POWER PLANTS, AIR QUALITY AND HEALTH:

The Case for Re-examining Ontario's Coal Policy

A REPORT TO THE ONTARIO POWER WORKERS' UNION

BY

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EXECUTIVE SUMMARY

The economic consequences of closing the province's thermal power plants may be significant. Nevertheless, if the health consequences of operating those plants are even more severe, the province may decide it is the right tradeoff to make. But the decision should not be made before hearing all sides of the debate and undertaking a level of due diligence appropriate to such a costly and far-reaching investment decision. That due diligence includes close scrutiny of the statistical claims that have recently been made concerning the health consequences of coal power plant emissions. This report offers a brief critique of this aspect of the reasoning behind the decision to close the power plants. The following questions are addressed.

1. How has Ontario air quality changed in recent decades?
2. How do power generation plants affect Ontario's air quality?
3. What is the scientific basis for connecting air pollution with disease and mortality in Ontario?
4. What are the main sources of uncertainty in these studies?

The findings are as follows.

1. ***Air quality in Ontario has improved substantially since the early 1970s.***

Most types of air contaminants have been reduced over the past three decades. Recent progress has been lacking in some areas, such as ground-level ozone, although even on that issue long-term progress has been made. Fine particulate matter has been recently identified as a specific potential threat to lung and cardiac health. Unfortunately long term data are not available, though total particulate levels show substantial reductions. The introduction of the smog alert system in the early 1990s may have created a the perception that air quality is worse now than in earlier decades.

2. ***Power plants play a small role in Ontario air quality, and have little impact on severe air quality episodes.***

The air contaminants usually associated with power plants were serious problems back in the 1960s and 1970s. Investments in pollution control and the switch to low-sulphur coal yielded substantial reductions in these pollution emissions. While the power generation sector continues to be a factor in general air pollution, the current focus is on improving pollution levels primarily associated with motor vehicle use, as well as pollutants entering Ontario from the US northeast. Acute air quality episodes in Ontario have been shown to be almost entirely unrelated to power production.

3. ***The scientific basis for linking health to air pollution consists primarily of correlation studies that suggest a small increased risk of mortality and morbidity from air pollution episodes.***

Epidemiological studies have compared air quality readings with model-generated estimates of "excess" mortality and morbidity rates. While results vary across studies, some have found a positive correlation. These correlations are usually reported as "relative risk" ratios, which have been used in simulation models to infer what proportion of observed deaths can be attributed to contemporaneous air pollution levels. The most prominent simulation models in the Ontario debate were from the Ontario Medical Association and the Toronto Public Health Board.

4. ***The air pollution-health link remains highly uncertain; strong evidence exists against it.***

Epidemiological studies have found evidence of both positive and negative correlations between pollution and health. The negative correlations are routinely explained away on the grounds that correlation is not the same as causality, yet the *positive* correlations are cited as evidence of causality. Such contradictory findings suggests that there may be problems of measurement and/or inadequate controls for confounding factors. The commonly-cited studies by the Toronto Public Health Board and the Ontario Medical Association can be shown to lack credibility. Laboratory studies on the medical effects of pollution conflict with the implied toxicity suggested by the epidemiological studies, suggesting that there are no significant toxicities even at concentrations of air pollution much higher than commonly experienced. This contradiction between epidemiological correlations and experimental results was noted by Health Canada in a recent science assessment. New, more comprehensive statistical procedures are showing the epidemiological results to be weaker than earlier thought. And claims that air pollution causes asthma have not been supported by medical research. Taken together these findings suggest that the disease and death rates attributable to levels of ambient air pollution in Ontario are smaller and less certain than has recently been asserted.

Against this background it must be pointed out that coal provides a reliable, low-cost source of electricity to Ontario. At a time of constrained supply and rising prices, there is considerable social benefit to having this power generation capacity on-line.

These considerations lead to the conclusion that thermal power plants are a net benefit to society, and shutting them down at this time is not in the public interest.

It must be emphasized that the economically-risky path of closing down a quarter of the province's generating capacity, or even talking as if it is going to be done shortly, is irresponsible, without first having undertaken extensive due diligence on all aspects of the decision, something which has not been done. In light of the substantial economic costs of abandoning the coal component of Ontario's power supply, and the evidence that Ontario's coal-plant emissions are unlikely to be the threat to life and health that has been claimed, the Ontario government should put on hold the planned phase-out of the thermal power plants by 2007, pending an exhaustive review of the real costs and benefits.

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Introduction

Ontario is a beautiful province, especially in summer. But many people have become concerned that there is a deadly health risk hidden in the fresh air we breathe. In recent years, claims have been made that ambient air pollution in Ontario kills thousands of people and sends tens of thousand more to hospital with coronary and lung problems.[1] The proponents of these fears can point to some statistical studies from Canada, the US and elsewhere that appear to show correlations between variations in air contaminant levels and the rate of mortality or morbidity (disease) in a region. As a result of these fears, attention has turned to major emitters, including the power plants that supply our electricity. The newly-elected Liberal government has pledged to shut down all the coal-fired power plants—representing a quarter of the province's generating capacity—by 2007.

This dramatic initiative is of concern to many groups. Workers at these plants are naturally concerned about the loss of jobs to US power plants, which will have to step in to supply electricity to Ontario, at least in the short- to medium-term. Major industrial power consumers, as well as suppliers to residential markets, are also concerned about the possibility that the power supply in Ontario could be severely compromised, and that replacement capacity will be costlier and less reliable when it eventually becomes operational. These concerns will have impacts on industrial investment decisions in all major economic sectors, both now and far into the future.

The economic consequences of closing the province's thermal power plants may be significant. Nevertheless, if the health consequences of operating those plants are even more severe, the province may decide it is the right tradeoff to make. But the decision should not be made before hearing all sides of the debate and undertaking a level of due diligence appropriate to such a costly and far-reaching investment decision. That exercise in due diligence should not treat any part of the statistical evidence concerning the environmental consequences of coal power as exempt from independent, 3rd-party review, including the claims about the health consequences of coal-based air pollution emissions. This report offers a preliminary critique the decision to close the power plants. The following questions are addressed.

1. How has Ontario air quality changed in recent decades?
2. How do power generation plants affect Ontario's air quality?
3. What is the scientific basis for connecting air pollution with disease and mortality in Ontario?
4. What are the main sources of uncertainty in these studies?

[1] See, for instance, Basrur (2000), Ontario Medical Association (2001), David Suzuki Foundation (1998).

1. How has Ontario air quality changed in recent years?

It is commonly asserted that air quality in Ontario is bad and getting worse.[2] However the data show that the most commonly-measured forms of air pollution are not getting worse, and in some cases have improved dramatically. Data gathered by Environment Canada's National Air Pollution Surveillance System (NAPS) show the following patterns for Ontario cities.[3]

- In every city, carbon monoxide (CO) and lead levels are extremely low compared to historical levels, and are well below the current safe standard.
- Likewise sulphur dioxide (SO_2), nitrogen dioxide (NO_2) and total suspended particulate (TSP) levels have fallen to the point where they are routinely below the Environment Canada recommended standards, with only occasional, temporary departures above the standard in some places.
- Only ozone regularly goes above the clean air standard, on an irregular, seasonal basis.

Ozone chemistry is highly complex, and many difficulties remain in constructing models to explain its formation and transport.[4] Ozone is not directly emitted by polluters but is a product of sunlight acting on natural and anthropogenic compounds (called ozone precursors) in the air. Since ambient ozone levels tend to be the same or worse in rural areas as it is in cities (see Figure 1), we should not expect that city air will ever be ozone-free, regardless of how much local emissions control is undertaken.

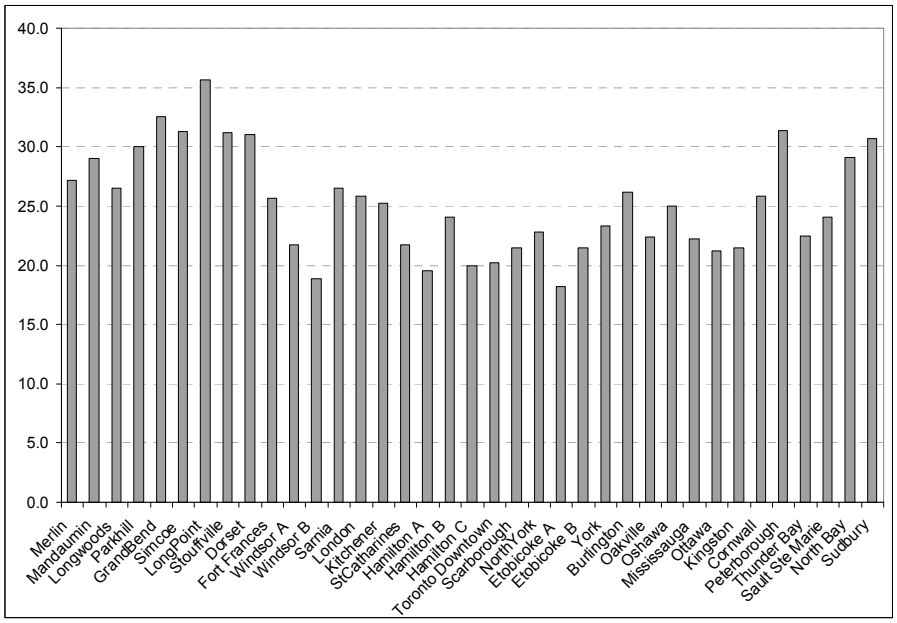

Figure 1: Average Ozone concentrations in Ontario locations, 1999 (parts per billion). All data from Ontario Ministry of the Environment (2003). Note rural monitoring stations are on the left end of the graph.

[2] See, for example, the Ontario Clean Air Alliance http://www.cleanair.web.ca/resource/submarine.pdf.
[3] The data are shown in Appendix B. See also Fraser Institute (2003).
[4] See, for example, Stein, Lamb and Draxler (2000).

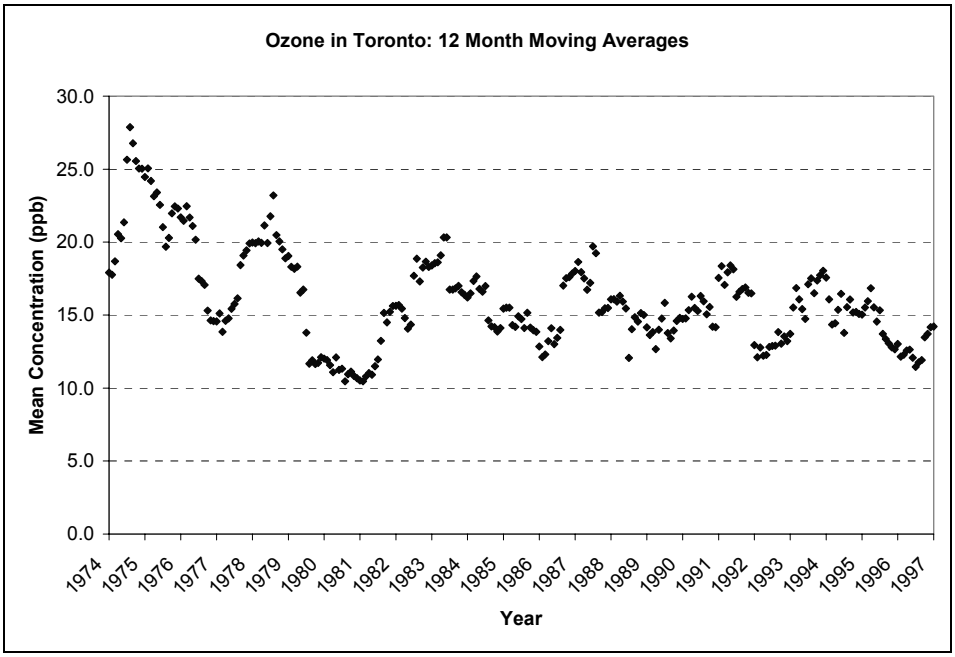

Figure 2: Ozone concentrations (12 month moving averages) in Toronto, 1974-1997. Source: Environment Canada NAPS data (see Appendix B).

Even still there has been progress on ozone. In the most populous city (Toronto) average ozone concentrations have been slowly declining since the 1970s, as seen in Figure 2, which shows 12-month moving averages back to the early 1970s. Notice the strongly cyclical nature of ozone levels, which may relate in part to meteorological conditions and the solar cycle. Updated data (to 2001) at the Ontario Environment Ministry website[5] confirm that the long-term downward trend has been maintained, though the 1995-2000 interval shows a small cyclical increase.

Overall, the claim that air pollution is getting worse is not supported by the available data. Looking at the actual pollution levels in urban air, the situation today is substantially improved compared to that in the 1960s and 1970s, even though the economy has grown considerably over the intervening years.

One potential complication to this story is the presence of sulphate aerosols in the atmosphere. These are ultrafine particles formed when SO_2 reacts with either a hydroxyl radical (OH) or hydrogen peroxide (H_2O_2).[6] Sulphates have been implicated in some epidemiological studies as potential threats to health (see Sections 3 and 4 below). The availability of the reactants depends on the abundance of Volatile Organic Compounds (VOCs) and Nitrogen Oxides (NO_x) in the air, which vary naturally by season and are also emitted by stationary and mobile pollution sources. While concentrations of the precursor compounds (SO_2 and NO_x,) have been measured for a long time, sulphate levels have not been directly measured except recently. Hence it is not possible to say if levels today are higher or lower than in earlier decades, nor can we establish trend lines. However the fact that the precursor compounds are limiting factors in sulphate formation and have themselves declined, and the fact that total suspended particulates

[5] http://www.ene.gov.on.ca/envision/techdocs/4521e_appendix.htm#10
[6] See Stein and Lamb (2003).

(TSP) have declined, provide indirect evidence that sulphates have likely declined as well. Also, the toxicological evidence for an effect of fine particulates is not strong (see Section 4.3).

One reason that people perceive worsening air quality is that in 1993 Ontario institute a system of smog alerts. In the years after 1995 there was an increase in the number of smog episodes as ground-level ozone went through a cyclical increase. While the long term trend towards improved air quality has not reversed, recent years have been associated with higher numbers of smog advisories than the mid-1990s, and of course adults don't remember smog alerts from their childhood since the system did not exist back then, though smog certainly did.

2. How do power generation plants affect Ontario's air quality?

Although people often speak about air pollution as "a" problem, it is actually many different problems, of varying origins and degrees of concern.

SO_2, CO and NO_x

According to Ontario government data, only about one-quarter[7] of current sulphur dioxide emissions are generated by utilities. Ambient concentration levels are very low across Ontario now, having fallen over 80 percent since the early 1970s. There is no monitoring site in the province—even at Sudbury—where SO_2 levels are considered a problem compared to federal and provincial air quality standards.

Carbon monoxide mostly comes from transportation sources (85 percent). As with SO_2, ambient levels are very low compared to the early 1970s and there is no monitoring location in the province that shows a CO concentration problem, even in cities.

NO_x is also mostly from mobile sources like cars and buses (63 percent). Utilities are responsible only for about 15 percent. At high concentrations NO_x can be a direct threat to health, but the main concern about NO_x at ambient levels is its role in ozone and sulphate formation.

Total Suspended Particulates

The category "particulates" includes not just soot from smokestacks, but particles formed of every substance known to man, including viruses, bacteria, moulds, pollen fragments from thousands of flowering plants, insect fragments, wind-eroded dust from exposed soil and sand, and chemical compounds from all sources.[8]

The US Environmental Protection Agency[9] provides detailed source breakdowns for American emissions (see Figure 3). While comparable data do not exist for Canada the shares are likely similar. The most recent data are from 1998, and show that on a national basis, natural sources dwarf all others combined. Natural sources are responsible for 5,307,000 short tons of emissions, followed by residential wood burning fireplaces a distant second at 411,000 short tons. Coal consumption for industry and electric power generation comes next at 362,000 short tons. Since the US economy is more coal-intensive than Canada's the fraction from thermal plants may be even lower here.

[7] See http://www.ene.gov.on.ca/envision/techdocs/4521e.htm#4 for data in this section.
[8] See Green et. al (2002).
[9] See http://www.epa.gov/ttn/chief/trends/index.html for these data.

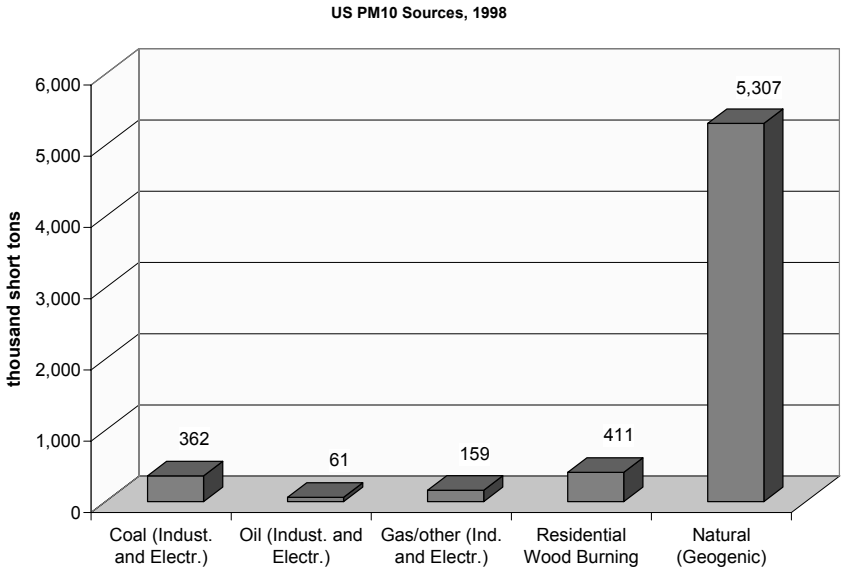

Figure 3: Major Particulate (PM10) Emission Sources in the US, 1998. Source: US Environmental Protection Agency http://www.epa.gov/ttn/chief/trends/index.html.

Ozone

Ground-level ozone has remained relatively steady on a long term basis since the 1970s, subject to multi-year cyclical fluctuations (see Figure 2), despite the growth of power production in Ontario over that time. Since ozone is formed by a photochemical reaction in the air involving both manmade and natural precursors, its level is harder to control and its origins are not straightforward. In Ontario, most of the precursor compounds come from the US Northeast.

The specific role of Ontario's power plants in Ontario air quality was studied recently in a report by RWDI Consultants.[10] The RWDI model simulates the US Northeast/Southern Ontario airshed, computing airflows and pollution loads using data on point and nonpoint-source emissions and meteorological inputs. The model was tuned to replicate a severe air quality episode in July 1999, then to simulate what the change in ozone and smog formation during that episode would have been had the coal plants not operated. The results showed that there would have been almost no difference: the reduction in ozone formation across the air shed would have been imperceptibly small. This indicates that most Ontario ozone is attributable to sources other than the thermal power plants, such as US NO_x emissions, transportation and natural (meteorological) conditions.

[10] RWDI (2004).

3. What is the scientific basis for connecting air pollution with disease and mortality in Ontario?

Efforts to discern a link between air pollution and health outcomes go back many years. In the past fifteen years there has been a large amount of empirical literature on the subject, using statistical analysis to see whether observed pollution levels correlate with health outcomes like premature death, lung disease, coronary disease, infant mortality, low birth weight, asthma, and so forth. Studies have differed based on whether they examined one location or multiple locations simultaneously, one air pollutant or many at once, the length of time studied, the covariates used to control for spurious effects, and the analytical techniques.

A common approach has been to start with data on mortality or morbidity (disease) and distinguish between the "expected" number of deaths and the "excess" number of deaths. The latter count would then be compared to pollution levels and other variables to see if there is a correlation. Estimating the "expected" number of deaths requires a combination of statistical know-how and subjective judgment. Researchers construct a trend model that tries to establish a baseline level of mortality through the time period of the data. The difference between the baseline mortality level and the observed level is then called the "excess" or premature death rate. Similar detrending methods can be used on any type of health data, such as disease diagnoses, hospital admissions, etc.

Among the findings in the most recent and most comprehensive studies are the following.

- There appears to be a small, positive and statistically significant correlation between exposure to particulate pollution and the risk of premature death in at-risk populations. Evidence from the United States suggests that every increase of 10 $\mu g/m^3$ in the concentration of particulates is correlated at the national level with in increase in mortality risk of about 0.5 percent,[11] and the effects appear even at ambient levels near zero.
- Elevated carbon monoxide, NO_x and ozone levels each correlate with a small, positive and statistically significant increase in cardiac illness and mortality.[12]

The mortality risk can be expressed in several different ways. If, say, there is a background risk of dying from cardiac disease of 0.00040 percent across the whole population, and elevated pollution levels cause this to rise to 0.00042 percent, the *absolute* risk has gone up by (0.00042 - 0.00040 =) 0.00002 percent, but the *relative* change in risk is (0.00042/0.00040 =) 1.05, which is described as a "relative risk increase" of 5 percent. So the same pollution can be described as causing an increased death risk of 5 percent, or 2 one-hundred thousandths of one percent. In practice, relative risks are used, so reported risk rates tend to sound large.

On the basis of epidemiological findings, the Ontario Medical Association[13] and the Toronto Board of Health[14] have both claimed that thousands of lives are lost each year in Ontario due to current air pollution levels. The OMA estimates that 1,900 Ontarians die annually from air pollution and 13,000 go to Emergency wards. The Toronto Board of Health estimates that about 1,000 deaths and 5,500 hospitalizations occur in Toronto alone due to air pollution.

[11] Dominici et. al (2002).
[12] Burnett et. al (1997), (1998).
[13] OMA (2001).
[14] Basrur (2000).

4. What are the main sources of uncertainty in these studies?

4.1 Correlation, Causality and Significance

It is commonplace to point out that correlation does not prove causality. In the case of air pollution and health risks, this must be emphasized. A simple example illustrates why. The prominent and widely-respected study by Dominici et. al (2002) estimated dose-response curves relating PM_{10} exposure and mortality risk in 88 US cities. The relative risk coefficients are graphed in Figure 2 of that paper, and show that in 20 of the 88 cities, the effect is negative: increased particulate pollution is associated with *reduced* mortality risk. Likewise the mortality effects when averaged across seven US regions are summarized with estimated dose-response curves (Figure 5 of that paper). In two of the largest regions (the US Southwest and Southeast) the graphs are negative below 60 $\mu g/m^3$, (indicating the risk of mortality declines in response to pollution exposure). In the Upper Midwest the effect is positive at 60 $\mu g/m^3$ but then declines to zero by 100 $\mu g/m^3$, suggesting high pollution levels are safer than low pollution levels.

Researchers set aside anomalous results like these by emphasizing the difference between correlation and causality. No one thinks pollution improves the health of a population, or that higher pollution levels are safer than lower levels, so such results are assumed to be spurious. In the survey used by the authors of the technical report for the Ontario Medical Association,[15] negative coefficients were simply deleted from the sample, again on the assumption that the epidemiological models only establish correlations, not causality, and the negative correlations were obviously spurious.

It is clearly inconsistent, therefore, for the OMA and the Toronto Public Health Board to turn around and use the *positive* correlation coefficients in their models as evidence of *causal* connections between pollution and health effects. This amounts to cherry-picking the science for results that support a preferred conclusion.

The conflation of correlation and causality is even more of a concern since the estimated effects are so small. For example, in the Toronto Public Health study, four papers were cited as providing evidence of the relative risk effect of carbon monoxide on mortality, with estimates of 1.0 percent, 1.3 percent, 4.3 percent and 4.8 percent.[16] These estimates are all very small: the relative risk ratios range from 1.01 to 1.05, where 1.00 implies no effects at all. It is worth noting that the first two estimates could not be deemed statistically significant without implying the last two numbers are impossibly high.[17] In other words they cannot all be equally likely, yet for the purpose of the Toronto study the simple average of all four coefficient was used.

The coefficients that link health responses to pollution emissions tend to follow this pattern of being small and inconsistent across locations. Referring again to Dominici et. al (2002), the PM_{10}-mortality relative

[15] DSS Management Consultants (2000), pages D-13, D-18, D-21.
[16] Basrur (2000), p. 10.
[17] Statistical significance in a model based on a symmetric Normal approximation refers to the ability to rule out the possibility that the coefficient actually has a value of zero, but by random chance a nonzero value was found in the data. If the confidence interval is small enough not to encompass zero when the coefficient is 1.0 or 1.3, then symmetrically it cannot encompass values above 2.0 or 2.6, respectively, unless the distribution is assumed to be highly asymmetric. But this would be inconsistent with the Normal approximation.

risk increase ranges from about -3 percent (per +10 μg/m^3 exposure) in Little Rock, Arkansas to about +3.2 percent in Topeka, Kansas. The national average of city results is about +0.5 percent, and this is the number most often quoted. But in any estimation exercise where a small effect is being measured in a noisy data set, the coefficient estimates need to be viewed with caution. Statistical significance is much more difficult to establish than is often realized by people with only limited training in statistics. In the case of pollution epidemiology there is good reason to doubt the statistical significance of existing coefficient estimates, as will be explained further in section 4.4 below.

4.2 Quality Control in the OMA and Toronto Public Health Studies

The Toronto Public Health (Basrur 2000) and the Ontario Medical Association (OMA 2001, DSS Consultants 2000) studies have attracted a great deal of attention and have no doubt influenced thinking by policy makers. It should be noted that the papers were not published in academic journals, but instead were merely posted on public web sites belonging to the agencies that commissioned or produced the papers. Considering the economic consequences of shutting down a quarter of the province's power generation capacity and building new plants to replace them, if the debate keeps coming back to these two studies as the principal rationale, then there is a positive obligation on policy makers to conduct rigorous due diligence by assessing the robustness of the results.

The Toronto Public Health study ("The Air Pollution Burden of Illness in Toronto" or APBIT) surveys a list of pollution epidemiology papers, and provides the reader with a good amount of detail about the coefficient estimates used for their model. The model is based on selectively reinterpreting positive correlation coefficients as causal functions, yielding the three equations presented in Appendix A. When applied to pollution data from the mid-1990s these equations generate predictions of the percentage of death and disease in Toronto can be attributed to air contaminants. When the percentages are multiplied by observed levels of death and disease the result is the large-sounding numbers of air pollution casualties.

How plausible are the three equations? Recent air pollution levels are low compared to the 1960s. A simple way to test the credibility of the APBIT model is to plug in observed air pollution levels back to the 1960s and see if the results remain reasonable. If the model accurately describes causal relationships rooted in actual physiological and medical responses, it ought to work not only for present data but for the recent past as well. But the results for previous decades are not merely implausible, they are numerically impossible. This calls into question the accuracy of the model and the meaningfulness of the data it generates.

Figures 4—6 show the results of applying the APBIT model to air pollution rates from 1965 to 1997 in downtown Toronto, as measured at the Bay/Wellesley monitoring station. NO_x and O_3 levels prior to 1973 are not available. The NO_x series was filled back to 1965 with the post-1973 sample mean, while the ozone series was filled with 30.0, which due to the threshold is effectively zero in the model. This likely understates the true value since there has been a downward trend over time (see Figure 2).

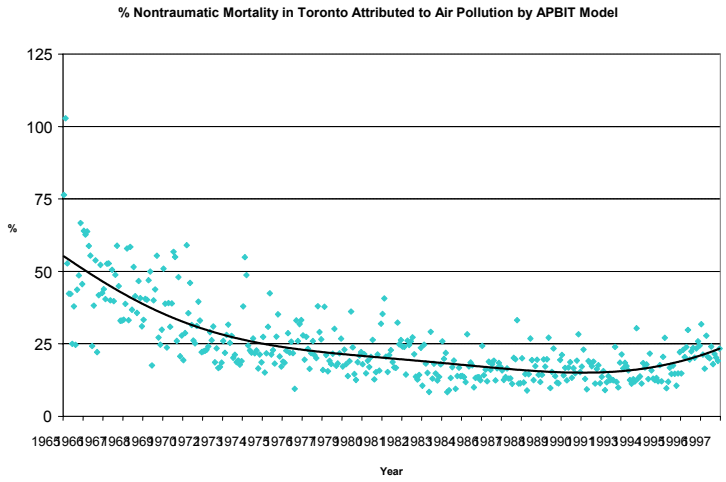

Figure 3: APBIT Model attribution of mortality in Toronto, extended back to 1965.

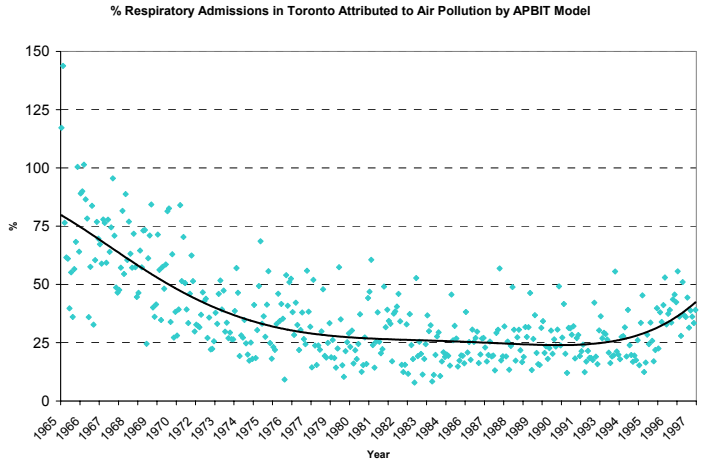

Figure 4: APBIT Model attribution of respiratory disease in Toronto, extended back to 1965.

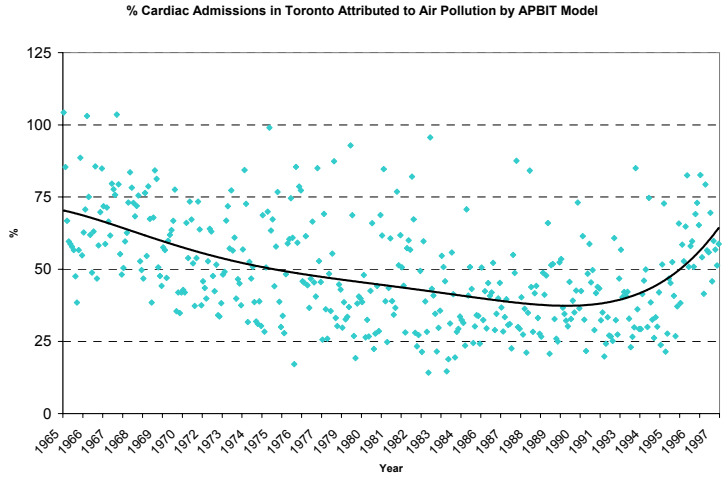

Figure 5: APBIT Model attribution of cardiac disease in Toronto, extended back to 1965.

The diamonds mark the monthly point estimates and the dark lines are 6th-order polynomial curves to show a smoothed mean. From Figure 4 we can see that while the implied death proportions are less than 10 percent in the mid-1990s, going back to the mid-1960s the model frequently attributes over 50 percent of deaths, and in one case over 100 percent of deaths, to air pollution. Likewise respiratory and cardiac admission attribution rates go over 100 percent at some points in the 1960s. These numbers are, needless to say, impossible. Cardiac admissions are especially attributed to air pollution: for much of the sample (including the most recent years) the model says that over 50 percent of hospital admissions for cardiac problems are due to air pollution.

With percentage attributions this high, the "body count" becomes high simply because these percentages are then multiplied by actual observed totals. For instance the model claims that in the year 1965, just over 50 percent of all non-traumatic deaths were caused by air pollution. Not old age, cancer, heart disease, stroke, pneumonia, or any of the other things people thought at the time. It even says that in February 1965, more people died of air pollution than there were deaths. The model clearly lacks basic credibility.

The OMA study was prepared by DSS Consulting with the assistance of a team of outside experts. It makes reference to a larger set of results from the environmental epidemiology literature than did the APBIT study, but the focus was only on ozone and particulates. As with the APBIT the core of the model is the set of correlation coefficients (therein called "E/RF" coefficients – Exposure/Response Functions), also inappropriately used to signify causality, after arbitrarily deleting[18] the negative ones. The OMA model goes on to assign economic values to estimates of mortality, morbidity, lost productivity and direct health care costs.

The illness and mortality risk data used to generate the E/RF functions are provided in Appendix D of the DSS paper. The E/RFs themselves are drive the model results. Unfortunately the particulate E/RFs are not reported, while for ozone, the tables presenting the E/RFs are full of numerical errors. Rather than trying to explain them, the tables (from the report's Appendix D, pages D-27-29) are simply reproduced below with the problem rows identified by arrows.

[18] DSS Consulting (2000) pages D-13, D-18, D-21.

Table D.5 - Exposure/Response Functions for Ozone

	ILLNESS CATEGORY											
	PREMATURE MORTALITY						HOSPITAL ADMISSIONS					
	Respiratory			Cardio-vascular			Asthma			COPD		
	Age Group 1	Age Group 2	Age Group 3	Age Group 1	Age Group 2	Age Group 3	Age Group 1	Age Group 2	Age Group 3	Age Group 1	Age Group 2	Age Group 3
ICD-9 Codes												
AGE-DEPENDENT RISK GROUP												
Start Age	0	18	65	0	18	65	0	18	65	0	18	65
Stop Age	17	65	99	17	65	99	17	65	99	17	65	99
O_3												
LINEAR												
Central Illness Rate [1]	-	-	-	-	-	-	#VALUE!	#DIV/0!	#DIV/0!	-	#DIV/0!	#DIV/0!
Probability Weight	-	-	-	-	-	-	50%	50%	50%	-	50%	50%
Central Effect Threshold	-	-	-	-	-	-	-	-	-	-	-	-
Probability Weight	-	-	-	-	-	-	-	-	-	-	-	-
Lower Illness Rate	-	-	-	-	-	-	#VALUE!	#DIV/0!	#DIV/0!	-	#DIV/0!	#DIV/0!
Probability Weight	-	-	-	-	-	-	25%	25%	25%	-	25%	25%
Lower Threshold Value	-	-	-	-	-	-	-	-	-	-	-	-
Probability Weight	-	-	-	-	-	-	-	-	-	-	-	-
Upper Illness Rate	-	-	-	-	-	-	#VALUE!	#DIV/0!	#DIV/0!	-	#DIV/0!	#DIV/0!
Probability Weight	-	-	-	-	-	-	25%	25%	25%	-	25%	25%
Upper Threshold Value	-	-	-	-	-	-	-	-	-	-	-	-
Probability Weight	-	-	-	-	-	-	-	-	-	-	-	-

[1] All risks are expressed as the risk per 1,000,000 people exposed.

Table D.5 - Exposure/Response Functions for Ozone (*continued*)

	ILLNESS CATEGORY											
	HOSPITAL ADMISSIONS											
	Pneumonia			Coronary Artery Disease			Dysrhythmias			Congestive Heart Failure		
	Age Group 1	Age Group 2	Age Group 3	Age Group 1	Age Group 2	Age Group 3	Age Group 1	Age Group 2	Age Group 3	Age Group 1	Age Group 2	Age Group 3
ICD-9 Codes												
AGE-DEPENDENT RISK GROUP												
Start Age	0	18	65	0	18	65	0	18	65	0	18	65
Stop Age	17	65	99	17	65	99	17	65	99	17	65	99
O_3												
LINEAR												
Central Illness Rate (a)	#VALUE!	#DIV/0!	#DIV/0!	-	-	-	-	#DIV/0!	#DIV/0!	-	-	-
Probability Weight	50%	50%	50%	-	-	-	-	0.55	0.51	-	-	-
Central Effect Threshold (b)	-	-	-	-	-	-	-	-	-	-	-	-
Probability Weight	-	-	-	-	-	-	-	-	-	-	-	-
Lower Illness Rate (a)	#VALUE!	#DIV/0!	#DIV/0!	-	-	-	-	#DIV/0!	#DIV/0!	-	-	-
Probability Weight	25%	25%	25%	-	-	-	-	0.25	0.25	-	-	-
Lower Threshold Value (b)	-	-	-	-	-	-	-	-	-	-	-	-
Probability Weight	-	-	-	-	-	-	-	-	-	-	-	-
Upper Illness Rate (a)	#VALUE!	#DIV/0!	#DIV/0!	-	-	-	-	#DIV/0!	#DIV/0!	-	-	-
Probability Weight	25%	25%	25%	-	-	-	-	0.2	0.24	-	-	-
Upper Threshold Value (b)	-	-	-	-	-	-	-	-	-	-	-	-
Probability Weight	-	-	-	-	-	-	-	-	-	-	-	-

Table D.5 - Exposure/Response Functions for Ozone (*continued*)

	ILLNESS CATEGORY														
	EMERGENCY ROOM VISITS						DOCTOR'S OFFICE VISITS								
	Respiratory			Cardio-vascular			Child Bronchitis			Chronic Respiratory Disease			Chronic Bronchitis		
	Age Group 1	Age Group 2	Age Group 3	Age Group 1	Age Group 2	Age Group 3	Age Group 1	Age Group 2	Age Group 3	Age Group 1	Age Group 2	Age Group 3	Age Group 1	Age Group 2	Age Group 3
ICD-9 Codes															
AGE-DEPENDENT RISK GROUP															
Start Age	0	18	65	0	18	65	0	18	65	0	18	65	0	18	65
Stop Age	17	65	99	17	65	99	17	65	99	17	65	99	17	65	99
O_3															
LINEAR															
Central Illness Rate (a)	-	-	#DIV/0!	-	-	#DIV/0!	-	-	-	-	-	-	-	-	-
Probability Weight	50%	50%	50%	-	50%	50%	-	-	-	-	-	-	-	-	-
Central Effect Threshold (b)	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Probability Weight	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Lower Illness Rate (a)	-	-	#DIV/0!	-	-	#DIV/0!	-	-	-	-	-	-	-	-	-
Probability Weight	25%	25%	25%	-	25%	25%	-	-	-	-	-	-	-	-	-
Lower Threshold Value (b)	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Probability Weight	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Upper Illness Rate (a)	#DIV/0!	#DIV/0!	#DIV/0!	-	#DIV/0!	#DIV/0!	-	-	-	-	-	-	-	-	-
Probability Weight	25%	25%	25%	-	25%	25%	-	-	-	-	-	-	-	-	-
Upper Threshold Value (b)	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Probability Weight	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-

These Tables are the numerical core of the model used in the OMA study. While the numerous errors likely did not affect the actual computations in the Report, it is noteworthy that this document has been on the OMA web site for 5 years,[19] and has been repeatedly cited by OMA officials as the basis for its conclusions. It is obvious no one proofread the paper: not the consultants, not the OMA executive, not the provincial government, not anyone who has used this study.

These points should suffice to show that neither the OMA nor the Toronto Public Health models can be taken at face value as an adequate basis for costly and far-reaching public investment decisions worth tens of billions of tax dollars. Instead both papers should be set aside pending a proper due diligence audit of the quality of the underlying work and the validity of their conclusions.

4.3 Epidemiology versus Toxicology

As mentioned above, evidence from US cities (all from the same study) finds mortality risk changes due to elevated fine particle levels ranging from -3 percent to +3 percent depending on the location. But why would the effect differ across locations? If an actual human physiological response is being accurately measured, it should be nearly the same everywhere. This raises the question of whether the epidemiological models adequately control for confounding effects. If they do not, they are at risk of assigning the blame for mortality and morbidity to factors (in this case airborne particulates) that might actually be medically irrelevant.

Hence it is important to check the results of epidemiological studies against those of experimental toxicology. In experimental studies, people and animals are exposed to controlled levels of air contaminants, while scientists monitor cardiac and lung functions for responses. There has been considerable work on this, recently summarized by Laura Green, Ph.D., a consulting toxicologist, and her colleagues in a pair of papers (Green et. al 2002, 2003) in the journal *Regulatory Toxicology and Pharmacology*.

[19] See http://oma.org/phealth/smogmain.htm#program.

Laboratory studies have shown that high ozone levels cause respiratory irritation in asthmatics, but have not yielded evidence of toxicity or respiratory problems (even for asthmatics) from exposure to particulate levels at or even much higher than observed ambient levels, although asthmatics show some response to highly acidic aerosols. Studies on hamsters, rats and dogs involving exposure to particulates up to 1,000 ug/m^3 have not produced evidence of interference with cardiac function. Repeated tests of the effects of particulates in commonly observed ambient concentrations have shown they do not harm health. Green and Armstrong (2003) conclude:

> It remains the case that no form of ambient PM—other than viruses, bacteria, and biochemical antigens—has been shown, experimentally or clinically, to cause disease or death at concentrations remotely close to U.S. ambient levels. This lack of demonstration is not for lack of trying: hundreds of researchers, in the U.S. and elsewhere, have for years been experimenting with various forms of pollution-derived PM, and none has found clear evidence of significant disease or death at relevant airborne concentrations.
>
> (p. 333)

This stands in contrast to the conclusions from some epidemiological studies, and calls into question their assertion that contemporary ambient pollution levels are lethal. The Health Canada Science Assessment[20] that provided background material for the development of new air quality guidelines also pointed out the mismatch between epidemiological and toxicological findings:

> Controlled human exposures to acidic and inert particles have not caused significant alterations in pulmonary function in healthy individuals at relatively high levels compared to those generally experienced in the environment. However, acidity has been shown to affect the slowing of mucociliary clearance at concentrations as low as 100 mg/m^3…There has been no convincing evidence suggesting that subjects with chronic obstructive pulmonary disease (COPD) or the elderly are susceptible populations in terms of pulmonary function responses, although some data show that the pulmonary deposition of ultrafine particles (mass median aerodynamic diameter [MMAD] 0.02 - 0.24 mm) in COPD patients was higher than in healthy subjects…
>
> **Overall, the clinical data does not lend much support to the observations seen in the epidemiology studies, particularly to the observations that high ambient particulate concentrations are associated with mortality** within hours or a few days at most. It does indicate one susceptible subpopulation, asthmatics, who currently comprise 5 to 8 percent of the population, a percentage that has been rising in the past decade in Canada as well as in other western countries.
>
> (http://www.hc-sc.gc.ca/hecs-sesc/air_quality/publications/particulate_matter_science_assessment/addendum/impacts.htm#7, emphasis added.)

Despite this, the Health Canada Science Assessment went with the epidemiological results and concluded that even low levels of exposure to particulates and ozone are hazardous to humans. The scientific advisory group to the Government of Alberta disputed this conclusion, arguing the following:

[20] See Health Canada (1997).

- No experimental dose-response curves have been presented for humans, animals or vegetation; statistical associations are being used as equivalent to dose-response curves and this is an incorrect approach.

- Relatively high ambient background levels of ozone are well documented, but have been ignored in interpreting the statistical studies.

- The results of controlled exposure experiments that clearly demonstrate effects only at much higher levels [than in ambient air] have been ignored.

Alberta Environment (1998).

4.4 New Evidence Concerning Model Uncertainty

It was noted above that epidemiological coefficient estimates often differ by place and by study. Results can also differ widely depending on the particular statistical model used. For instance, in a recent study of pollution and infant mortality in California (Currie and Neidell 2004), statistically significant effects between pollution and short gestation periods are observed, but when fixed effects due to community socioeconomic characteristics are controlled, the pollution effects disappear. The same thing happens when examining low birth weight and fetal death: the pollution effects disappear when other controls are added to the model.[21]

There are many covariates that should potentially be included in a statistical model, such as weather data (temperature, air pressure, wind), smoking rates, income levels, trends, lags, cross products and so forth. While it is possible to simply put everything in a model simultaneously, this is not necessarily the best approach, as statistical estimates get less precise the greater the number of coefficients that have to be estimated at once. But testing out all possible combinations of variables can be difficult, as the combinations may number in the trillions. Typically a study only reports one or several forms of the model. The results are referred to as "point estimates." Since a model may have trillions of possible specifications there could be trillions of potential point estimators, but the researcher cannot report them all.

Highly automated computational processes are now available which can evaluate the trillions of possible point estimates and assign to each one a probability based on the goodness-of-fit between the model and the data. Then a so-called *posterior* estimate can be reported in which the model-specific point estimates are averaged, each weighted by the probability it came from the correct model. The technique is called *Bayesian Model Averaging* or BMA.

Koop and Tole (2004) recently published one of the first BMA-based studies of air pollution and mortality in Toronto. They evaluated over 567 trillion model specifications and found that while some specifications can yield a positive relationship between air quality and premature death rates, the model specification that receives the most support from the data only includes weather variables. In other words while the data can potentially be mined to "prove" pollution causes mortality, more thorough analysis shows that pollution is unlikely to be correlated with mortality. When they looked for possible interaction effects (i.e. maybe pollution only matters in hot weather) they found nothing but a Table "composed of zeroes (to three decimal places)."

[21] The Currie and Neidell study did find a small but significant relative risk (0.014%) between increased carbon monoxide and infant mortality.

4.5 Air Pollution and Asthma: The Conclusion of the UK Government

There is credible evidence to support the view that ordinary, ambient "fresh" air is not a health hazard, even with current pollution sources operating. A number of commentators have suggested air pollution levels may be causing increases in the rates of asthma. Recently, the UK government's *Committee on the Medical Effects of Air Pollution* studied the question and concluded that this claim is unfounded:

> "For the most part, [healthy] people will not notice or suffer from any serious or lasting ill effects from levels of pollution that are commonly experienced in the UK, even when levels are described as 'high' or 'very high' according to the current criteria…Perhaps surprisingly, long term exposure to air pollution is unlikely to be a cause of the increased number of people now suffering from asthma in the UK."
>
> (http://www.advisorybodies.doh.gov.uk/comeap/statementsreports/healtheffects.htm)

Taken together these findings suggest that the disease and death rates attributable to levels of ambient air pollution in Ontario are smaller and less certain than has recently been asserted.

Conclusion: The need for due diligence and thorough cost-benefit analysis

Coal provides a low-cost, reliable component of the electricity supply for Ontario. At a time of constrained supply and rising prices, there is considerable social benefit to having this power generation capacity on-line. Removal of these generating plants will require large-scale replacement from higher-cost sources, including natural gas, nuclear and renewables. But high electricity prices, and increased risk of supply interruption, are themselves a threat to public health and welfare. Household electricity demand is "inelastic"[22] to both price and income, meaning that it exhibits relatively little response to price and wage increases. This is because basic electricity consumption is a necessity, and cannot easily be adjusted in response to price increases, which therefore translate into increased real costs of living. Income inelasticity implies energy costs are a relatively larger fraction of low-income household budgets than for high-income households. Hence increased electricity costs are regressive, hitting low-income groups proportionately harder. Higher electricity costs also create a barrier to new industrial investment. Blackouts, needless to say, are extremely costly and risky to public safety.

In addition, as was explained above, the environmental impact of coal power is much smaller than popularly supposed, and the evidence for a connection to human health is weak and contradictory.

These considerations lead to the conclusion that thermal power plants are a net benefit to society, and shutting them down at this time is not in the public interest.

It must be emphasized that the economically-risky path of closing down a quarter of the province's generating capacity, or even talking as if it is going to be done shortly, is irresponsible, without first having undertaken extensive due diligence on all aspects of the decision, something which has not been done. In light of the substantial economic costs of abandoning the coal component of Ontario's power supply, and the evidence that Ontario's coal-plant emissions are unlikely to be the threat to life and health that has been claimed, the Ontario government should put on hold the planned phase-out of the thermal power plants by 2007, pending an exhaustive review of the real costs and benefits.

[22] See Garcia-Cerutti (2000), Silk and Joutz (1997) for reviews and recent elasticity estimates.

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APPENDIX A: APBIT Model Equations

The coefficients listed in Basrur (2000) are used in a simple 3-equation linear model to predict health consequences of ambient air pollution as follows.

(1) $D_p = D*[0.08*(PM_{10} > 5.0) + 3.48*CO + 0.119*NOx + 0.225*SO_2 + 0.04*(Ozone > 30)]/100$

(2) $R_p = R*[0.17*(PM_{10} > 5.0) + 0.249*CO + 0.276*NOx + 0.11*(Ozone > 30)]/100$

(3) $C_p = C*[0.23*(PM_{10} > 5.0) + 1.69*CO + 0.44*NOx + 0.45*(Ozone > 30)]/100$

(1): D denotes the total non-traumatic mortality in Toronto each year, and D_p denotes the total deaths attributable to pollution. The items in square brackets are the coefficients drawn from epidemiological studies multiplied by the observed ambient pollution levels. The model assumes there are threshold effects for PM_{10} and ozone. PM_{10} levels below 5 ppb are ignored, as are ozone readings below 30 ppb. The expression in square brackets yields a percentage.

(2): R is the total observed hospital admissions for respiratory ailments and R_p is the number attributable to pollution.

(3): C denotes the observed total hospital admissions for cardiac problems and C_p denotes the number attributable to pollution.

These three equations were used to determine the "Air Pollution Burden of Illness" for Toronto, as the sum of $D_p+R_p+C_p$. They imply, for example, that in the City of Toronto (i.e. the core, pre-amalgamation), out of 6,456 non-traumatic deaths in 1995, 542 were attributable to pollution. For Metro Toronto as a whole, 1,356 out of 16,615 deaths were due to pollution. Thus, about 8 percent of deaths in 1995 were attributed to air pollution. The model attributes 11 percent of respiratory admissions and 26 percent of cardiac admissions to air pollution levels.

Equations (1-3) can be rearranged slightly to yield predictions of the proportion of deaths rather than absolute levels. For (1), simply divide both sides by D and multiply by 100, yielding:

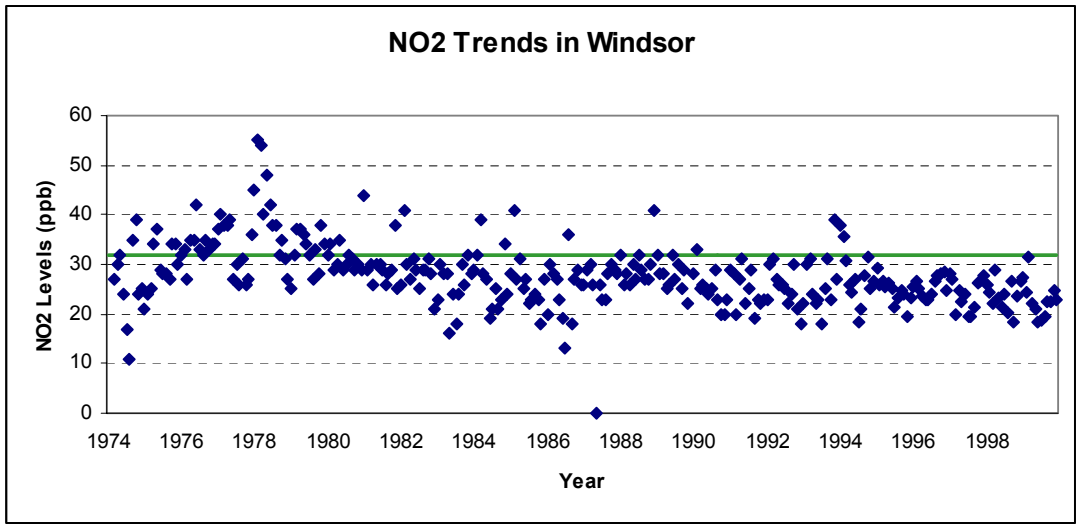
$$100\frac{D_p}{D} = [\cdots].$$

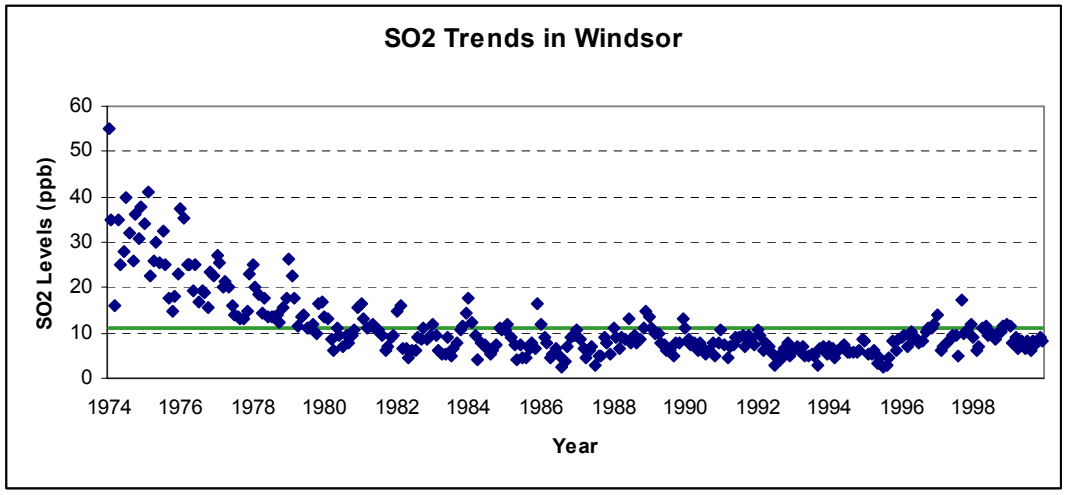
The expression on the right side is the formula in square brackets from (1), and the expression on the left side is the percentage of deaths due to pollution. The same operation can be applied to (2) and (3), yielding percentages of respiratory and cardiac admissions attributable to air pollution levels.

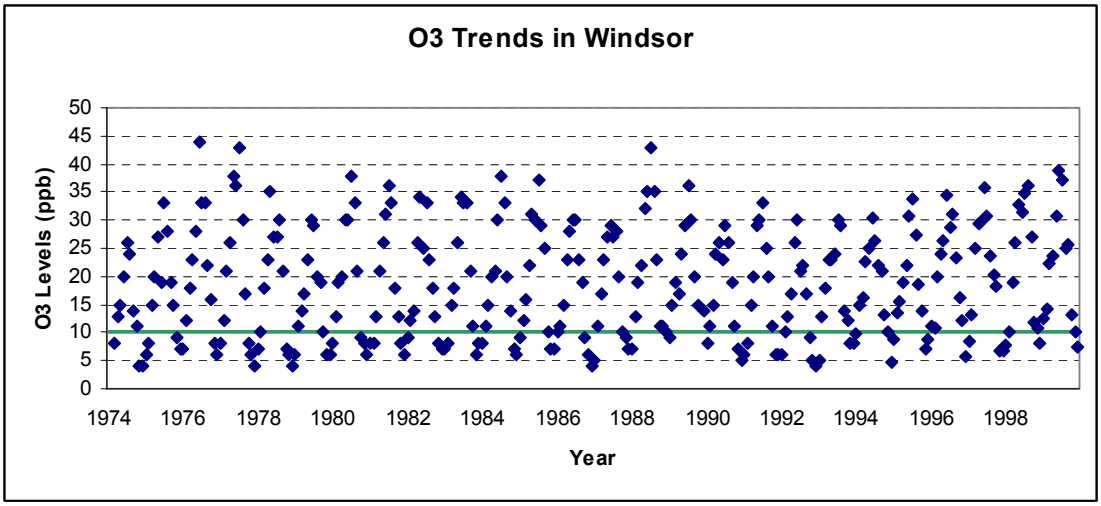
APPENDIX B: Air Contaminant Concentrations in Ontario

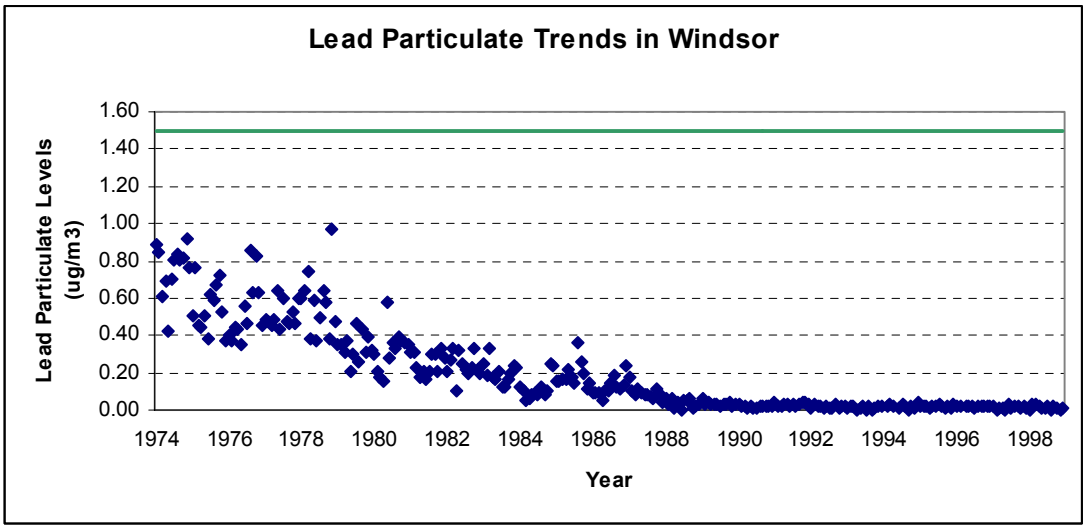
(Green line – Environment Canada Standard)

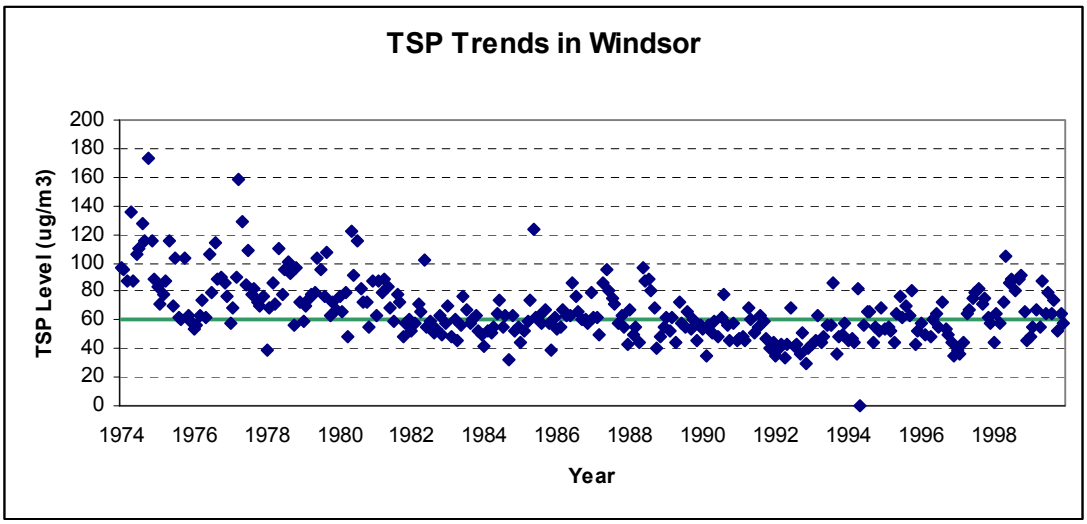
Windsor



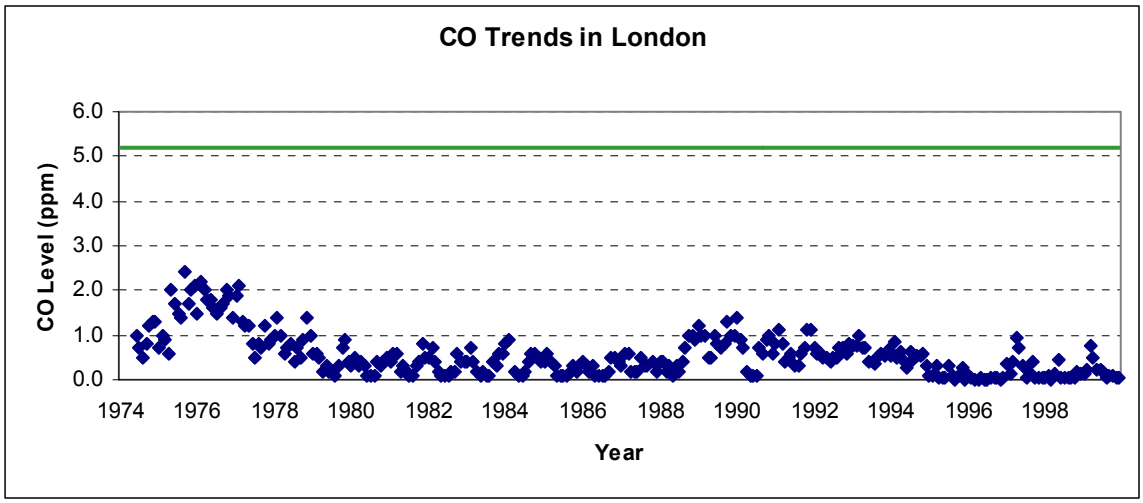


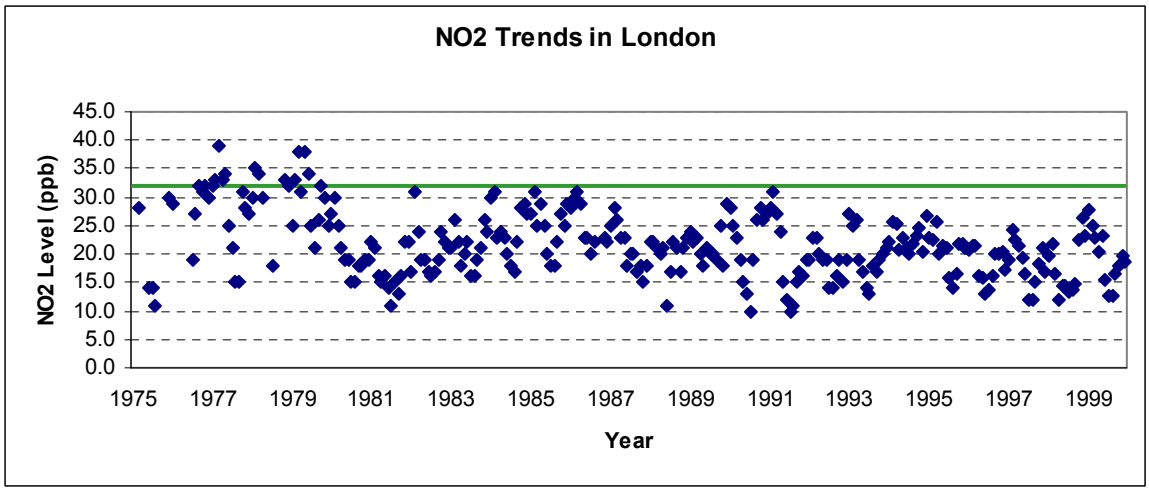

O3 Trends in Windsor
O3 Levels (ppb)
50
45
40
35
30
25
20
15
10
5
0
1974
1976
1978
1980
1982
1984
1986
1988
1990
1992
1994
1996
1998
Year

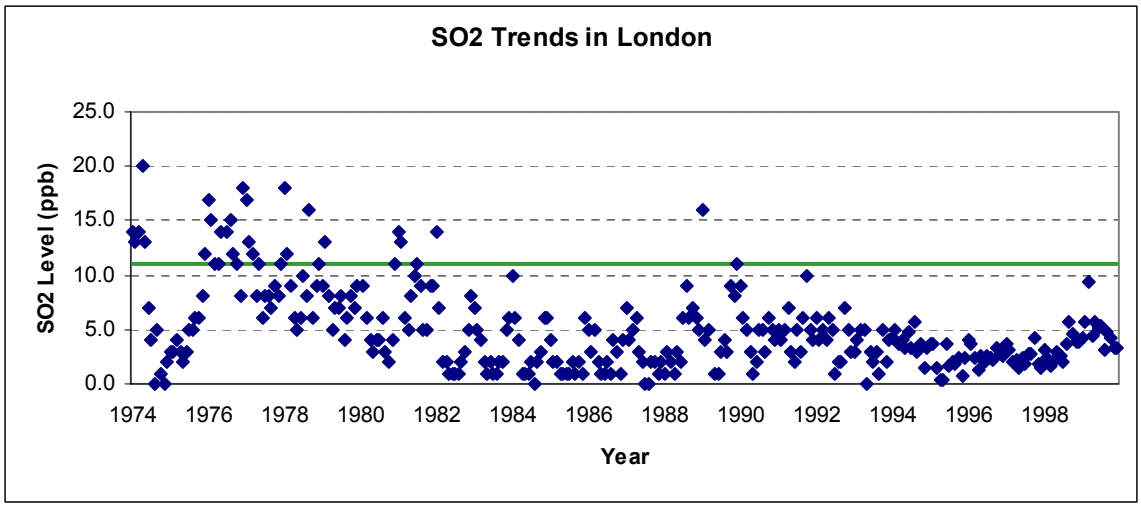

Lead Particulate Trends in Windsor
Lead Particulate Levels (ug/m3)
1.60
1.40
1.20
1.00
0.80
0.60
0.40
0.20
0.00
1974
1976
1978
1980
1982
1984
1986
1988
1990
1992
1994
1996
1998
Year

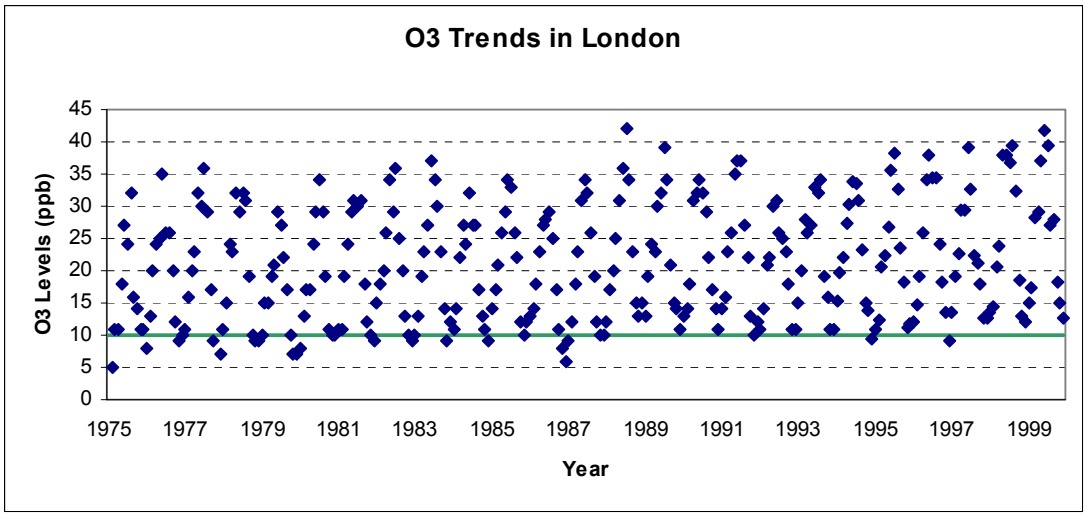

TSP Trends in Windsor
TSP Level (ug/m3)
200
180
160
140
120
100
80
60
40
20
0
1974
1976
1978
1980
1982
1984
1986
1988
1990
1992
1994
1996
1998
Year

London


CO Trends in London
CO Level (ppm)
6.0
5.0
4.0
3.0
2.0
1.0
0.0
1974 1976 1978 1980 1982 1984 1986 1988 1990 1992 1994 1996 1998
Year


NO2 Trends in London
NO2 Level (ppb)
45.0
40.0
35.0
30.0
25.0
20.0
15.0
10.0
5.0
0.0
1975 1977 1979 1981 1983 1985 1987 1989 1991 1993 1995 1997 1999
Year


SO2 Trends in London
SO2 Level (ppb)
25.0
20.0
15.0
10.0
5.0
0.0
1974 1976 1978 1980 1982 1984 1986 1988 1990 1992 1994 1996 1998
Year


O3 Trends in London
O3 Levels (ppb)
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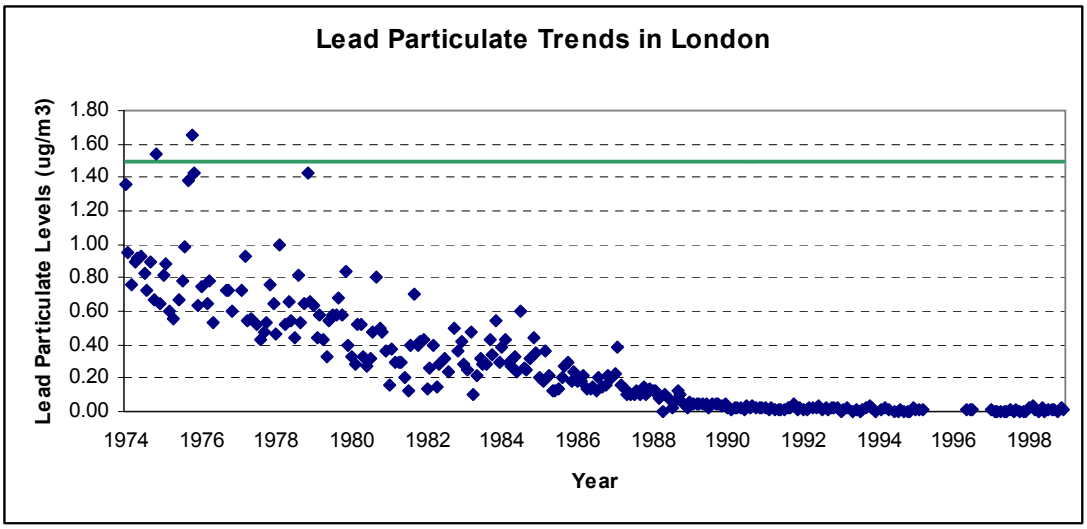
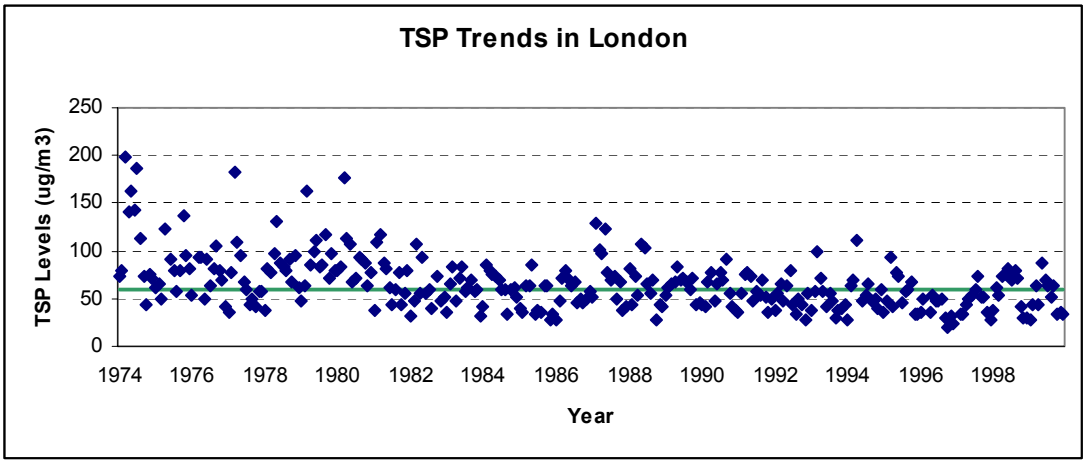
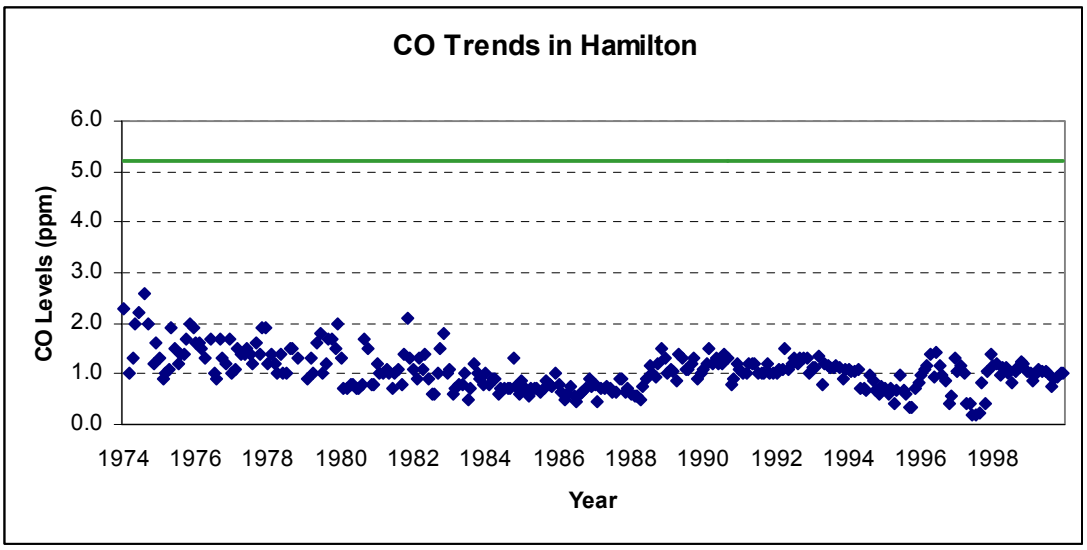
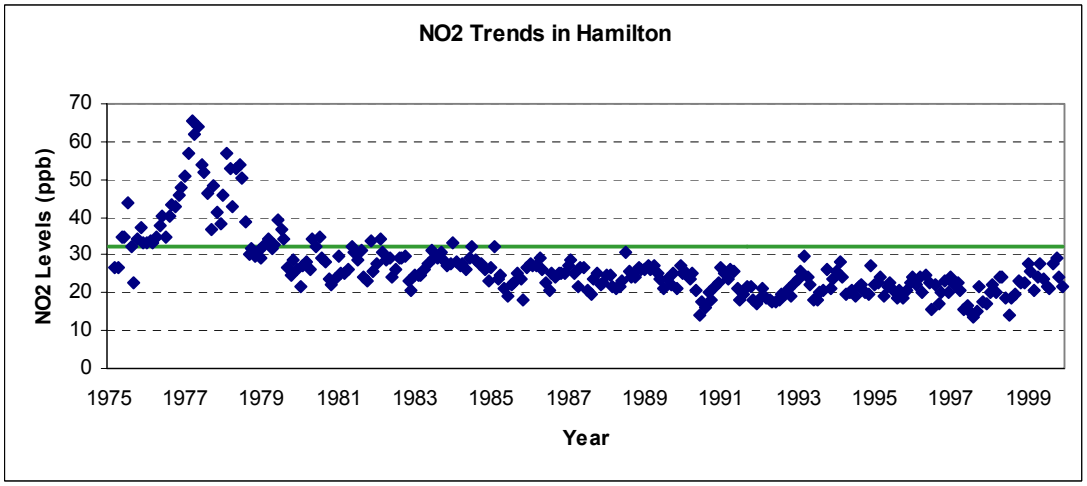
40
35
30
25
20
15
10
5
0
1975 1977 1979 1981 1983 1985 1987 1989 1991 1993 1995 1997 1999
Year

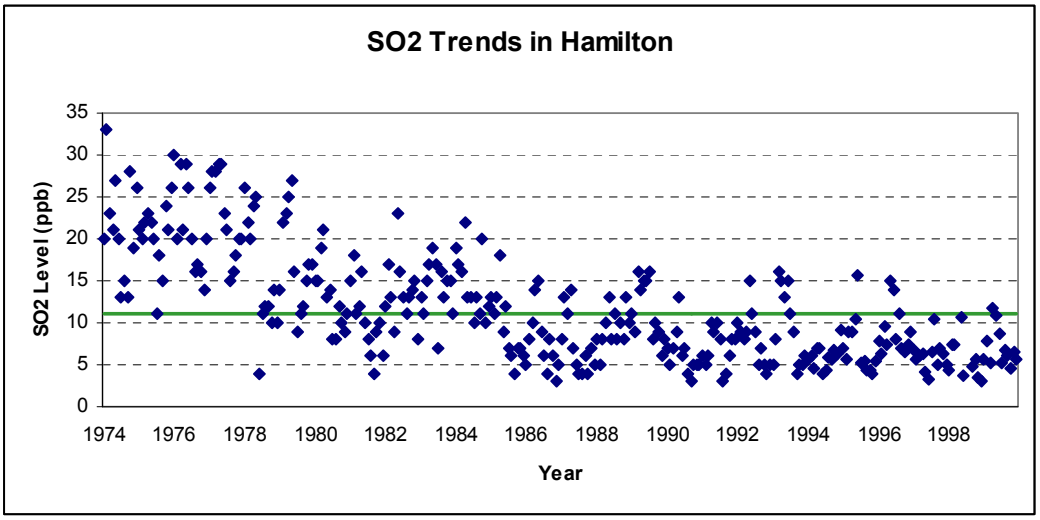

Lead Particulate Trends in London
Lead Particulate Levels (ug/m3)
1.80
1.60
1.40
1.20
1.00
0.80
0.60
0.40
0.20
0.00
1974 1976 1978 1980 1982 1984 1986 1988 1990 1992 1994 1996 1998
Year

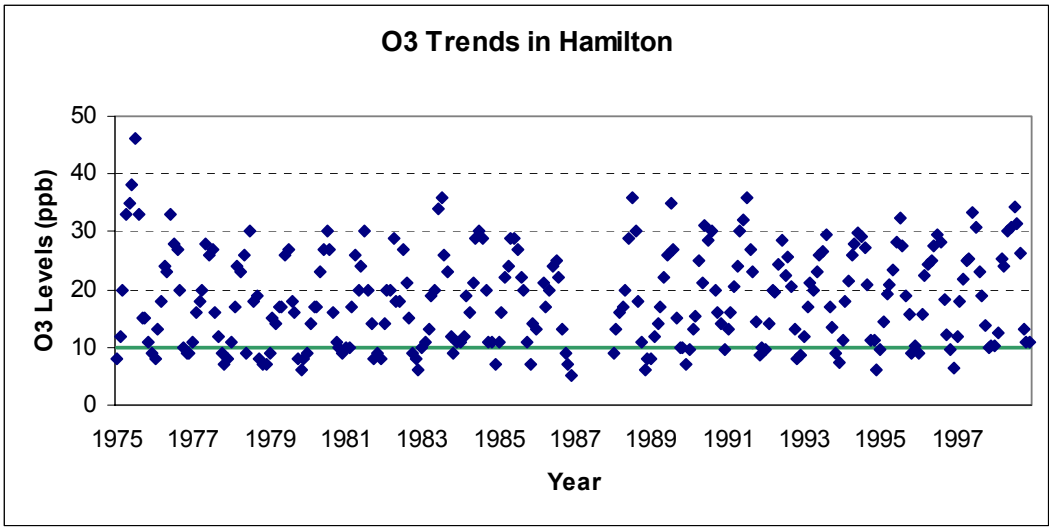

TSP Trends in London
TSP Levels (ug/m3)
250
200
150
100
50
0
1974 1976 1978 1980 1982 1984 1986 1988 1990 1992 1994 1996 1998
Year

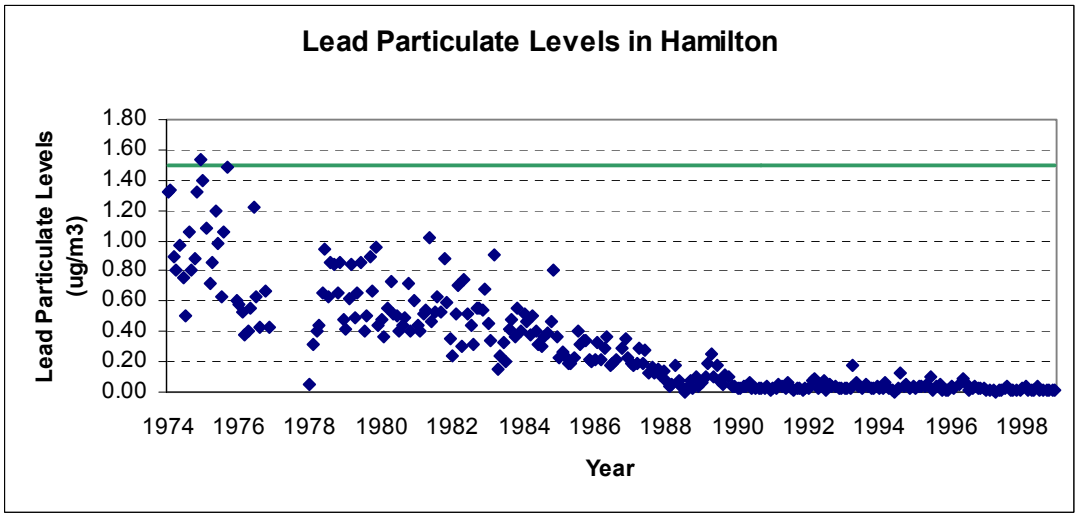
Hamilton

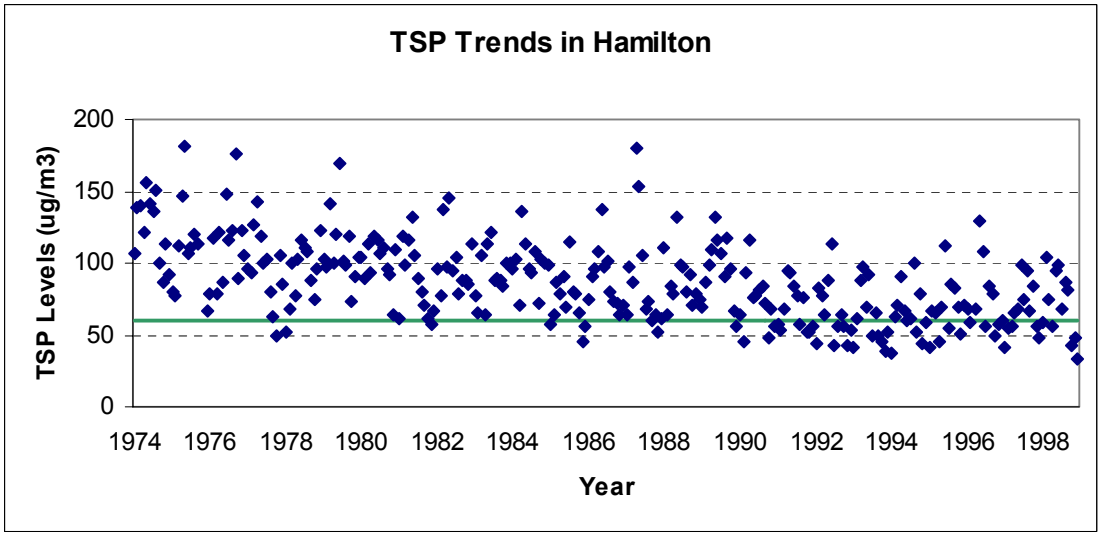

CO Trends in Hamilton
CO Levels (ppm)
6.0
5.0
4.0
3.0
2.0
1.0
0.0
1974 1976 1978 1980 1982 1984 1986 1988 1990 1992 1994 1996 1998
Year


NO2 Trends in Hamilton
NO2 Levels (ppb)
70
60
50
40
30
20
10
0
1975 1977 1979 1981 1983 1985 1987 1989 1991 1993 1995 1997 1999
Year

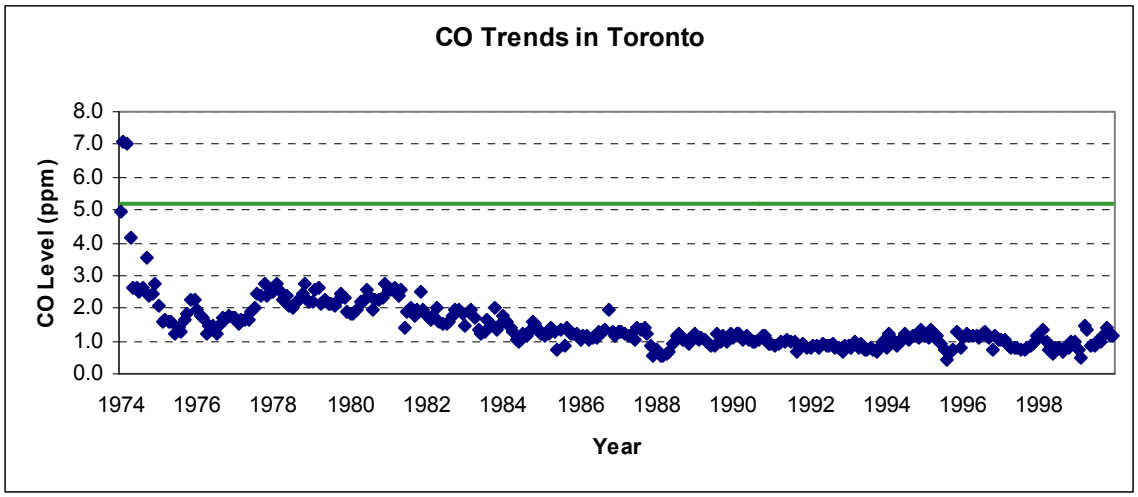

SO2 Trends in Hamilton
SO2 Level (ppb)
35
30
25
20
15
10
5
0
1974 1976 1978 1980 1982 1984 1986 1988 1990 1992 1994 1996 1998
Year

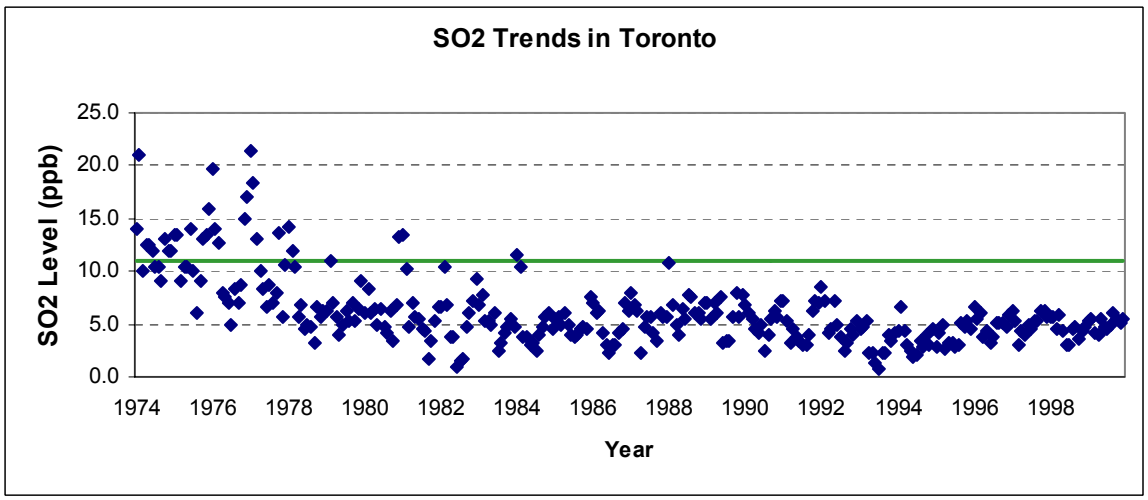

O3 Trends in Hamilton
O3 Levels (ppb)
0
10
20
30
40
50
1975
1977
1979
1981
1983
1985
1987
1989
1991
1993
1995
1997
Year

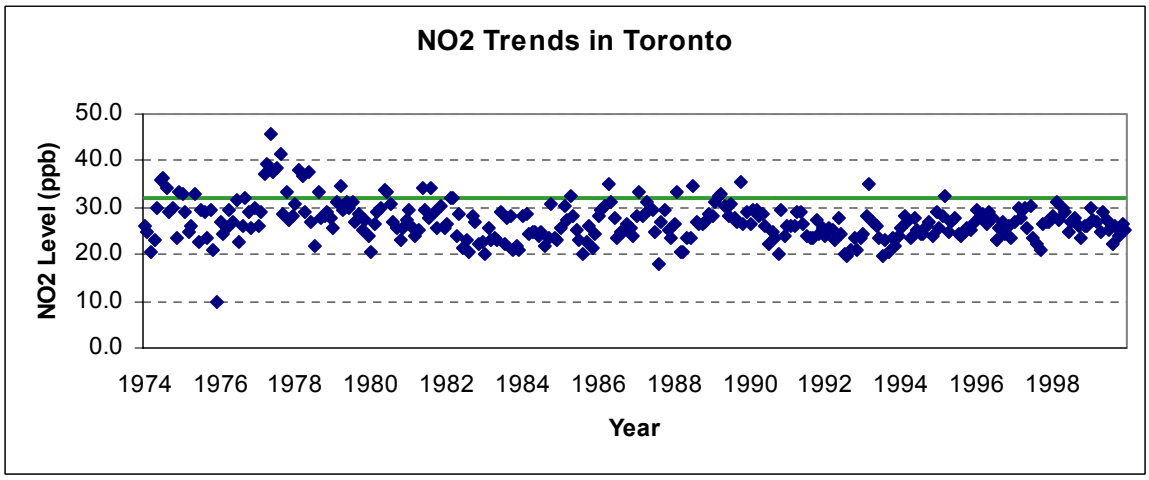

Lead Particulate Levels in Hamilton
Lead Particulate Levels (ug/m3)
0.00
0.20
0.40
0.60
0.80
1.00
1.20
1.40
1.60
1.80
1974
1976
1978
1980
1982
1984
1986
1988
1990
1992
1994
1996
1998
Year

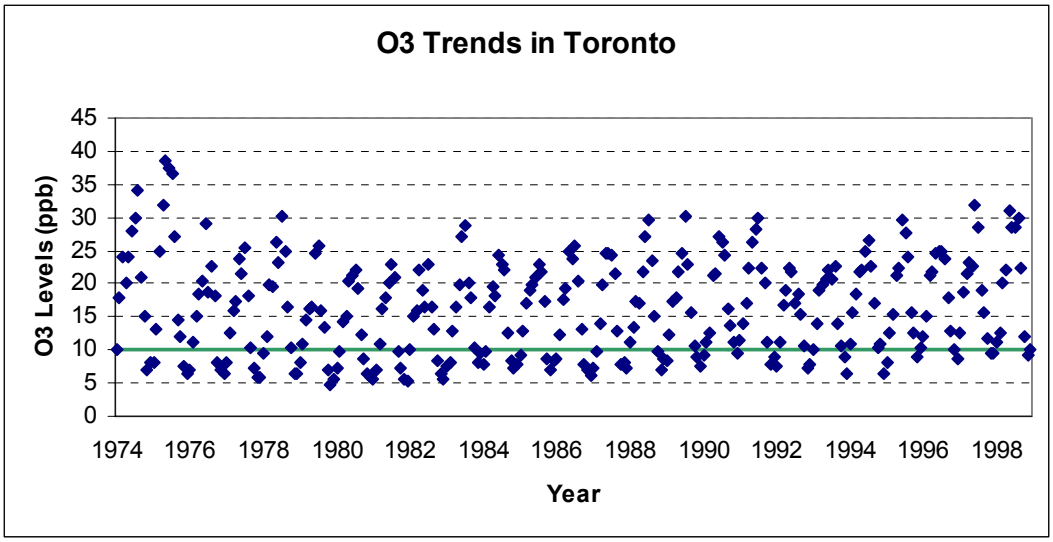

TSP Trends in Hamilton
TSP Levels (ug/m3)
0
50
100
150
200
1974
1976
1978
1980
1982
1984
1986
1988
1990
1992
1994
1996
1998
Year

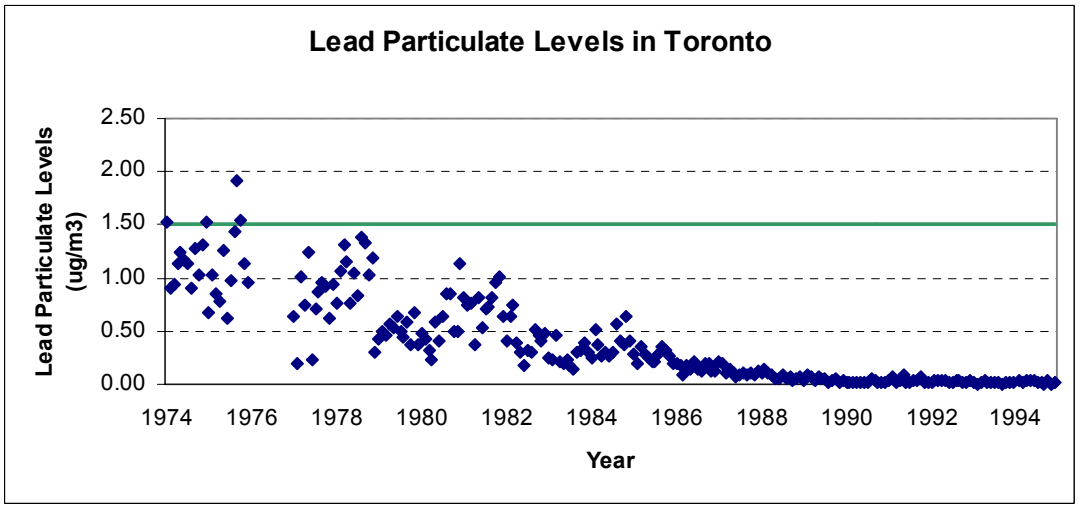
Toronto

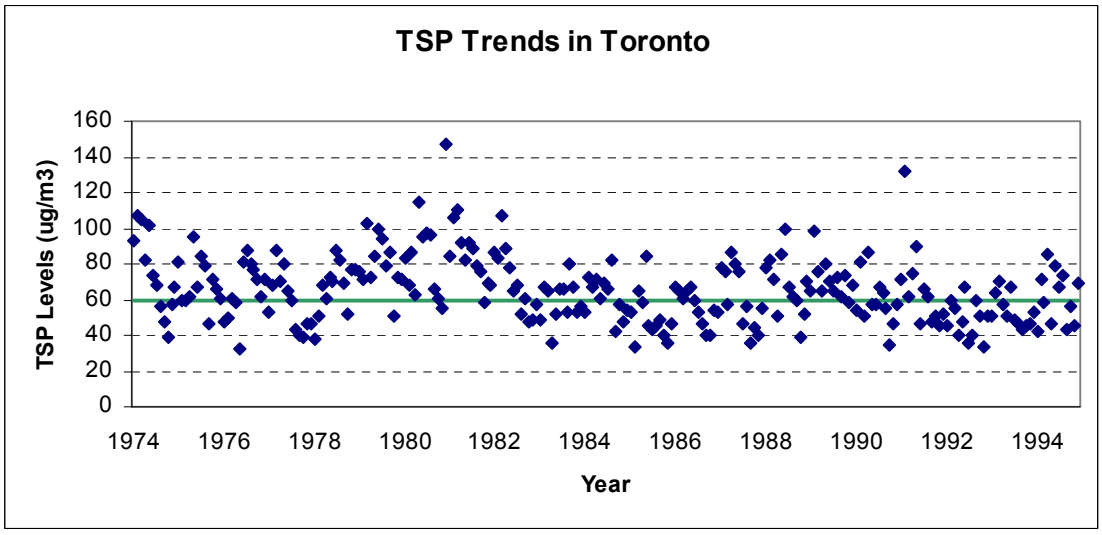

CO Trends in Toronto
CO Level (ppm)
8.0
7.0
6.0
5.0
4.0
3.0
2.0
1.0
0.0
1974 1976 1978 1980 1982 1984 1986 1988 1990 1992 1994 1996 1998
Year


SO2 Trends in Toronto
SO2 Level (ppb)
25.0
20.0
15.0
10.0
5.0
0.0
1974 1976 1978 1980 1982 1984 1986 1988 1990 1992 1994 1996 1998
Year

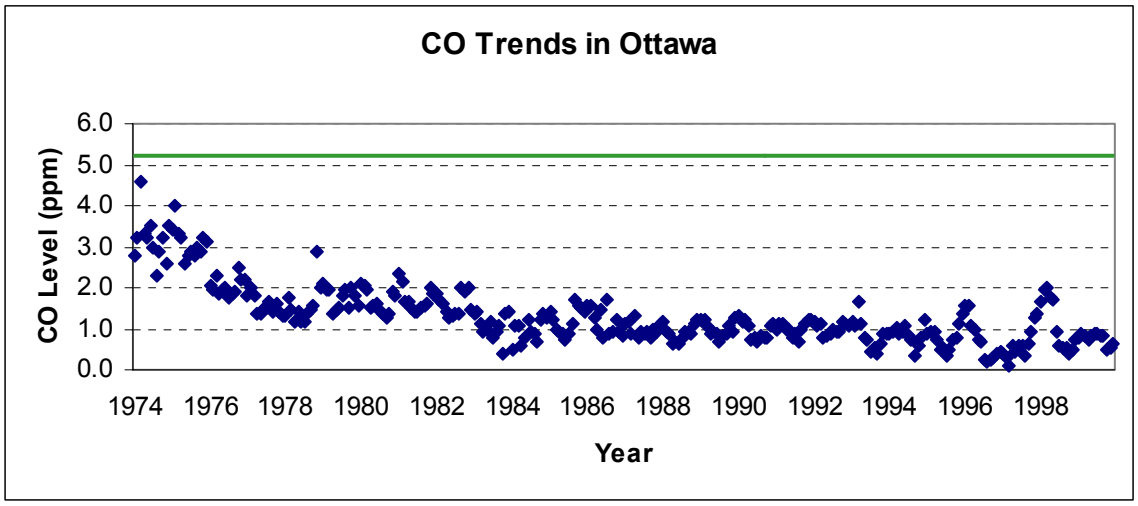

NO2 Trends in Toronto
NO2 Level (ppb)
50.0
40.0
30.0
20.0
10.0
0.0
1974 1976 1978 1980 1982 1984 1986 1988 1990 1992 1994 1996 1998
Year

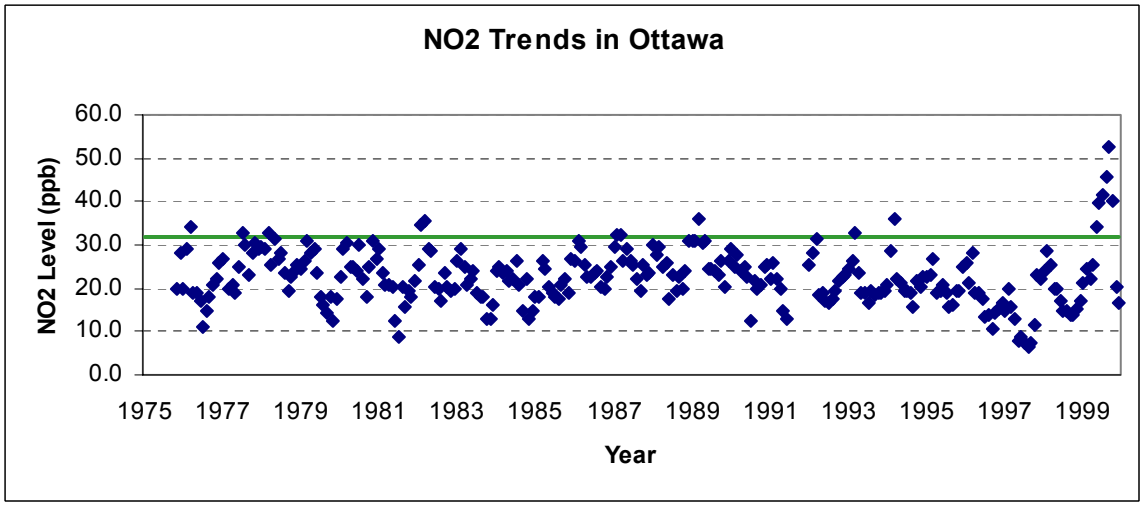

O3 Trends in Toronto
O3 Levels (ppb)
45
40
35
30
25
20
15
10
5
0
1974 1976 1978 1980 1982 1984 1986 1988 1990 1992 1994 1996 1998
Year

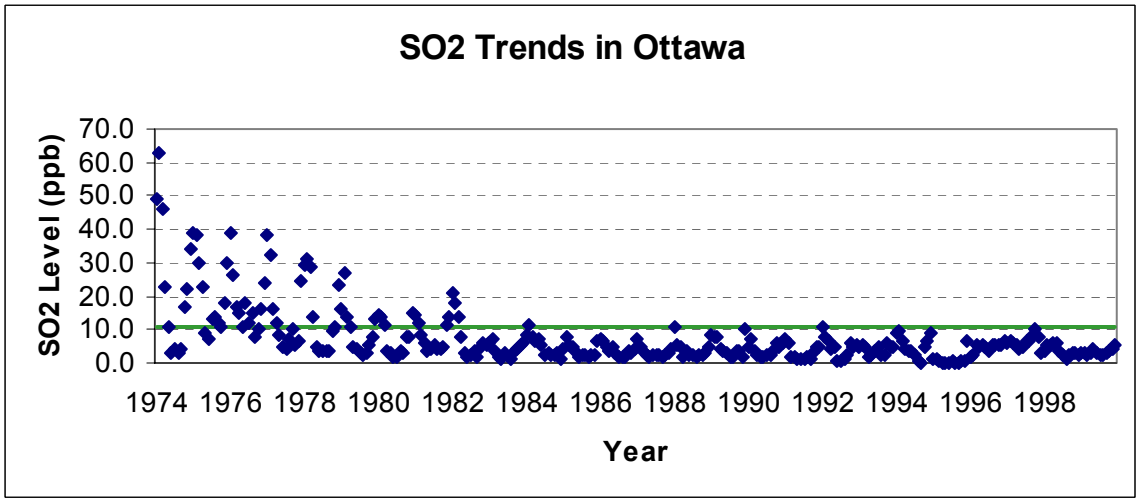

Lead Particulate Levels in Toronto
Lead Particulate Levels (ug/m3)
2.50
2.00
1.50
1.00
0.50
0.00
1974 1976 1978 1980 1982 1984 1986 1988 1990 1992 1994
Year

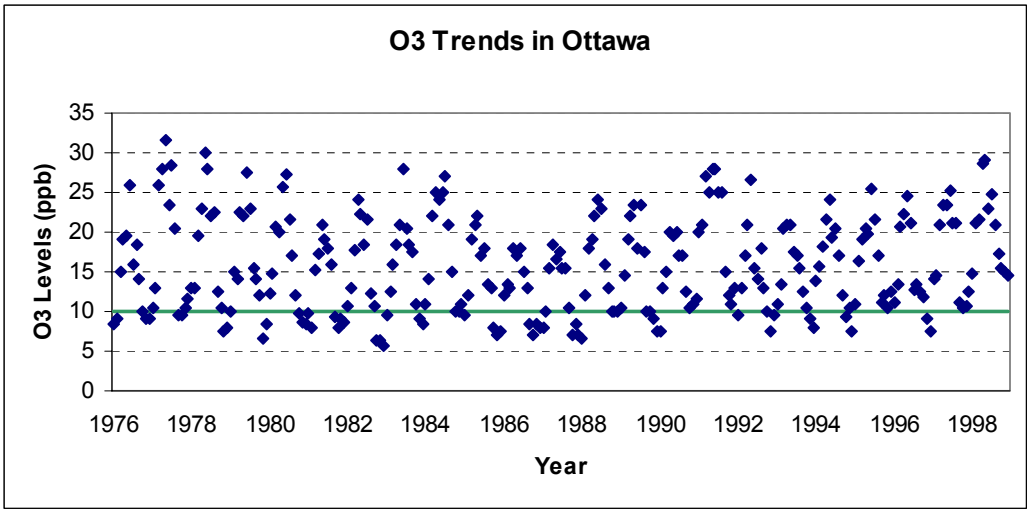

TSP Trends in Toronto
TSP Levels (ug/m3)
160
140
120
100
80
60
40
20
0
1974 1976 1978 1980 1982 1984 1986 1988 1990 1992 1994
Year

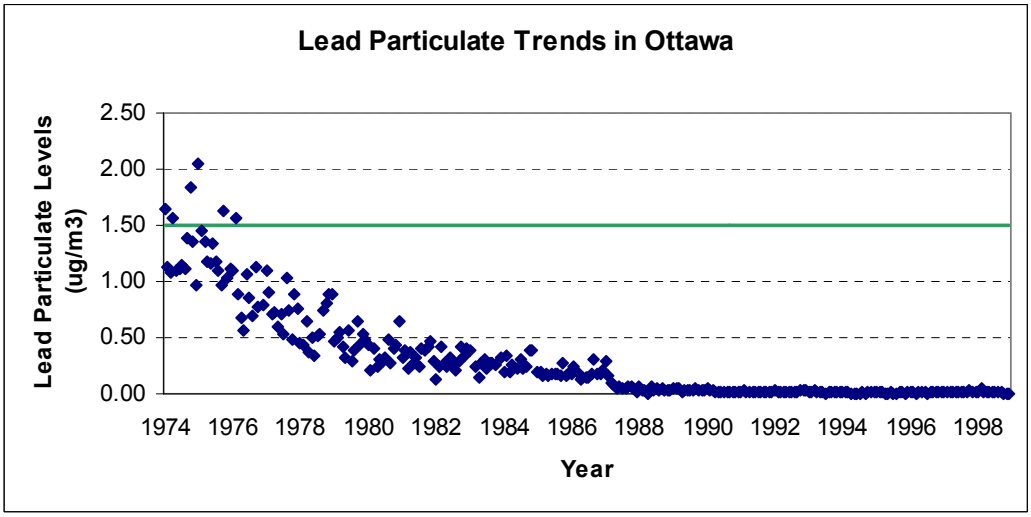
Ottawa

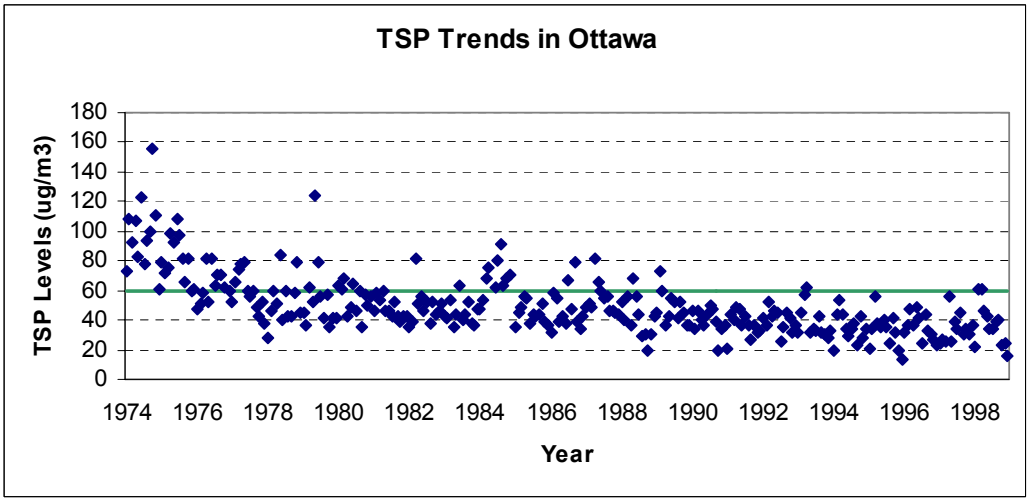

CO Trends in Ottawa
CO Level (ppm)
6.0
5.0
4.0
3.0
2.0
1.0
0.0
1974 1976 1978 1980 1982 1984 1986 1988 1990 1992 1994 1996 1998
Year


NO2 Trends in Ottawa
NO2 Level (ppb)
60.0
50.0
40.0
30.0
20.0
10.0
0.0
1975 1977 1979 1981 1983 1985 1987 1989 1991 1993 1995 1997 1999
Year


SO2 Trends in Ottawa
SO2 Level (ppb)
70.0
60.0
50.0
40.0
30.0
20.0
10.0
0.0
1974 1976 1978 1980 1982 1984 1986 1988 1990 1992 1994 1996 1998
Year


O3 Trends in Ottawa
O3 Levels (ppb)
35
30
25
20
15
10
5
0
1976 1978 1980 1982 1984 1986 1988 1990 1992 1994 1996 1998
Year


Lead Particulate Trends in Ottawa
Lead Particulate Levels (ug/m3)
2.50
2.00
1.50
1.00
0.50
0.00
1974 1976 1978 1980 1982 1984 1986 1988 1990 1992 1994 1996 1998
Year


TSP Trends in Ottawa
TSP Levels (ug/m3)
180
160
140
120
100
80
60
40
20
0
1974 1976 1978 1980 1982 1984 1986 1988 1990 1992 1994 1996 1998
Year

ABOUT THE AUTHOR:

Professor McKitrick holds a BA in economics from Queen's University, and an MA and Ph.D. in economics from the University of British Columbia. He was appointed Assistant Professor in the Department of Economics at the University of Guelph in 1996 and Associate Professor in 2000. His area of specialization is environmental economics and policy analysis. He has published scholarly articles in *The Journal of Environmental Economics and Management*, *Economic Modeling*, *The Canadian Journal of Economics*, *Environmental and Resource Economics*, *Climate Research* and other journals, as well as commentaries in newspapers and other public forums. His current research areas include empirical modeling of the relationship between economic growth and pollution emissions; the impact of economic activity on the measurement of surface temperatures; and the climate change policy debate. Professor McKitrick has made invited academic presentations in Canada, the US and Europe, as well as professional briefings to the Canadian Parliamentary Finance Committee, and to government staff at the US Congress and Senate. In the fall of 2002 he was appointed as a Senior Fellow of the Fraser Institute in Vancouver B.C.