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February 25, 2009

Re: Human Health Effects of the Air Pollution Generated by the Windsor Essex Parkway as Compared to a Tunneled Alternative

Dear Mr. Estrin,

As requested, please find enclosed my expert opinion in relation to the human health impacts of the air pollution that will be generated by the Windsor-Essex Parkway ("Parkway"), as compared to a tunneled alternative.

Executive Summary

As set out below in detail, my review has determined that the Windsor Essex Parkway ("Parkway"):

- Will generate significant negative health impacts for people living, working, playing and going to school adjacent to the Parkway as proposed;
- The proposed Parkway's negative human health impacts can be estimated on both a short and long-term basis, and include:
 - increased risk of asthma attacks and difficulty breathing;
 - increased risk of emergency room visits and hospitalization;
 - increased risk of stroke;
 - increased risk of heart attack;
 - increased risk of premature death;
 - increased risk of all causes of death;
- My review of the DRIC Human Health Risk Assessment indicates that it is significantly deficient for the following reasons:
 - The Parkway's negative health impacts have not been assessed due to the incorrect premise stated in the DRIC Air Quality Assessment TEPA (December 2008) that particulate matter is "not considered a health-based contaminant";
 - Both PM_{2.5} and PM₁₀ are indeed health-based contaminants, with known increases in the risk of negative human health impacts per unit of increased pollution;
 - These negative human health impacts from the Parkway will be added to existing poor air quality that already places residents in Windsor, Ontario at risk;
 - The negative human health impacts of PM₁₀ outside the ROW, for the homes, daycares, schools and old folks' homes located adjacent to the access road have not been evaluated;

- Human health impacts from all of the pollutants that will be present within the Parkway's trails and greenspace have not been evaluated, despite excessively high pollution levels in an area that will be frequented by the human health "receptors" at greatest risk – babies, toddlers, children, pregnant mothers, grandparents and people with pre-existing medical conditions;
 - The Parkway's greenspace is designed to invite people to exercise and recreate, despite levels of pollutants so high that the risk of heart attack may increase as much as 563% after a 2 hour exposure in the highest exposure locales;
 - The Parkway's negative health impacts from PM_{2.5} have been dismissed on the basis that the impacts are similar to the No Build alternative, and it is not made clear that these negative health impacts could be avoided through the use of tunneling;
 - The potential human health benefit of a tunneled roadway, which could be used to effectively shield adjacent homes and sensitive individuals from the negative human health impacts of inhalable and respirable particulate matter resulting from the roadway has not been discussed.
- I therefore conclude that the Parkway design results in significant acute and chronic adverse risks to the public health of those living, working, or recreating adjacent to the Parkway as a result of exposure to PM_{2.5} and PM₁₀. These negative impacts could be avoided by the use of real tunneling, to shield these sensitive receptors from exposure to these contaminants, and bring some relief from the elevated PM exposures to which residents are presently exposed in Windsor, Ontario.

For ease of reference, this opinion is divided into the following sections:

- Background and Qualifications
- Scope of Work
- Background on Particulate Matter Air Pollution
- Air Quality Criteria for Particulate Matter
- The Impact of the Proposed Parkway on Particulate Air Pollution in Windsor
- DRIC's Evaluation of the Human Health Impacts
- Particulate Matter Impacts on Human Health
- Short Term (Acute) Health Impacts
- Long Term (Chronic) Health Impacts
- Parkway Impacts on Human Health
- Conclusions

Background & Qualifications

I am a full Professor at the New York University (NYU) School of Medicine in the Department of Environmental Medicine. I have also served as the Deputy Director of the NYU Particulate Matter Health Research Center, beginning in 2002.

I received my undergraduate degree in Environmental Engineering from Brown University in 1974, and my Doctorate of Science (Sc.D.) in Environmental Health Sciences from the Harvard University School of Public Health in 1983. I have served on the New York State Department of Environmental Conservation's Air Management Advisory Committee from 1991 to 1996, and 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 human health effects of inhaled air pollutants, particularly in relation 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 multiple times in recent years to provide testimony before them regarding the human health effects of air pollution.

I presently serve as an advisor to the U.S. EPA regarding the human health effects of air pollution as a member of the Clean Air Scientific Advisory Committee (CASAC) panel on Sulfur Oxides and Nitrogen Oxides. A copy of my professional curriculum vitae is attached to this letter, and it accurately represents my relevant education, training, and experience.

Scope of Work

I was retained to provide an expert opinion in relation to human health effects of the air pollution levels modelled in the TEPA Air Quality Impact Assessment (December 2008) and the conclusions set out in this regard within the DRIC TEPA Human Health Risk Assessment, with a particular focus on particulate matter exposure. In the course of preparing my opinion, I reviewed reports and background material including, but not limited to, the following reports:.

- i. the Canada-United States-Ontario-Michigan Border Transportation Partnership's May 2005 DRIC Environmental Assessment Study: Environmental Overview Paper-Canadian Existing Conditions Volume 1;
- ii. the Canada-United States-Ontario-Michigan Border Transportation Partnership's May 2008 DRIC Environmental Assessment Study: Practical Alternatives Evaluation Working Paper: Air Quality Impact Assessment ("May Report"); and,
- iii. the Canada-United States-Ontario-Michigan Border Transportation Partnership's December 2008 DRIC Environmental Assessment Study: Air Quality Impact Assessment, Technically and Environmentally Preferred Alternative ("December Report"); and,
- iv. the Canada-United States-Ontario-Michigan Border Transportation Partnership's December 2008 DRIC Environmental Assessment Study: Human Health Risk Assessment, Technically and Environmentally Preferred Alternative ("HHRA").

My opinion addresses the public health impacts of emissions of particulate matter (PM) generally and, specifically, the public health impacts of fine particle (PM_{2.5}) and thoracic particle (PM₁₀) exposures resulting from vehicles driven on roads, tunnels, and bridges like those being proposed by the DRIC. Please note, in relation to terminology, that the Ministry of the Environment for Ontario ("MOE") also refers to fine particulate or PM_{2.5} as "respirable particulate", and thoracic particle or PM₁₀ as "inhalable particulate".¹

Background on Particulate Matter Air Pollution

Particulate matter includes aerosols, smoke, fumes, dust, fly ash and pollen. The greatest effect on health is from particles 10 micrometers or less in diameter, which can enter the lung and then have the potential to cause numerous adverse health effects, including the aggravation of bronchitis, asthma and other respiratory diseases.

Particles in the atmosphere have therefore been characterized according to size. The broadest category is Total Particulate Matter or Suspended Particulate Matter, which measures all particles smaller than approximately 50 microns in diameter. The next category is PM₁₀, which measures only those particles less than or equal to 10 micrometers in size, which are termed thoracic or inhalable particulate matter (PM₁₀). The very smallest particulate matter category is fine particulate matter or respirable particulate matter (PM_{2.5}), which measures only those particles less than or equal to 2.5 micrometers in diameter. To provide a point of comparison, fine particulate matter is "approximately 30 times smaller than the average diameter of a human hair".² PM₁₀

¹ Ontario Ministry of the Environment, "Chapter 3: Fine Particulate Matter", Air Quality in Ontario, 2000.

² Ontario Ministry of the Environment, "Chapter 3: Fine Particulate Matter", Air Quality in Ontario, 2007, at pg. 13.

measurements are inclusive of PM_{2.5}, and measures of total particulate or PM includes both PM₁₀ and PM_{2.5}.

In relation to the particulate matter fractions (or sizes) of greatest health concern, PM₁₀ is called “inhalable” particulate because it is comprised of those particles that are small enough to pass through the trachea and into the lung. PM_{2.5} are the smallest of the PM₁₀ particles, usually comprising on the order of 60 % of the PM₁₀, and it is called “respirable” because it can penetrate into the deepest recesses of the lung.

Among the major anthropogenic sources of airborne PM₁₀ particles are a variety of industrial sources, motor vehicles exhausts, residential wood combustion, and road dust generated by motor vehicle traffic. PM_{2.5} a subset of PM₁₀, is primarily formed in the atmosphere from chemical reactions, in addition to direct emission by fossil fuel combustion sources, including exhaust from cars and trucks. People with preexisting cardiovascular or lung disease, as well as the very young and old, are among those considered to be at the most risk for the most severe effects of both PM₁₀ and PM_{2.5} particulate matter. Particle air pollution is also responsible for environmental “welfare” effects, including corrosion, soiling, damage to vegetation, and reductions in visibility.

Air Quality Criteria for Particulate Matter

As a result of the growing concern for health effects due to breathable particulate matter, Ontario introduced, in November 1997, an interim thoracic particulate matter (PM₁₀) criterion of 50 µg/m³ on a 24-hour basis.³ Shortly thereafter, in 2000, the Canadian Council of Ministers of the Environment (CCME) also developed a Canada-Wide Standard (CWS) for PM_{2.5} as a result of the growing awareness of this portion of PM’s adverse effects on human health and the environment.⁴ As referenced in the Guidance Document on Achievement Determination, the CWS for PM_{2.5} is 30 micrograms per cubic meter (µg/m³), based on the 98th percentile ambient measurement annually averaged over three consecutive years. Jurisdictions are required to meet the CWS for PM_{2.5} by year 2010, with reporting commencing in 2011.⁵ Ontario has committed to achieving the CWS, as set out in the Ontario Clean Air Action Plan (2004).⁶

The PM₁₀ and PM_{2.5} criteria levels set out above will be used in the following discussion as “yardsticks” for assessing the air pollution data modelled and monitored by DRIC.

The Impact of the Proposed Parkway on Particulate Air Pollution in Windsor, Ontario

Air quality monitoring data provided in the above-noted DRIC reports, and data reported in recent Ontario Ministry of the Environment’s annual Air Quality in Ontario reports, indicate that high levels of PM₁₀ already exist in Windsor, Ontario, and that these pollutant exposures will be exacerbated in locales near the proposed Parkway alternative.⁷

For example, as shown in Figure 1, in the years when PM₁₀ measurements were available across the province, Windsor has been the city with the highest PM₁₀ levels in Ontario, with ambient levels higher than Hamilton.

³ Supra note 1, at pg. 15.

⁴ Canadian Council of Ministers of the Environment, Canada Wide Standards for Fine Particulate Matter and Ozone, 2000, at pg. 4, available at http://www.ccme.ca/assets/pdf/pmozzone_standard_e.pdf.

⁵ Supra note 2 at pg.17.

⁶ Ontario Ministry of the Environment, “Clean Air Action Plan: Protecting Environmental and Human Health in Ontario”, June 21, 2004.

⁷ See, for example, : <http://www.ene.gov.on.ca/en/publications/air/index.php#9a>).

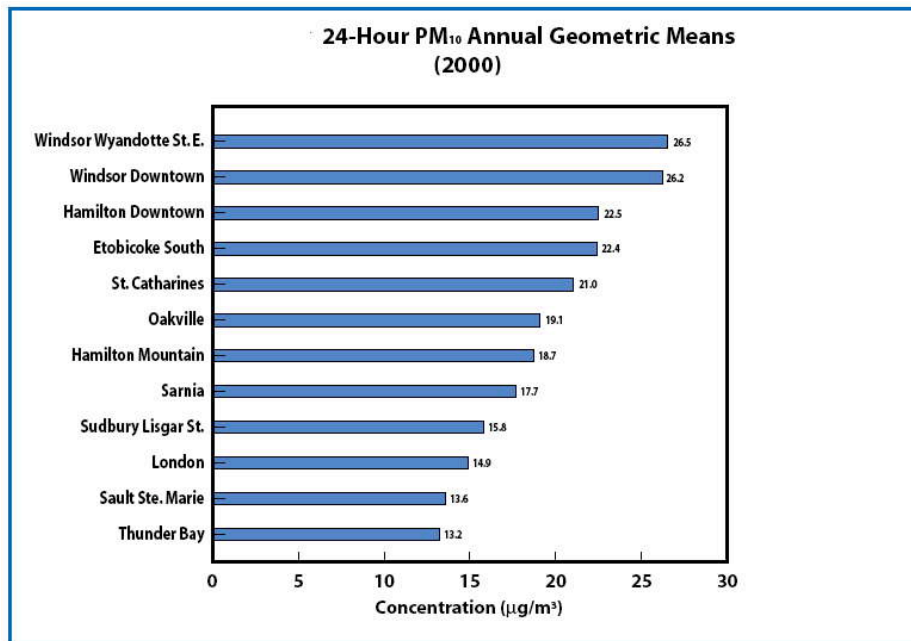


Figure 1. Annual Levels of PM₁₀ in 2000 (OME, Air Quality in Ontario 2001 Report)

While PM₁₀ impacts were not modelled in the May 2008 DRIC report, the report did note that the only alternative with real tunneling, Alternative 3 (“Tunneled Alternative”) was not predicted to have any exceedances of PM_{2.5}, even as close as 50 meters of the edge of the right-of-way (“ROW”). DRIC also observed that the Tunneled Alternative showed air pollution reductions over No Build within the first 100 m from the edge of the ROW:

“Reductions for Alternative 3, the Tunnel, are attributable to emission sources being covered and vented at tunnel portals, with this effect generally limited to within 50 -100 m of the ROW...Exceedances of the CWS PM_{2.5} 24-hour standard are generally predicted to occur under certain conditions within 50 m of ROW for all alternatives in all horizon years, except for Alternative 3 which is not predicted to have exceedances.”⁸

It is logical that the advantages of the Tunnel option over the Parkway would be most apparent in the first 100 meters from the right-of-way, as the air quality impact of a roadway’s emissions declines exponentially with distance, with the greatest impacts being felt in the first 100 meters downwind of a roadway. It is also clear that DRIC has not reported results between 100 m and 250 m from the edge of the ROW, so that protective effects from tunneling in relation to receptors located within 101-249 m from the edge of the ROW are also not set out in the December Report.

Furthermore, the DRIC December Report also notes that the PM₁₀ road dust emissions generated by traffic are some 7 times higher than those as PM_{2.5}, indicating that the advantages of the Tunnel option vs. the Parkway would have been far more apparent had the DRIC report also compared the PM₁₀ pollution caused by the alternatives in the May Report, rather than using PM_{2.5} as the basis for comparison.⁹ This is evident looking at the December Report, which shows a much higher maximum value of the former (PM₁₀=114 ug/m³) than for the latter (PM_{2.5}= 30 ug/m³).

The December 2008 DRIC air quality and health effects reports are also incomplete, in that they do not discuss the protective or mitigating effect that a tunnel would have in relation to particulate exposures, in strong contrast to the air quality deterioration modeled by DRIC for both the Parkway and No Build in the December

⁸ DRIC Draft Practical Alternative Working Paper: Air Quality Impact Assessment, May 2008, at pg. 48, emphasis added.

⁹ DRIC Draft Practical Alternative Working Paper: Air Quality Impact Assessment, May 2008, Table 4 “Paved Road Parameters”, pg. 155.

Report. In DRIC's May Report, as quoted above, the Tunneled Option was shown to eliminate 100% of the failures of PM_{2.5} criteria, in areas that were fully tunneled. In comparison, the Parkway and No Build were both indicated to generate air quality criteria failures. Clearly, the protective effect of the Tunnel option would have shown even more clearly in May 2008 had PM₁₀ been used to evaluate the various options for selection as the preferred alternative.

It is my opinion that, from the perspective of human health impacts, the Tunnel alternative is the most appropriate comparison with the other access road alternatives, not the "No Build" option, as incorrectly assumed in the DRIC reports. By comparing the impacts of the Parkway with the No Build option alone, DRIC has not described the fact that *both* the Parkway and No Build allow air quality to deteriorate to the point where it regularly exceeds (or fails) to meet Ontario's criteria for particulate matter. If all three alternatives had been included, the reader would clearly see that only the Tunneled Alternative would prevent the deterioration of air quality modeled for both the No Build and the Parkway, which is what will generate human health impacts of concern in the HHRA.

Thus, from an air quality and human health impact perspective, the fully Tunneled Option is the more appropriate baseline against which the environmental and health impacts of the various build options should have been compared, not the "No Build" option. Unfortunately, the December Report and the HHRA both fail to provide comparisons with the much lower impact Tunneled Option for either PM_{2.5} or PM₁₀, masking both the unnecessary negative human health impacts that will result from the Parkway versus the human health benefits (i.e., reductions versus what is prevailing today) that could be expected from choosing the Tunneled Option alternative.

DRIC's Evaluation of the Human Health Impacts

DRIC's December Report shows that there are many days of high PM₁₀ concentrations near the proposed Parkway. As a result, there are increases in adverse health effects risks associated with those elevated PM₁₀ exposures. However, the DRIC HHRA focuses only on PM_{2.5}, ignoring any possible health effects from the high levels of PM₁₀ both outside the ROW, in relation to residential receptors, daycares, schools and old-folks homes that exist adjacent to the Parkway, and inside the Parkway's greenspace that are indicated in its companion air quality report.

In fact, DRIC's December Report asserts that "PM₁₀ is **not considered a health based contaminant**".¹⁰ I disagree with that statement. As set out in detail in the following section, PM₁₀ is without question a contaminant of health concern, and one that has an air quality criterion that was established as a result of concern regarding human health impacts. To be more accurate, DRIC should have observed that the HHRA reviewed the human health impacts of PM₁₀ set out in the scientific literature, but instead remarkably failed to evaluate, in light of this literature, the significant levels of PM₁₀ pollution predicted for the Parkway.

It is revealing to view, in the December Report, just how far out from the Parkway that impacts reach that are still above the PM₁₀ criteria level (50 ug/m³), and how much the roadway contributes as an increment over background. For example, Table 4.18 on pg. 59 of the December DRIC air quality report shows hundreds of exceedances of the PM₁₀ criteria all along the proposed Parkway, while Table 4.17 of this report shows routine exceedances occurring from as far as 290 meters away from the Parkway.¹¹ Despite citing toxicological and epidemiological literature to the contrary (e.g., see pages 29 and 30 of the report), the December health effects document inappropriately ignores the considerable potential for human health consequences from such repeatedly high levels of PM₁₀ over a wide swath of land adjacent to each side of the proposed Parkway.

¹⁰ DRIC Air Quality Impact Assessment TEPA, December 2008 at pg. 53.

¹¹ DRIC Air Quality Impact Assessment TEPA, December 2008 at Table 4.16 at pg. 57.

The December DRIC air quality report also considers the PM_{2.5} impacts, and states on page 61 that: “PM_{2.5} concentrations are typically not expected to exceed the Canada Wide Standard (CWS) and changes between the TEPA and No Build scenarios are generally within 10% of each other except in locations where significant additional traffic is increased (Plaza B1).” In other words, the Parkway (TEPA) provides little improvement over, and sometimes is worse than, the No Build alternative. Table 4.20 shows that, under the TEPA Parkway option the PM_{2.5} levels would reach up to a maximum of 30 ug/m³ (equal to the criteria for PM_{2.5}), compared to a background of 21 ug/m³. Significant adverse health consequences should be expected to result from this pollution, whether generated by the No Build or the Parkway. It is noteworthy that DRIC’s HHRA uses 7 ug/m³ as the health-based threshold for negative impacts (pg. 28, HHRA), and the literature discussed below measures impacts in increments as small as one microgram (ug/m³), such that an increment of 9 ug/m³ PM_{2.5} over background (from 21 to 30 ug/m³) would have an impact on the adverse human health risks associated with particulate matter exposures in the vicinity of the roadway.

As was the case with PM₁₀, no information is given as to the comparative extent of improvement that might be attained by the Tunneled Option, as compared to either the Parkway or No Build

Particulate Matter Impacts on Human Health

Inhalable and respirable particulate matter have both acute and long-term negative impacts on human health. In general terms, increasing exposure to particulate matter increases the risk of negative human health impacts, as discussed below, and decreasing exposure to particulate matter provides human health risk benefits.

To illustrate the benefits of decreasing exposure, a very recent study published in the New England Journal of Medicine on January 22, 2009, demonstrated that decreasing exposure to particulate matter by just 10 ug/m³ increased life expectancy by an average of 7 months.¹² In metropolitan areas, decreases of 13 –14 ug/m³ in the annual average PM_{2.5} was estimated to increase life expectancy by as much as 9.8 months.¹³ These increases were indicated even after accounting for other important co-factors such as smoking and socio-economic status. The documentation of the adverse human health effects of air pollution includes impacts demonstrated by both controlled chamber exposures and observational epidemiology, which shows consistent associations between particulate matter air pollution and adverse impacts across a wide range of human health outcomes (e.g., see: U.S. Air Quality Criteria for Particulate Matter (October, 2004) EPA/600/P-99/002aF). Amongst this evidence, observational epidemiology studies, such as the ones I have conducted, have shown some the most compelling and consistent evidence of adverse effects by air pollution.

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 shown confirmatory associations between ambient air pollution exposures and increased adverse health impacts, including, but not limited to:

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

The fact that these effects have both been shown so consistently across outcomes and from place to place across the nation, and around the world, indicates these associations to be causal.

¹² C. Arden Pope, M. Ezzati, D. Dockery. “Fine Particulate Air Pollution and Life Expectancy in the United States”, New England Journal of Medicine: Volume 360:376-386, see “Discussion” at pg. 7 of 11 (online version).

¹³ Ibid.

Particulate matter is one of the ambient air pollutants most carefully studied in recent years. Inhalable and fine particulate matter (PM_{10} and $PM_{2.5}$), such as that resulting from vehicular emissions, can bypass the defensive mechanisms of the lung, and become lodged in the lung, where they can cause a variety of health problems. Indeed, the latest evidence indicates that short-term exposures can increase the risk not only of respiratory damage, but also of cardiac effects, including heart attacks. Moreover, long-term exposure to fine particles increases the risk of death, and has been estimated to take many months from the life expectancy of people living in the most polluted cities, relative to those living in cleaner cities.

In my own research, I have found that acute increases in ambient air pollution are associated with increases in the number of daily asthma attacks, hospital admissions, and mortality. In particular, I have found that particulate matter air pollution is associated with increased numbers of respiratory hospital admissions in New York City, Buffalo, NY, and Toronto, Ontario, as well as with mortality in cities such as Chicago, IL and Los Angeles, CA, even at levels below the current PM standards. My results have been confirmed by other researchers considering locales elsewhere in North America and throughout the world (e.g., see: Schwartz, J. (1997) Health effects of air pollution from traffic: ozone and particulate matter. Health at the Crossroads: Transport Policy and Urban Health, T. Fletcher and A.J. McMichael Eds., John Wiley and Sons Ltd., New York, NY.)

In particular, there is growing scientific evidence indicating that particulate matter (PM) air pollution emitted by traffic is among the more important contributors to the toxicity of PM. An increasing body of evidence indicates that traffic-related exposures and residential proximity to vehicular traffic are associated with increased respiratory conditions and symptoms in children, including increased prevalence of asthma, wheezing, recurrent respiratory illnesses, and hospital admissions for asthma. In my own research of elementary children in the South Bronx in New York City (which is an area adjacent to the Major Deegan Interstate highway and to the Triboro Bridge) indicates that there is a statistically significant increase in children's asthma symptoms, as well as a reduction in their lung function, on days with elevated levels of elemental carbon soot (such as that emitted by diesel vehicles). This particular research has led to a recent article on the effects of diesel pollution on children with asthma in the New York Times (October 29, 2006) entitled "A Study Links Trucks' Exhaust to Schoolchildren's Asthma" and a subsequent New York Times editorial (11/19/06) entitled "Black Soot and Asthma" in which the editors called upon policymakers to reduce this problem by "declaring war on poisonous diesel fumes." As a result of this research, we have found, for example, that there is a 40 to 45% increase in the occurrence of wheeze and shortness of breath symptoms among urban children with asthma on days with an increase of only 3 ug/m^3 of elemental carbon soot (EC), which represents a large fraction of the fine particles ($PM_{2.5}$) emitted by automobiles and trucks. Thus, assuming approximately one-third of the traffic $PM_{2.5}$ is as EC, for a day that is some 9 ug/m^3 higher in $PM_{2.5}$ due to roadway emissions, it could be expected that children with asthma in that locale would experience on the order of a 40% increase in their risk of these asthma symptoms vs. if they were not so exposed to this roadway-associated fine particle PM.

Among the particulate matter (PM) that is emitted by vehicles and other sources, the smaller, inhalable, particles have been indicated to be of the greatest health significance. Nasal hairs, sneezing, and the lung's mucociliary clearance system are among the lung's natural defenses against particles. But smaller particles can get past these defenses, and penetrate into the deepest recesses of the lung, making them especially dangerous to human health. The United States Environmental Protection Agency ("EPA") recognized the adverse health effects of small particulate pollution as early as 1987 when, pursuant to its authority under the Clean Air Act, it promulgated a National Ambient Air Quality Standard ("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."

Short Term (Acute) Health Impacts

Both PM₁₀ and PM_{2.5} exposures have been quantitatively associated with a range of adverse human health risks via epidemiology, ranging from morbidity to mortality in severity, and from acute to chronic in timespan. One of the PM₁₀ acute health effects studies noted in the DRIC HHRA was the National Mortality and Morbidity Air Pollution Study (NMMAPS) that evaluated data from 90 large U.S. cities (Dominici et al 2003). This study showed that cardiopulmonary mortality increased immediately (within 1 to 2 days after air pollution exposure) by 0.21% for each 10 ug/m³ increase in PM₁₀.

As noted in the December DRIC health effects report: “The importance of this is that the particulate matter exposures that North Americans breathe on an almost daily basis have a measurable impact in our daily mortality total.” Zanobetti et al. (2000) also performed a multi-city analysis that found, for each increase of 10 ug/m³ of 24-hour PM₁₀, chronic obstructive pulmonary disease (or “COPD”) hospitalization rates increased by 2.5 %, pneumonia rates increased by 1.95 % and cardiovascular disease rates increased by 1.27 %.

Similarly, Wellenius et al. (Stroke. 2005 Dec;36(12):2549-53.) evaluated the association between daily levels of PM₁₀ and hospital admission for ischemic stroke among Medicare recipients (age > or =65 years) in 9 US cities. This study found that a one day 23 ug/m³ increase in PM₁₀ was associated with a 1.03% (95% CI, 0.04% to 2.04%) increase in ischemic admissions on the same day. Dominici et al. (2006) also examined hospital admissions associations with short-term exposure to PM_{2.5}, finding that heart failure had a 1.28 % increase in risk per 10 ug/m³ increase in same-day PM_{2.5} concentration.

Peters et al. (Circulation. 2001 Jun 12;103(23):2810-5) have found that elevated concentrations of PM in the air can dramatically increase the risk of Myocardial Infarctions (MI's), or heart attacks, within a few hours, and extending 1 day after PM exposure. The Harvard University team found that a 66 percent increase in the risk of MI was associated with an increase of 30 ug/m³ PM₁₀ during a 24-hour period prior to the onset of MI, and a 51 percent increase in MI risk to be related to an increase of 40 ug/m³ in the 2-hour average PM_{2.5} before the MI onset. This study makes clear that even brief exposures to elevated PM₁₀ concentrations are associated with large increases in the risk of very severe health effects in susceptible members of the public. Clearly, short-term increases in either PM₁₀ or PM_{2.5} are associated with increased acute risks of severe morbidity and mortality effects.

Long Term (Chronic) Impacts

Long-term particulate matter is also associated with increased risk of chronic disease and mortality. For example, as noted in the December DRIC health effects report: “The American Cancer Society and the Harvard Six Cities studies are both landmark cohort studies that have shown a link between PM_{2.5} and mortality.” The 2002 re-analysis of the American Cancer Society data indicated the long-term all-cause, cardiopulmonary, and lung cancer mortality were increased by a 4%, 6%, and 8%, respectively, for each 10 ug/m³ increase in fine particulate matter (Pope et al. 2002). I was the Senior (last) author, and Principal Investigator of this 2002 NIH-funded reanalysis of the long-term (chronic) cumulative exposures to PM. Not discussed in the DRIC HHRA, however, was that, in this study, PM₁₀ yielded an effect estimate of a 10% increase in cardiopulmonary mortality per 29 ug/m³ increase in PM₁₀. In another study that I was involved with, cardiovascular causes of death increased by 8-18% per 10 ug/m³ of PM_{2.5}.¹⁴ In addition, a recent U.S. EPA Particulate Matter Expert Elicitation reported results consistent with a 1% effect on mortality per ug/m³ of PM_{2.5}, indicating that the Pope et al (2002) results may provide a low estimate of the mortality effects of long-term PM air pollution exposure (Roman et al., Environ. Sci. Technol. 2008 Apr. 1;42(7):2268-74.). Overall, it is clear that both PM_{2.5} and PM₁₀ exposures are associated with significant long-term adverse implications to health and mortality.

¹⁴ C. A. Pope, R. T. Burnett, G. D. Thurston, M. J. Thun, E. E. Calle, D. Krewski, J. J. Godleski. “Cardiovascular Mortality and Long-Term Exposure to Particulate Air Pollution”, Circulation: January 6/13, 2004.

Parkway Impacts on Human Health

The DRIC HHRA inappropriately ignores potential PM₁₀ effects on morbidity and mortality, both inside the Parkway's greenspace and for receptors outside the Parkway ROW. The HHRA also dismisses the potential health effects of the Parkway associated PM_{2.5} exposures. The DRIC report concludes (on page ES-4) that:

“Predicted concentrations of gaseous air pollutants, fine particulate matter, and Volatile Organic Compounds for the Future “No Build” and TEPA scenarios are not much different from each other and background. Thus, the TEPA does not result in an increased health risk over the Future “No Build” or background scenarios. This conclusion supports the findings of the Air Quality Impact Assessment.”

Thus, the DRIC HHRA completely ignores any possible impact of the many PM₁₀ exceedances (or failures of the air quality criterion) found in the companion December Report, and does not consider the potential health benefits of significantly *improving* PM_{2.5} and (especially) PM₁₀ levels over those that the Parkway option would generate, through the use of tunneling.

As outlined above, the adverse human health risks associated with breathing the levels of PM₁₀ and PM_{2.5} particulate matter air pollution predicted in these documents are serious and well documented, and should not be dismissed. This is true even at sites predicting PM_{2.5} and PM₁₀ levels at or below the current Canadian air quality guidelines, as these standards are no guarantee of an absence of adverse effects. DRIC implicitly acknowledges this point in the HHRA when it chooses a level of 7 ug/m³ for PM_{2.5} as the health-based threshold for measuring impacts, in contrast to the air quality criterion of 30 ug/m³. The fact that the health-based threshold is lower than the criterion means that health impacts will occur even if the criterion is not exceeded.

In the DRIC HHRA, it is acknowledged (on pages ES-4 and 53) that “Fine particulate matter (PM_{2.5}) background concentrations in the Windsor area are relatively high and are above health based toxicity reference values.” For this reason, there is a public health imperative to not only match the “No Build” option, but if possible to lower levels below what would prevail without the new project by shielding homes and other sensitive receptors, such as daycares, homes for older adults, and schools, from exposure to harmful levels of particulate matter. But in Table 5.3 of this report, the Parkway Health Hazard Quotient is shown to be well above 1.0, and as high or higher than the do nothing “No Build” option in roughly two-thirds (29 of 42) of the locations considered in this DRIC study table. This means that the Parkway option represents a health risk that is as serious as, or in some cases worse than, the No Build scenario.

Therefore, the alternative really needed to provide public health improvement in Windsor is a tunneled option, which would lead to PM_{2.5} and PM₁₀ levels lower than either the No Build or the Parkway options for sensitive receptors and homes adjacent to the access road and would thereby act to reduce the prevailing problematic levels of particulate matter to which those receptors are exposed.

It is possible to get order of magnitude estimates of some of the potential health benefits of choosing the Tunneled Option (vs. the Parkway option) by comparing the reported Parkway impacts with the background level of pollution, which is roughly what the Tunneled Option would likely achieve over most of its length, by eliminating emissions from each open section). This assumption is supported by an examination of the May 2008 report (i.e., in Tables 4.2, 4.6, 4.8, and 4.10), which indicates that the total maximum PM_{2.5} concentration is substantially reduced only by the Tunneled Option. (It is of note that this is not true for Table 4.12, but it is explained on page 62 that there is no tunnel for Alternative 3 at that point in the road, again confirming the benefits of a tunnel vs. open air depressed roadways).

The DRIC December Report assumed 24-hr and annual average background PM_{2.5} levels of 21 and 10 ug/m³, respectively (as set out in Table 3.1 in the HHRA), while the 24-hr average background PM₁₀ was assumed to

be 42ug/m³ (Table 4.18, December Report). The maximum reported concentration of PM_{2.5} for the Parkway is 30 ug/m³, which is an increase of 9 ug/m³ over the background (Table 4.20, December Report).

GreenLink Windsor proposes three tunnels of 1 km or more in length; unlike the Tunneled Option that DRIC studied, they do not use ventilation towers. Since GreenLink would vent the PM pollution elsewhere (presumably away from the public), it may well be that the estimated acute PM_{2.5} effects of the roadway (for either No Build or the Parkway options) would be reduced by nearly 100% for those locations along the ROW that are shielded by GreenLink tunneling. In relation to the overall acute impacts of PM_{2.5} adjacent to the roadway, tunneling might effectively prevent the increased PM levels that the Parkway would bring.

It is also especially relevant to consider the risks that would be taken by residents who visited the greenspace trails and parks over the Parkway, as the nearby tunnel portal concentrations of PM₁₀ would be extremely high (calculated to reach as high as 484 ug/m³ in Table 4.2.3 of the December air quality report). These are very high PM₁₀ concentrations, and are likely associated with very high increases in risk to park users.

For example, based upon the Peters et al. (2001) report, this concentration over a 2-hr period would be associated with an approximate $(484 \text{ ug/m}^3 \text{ max.} - 42 \text{ ug/m}^3 \text{ background}) * 51\%/40 \text{ ug/m}^3 = 563\%$ increase in acute risk of heart attack during a two hour visit to the park over the highway. Now the concentration in the park may well be expected to be somewhat lower than estimated at the portal, but we are applying the maximum 24 hour concentration here, and the 2-hr maximum can be expected to be much higher than that averaged over an entire 24-hour period, so these effects will tend to cancel each other out. At a minimum, it is safe to say that visitors to the parks over the highway would be substantially increasing their cardiac risk during that visit.

Conclusions

Overall, PM_{2.5} and PM₁₀ concentrations in the vicinity of Windsor already approach or exceed the applicable air quality criteria for these air pollutants. Therefore, any action that increases the amount and dispersion of PM₁₀ and/or PM_{2.5} emissions have the potential to worsen this pollution's prevailing adverse impacts on human health.

As a result, the proposed added impacts from fine and thoracic particulate matter (PM_{2.5} and PM₁₀) to the residents of Windsor, as compared to the much lower impacts of the Tunneled Option, are of the type that would have especially adverse health implications when breathed in conjunction with already existing high background levels of particulate matter pollution.

While the adverse human health impacts of breathing these traffic emissions of particulate matter would occur even if the ambient levels were below the ambient air quality standards (since a threshold below which no effects occur has yet to be identified), the impacts are even more onerous when added to the already elevated existing pollution levels.

I therefore conclude that the Parkway design results in significant acute and chronic adverse risks to the public health of those living, working, or recreating adjacent to the Parkway, as a result of exposure to PM_{2.5} and PM₁₀. These negative impacts could be avoided by the use of real tunneling, to shield these sensitive receptors from exposure to these contaminants.

Sincerely,



Dr. George D. Thurston, Sc.D.
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Chester, NY 10918

BIOGRAPHICAL SKETCH

Provide the following information for the key personnel and other significant contributors in the order listed on Form Page 2.
Follow this format for each person. **DO NOT EXCEED FOUR PAGES.**

NAME George D. Thurston	POSITION TITLE Professor
eRA COMMONS USER NAME THURSTON	

EDUCATION/TRAINING *(Begin with baccalaureate or other initial professional education, such as nursing, and include postdoctoral training.)*

INSTITUTION AND LOCATION	DEGREE <i>(if applicable)</i>	YEAR(s)	FIELD OF STUDY
Brown University, Providence, RI	Sc.B.	1974	Environ. Engineering
Brown University, Providence, RI	A.B.	1974	Environ. Studies
Harvard University, Cambridge, MA	M.S.	1978	Environ. Health Sci.
Harvard University, Cambridge, MA	Sc.D.	1983	Environ. Health Sci.

A. Positions and Honors

Positions and Employment

- 1974-1977 Engineer, Hittman Associates, Inc., Energy and Environmental Systems Department, Columbia, MD
- 1978-1982 Researcher, Harvard University School of Public Health, Dept. of Environ. Health, Boston, MA
- 1982-1984 Research Fellow, Harvard University, Kennedy School of Government, Health & Environmental Policy Center, Cambridge, MA
- 1984-1987 Research Assistant Professor, Department of Environmental Medicine, New York University School of Medicine, New York, NY
- 1987-1993 Assistant Professor, Department of Environmental Medicine, New York University School of Medicine, New York, NY
- 1993-2007 Associate Professor (Tenured), Department of Environmental Medicine, New York University School of Medicine, New York, NY
- 2002-pres Deputy Director, NYU Particulate Matter Center, NYU School of Medicine, New York, NY
- 1995-2004 Director, Community Outreach and Education Program, NIEHS Center of Excellence, New York University School of Medicine, New York, NY
- 2007-pres Affiliated Faculty, Environmental Studies Program, College of Arts and Sciences, New York University, New York City, NY.
- 2007-pres Full Professor (Tenured), Department of Environmental Medicine, New York University School of Medicine, New York, NY

Other Experience and Professional Memberships

- U.S. EPA Clean Air Science Advisory Committee (CASAC). Sulfur Oxides and Nitrogen Oxides Committee member (2007-present).
- American Thoracic Society (Environ. and Occup. Health Program Committee)
- International Society for Environmental Epidemiology
- International Society of Exposure Analysis (Associate Editor: J. Exp. Anal. Environ. Epidemiol. (1993-2008))
- National Academy of Sciences, Committee on Health Effects of Waste Incineration (1/95-1/2000)
- ATS/ALA National Air Conservation Commission Member (1997-2000) and National Clean Air Committee (2005-present).
- Chairman, Health and Environmental Impact Assessment Panel, Joint Canadian Industry/Government Study of Sulphur in Gasoline and Diesel Fuels (9/96-6/97)

B. Peer-reviewed publications

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- Thurston, G.D. and Laird, N. Letters: Tracing aerosol pollution. Science 227:1406-1408 (1985).
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