

Air Pollution, Health and Mortality: Separating Fact from Fiction

Ross McKittrick
Associate Professor of Economics
University of Guelph
Guelph, Ontario N1G 2W1

Presentation to the Association of Major Power Consumers of Ontario

Toronto, April 30 2004

Air Pollution, Health and Mortality: Separating Fact from Fiction

1 Big Fears and Bold Plans

In the last few years, Ontarians have been repeatedly told that the people who supply our electricity are mass murderers. This eye-popping accusation does not come from the radical fringe, but from banks, churches, industry groups, unions, Environment Canada, school groups, the Ontario Medical Association (OMA), and numerous others, via the umbrella group the Ontario Clean Air Alliance (OCAA).¹ Here is what the OCAA says about our coal-fired power plants:

The Ontario Medical Association estimates that air pollution costs Ontario more than \$10 billion per year in health care costs, lost work time and other quantifiable expenses, as well as killing an estimated 2,000 Ontarians each year. The OMA has declared air pollution "a public health crisis" in Ontario. Coal-fired power plants are the single largest industrial contributors to this crisis. (<http://www.electricitychoices.org/coal.html>)

The pamphlet from which this statement is quoted has a photo on the front that juxtaposes a smokestack with a child wearing a respiratory mask:



Figure 1: "Dirty Coal, Dirty Power, Dirty Air." (Ontario Clean Air Alliance)

¹ The list of backers is at <http://www.cleanair.web.ca/aboutus/sponsors.html>.

To put the claim in perspective: The OCAA says that air pollution in general, and the operators of coal-fired power plants in particular, allegedly kill more people in Ontario *every 18 months* as Osama bin Laden killed in the USA on 9/11. The Toronto Board of Health has repeated this accusation, claiming that in the City of Toronto alone, air pollution is responsible for over 1,000 premature deaths and about 5,500 hospitalizations each year (Basrur 2000). The two claims are not really independent, for they both arise from the same model-based extrapolations on the same thin statistical foundations. They are not statements of observed fact, they are conjectures that have been accepted uncritically by policymakers and commentators.

No one wants people to suffer from aggravated respiratory disease. Where identifiable harm exists I have written extensively on the design and implementation of effective market-based measures, such as emission pricing and tradable permits, to efficiently remediate real harms. But the sort of irresponsible rhetoric engaged in by the OCAA doesn't help to identify and remediate verifiable harms to human health. Rather, OCAA's rhetoric verges on hatemongering, which can only impede reasoned consideration of the future of our energy supply, precisely when dispassionate inquiry is most needed.

The specific claims examined in this paper are as follows:

- *air pollution is bad and getting worse*
- *air pollution mainly comes from power plants burning coal*
- *air pollution is a leading cause of mortality and disease*

The first two can be dealt with by looking at available data. The latter requires consideration of existing scientific literature, especially the widely cited studies from the OMA (2000) and the Toronto Board of Health (2000), as well as consideration of what additional studies ought to be undertaken to fill in the gaps in the current literature.

This discussion takes place in the context of the recent Ontario government decision to shut down the coal-fired power plants operated by Ontario Power Generating Corp.² This paper will end with some comments about how such decisions ought to be made.

2 Fictions and Facts

2.1 "Air pollution is bad and getting worse"

Let's first look at whether air pollution is getting worse. The OCAA says it is, though they are characteristically thin on evidence. In their report "Expanding Exports, Increasing Smog" they state:

² See, e.g., Ontario Ministry of Energy news release at http://www.energy.gov.on.ca/index.cfm?fuseaction=english.news&body=yes&news_id=50.

Between 1995 and 2000, production of coal-fired electricity grew by almost 150% in Ontario. This increased use of dirty coal to produce electricity brought with it a major increase in smog-causing emissions from Ontario Power Generation's five coal-fired power plants. These emissions have contributed to steadily worsening air quality in Ontario and regularly reach as far as the Atlantic provinces.

(<http://www.cleanair.web.ca/resource/submarine.pdf>)

The OMA (2001) makes a similar claim: "This review finds a worrying situation in the recent trends in Ontario's air quality."

The Appendix contains graphs displaying Ontario air pollution concentration levels for Windsor, London, Hamilton, Toronto and Ottawa, from 1973 to 1999 (the most recent year I could obtain as of last summer). The criterion air contaminants are carbon monoxide (CO), nitrogen oxides (NO, NO₂, collectively "NO_x"), sulphur dioxide (SO₂), lead, ozone (O₃) and total suspended particulates (TSP). The data are compiled by Environment Canada through the National Air Pollution Surveillance (NAPS) system. In each graph the horizontal green line represents the Environment Canada desirable standard except for lead, where the US Environmental Protection Agency standard is shown.

The data reveal that there is no pattern of rising air pollution. In every city, CO and lead levels are trivial compared to historical levels and low compared to the current safe standard (Windsor CO is not shown—data n/a). Likewise SO₂, NO₂ and TSP levels have fallen to the point where they are routinely within the Environment Canada recommended standards, with only occasional, temporary departures above the standard in some places. Only ozone regularly goes above the standard, but on an irregular, seasonal basis. Ozone chemistry is highly complex, and still poorly understood. 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 average ozone levels tend to be the same in rural areas as it is in cities,³ we should not expect that city air will ever be ozone-free, regardless of how much pollution control is undertaken. Ozone is quite difficult to control. Fortunately, it is primarily a summertime phenomenon that only becomes acute in temporary episodes. Since these episodes are known to be seasonal and temporary, people can to some extent mitigate their risk by following public health advisories, by remaining indoors and by using air conditioning—assuming the electricity is available and affordable.

Even in the case of ozone there has been progress. In the most populous city (Toronto) average ozone concentrations have been slowly declining since the 1970s, which is made visible by examining 12-month moving averages (see Figure 2). Notice the strongly cyclical nature of ozone levels, which may relate in part to meteorological conditions.

³ The data are in Table 5, Air Quality in Ontario 2001, Data Appendix.

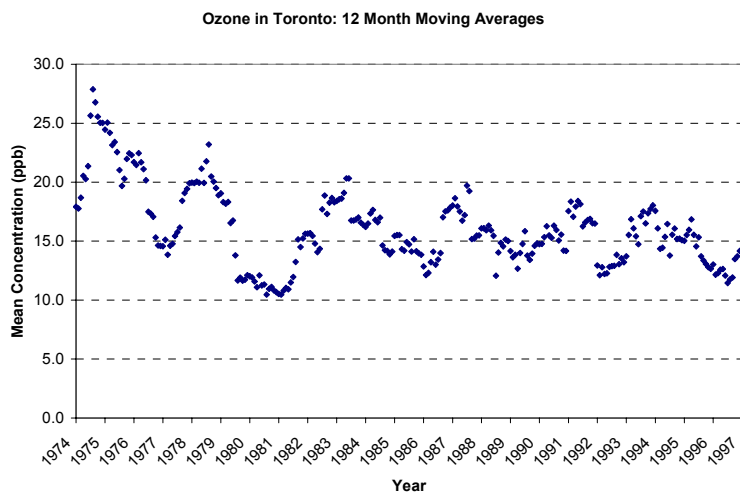


Figure 2: Ozone concentrations (12 month moving averages) in Toronto, 1974-1997. Source: Environment Canada NAPS data (see Appendix).

The data shown in the Appendix Tables end at 1999. Updated data (to 2001) are available at the Ontario Environment Ministry website⁴ and confirm that the long-term improvement trends have not reversed. There was a small rise in ozone from 1995-2001 (see Figure 5 below) but not enough to reverse the long term trend of Figure 2.

Overall, the claim that air pollution is getting worse is not supported by the available data. Indirect measures (such as “bad air days”) are unreliable as the standard has changed over time. 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.

2.2 “Air Pollution Mainly Comes From Power Plants Burning Coal”

Air pollution is not “one” thing—there is a long list of gases and aerosols of concern. So not surprisingly it does not come from “one” place.

SO₂, CO and NO_x

Just over half⁵ of current sulphur dioxide emissions are attributed to smelters and refineries. Utilities generate about one-quarter of emissions. 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 SO₂ levels are in violation of air quality standards.

⁴ http://www.ene.gov.on.ca/envision/techdocs/4521e_appendix.htm#10

⁵ See <http://www.ene.gov.on.ca/envision/techdocs/4521e.htm#4> for data in this section.

Carbon monoxide mostly comes from transportation sources (85 percent). As with SO₂, ambient levels are very low compared to the early 1970s and there is no location in the province with a CO concentration problem, even in cities (see charts in Appendix).

NO_x is also mostly from transportation (63 percent), with utilities responsible for 15 percent. The main concern about NO_x is its role in ozone 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 (Green et. al. 2002). Some of it, of course, comes from smokestacks. In the annual “Air Quality in Ontario” Report published by the Ontario Environment Ministry, industrial sources are highlighted, creating the impression that these predominate.

The US Environmental Protection Agency provides more detailed source breakdowns for American emissions. The most recent data are from 1998, and show that on a national basis natural sources dwarf the rest (5,307,000 short tons), followed by residential wood burning fireplaces (411,000 short tons). Use of coal for industry and electric power generation comes next at 362,000 short tons. To the extent that particulates are a health hazard, Mother Nature is the number one polluter, followed by users of that popular “renewable” energy source, wood. However, the health effects of particulates is a complicated topic, as will be discussed further below.

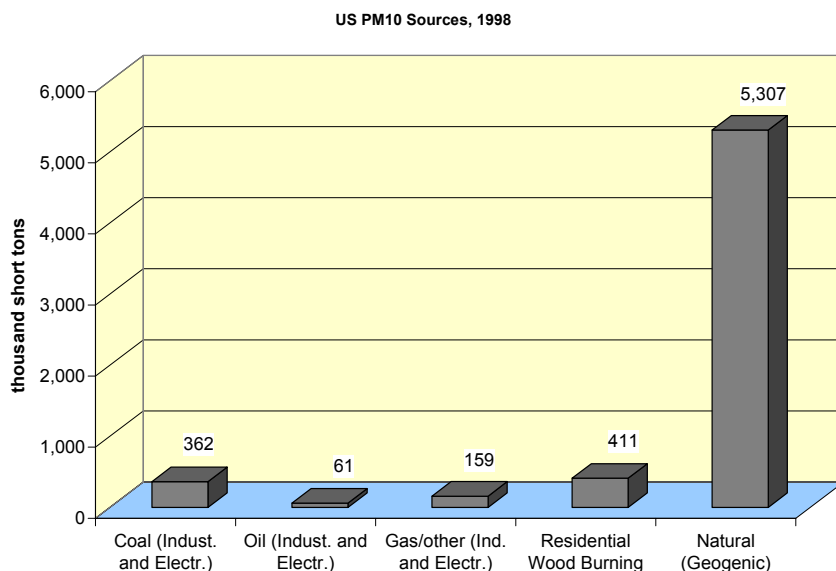


Figure 3: Major Particulate (PM10) Emission Sources in the US, 1998. Source: US Environmental Protection Agency <http://www.epa.gov/ttn/chief/trends/index.html>.

Ozone

We can evaluate the specific claim from the OCAA (see above) regarding the ramp-up of coal-based power production between 1995 and 2000 causing increased smog by comparing the ground-level ozone readings across the province⁶ in 1995 and 2000, to see if the 150% production increase had an effect. The data are shown in Figure 4. There was little change in ozone levels between these two years. Some locations rose slightly while others fell. The average for all locations hardly moved: it was 23.4 parts per billion (ppb) in 1995 and 23.6 ppb in 2000.

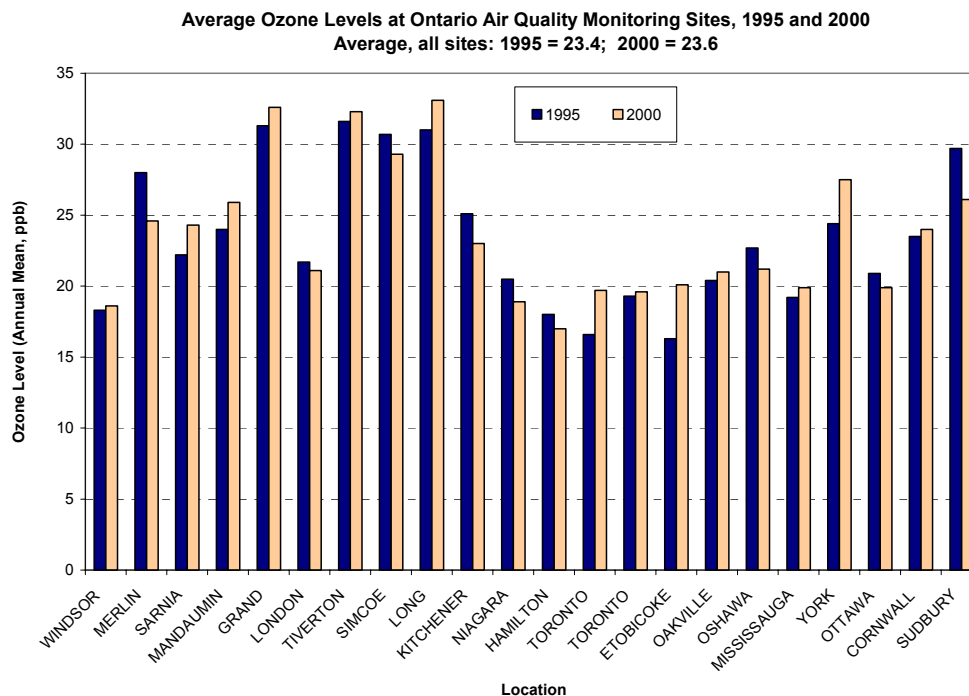


Figure 4: Ozone readings in Ontario, 1995 and 2000. Source: Air Quality in Ontario 2001 Data Appendix, Table 16.

However 2000 was a ‘dip’ compared to other years, which show a slight recent trend increase. The average for 1999 was 25.8 and for 2001 it was 25.7. Mother Nature is again a potential culprit. Ground level ozone is produced by sunshine acting on the boundary layer atmosphere. The process is exceedingly complex and depends on numerous factors other than solar flux, including cross-border pollution transport. Solar output is measured in a number of ways, including sunspot counts and F10.7 cm radio flux (both of which are correlated). The intensity of solar flux goes through irregular 11-year cycles. The last solar minimum was in 1995, following which solar output rose to the most recent maximum (indeed a double maximum) in 2001.⁷ So, all other things being equal, we would expect ozone levels to rise over the latter half of the 1990s even without increased air pollution emissions. The plot of annual Ontario ozone averages and solar flux over the 1995-2001

⁶ Air Quality in Ontario 2001 Data Appendix, Table 16.

⁷ See <http://www.drao-ofr.hia-ihc.nrc-cnrc.gc.ca/icarus/www/maverplot.html>

interval is in Figure 5. Note that the ozone level changes are still small (one-tenth of the scale on Figure 2) and the level for 2000 is, if anything, rather low compared to what we might expect at that point in the solar cycle.

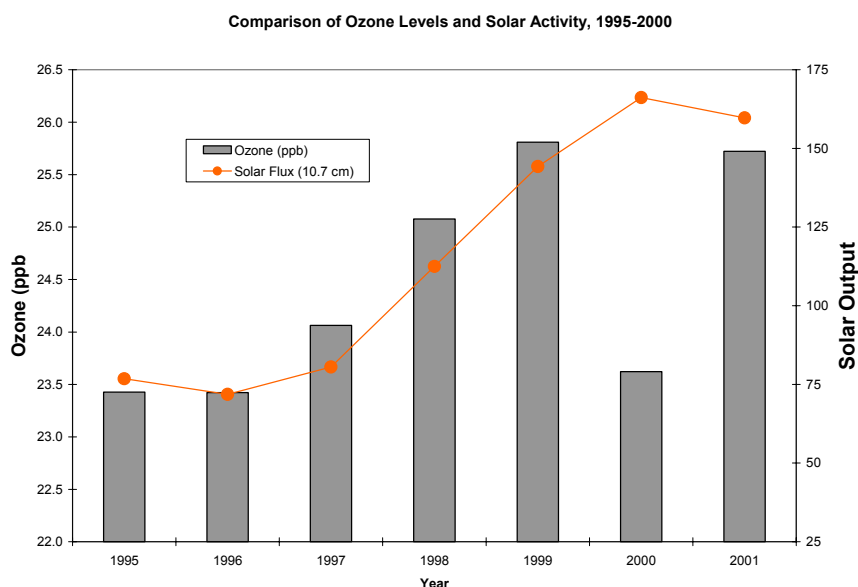


Figure 5: Ontario Average Ozone Levels (gray bars) and Solar Flux (orange circles) 1995-2001. Ozone data as for Figure 2. Solar flux data: Australian Space Weather Agency <http://www.ips.gov.au/Main.php?CatID=5>.

In sum, it is simplistic to assert that air pollution is rising and coal plants are to blame - the facts do not support the claim. Air pollution is generally falling, not rising, with the possible exception of ozone. Particulates come from many sources, including natural ones, and even among anthropogenic sources, coal-using utilities are not the biggest contributor. Ozone is a complex phenomenon only indirectly associated with air emissions, and the natural solar cycle is potentially a contributor to recent changes in observed levels.

2.3 “Air Pollution is a Leading Cause of Mortality and Disease”

It is very difficult to tie air pollution to mortality or morbidity (i.e. lung and cardiac disease). There are three distinct types of studies that attempt to do this: epidemiological, modeling and toxicological. Epidemiological studies look at time series data to see if death rates or disease incidence can be correlated with observed pollution rates. Disease incidence is usually measured by looking at hospital admissions, but may also be looked at in terms of asthma rates in children and so forth. These studies publish results expressed as “relative risk ratios” (RR). For example, an RR of 1.05 per 10 ppb would be interpreted as saying that, for a specified contaminant, a 10 ppb

increase in the ambient level is associated with a 5% increase in the risk of a specified outcome (e.g. admission for lung disease). Modeling studies take epidemiological results and use them to attribute portions of observed deaths or diseases to air pollution. Toxicologists use controlled experiments to detect a dose-response relationship.

Epidemiology

The field of environmental epidemiology has tentatively put forward claims of a small but significant positive effect of air pollution on disease and premature deaths. It is important to note, first, that the deaths are notional ones, as indicated by the adjective “premature.” The papers that search for pollution-mortality correlations begin with a model of the “expected” number of deaths on a given day, as a function of weather, population, trends, and other exogenous factors. Then the difference between the actual death rate and the expected death rate is computed, and this difference is correlated to pollution. The result is an RR measure for each pollutant.

Since the model depends on accurate estimates of an unobservable variable, namely the “expected death rate,” results are sensitive to the model specification and the particular data set examined. Researchers have reported a wide range of results, sometimes even in the same study. One of the largest studies of the pollution-mortality relationship in the US was by Dominici et. al. (2002). They studied 88 cities across the US over the 1987 to 1994 interval, estimating RR levels for small particulates in each. As shown in Figure 6 (which reproduces their Figure 2) the results do not indicate a uniformly positive relationship between pollution and mortality. The large (horizontal) uncertainty bars show that the effects are not statistically significant in most (78 out of 88) cities. In some cities in fact, higher air pollution is associated with *lower* mortality—a finding which raises pertinent questions about what exactly is being measured here. The small, bold-faced box-whisker plots at the bottom of each section show the average of the city-specific point estimates. These are small, positive and statistically-significant, although note that since they refer to averages across cities they measure effects no one actually experiences, since people only live in one city at a time.

Modeling

A second type of study is based on use of spreadsheet models. Prominent examples include the work of the Ontario Medical Association (2001) and the Toronto Board of Public Health (Basrur 2001). In these, the researcher surveys some of the published results and finds coefficients that relate pollution levels to mortality or disease. For instance, a user of the Dominici et. al. study might take from their paper an RR value of 1.0043 for a 10 $\mu\text{g}/\text{m}^3$ increase in particulates,⁸ which implies a 0.043% increase in mortality per 1 $\mu\text{g}/\text{m}^3$ increase. This can be converted into an absolute relationship by extrapolating back to zero, then a function can be approximated that maps an observed level of particulates to a predicted percentage risk of premature death. This, in turn, can be used with local data on non-traumatic deaths to attribute a certain number of them to pollution.

⁸ $\mu\text{g}/\text{m}^3$ refers to micrograms per cubic meter, the standard unit of measurement for particulate pollution.

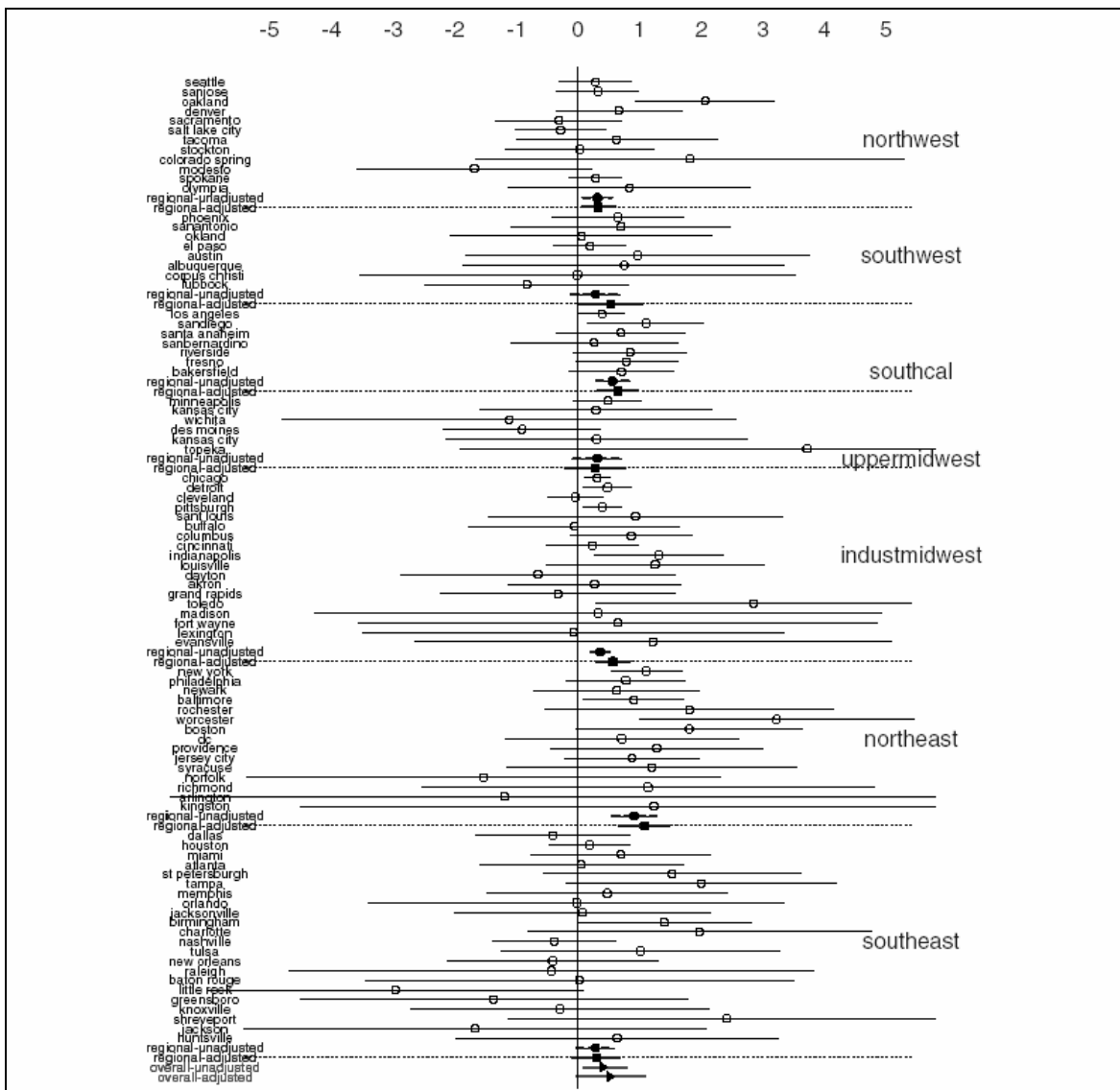


Figure 2. MLEs and 95% Confidence Intervals of the Log-Relative Rates of Mortality per 10 $\mu\text{g}/\text{m}^3$ increase in PM_{10} for Each Location. The solid and the square circles with the bold segments denote the posterior means and 95% posterior intervals of the pooled regional effects without and with covariate adjustments. At the bottom, marked with triangles and bold segments, are the overall effects for PM_{10} for all the cities without and with covariate adjustments.

Figure 6: Reproduced from Dominici et. al. (2002). Each line shows the point estimate (square) and confidence interval (line) of the percentage increase in mortality associated with a 10 $\mu\text{g}/\text{m}^3$ increase in PM_{10} . The dark-shaded lines at the bottom of each section show regional averages.

A major risk in modeling studies is that the survey of mortality/disease coefficients may suffer from publication bias: researchers who do not get significant results are less likely to publish their results. Also, in the case of air pollution data, many studies done in the latter half of the 1990s are now known to have overestimated coefficients because of improper settings in a popular statistical

package.⁹ A large project is underway¹⁰ by researchers in the field to re-estimate their results, and already some major regulations planned by the Environmental Protection Agency have been put on hold because the new results show lower mortality coefficients.

The Toronto Public Health study surveyed much the same list of papers as did the OMA, and their model boils down to the following three equations:

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

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

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

(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 by the Board of Health to determine the “Air Pollution Burden of Illness” for Toronto, as the sum of $D_p + R_p + C_p$. They estimated, 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:

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

⁹ Andrew Revkin, “Data revised on soot in air and deaths” New York Times, June 5 2002 p. A23.

¹⁰ See http://www.biostat.jhsph.edu/biostat/research/nmmaps_faq.htm

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.

One way to evaluate the plausibility of Board of Health model is to plug in observed air pollution levels back through the early 1970s and see if the results change much. The most basic test of the model's plausibility is that it should never attribute more than 100 percent of the actual observed deaths or hospital admissions to air pollution. Unfortunately it fails this test.

Figures 7-9 show the results of applying the Toronto Public Health model (Air Pollution Burden of Illness in Toronto, or APBIT) to air pollution rates from 1965 to 1997 in downtown Toronto, as measured at the Bay/Wellesley monitoring station. NO_x and O₃ levels prior to 1973 are not available. The NO_x series was filled 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).

The diamonds mark the monthly point estimates and the dark lines are 6th-order polynomial curves to show a smoothed mean. From Figure 7 we can see that while the model regularly returns death proportions of 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 in the 1960s. Cardiac admissions are heavily 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.

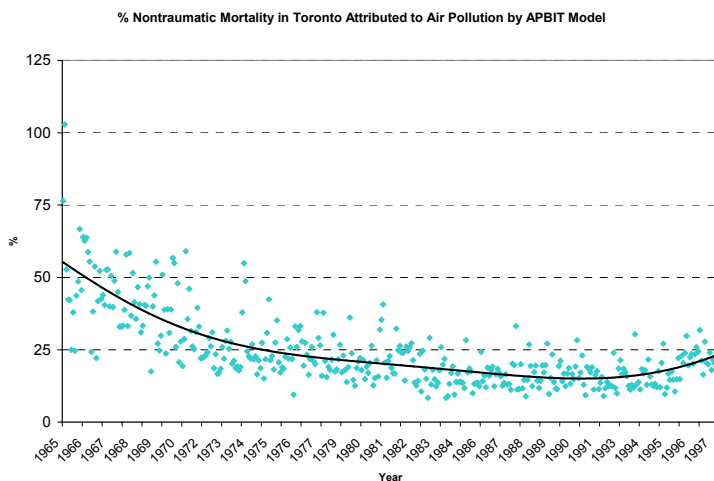


Figure 7: APBIT Model attribution of mortality in Toronto

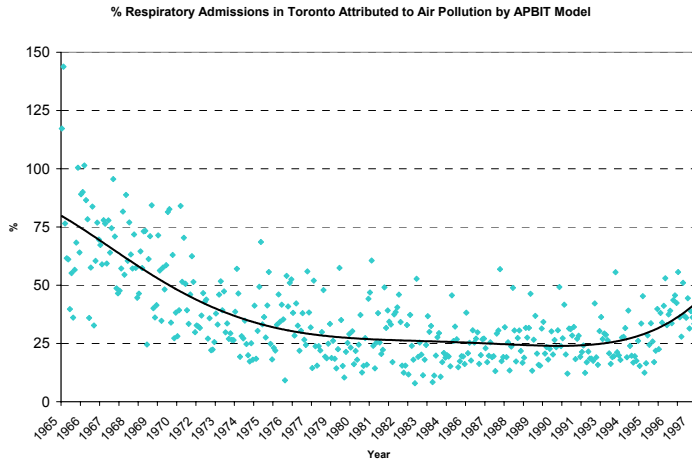


Figure 8: APBIT Model attribution of respiratory disease in Toronto

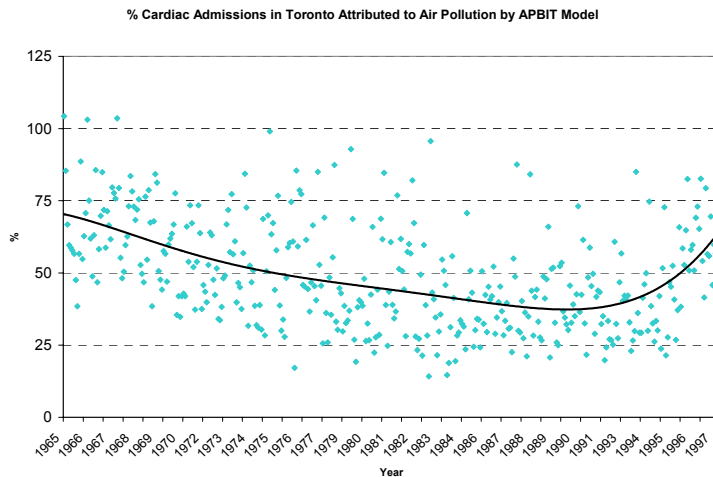


Figure 9: APBIT Model attribution of cardiac disease in Toronto

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-trauma 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 is obviously implausible. It works by making unstable and exaggerated extrapolations from a limited set of epidemiological coefficients. It is enough to examine its predictions in earlier decades to see this, but additional evidence comes from recent studies of *model uncertainty*. From Figure 6 it is clear that results can differ widely depending on the place studied. But results also

differ widely depending on the regression model used. While there might be a simple correlation between time series data on air pollution and hospital admissions for breathing difficulties, this correlation may not show up once other covariates are included, such as weather data (temperature, air pressure, wind), smoking rates, income levels, or if the model is slightly respecified to include trends, lags and so forth. 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. These 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 that it is the “correct” one based on the way the model fits 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 found that while some specifications can yield a positive relationship between air quality and premature death rates, the most likely model specification only includes weather variables. In other words while the data can be analyzed to “prove” pollution causes mortality, more thorough analysis shows this is unlikely to be true. They evaluated over 567 trillion model specifications and concluded there is no significant effect on mortality from air pollution. 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).”

The study by Koop and Tole is the most comprehensive study of the air pollution-mortality connection in Toronto to date. I have begun a federally-funded multi-city study which will look at the same question for 13 cities across Canada, using air pollution and respiratory admission rates matched to income levels, smoking rates, demographics, weather and other covariates. The availability of much larger data sets and sufficient computational power is now making it possible to advance environmental epidemiology past the stage of offering selective point estimators.

Toxicology

The final approach to the air pollution/health connection is experimental toxicology. Green et al. (2002) provide a review of the literature assessing the toxicology of fine particulate matter. Experiments have shown that high ozone levels cause respiratory irritation in asthmatics. But lab studies of particulates have not found evidence of toxicity or respiratory problems (even for asthmatics) from exposure to particulate levels much higher than observed ambient levels, although asthmatics show some response to highly acidic aerosols. This review emphasizes that the term “particulates” is toxicologically meaningless since it encompasses such a wide range of compounds. The solubility of a compound is one of the key determinants of its toxicity, yet particulate standards make no distinction between soluble and insoluble kinds. Studies on hamsters, rats and dogs involving exposure to particulates up to 1,000 $\mu\text{g}/\text{m}^3$ 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: indeed the matter is sufficiently settled that it is no longer actively investigated.

The contrast between the results of the toxicology literature and that of the epidemiological literature can be illustrated by applying the concept of lethal potency to ambient fresh air. Hazardous gases are assigned coefficients called the unit risk factor (URF), which measures the carcinogenic potential of long term exposure to inhaled gases. Green et. al. take a RR factor reported in the epidemiological literature for sulfates and compute the implied URF. The result is that sulfate levels in ordinary fresh air allegedly have 1.7 times more potency as a lung carcinogen than directly inhaled coke oven emissions. As the authors ask, rhetorically, “How plausible is this?” (p.328).

By looking at the literature on medical toxicology, not just selected studies from the epidemiology literature, it is difficult to conclude that ordinary, ambient “fresh” air is a health hazard, even with current pollution sources operating. It is noteworthy that the UK government’s *Committee on the Medical Effects of Air Pollution* also studied the issue and concluded:

“For the most part, 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/index.htm>)

3 Should Ontario Shut Down the Coal Plants?

The way to answer this question is compare the costs and the benefits. Any such exercise must include the costs of replacement power sources, the risks of blackouts, the deterrent to investment if Ontario is persistently undersupplied with electricity or power costs shift permanently upward, the social effects of higher electricity prices including the adverse health impacts if poor people cannot afford to operate air conditioning systems during bad air episodes, and so forth.

To the extent that benefits have been tallied they seem to presuppose a lot of fictional claims about air pollution trends, sources and impacts, as discussed in the previous section. Despite perceptions to the contrary, air pollution is not a growing problem in Ontario, and the marginal health benefits of reducing coal-plant emissions are sketchy, likely minuscule and probably non-existent. Trendy alternate energy sources, such as wind or solar power, are infeasible on a large scale¹¹ and carry their own steep ecological price tag since they are very land-intensive if they are implemented on more than a token scale. Encouraging homes to use wood for lighting or heating may have a quaint, rustic charm about it, but fireplaces and woodstoves are just as much a pollution source as the

¹¹ Hoffert et. al. (2002)

smokestacks of Nanticoke. Demand-side management somehow remains a popular idea yet it is a longstanding costly failure and cannot be relied upon to balance supply and demand.

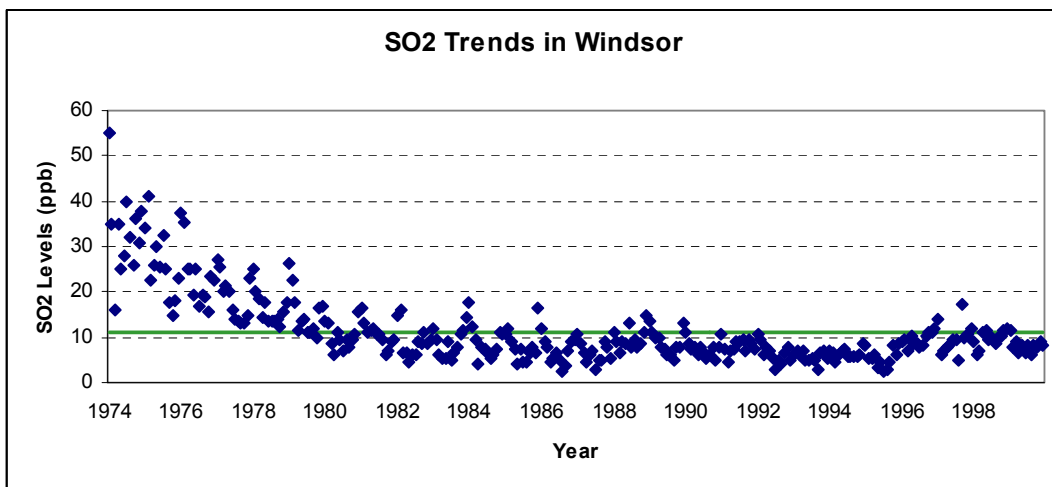
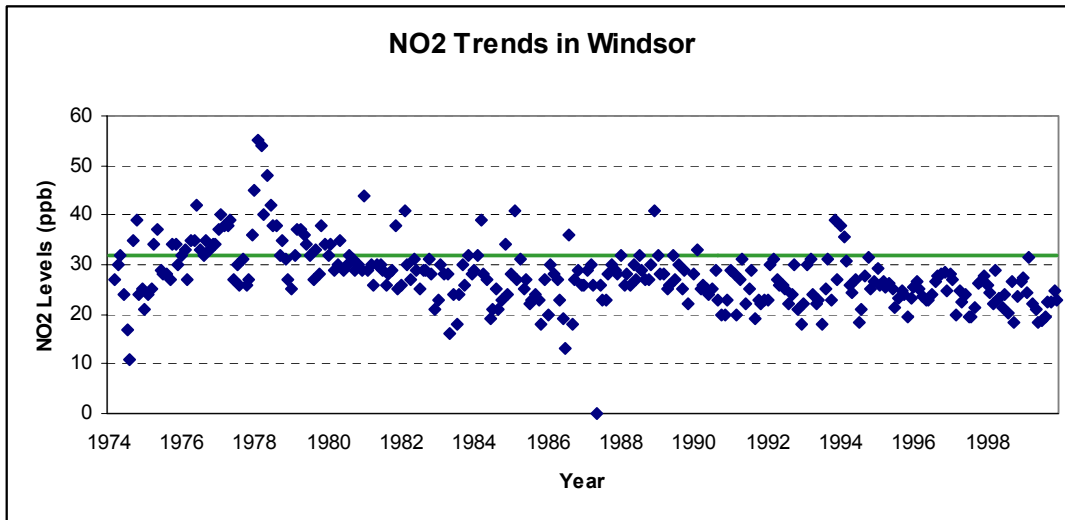
Based on these considerations it seems unlikely that the benefits of the proposed shutdown of the coal plants could even remotely offset the costs. That said, the most important thing is that the final decision be based on a full, careful and dispassionate appraisal of the costs and benefits, based on facts not fictions.

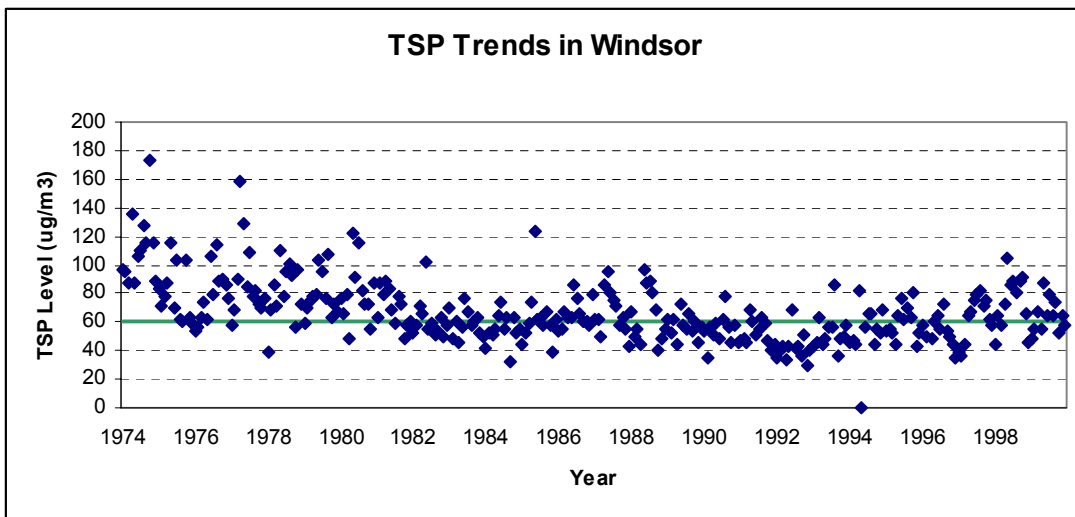
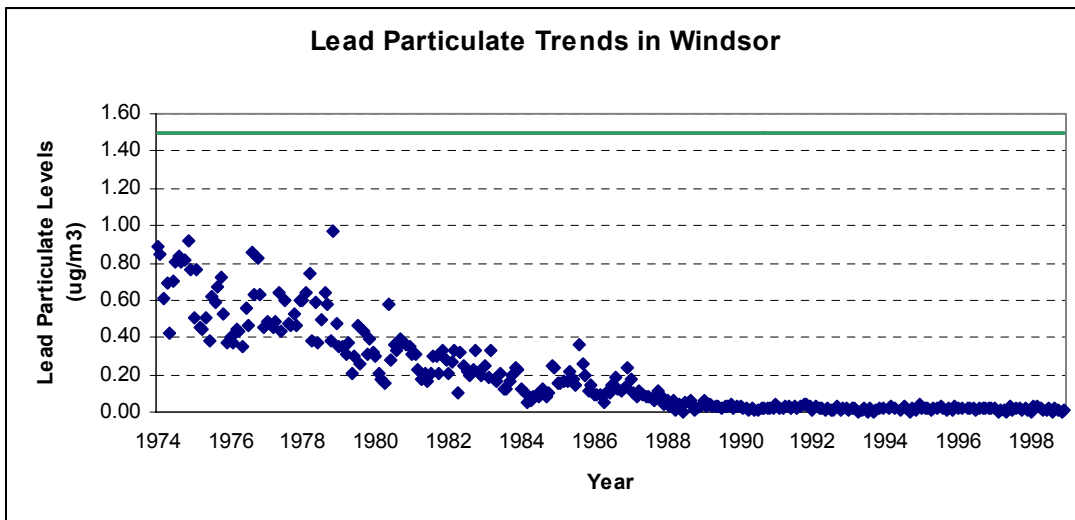
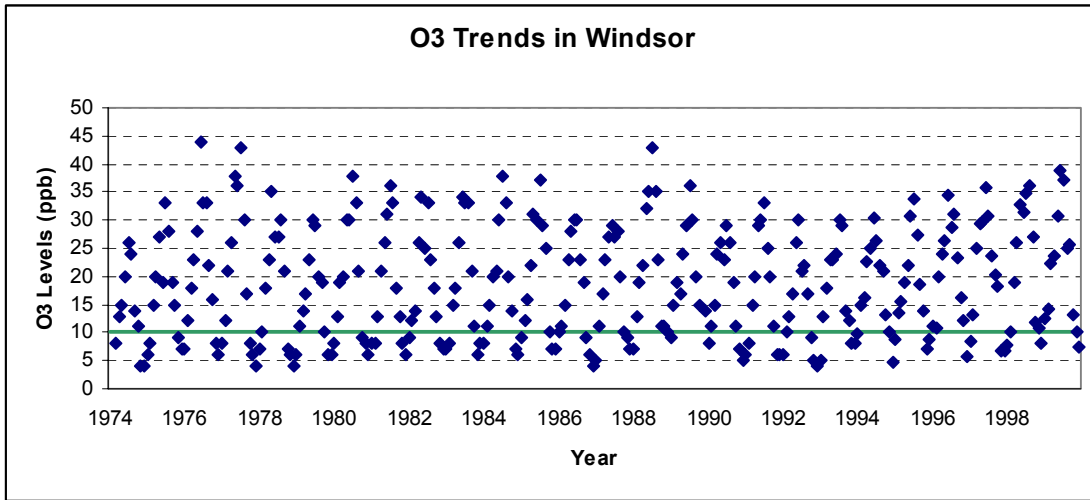
References

- Basrur, Sheela (2000) “Air Pollution Burden of Illness in Toronto.” Toronto Public Health, May 2000
- Dominici, Francesca, Michael Daniels, Scott L. Zeger, and Jonathan M. Samet (2002) “Air Pollution and Mortality: Estimating Regional and National Dose–Response Relationships.” *Journal of the American Statistical Association* 97(457) 1—12.
- Green, Laura C., Edmund A. C. Crouch, Michael R. Ames, and Timothy L. Lash (2002). “What’s Wrong with the National Ambient Air Quality Standard (NAAQS) for Fine Particulate Matter (PM_{2.5})?” *Regulatory Toxicology and Pharmacology* 35: 327—337.
- Hoffert, Martin and 16 others (2002) “Advanced Technology Paths to Global Climate Stability: Energy for a Greenhouse Planet.” *Science* 298, November 2002, 981—987.
- Koop, Gary and Lise Tole (2004) “Measuring the Health Effects of Air Pollution: To What Extent Can We Really Say That People are Dying from Bad Air?” *Journal of Environmental Economics and Management* 47: 30—54.
- Ontario Medical Association (OMA) (2000). “Illness Cost of Air Pollution.” Prepared by DSS Management Consultants, June 2000.
- Ontario Medical Association (OMA) (2001). “Ontario’s Air: Years of Stagnation” <http://oma.org/phealth/report/airq001.pdf>
- Ontario Ministry of the Environment (2003). Air Quality in Ontario 2001. <http://www.ene.gov.on.ca/envision/techdocs/>

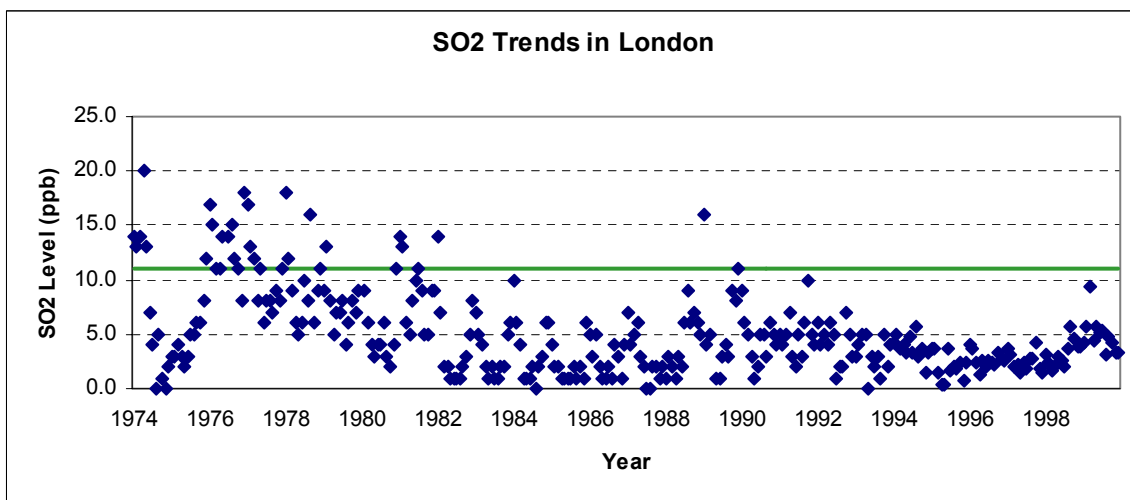
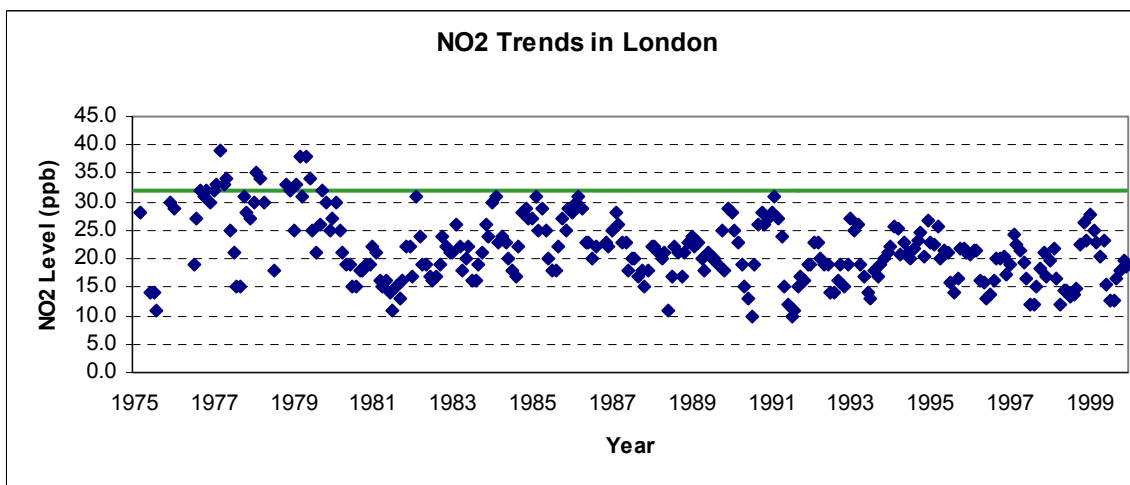
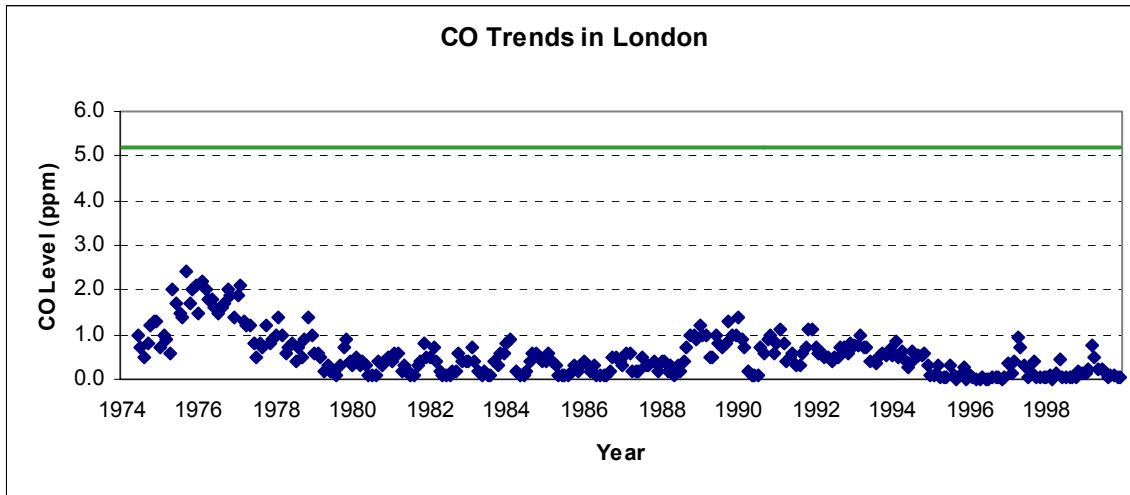
**APPENDIX: Air Contaminant Concentrations in Ontario
(Green line – Environment Canada Standard)**

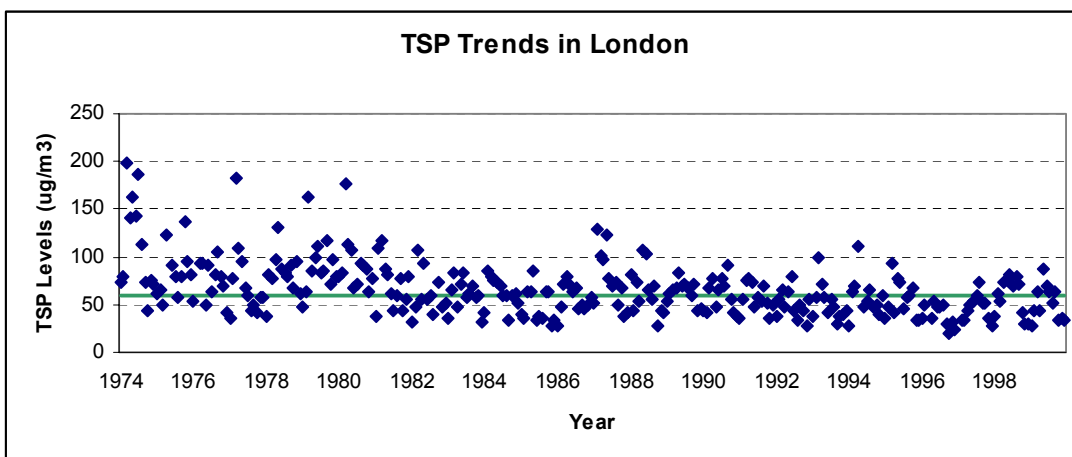
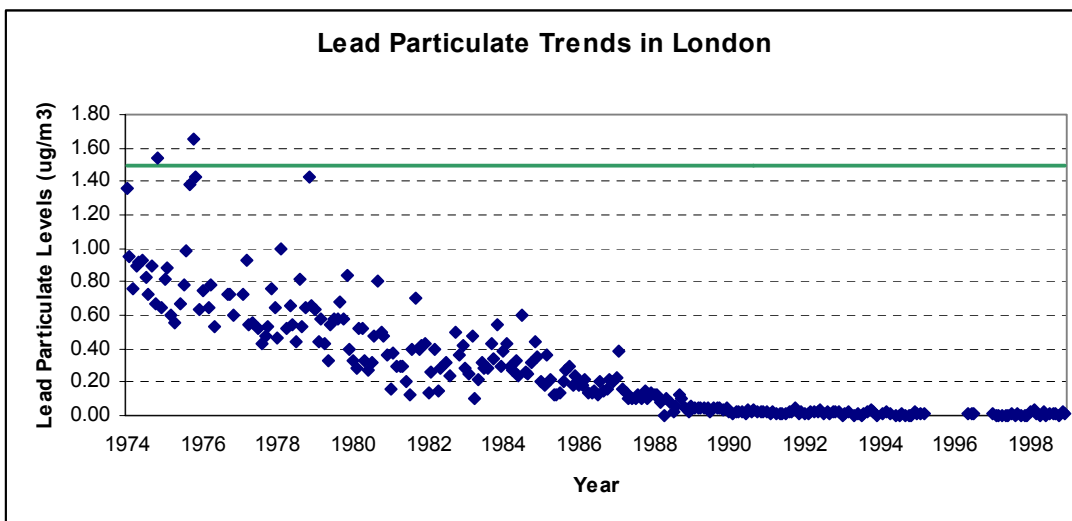
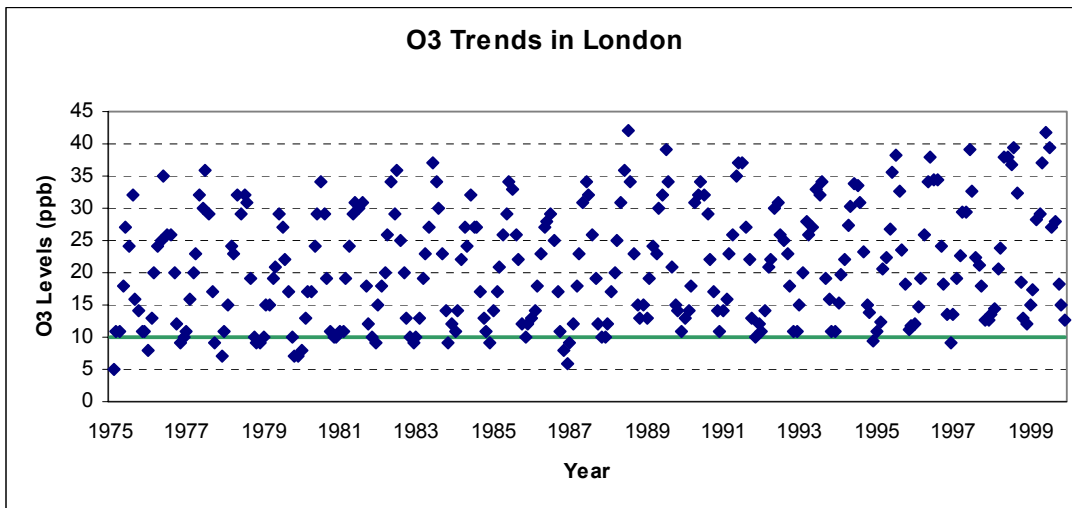
Windsor



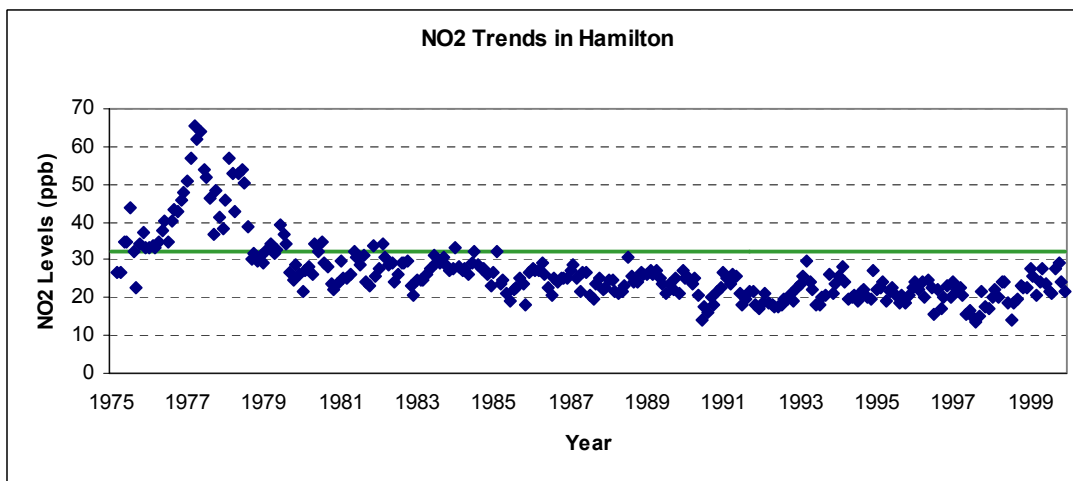
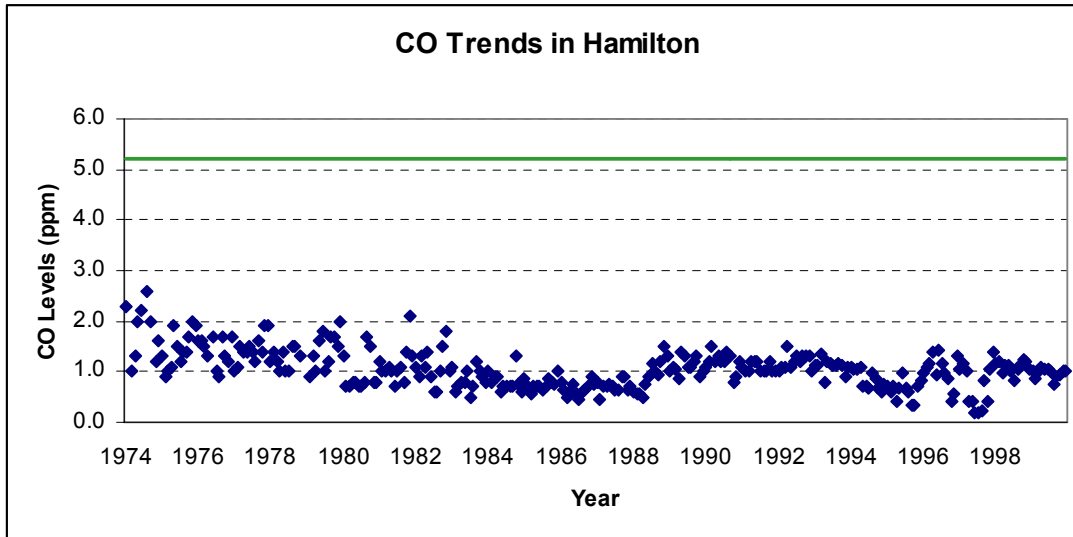


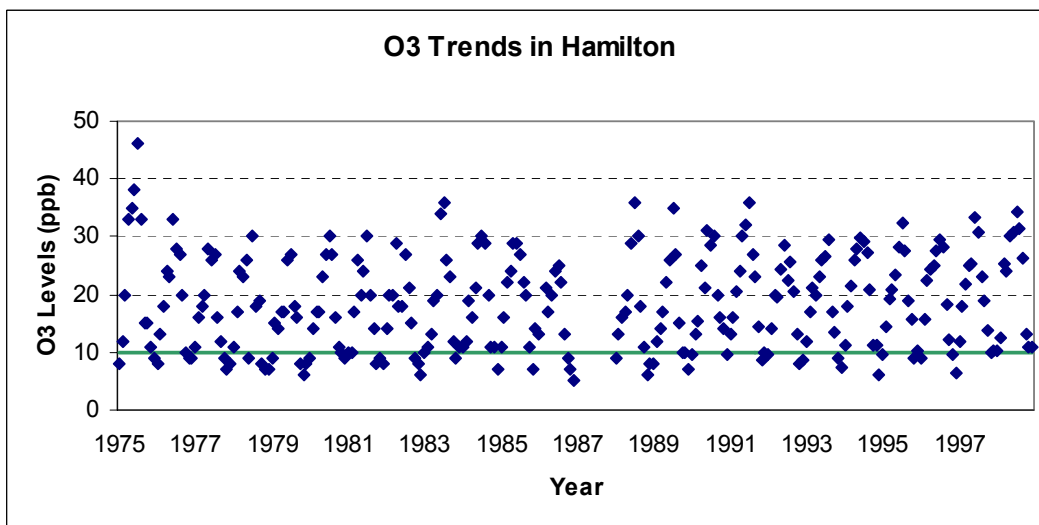
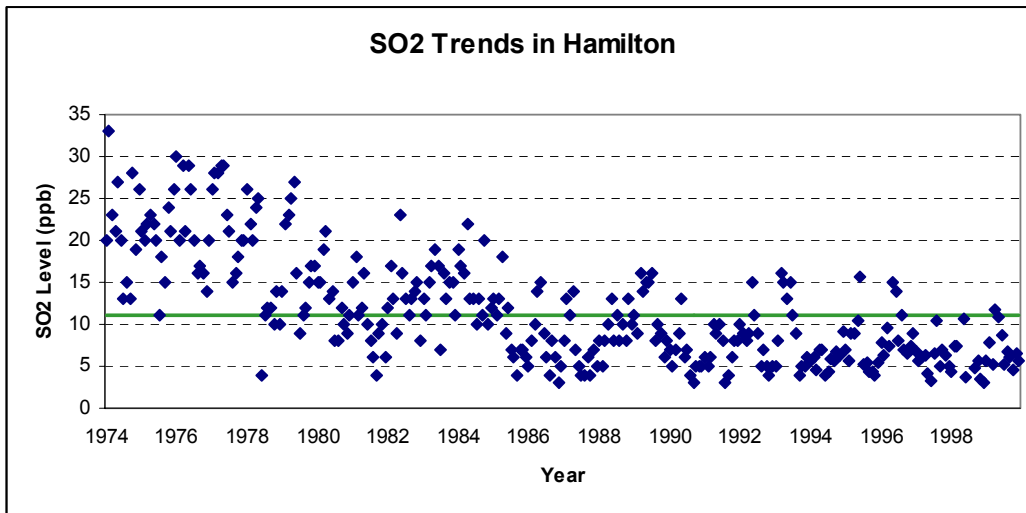
London

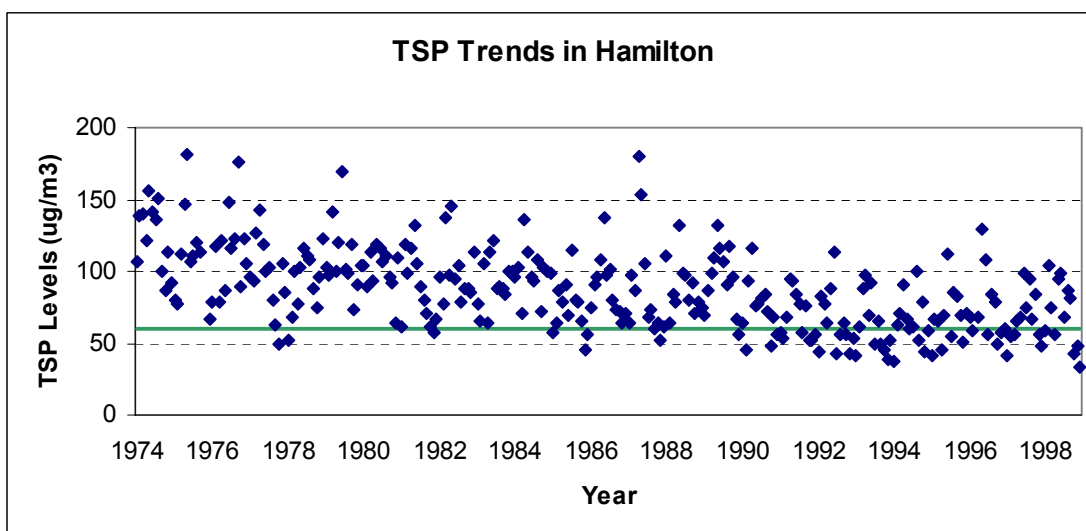
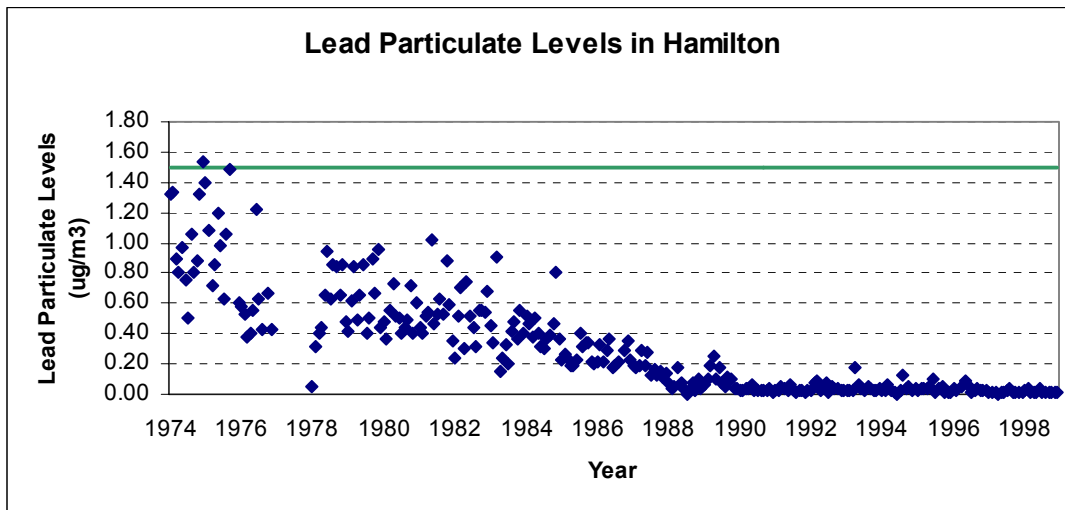




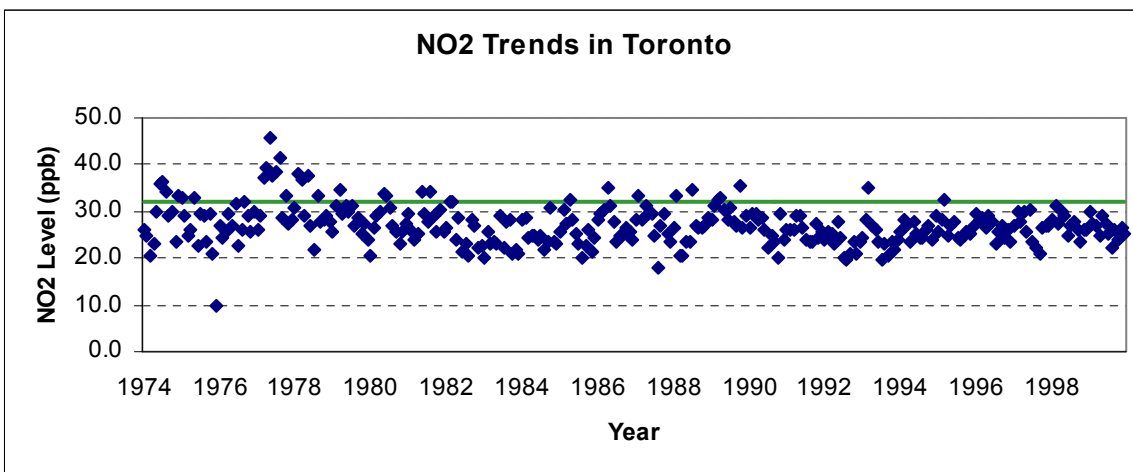
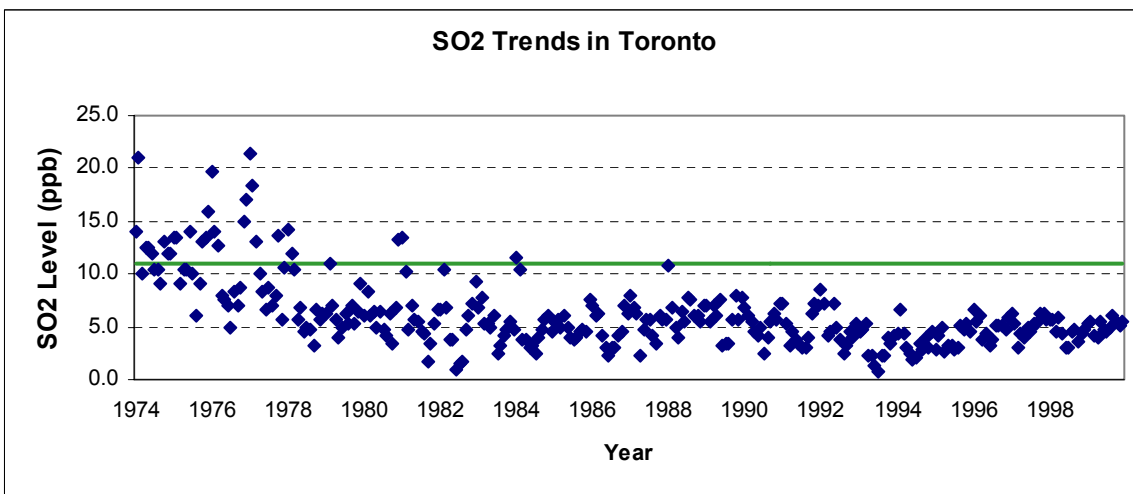
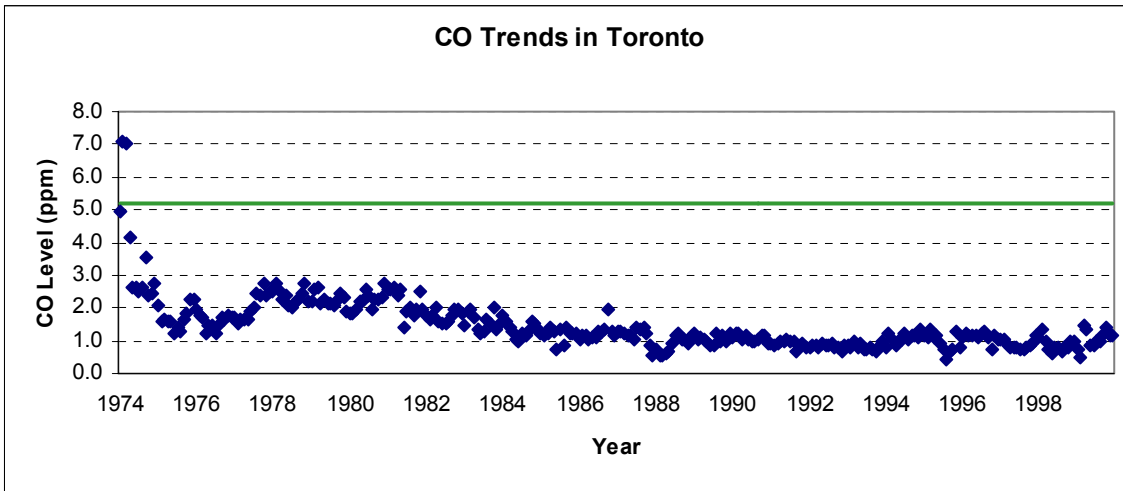
Hamilton

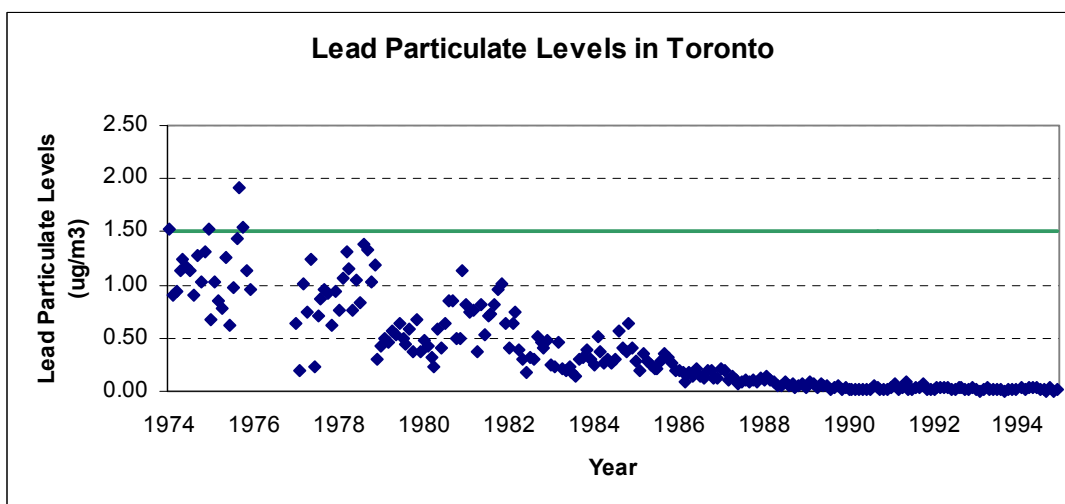
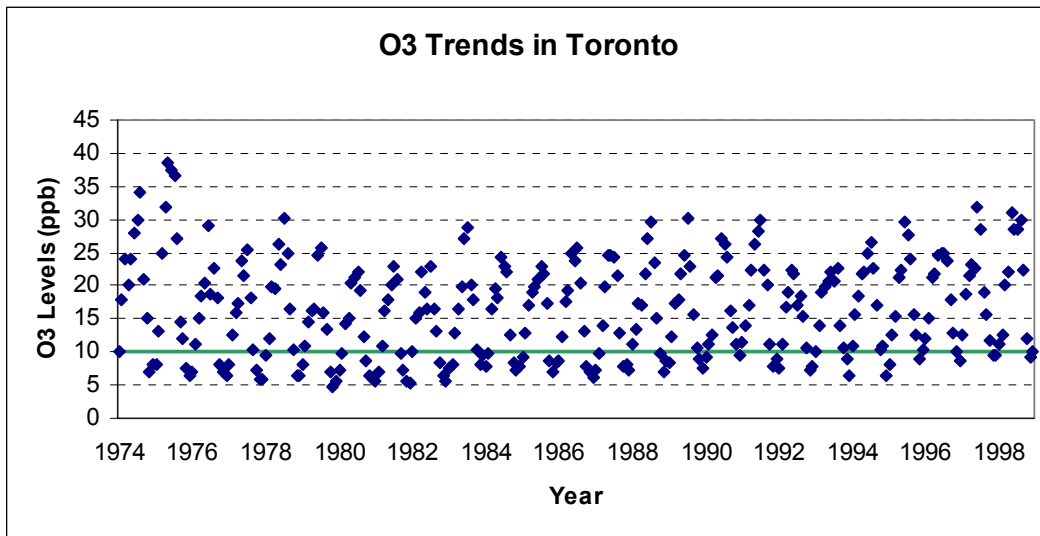


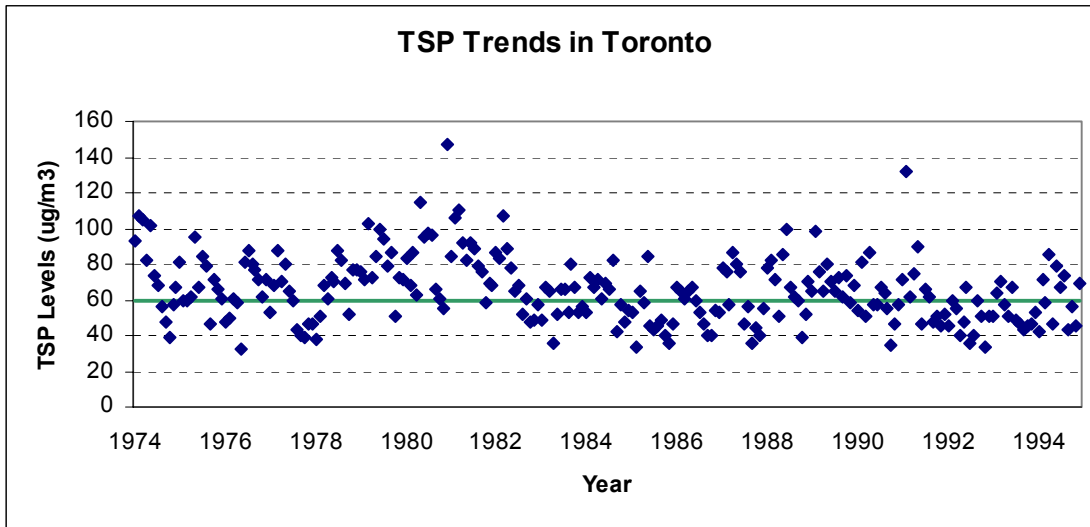




Toronto







Ottawa

