ability of the immune system to defend against infection. Increased susceptibility to bacterial infection has been reported in mice at below 80ppb ozone for a single 3-hr exposure (Ehrlich *et al.* 1977). Related alterations of the pulmonary defenses caused by short-term exposures to O3 include impaired ability to inactivate bacteria in rabbits and mice (Coffin and Gardner 1972; Ehrlich *et al.* 1979) and impaired macrophage defense mechanisms in the lung (Dowell *et al.* 1970; Goldstein *et al.* 1971; McAllen *et al.* 1981; Amoruso *et al.* 1981). Thus, the biological plausibility of the

adverse air pollution health effects associations found by epidemiological studies is supported by a body of controlled exposure animal studies.

The O_3 - morbidity associations indicated by the above-presented epidemiological studies are also supported by a large body of data from controlled human exposure studies that give consistent and/or supportive results, and that have demonstrated pathways by which ozone can damage the human body when breathed. Clinical studies have demonstrated decreases in lung function, increased frequencies of respiratory symptoms, heightened airway hyper-responsiveness, and cellular and biochemical evidence of lung inflammation in healthy exercising adults. For example, in controlled exposure studies, McDonnell *et al.* (1991) and Devlin *et al.* (1991) found that prolonged controlled exposures of exercising men to levels of ozone common in present-day U.S. (only 80 ppb) resulted in significant decrements in lung function, induction of respiratory symptoms, increases in nonspecific airway reactivity, and cellular and biochemical changes in the lung.

Ozone exposure has also been shown to have adverse effects on athletic performance. Epidemiological evidence compiled more than three decades ago suggested that the percentage of high school track team members failing to improve performance increased with increasing oxidant concentrations the hour before a race (Wayne *et al.* 1967). Controlled exposure studies of heavily exercising competitive runners have demonstrated decreased function at 200 to 300 ppb (Savin and Adams 1979; Adams and Schelegle 1983). More recent studies have shown reduced athletic performance at even lower O_3 concentrations. Schlegle and Adams (1986) exposed 10 young male adult endurance athletes to 120, 180, and 240 ppb O3 while they exercised for 60 minutes. Although all 10 completed the protocol for filtered (clean) air exposure, 1, 5, and 7 of them could not complete it for the 120, 180 and 240 ppb O3 exposures, respectively, indicating that higher O3 concentrations made exercising more difficult.

Another study considers a broadly relevant case showing the benefits of cleaner air. During the Atlanta Summer Olympics of 1996, traffic-related ozone and PM declined significantly as a result of the alternative mass transportation strategy implemented to reduce road traffic during the Games (Friedman *et al.*, 2001). These improvements were correlated with changes in the rate of children's hospital admissions. Compared to a baseline period, traffic related ozone and PM10 levels declined by 28% and 16%, respectively.

Concentrations of both PM and ozone also rose noticeably after the end of the Olympics. The study showed a significant reduction in asthma events associated with these pollution improvements. This study supports the hypothesis that improvements in acute air pollution can provide immediate health benefits.

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

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

Emergency Room Visits and Hospital Admissions are also increased by O3 air pollution. Cody *et al.* (1992) analyzed data on New Jersey hospital emergency department (ED) visits for asthma, bronchitis, and finger wounds (a non-respiratory control) for the period May through August for 1988 and 1989, finding that, when temperature was controlled for in a multiple regression analysis, a highly significant relationship between asthma visits and ozone concentration was identified. In addition, a 5-year retrospective study by Weisel *et al.* (1995) of the association between ED visits for asthma with mean ambient ozone levels was conducted for hospitals located in central New Jersey. An association was identified in each of the years (1986-1990), and ED visits occurred 28% more frequently when the mean ozone levels were greater than 60 ppb O3, as compared to when they were less than 60 ppb O3.

Epidemiological evidence has accumulated over recent years indicating a role of O3

in daily hospital admissions. As displayed in Figure 11, time-series studies conducted in the U.S. have shown increased risk of hospital admissions (Relative Risk>1.0) at higher O3 levels, even after accounting for the effects of PM (Schwartz, J. in Health at the Crossroads, 1997). This work has now been expanded to consider 36 cities across the U.S., finding that, during the warm season of the year, the 2-day cumulative effect of a 5-ppb increase in O3 was an estimated 0.3% increase in the risk of chronic obstructive pulmonary disease admissions, and a 0.4% increase in the risk of pneumonia admissions (Medina-Ramon *et al.*, 2006).

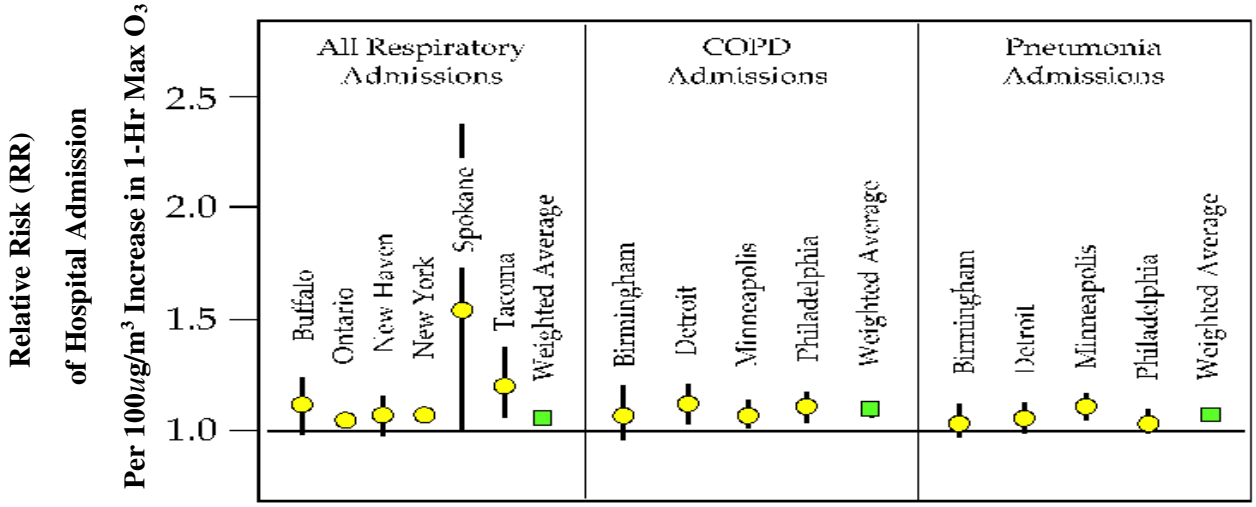

Figure 11. Studies of air pollution in many cities have shown increased risk of respiratory hospital admission (RR >1.0) on days of high ozone air pollution (Source: Schwartz, J. in Health at the Crossroads, 1997).

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

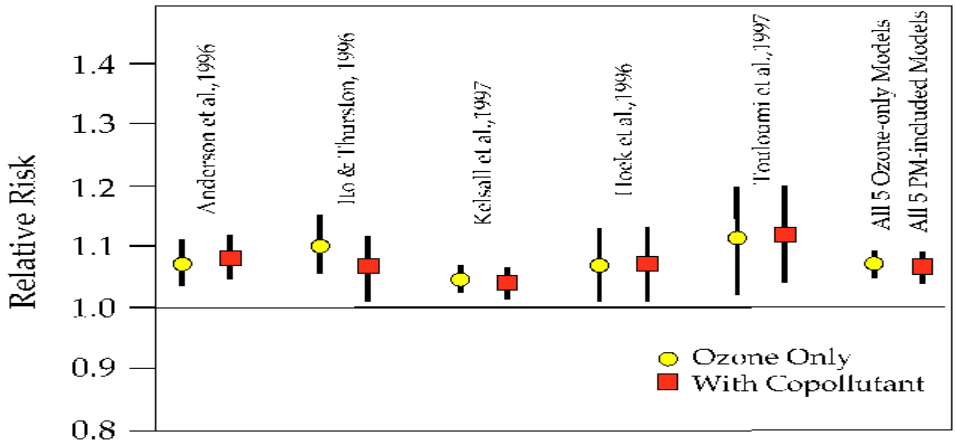

Figure 12. Studies indicate an increased risk of mortality (RR >1.0) at higher ozone concentrations, even after considering the effects of PM. (Source: Thurston and Ito, 2001)

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

More recently, mortality effects from long-term exposure to ozone air pollution has now been confirmed in a major cohort study (Jerrett et al, 2009; Turner et al, 2016). In Jerrett et al, data from the study cohort of the American Cancer Society Cancer Prevention Study II were correlated with air-pollution data from 96 metropolitan statistical areas in the United States. 448,850 subjects, with 118,777 deaths in an 18-year follow-up period were considered. Data on daily maximum ozone concentrations were obtained from April 1 to September 30 for the years 1977 through 2000. Data on concentrations of fine particulate matter (PM2.5) were obtained for the years 1999 and 2000. Associations between ozone concentrations and the risk of death were evaluated with the use of standard and multilevel Cox regression models. In single-pollutant models, ozone was associated with the risk of death from respiratory causes. The estimated relative risk of death from respiratory causes

that was associated with an increment in ozone concentration of 10 ppb was 1.040 (95% confidence interval, 1.010 to 1.067). The association of ozone with the risk of death from respiratory causes was insensitive to adjustment for confounders and to the type of statistical model used. In a follow-up analysis of this same database, Turner et al (2016) improved ozone exposure estimates by employing estimates of O3 concentrations at the participant's residence, as derived from a hierarchical Bayesian space–time model. In two-pollutant models, adjusted for PM2.5, significant positive associations remained between O3 and all-cause (hazard ratio [HR] per 10 ppb, 1.02; 95% confidence interval [CI], 1.01–1.04), circulatory (HR, 1.03; 95% CI, 1.01–1.05), and respiratory mortality (HR, 1.12; 95% CI, 1.08–1.16) that were unchanged with further adjustment for NO2.

Exposures to nitrogen oxides themselves have also been associated with adverse human health effects, in addition to leading to the formation of PM2.5 and ozone. As concluded in a U.S. EPA Risk and Exposure Assessment Report for NOx (EPA-452/R-08-008a), research studies have provided scientific evidence that is sufficient to infer a similar relationship to also exist between short-term (e.g., daily) NO2 exposure and adverse effects on the respiratory system. This finding is supported by the large body of recent epidemiologic evidence as well as findings from human and animal experimental studies. These epidemiologic and experimental studies encompass a number of endpoints including ED visits and hospitalizations, respiratory symptoms, airway hyperresponsiveness, airway inflammation, and lung function (U.S. EPA, 2008).

D. CONCLUSIONS

It is my conclusion that additional emissions from the proposed facility will add to the existing levels of PM2.5 and nitrogen oxides in the vicinity of the facility, and, because no threshold of air pollution effects has yet been found, any incremental air pollution exposures add an incremental adverse health risk to residents near a source of fossil fuel combustion air pollution. Also, such 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 PM2.5 and other air pollutants 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 PM2.5, it will cause an increase in the risk of adverse health effects among those who breathe that pollution, and especially for the socio-economically disadvantaged populations living within the most affected areas immediately surrounding the facility. Furthermore, in addition to the effects of PM2.5, the proposed facility's emissions of nitrogen oxides will also contribute to the increases in health risks from added local air pollution, as well as to the downwind formation of, and exposures to, ozone air pollution, and to associated downwind increases in adverse human health effects caused by those incremental O3 exposures. This, this proposed facility will have both local and downwind adverse human health consequences.

Although the state's air quality modeling report concludes that " the results of the air quality modeling analysis demonstrate that the proposed Buckingham Compressor Station Project does not cause or contribute to any exceedance of the NAAQS for NO2, PM2.5, PM10 and CO", this does not mean there are no health impacts, as there are no known thresholds of effects, as documented in this report. Quite the opposite, the report results indicate to me that the adverse human health effects of long-term (annual) exposures to PM2.5 at these locations will rise by at least 21%, while the adverse human health effects of short-term (24-hr) exposures to PM2.5 will rise by at least 44%. The reason the rise in risk will likely be higher than these percentages indicate is that, as discussed in this report, the PM2.5 from fossil fuel combustion has much greater health impacts (up to 5 times higher) than most other types of PM2.5 mass.

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