DOES AIR POLLUTION CAUSE RESPIRATORY ILLNESS? A NEW LOOK AT CANADIAN CITIES

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ABSTRACT

It is routinely asserted that urban air pollution is a major cause of acute respiratory conditions, leading to thousands of hospitalizations each year. The claim is based on inferences from partial correlations between ambient air pollution levels and hospitalization rates. Yet questions persist about the statistical robustness of the epidemiological findings, and controlled experiments have not confirmed the statistical findings. In this paper we present and analyze a new monthly data base showing concentrations of five major air contaminants in 11 large Canadian cities from 1974 to 1994, matched with monthly hospital admission rates by age group for all lung diagnostic categories; as well as a comprehensive set of socioeconomic and meteorological covariates. We compare two estimation approaches: model selection and Bayesian model averaging. Almost all of our estimates of the health effects of air pollution are insignificant. Two pollutant types have significantly negative coefficients, indicating, if interpreted in the standard way, that these pollutants are actually beneficial for health. We do not claim this, but we conclude that the perceived statistical relationship between air pollution and health is not robust.

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DOES AIR POLLUTION CAUSE RESPIRATORY ILLNESS? A NEW LOOK AT CANADIAN CITIES, 1974-1994

1. Introduction

Many studies—numbering in the hundreds—have been published asserting a significant connection between ambient air pollution levels and health. Amidst considerable variation in methodology, time and place of study, types of pollutants and measures of health outcomes, a view has emerged that air pollution exerts a small but significant effect on health. Dependent variables are, typically, modified counts of daily mortality, daily hospital admissions for respiratory and/or cardiac disease, or emergency room visits. Explanatory variables typically include one or more pollutants, meteorological variables and other variables intended to capture fluctuations in health outcomes which are unrelated to pollutants. Regression methods (or similar statistical methods) are used to measure the effect of pollution on health, after controlling for the other confounding variables. Intuitively, these methods provide an estimated baseline "expected" level of hospital admissions (or deaths), leaving behind "excess" admissions. The methods investigate whether these excess admissions are related to pollution levels.

As we shall elaborate on below, the enormous number of potential confounding variables implies that a huge number of models could be used to estimate expected admissions. To use some statistical jargon, model uncertainty is potentially an extremely important issue in this literature. In this paper, we investigate the two major competing ways of dealing with model uncertainty: model selection (i.e. selecting a single model) and model averaging. In previous work Koop and Tole (2004) argued that there were many theoretical arguments in favour of model averaging and, in an empirical exercise using daily time series data for Toronto, found that the use of model averaging led to a greater degree of uncertainty about the magnitude of the health effects of air pollution. Of particular importance was the finding that the hypothesis "air pollution has no effect on mortality" was not an implausible one.

The present paper investigates both model averaging and selection in a new and extensive data set involving many Canadian cities. We argue that results derived using a variety of different statistical methodologies and specifications are more convincing than those derived from a single methodology and specification. In this spirit, we note that we can present a model

specification and a data sub-period in which pollution apparently yields a significant negative effect on health. But we can also show that this result is not robust. Use of a long panel that stretches back into an earlier interval with generally higher and more varied air pollution levels, and formal treatment of model uncertainty, undermines the statistical evidence of negative health effects of pollution. In most cases, the health effects associated with individual pollutants are not significant. In the few cases where we do find significant health effects (e.g. TSP and O3), they are usually associated with negative coefficients (indicating, counter-intuitively, that these pollutants are actually beneficial for health). In the last part of the paper, we provide some explanation for these findings but emphasize our overall conclusion that (at least with this data set) there is no evidence of negative health effects of current levels of air pollution in Canadian cities.

2. Literature Review

With very few exceptions [e.g. Clyde (2000), Clyde and DeSimone-Sasinowska (1997) and Koop and Tole (2004)], the existing literature on air pollution health effects have used model selection methods. Some major recent reviews, such as Stieb (2002) and Basrur (2000), conclude that air pollution has an impact on health. However the findings do not exhibit the degree of consistency across studies one would expect if the measured phenomenon were based on a consistent physiological response, nor do they identify the types of contaminants that can be associated with specific risks. Studies using premature mortality in North American cities as the dependent variable include Burnett et al. (1998), Goldberg et al. (2001a, b), Samet et al. (2000) and Domenici et al. (2002). Burnett et al. find significant mortality risks from Nitrogen Dioxide (NO_2), ground-level ozone (O_3), Sulphur Dioxide (SO_2) and Carbon Monoxide (CO) in a panel of 11 cities. Goldberg et al. (2001 a) find effects in Montreal for the Coefficient of Haze (COH) and SO_2, but not fine or ultra-fine Particulate Matter[1] (PM_{10} or $PM_{2.5}$). Samet et al. studied 20 US cities and find PM_{10} and O_3 significant, but find CO, SO_2 and NO_2 insignificant. Dominici et al. estimated dose-response curves relating PM_{10} exposure and mortality risk in 88 US cities. While the nationally-pooled results suggest a small positive effect, the relative risk coefficients suggest that in 20 of the 88 cities, the effect is negative: increased particulate pollution is associated with *reduced* mortality risk.

The results for risk of admission to hospital are likewise conflicted. Bates (1983) studied data for 79 hospitals in Southern Ontario over the 1974—1978 interval and found summertime SO_2 and O_3 levels significant, but wintertime O_3 levels correlate to lower admission rates. Burnett et al. (1997a) used data for 16 Canadian cities from 1981 to 1991 and found a significant role for summertime O_3, but not in Montreal or Vancouver, two of the three largest cities. They also found no role for NO_2. Burnett et al. (1997b) found summertime NO_2 to be a significant risk factor in Toronto, while sulphates (SO_4) and airborne acidity (H^+) are not. Thurston (1994), in contrast, examined Toronto admissions data for 1986 and 1988 and concluded SO_4 and H^+ are significant, as is O_3, but not PM_{10}. Indeed the absence of effects for TSP and/or PM_{10} is commonly found in admissions data studies [Thurston (1994), Burnett et al. (1997b, 2001)] yet these pollutants are often cited as significant risk factors for mortality.

One of the reasons for this profusion of apparently contradictory results is the lack of a consistent approach to regression modeling. Published studies make use of many different specifications, including logs, lags, stepwise and pairwise introduction of variables, as well as numerous ad hoc specifications of the trend component used to convert observed health effects to an "unexplained" or "excess" residual. The necessity of addressing methodological issues has been raised in two recent re-analyses of data from Birmingham, Alabama. The seminal work of Schwartz (1993 and 1994) concluded a link exists between particulate matter and both admissions and excess mortality in this data set. Smith et al. (2000) replicated these results on a new version of the Birmingham data, but then showed they are not robust to minor respecification of the regression model and inclusion of lagged exposure levels. Clyde (2000) also reanalyzed Birmingham data using Bayesian Model Averaging (BMA) and found the data supported both a lower central relative risk estimate and wider posterior confidence intervals (implying insignificance)[2] than did the point estimators derived in Schwartz (1993, 1994). And as discussed above, Koop and Tole (2004) analyzed daily Toronto data over the 1992 to 1997 interval using BMA, and were unable to find a significant health effect for several different pollutants.

Another commonly-cited problem in reconciling earlier results is the possible role of missing confounders, i.e. socioeconomic covariates that may be excluded due to unavailability. In a recent study of pollution and infant mortality in California [Currie and Neidell (2005)], statistically significant effects between pollution and short gestation periods are observed, but

when fixed effects due to community socioeconomic characteristics are controlled for, the health effects of pollution 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, though a small but significant relative risk (0.014%) between increased carbon monoxide and infant mortality is found even with the socioeconomic covariates.

Laboratory-based toxicological studies have not provided uniform support for time series epistemology, adding to the questions of their robustness. High ozone levels have been shown to 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.

A recent Health Canada Science Assessment[3] that provided background material for the development of new Canadian air quality guidelines concluded that:

> 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.

An updated assessment from Health Canada in 2004[4] added only limited support for the small health effects estimated epidemiological studies, but also reported on a significant error discovered in a widely-used statistical algorithm, which added an upward bias to many of those published risk estimates. It restated the problem that epidemiological findings are not well-supported by experimental results but did not propose a resolution.

The study herein attempts to improve on previous statistical analyses in several ways. First, by assembling data that span 1974 to 1994[5] we are examining the longest pollution-health time series ever analyzed, at least that we are aware of. By comparison, the large study by Domenici et al. (2002) covered 88 cities, but the time span was only from 1987 to 1994. A database extending longer than one decade is important because in many Canadian and American cities there were large reductions in air pollution levels in the 1970s (see Figure 1). Studies that look at relatively short panels in recent years may have too little variance in pollution levels to permit clear identification of significant effects. If the small-but-significant effects found in short-panel studies are truly present in the data, they should emerge more strongly as we extend the data back in time into an interval with both higher average levels and larger annual and between-city variance (see Figure 2). Alternately, if there is a chance the existing results are overly sensitive to model selection bias and insufficient resolution in the data, then use of long time series back to the early 1970s will help identify this.

Second, the length of the time series allows us to control for a group of socioeconomic covariates, including income, local GDP, smoking rates and demographic changes. Third, by employing Bayesian Model Averaging techniques, we are able to test an exhaustive range of regression equation specifications, providing well-defined posterior parameter distributions rather than point estimates that are contingent on a specific regression formulation. Fourth, by considering separate age groupings we are able to examine whether air pollution effects are pronounced among certain groups thought to be especially at risk, namely young children and the elderly.

The price of these gains is that we are using monthly data rather than daily data, as is customary in this literature. Each row in our data set represents an average for that month, rather than for a 24 hour period. If there are aspects of the pollution-health nexus that are only visible when analyzing daily data then we run the risk of missing them. However, we believe the use of

monthly data is not too large a price to pay. Daily air pollution series are autocorrelated, so episodes of acutely higher air contamination tend to last more than a day, and can easily span a week or more. If the episode is acute enough to send people to the hospital, that will push up the monthly, as well as daily, hospital admission counts. Also, the monthly pollution data over the span we are examining exhibit substantial variability among cities and over time. If the health effects are so small that averaging admissions up to monthly frequency removes any visible correlation with average pollution, then the effects are too small to expect to be able to accurately measure anyway. Or, put another way, if there really is a daily effect in the data, it should be preserved in monthly data, given the length of time series we are able to analyze herein.

Although most of the studies of the health effects of air pollution do use daily time series data, it is worth noting that there are quite a few that use data at lower frequencies. In addition to the Currie and Niedell (2004) paper mentioned above, a couple other representative examples include Chay and Greenstone (2003) and Pope (1991).

3. Statistical Methods

All of the statistical methods used in this paper are regression based in the sense that we have a dependent variable which depends on explanatory variables. However, an important issue arises in studies of the health effects of air pollution since there are so many potential explanatory variables (e.g. pollutants and weather variables plus lags, dummy variables for month and city and, in our case various economic and social variables). When confronted with this situation, there are two main approaches taken in the literature. First, one can use model selection or hypothesis testing procedures to select a single set of explanatory variables and then present estimates based on this set. Second, one can average across various plausible models [e.g. Clyde (2000), Clyde and DeSimone-Sasinowska (1997), Koop and Tole (2004)]. This is not the place to reproduce the model averaging/model selection debate. Model selection is the more familiar approach and so we will not discuss it in detail. Model averaging is perhaps less familiar and, hence, it is worthwhile to digress briefly to explain the motivation for this approach.

The use of model averaging is partly motivated by the well-known problems associated with the presentation of results from a single model selected on the basis of a sequence of hypothesis tests. Most econometrics textbooks will provide a discussion of the issues associated with so-called pre-test estimators [e.g. Poirier (1995), pp. 519-523, Draper (1995) and Hodges (1987) are also useful references]. Here we provide a brief intuitive explanation. Note first that each time a hypothesis test is carried out, the possibility exists that a mistake will be made (i.e. the researcher will reject the better model for a not so good one). This possibility multiplies sequentially with each test done. So, for instance, a claim that a regression t-statistic of 2.0 means that a hypothesis is rejected at the 5% level of significance is spurious and, potentially vastly misleading, if the regression is selected on the basis of previous hypothesis tests. Second, even if a sequential hypothesis testing procedure does lead to the selection of the best model, standard decision theory implies that it is rarely desirable to present results for this model while ignoring all evidence from the not quite so good model(s). Generally, this is reflected in the common empirical wisdom that if one mines the data long enough one is bound to find something; however, one should not put too much trust in the finding.

Model averaging surmounts these problems by including information from every potential model. Results are a weighted average of results from every model where the weights are proportional to the support each model gets from the data. It is easier to implement these ideas in a Bayesian framework since it treats models (and parameters) as random variables. Suppose the researcher is entertaining R possible models, denoted by $M_1,..,M_R$ to learn about a parameter of interest, θ (e.g. the effect of a pollutant on health). If the models and parameters are treated as random variables then the posterior model probability, $p(M_r \mid Data)$, is the probability that the r^{th} model is correct, given the data. The logic of conditional probability tells us that this is a sensible measure of the evidence in favor of M_r. Similarly, $p(\theta \mid Data)$ should be used to summarize all the data evidence about θ. As described in the Technical Appendix, it is straightforward to calculate $p(M_r \mid Data)$ for the models considered in this paper. It is also straightforward to calculate a point estimate of θ, $E(\theta \mid Data, M_r)$, in every model. According to the rules of conditional expectation, it follows that:

$$E(\theta \mid Data) = \sum_{r=1}^{R} p(M_r \mid Data) E(\theta \mid Data, M_r).$$

In words, the overall point estimate of θ is the weighted average of the point estimates in every model. The weights in the weighted average are the posterior model probabilities, $p(M_r|Data)$ for r=1,..,R. This same logic applies to functions of θ. For instance, since

$$\text{var}(\theta \mid Data) = E\left(\theta^2 \mid Data\right) - \left[E(\theta \mid Data)\right]^2$$

we can use:

$$E\left(\theta^2 \mid Data\right) = \sum_{r=1}^{R} p(M_r \mid Data) E\left(\theta^2 \mid Data, M_r\right)$$

to help us calculate the posterior variance of θ. It can then be used to quantify uncertainty about θ. Precise formulae are provided in the Appendix. By way of intuition, we note that $E(\theta \mid Data, M_r)$ is similar to an OLS estimate and $p(M_r \mid Data)$ shares similarities with information criteria such as the Schwarz criteria or Akaike information criteria.

In this paper, we define our set of models by whether each includes or omits a potential explanatory variable. If K is the number of potential explanatory variables, this means we have 2^K models. We work with setups where K is roughly 40 (depending on the precise choice of potential explanatory variables) and, thus, the number of models we work with is huge indeed. Accordingly, we use an efficient algorithm referred to as Markov Chain Monte Carlo Model Composition (MC^3) to surmount the computational difficulties caused by the enormous model set. Details are given in the Technical Appendix. Suffice it to note here that we implement MC^3 in a standard way, drawing on the original paper of Madigan and York (1995) as implemented in Fernadez, Ley and Steel (2001). Chapter 11 of Koop (2003) provides an expository introduction.

In our empirical analysis, we present both Bayesian model averaging and model selection results. The latter involves selecting the single model with the highest value for $p(M_r \mid Data)$. This is analogous to using an information criteria to select a single model.

4. Empirical Results

In this section, we present results using data from 11 Canadian cities from January 1974 through March 1994. For the three biggest cities (Montreal, Toronto and Vancouver), we have separate data on inner city and suburbs and, accordingly, have data for 14 different urban areas. In all of our regression exercises, the dependent variable is a health outcome relating to a respiratory illness. The potential explanatory variables include one or more pollutants and some

weather, economic and social and city and monthly dummy variables. Given our focus on the potential medium term health effects of air pollution, we include a one month lag of the air pollutants and weather variables.

Our pollutants are CO, TSP, Sulphur Dioxide SO_2, Nitrogen Oxides (NOx) and O_3. We include the mean, minimum and maximum monthly temperature labeled Temp(mean), Temp(min) and Temp(max), respectively. We also have data on barometric pressure and wind speed (Press and Wind speed). We also include earnings, GDP and smoking, and include monthly and city dummy variables. Precise details of our data are provided in the appendix.

We have carried out a wide variety of statistical exercises and summarize them below. These differ in the choice of dependent variable and pollutants. We use two different dependent variables: hospital admissions in all respiratory categories and patient days (i.e. days spent in hospital) in all respiratory categories.[6] With regards to explanatory variables, we believe that any statistical analysis should begin with a wide list of potential explanatory variables (and then average over the models defined by the inclusion/exclusion of each potential explanatory variable in a BMA exercise or eliminate some by using statistical testing methods in a model selection exercise). The unsatisfactory alternative is to use some subjective procedure to exclude possible explanatory variables before looking at the data. For this reason, we prefer to include all of our pollutants (and one lag) as potential explanatory variables. After all, all of them may have an effect on hospital admissions. However, most papers in this literature use a single pollutant as an explanatory variable.[7] Accordingly, we also present empirical results for specifications that include only one pollutant at a time.

In summary, the results below are based on 24 different statistical exercises. That is, for each of two dependent variables (admissions and patient days) we consider six different sets of potential explanatory variables (one with all five pollutants plus the five pollutants each being included individually) and present two types of results (Bayesian model averaging and model selection).

It is worthwhile to digress briefly to discuss some of the properties of our data and econometric methods. With this data set, our regression methods enable us to draw on variations in our explanatory and dependent variables, both over time and across cities. Most of the intertemporal variation comes from our pollution and meteorological variables (and the monthly dummies). The economic and social variables exhibit some variation over time, but the much

larger source of variation is across cities. We find that even with the inclusion of these variables, we cannot adequately control for empirically relevant cross-city differences in the health-air pollution relationship. For this reason, we include city dummies in our specification. Note that the inclusion of city dummies will also account for demographic differences (e.g. population size or composition) between cities. It is also important to note that, to aid in interpretation, all of our dependent and explanatory variables (except for the intercept and dummy variables) have been standardized by subtracting their sample mean and dividing by their standard deviation. Thus, regression coefficients (of the sort presented in Tables 3 through 6) are interpreted as measuring the effect on the dependent variable of a one standard deviation change in the explanatory variable (holding other explanatory variables constant). So, for instance, if β is the coefficient on ozone, then we can say that "if ozone levels increase by one standard deviation, then hospital admissions will tend to increase by β standard deviations, holding other explanatory variables constant". Given our large number of potential variables and the wide variety of units in which they are measured, it is useful to adopt this standardization that ensures consistency of interpretation but does not affect the empirical results.

Table 1 presents a brief summary of the most important of our empirical results: those relating to the health effect of air pollution. This table presents the point estimate (i.e. the posterior mean) and measure of uncertainty associated with that estimate (i.e. the posterior standard deviation) for the cumulative effect of each pollutant on the health outcome. This cumulative affect is the standard multiplier (i.e. for any pollutant, we sum the coefficients of the current value and the lag). In the following discussion, we will use the term "significant" in an informal sense to denote that the point estimate is two standard deviations from zero.

Table 1 Here

When using patient days as the dependent variable, none of the pollutants has a significant effect on health. This holds regardless of whether we use BMA or simply select the single model that bests fit the data. When using hospital admissions as the dependent variable, our empirical findings are even more discouraging for a researcher hoping to find that air pollution in unhealthy. We are finding that health effects are either insignificant or negative (i.e.

actually imply pollutants are beneficial for health). This statement also holds regardless of whether we use BMA or model selection.

One particularly robust result is that O_3 has a negative and significant coefficient (although only when admissions are used as the dependent variable). Even after controlling for a host of plausible meteorological, social, economic and other explanatory variables, our results indicate ozone is negatively associated with hospital admissions and patient days. We stress that the significantly negative coefficient on O_3 occurs even when it is the only pollutant included as a potential explanatory variable. Thus, it is not the case that this finding is due to O_3 's interactions with other pollutants.

We leave the reader to interpret these findings in whatever manner she wants. It is possible (though counter-intuitive) that our regressions are indeed uncovering beneficial health effects from O_3 and TSP. What the physiological effect might be we are unable to say. It might be that during smog episodes, which typically involve elevated O_3 and TSP levels, individuals facing a health risk protect themselves by staying indoors, thus facing reduced actual (indoor) exposure compared to days with moderate outdoor pollution. It is also possible that these results are due to the omission of important explanatory variables. Similarly, with our large sample size, it is possible that even with a careful BMA exercise, these findings are spurious and merely reflect coincidental relationships driven by random error in the data. In light of these considerations we hesitate to draw strong conclusions from our results other than to say that there is no evidence that air pollution has a detrimental effect on human health in the Canadian cities we consider.

Koop and Tole (2004), in a study involving daily time series data for Toronto, found that Bayesian model averaging and selection results were, in some cases, quite different. We do not find such differences across methodologies here. In fact, BMA and model selection results are telling the same story in respect to the significance of health effects. However, it is worth noting that, with BMA, the posterior standard deviations tend to be somewhat larger. This is sensible. BMA provides us with a proper treatment of model uncertainty and the uncertainty we have regarding which model is the correct one spills over into the posterior for the coefficients. In contrast, model selection ignores model uncertainty by pretending that the selected model is the true one, thus yielding over-precise estimates. We would argue that BMA is the preferred

statistical methodology and, thus, that any researcher should report the BMA when advising policymakers.

Tables 3 through 6 are presented in an appendix. These contain more detailed results, providing point estimates and standard deviations for every coefficient for all of our 24 different statistical exercises. Results for all these different explanatory variables will have varying interest for readers. It is sufficient for our purposes to note that, as before, BMA and model selection approaches yield qualitatively similar results, but that BMA yields larger standard deviations. Although there are some differences across dependent variables, for the vast majority of coefficients a similar pattern holds across specifications. For instance, smoking is consistently positively related with respiratory problems. Many of the city dummies are consistently significant, indicating that including key variables that reflect city characteristics is insufficient to control for differences across cities.

As another test of the robustness of our results, we have re-done all of the empirical work underlying Table 1 using only data from January 1985 through March 1994. The motivation for this is that pollution levels tend to be much lower in the latter half of our data set. Hence, it is possible that the air pollution-health relationship differed between the first and last halves of our sample.

The key results using this sub-sample of the data are given in Table 2. Results are slightly different, but do not differ in a manner that suggests that air pollution is deleterious for health in either part of our sample. As before, when using patient days as the dependent variable we find little evidence that pollutants have any significant health effects at all. When we include all five pollutants as potential explanatory variables, we find nothing to be significant. When we include only O_3, we find the same statistically significant negative coefficient discussed previously (although only when we use model selection). When we include only CO, we find our only positive and significant coefficient on any of our pollutants in any of our statistical exercises. Since this would mean CO has a deleterious effect on health, this is potentially an important finding. However, it would be misleading, in the context of the rest of our findings, to report results based on the post-1985 subsample, using model selection and including only one air pollutant, knowing that the effect is only found with model selection (not BMA), it does not hold up when we include other pollutants as explanatory variables and it is not found in the full sample. Consequently, we hesitate to place much weight on this finding, and we caution against

placing too much weight on any findings that rely on model selection, short (or overly recent) panels, or those that are based on inclusion of pollutants one-at-a-time in a regression model. This would potentially call into question a large fraction of the epidemiological findings on air pollution and health effects.

When we use admissions as the dependent variable, the negative coefficient on O_3 which we found with the full sample still comes through using the sub-sample. However, there is less evidence that TSP is a significant explanatory variable and, interestingly, we now find NOx to have a negative and significant coefficient (i.e. counter-intuitively, this suggests NOx is actually good for health). However, as with all our results, not wanting to oversell them, we note only that, with this sub-sample, there is no evidence that air pollution has a detrimental effect on human health in the Canadian cities we consider.

Table 2 Here

5. Conclusions

In this paper, we have investigated the health effects of air pollution using a new, extensive, multi-city Canadian data set measuring hospital admissions for respiratory ailments, a wide variety of pollutant levels, and other variables, including meteorological, economic and social covariates. We use regression-based methods involving two dependent variables (hospital admissions and days spent in hospital) and various sets of potential explanatory variables containing different pollutants.

An important issue that our study addresses is model uncertainty. With so many explanatory variables potentially involved in the air pollution-health relationship, the set of possible regression models is enormous—numbering in the trillions. Thus, there are many ways that the standard model selection strategy used by health researchers could go wrong. For this reason, we investigate both model selection and model averaging strategies.

Although our model averaging strategy indicates a larger degree of uncertainty over the magnitude of our regression coefficients, overall our empirical findings are telling a consistent story: we can find no evidence that air pollution has a detrimental effect on either excess hospital admissions or time spent in hospital for the Canadian cities comprising our data set.

6. Endnotes

1. $PM_{2.5}$ denotes particulate matter smaller than 2.5 microns; PM_{10} denotes PM smaller than 10 microns.
2. We are using formally incorrect terminology here. Bayesians produce posterior credible intervals (not confidence intervals) and do not often use the term "insignificant". However, we adopt this more familiar classical terminology as being informally adequate and clearer to a non-Bayesian audience. We informally use the term "insignificant" to mean "the credible interval contains zero" or "the point estimate is less than two posterior standard deviations from zero".
3. See Health Canada (1997) http://www.hc-sc.gc.ca/hecs-sesc/air_quality/publications/particulate_matter_science_assessment/addendum/impacts.htm#7 .
4. Available at http://www.ccme.ca/assets/pdf/prrvw_pm_fine_rvsd_es_e.pdf.
5. We were not able to use post-1994 admissions data in this study: see Data Appendix for discussion.
6. In future work we plan on investigating dependent variables involving the various diagnostic sub-categories described in the Appendix. We also plan on using different age groups. Preliminary investigations indicate that results for individual age groups are qualitatively similar to those presented here.
7. One justification for this is to avoid problems associated with multicollinearity. We note, however, that that our five pollutants are not highly correlated with one another. The highest correlation (between CO and NOx) is only 0.36 and some of the correlations between pollutants are actually negative.

Table 1: Point Estimate of the Health Effect of Each Pollutant (posterior standard deviation in parentheses)				
	Dependent Variable is Admissions		Dependent Variable is Patient Days	
	BMA	Model Selection	BMA	Model Selection
All Pollutants Included as Explanatory Variables				
CO	-0.001 (0.004)	----	0.002 (0.051)	----
TSP	-0.102** (0.014)	-0.102** (0.013)	-0.011 (0.018)	----
SO2	0.000 (0.002)	----	0.000 (0.003)	----
NOX	-0.000 (0.003)	----	0.000 (0.004)	----
O3	-0.051** (0.020)	-0.048** (0.013)	-0.001 (0.006)	----
Pollutants Included One-at-a-time in Separate BMA Exercises				
CO	-0.019* (0.015)	-0.029* (0.010)	0.000 (0.002)	----
TSP	-0.082** (0.014)	-0.081** (0.011)	0.000 (0.003)	----
SO2	0.000 (0.002)	----	0.001 (0.004)	----
NOX	-0.004 (0.010)	----	0.000 (0.003)	----
O3	-0.051** (0.018)	-0.053** (0.012)	-0.005 (0.012)	----
Note: Entries of "----" for the Model selection case indicate that the corresponding pollutant was not included in the model selected as best. Point estimates denoted with **/* are two/one standard deviations from zero.				

Table 2: Point Estimate of the Health Effect of Each Pollutant Using Data from 1985 through 1994

(posterior standard deviation in parentheses)

	Dependent Variable is Admissions		Dependent Variable is Patient Days	
	BMA	Model Selection	BMA	Model Selection
All Pollutants Included as Explanatory Variables				
CO	-0.017 (0.020)	----	0.010 (0.019)	----
TSP	-0.006 (0.012)	----	0.000 (0.004)	----
SO2	0.001 (0.005)	----	0.002 (0.008)	----
NOX	-0.046** (0.019)	-.049 (0.012)	-0.017 (0.028)	----
O3	-0.083** (0.021)	-0.049** (0.012)	-0.013 (0.022)	----
Pollutants Included One-at-a-time in Separate BMA Exercises				
CO	-0.004 (0.010)	----	0.010 (0.018)	0.043** (0.014)
TSP	-0.021* (0.019)	-0.032** (0.011)	-0.001 (0.005)	----
SO2	0.001 (0.005)	----	0.001 (0.006)	----
NOX	-0.057** (0.015)	-0.069** (0.011)	-0.012 (0.022)	----
O3	-0.086** (0.018)	-0.080** (0.012)	-0.022 (0.025)	-0.044** (0.015)

Note: Entries of "----" for the Model selection case indicate that the corresponding pollutant was not included in the model selected as best. Point estimates denoted with **/* are two/one standard deviations from zero.

7. References

Basrur, S. [2000]. Air Pollution Burden of Illness in Toronto. May 2000, Toronto Board of Public Health, mimeo.

Brown, J. [2004]. *Environmental Indicators*. Sixth Edition. Vancouver, British Columbia: The Fraser Institute.

Burnett, R.T., J.R. Brook, W.T. Yung, R.E. Dales and D. Krewski. [1997a]. Association Between Ozone and Hospitalization for Respiratory Diseases in 16 Canadian Cities. *Environmental Research* 72: 24-31.

Burnett, R.T., S. Cakmak, J.R. Brook and D. Krewski [1997b]. The role of Particulate Size and Chemistry in the Association Between Summertime Ambient Air Pollution and Hospitalization for Cardiorespiratory Diseases. *Environmental Health Perspectives* **105**: 614-620.

Burnett, R.T., S. Cakmak and J.R. Brook [1998]. The Effect of the Urban Ambient Air Pollution Mix on Daily Mortality Rates in 11 Canadian Cities. *Canadian Journal of Public Health* 89(3): 52-156.

Burnett, R.., Smith-Doiron, Stieb, D., Raizenne, M.E., Brook, J.R., Dales, R.E., Leech, J.A., Cakmak, S. and D. Krewski. [2001] Association between ozone and hospitalization for acute respiratory diseases in children less than 2 years of age. *American Journal of Epidemiology*. 153(5): 444-452.

Chay, K. and Greenstone, M. [2003]. The Impact of Air Pollution on Infant Mortality: Evidence from Geographic Variation in Pollution Shocks Induced by a Recession. *Quarterly Journal of Economics*.118: 1121-1167.

Clyde, M. [2000]. Model Uncertainty and Health Effect Studies for Particulate Matter. *Environmetrics* 11: 745-764.

Clyde, M. and DeSimone-Sasinowska, H. [1997]. Accounting for Model Uncertainty in Poisson Regression models: Particulate Matter and Mortality in Birmingham, Alabama. Institute of Statistics and Decisions Sciences, Duke University, Discussion Paper 97-06.

Currie, J. and M. Niedell [2005]. Air Pollution and Infant Health: What can We Learn from California's Recent Experiment? *Quarterly Journal of Economics* 120: 1003-1030.

Dominici, F., Daniels, M., Zeger, S.L., and J. M. Samet. [2002]. Air Pollution and Mortality: Estimating Regional and National Dose–Response Relationships. *Journal of the American Statistical Association* 97(457):1-12.

Draper, D. (1995). Assessment and propagation of model uncertainty (with discussion). *Journal of the Royal Statistical Society, Series B*, 56: 45-98

Fernandez, C., Ley, E. and M., Steel. [2001]. Benchmark Priors for Bayesian Model Averaging. *Journal of Econometrics* 100: 381-427.

Goldberg, M.S., Burnett, R.T., Bailar, J.C, Brook, J., Bonvalot, Y., Tamblyn, R., Singh, R. and M.F. Valois. [2001a]. The Association Between Daily Mortality and Ambient Air Particle Pollution in Montreal, Quebec 1. Non-Accidental Mortality. *Environmental Research* 86(1): 12-25.

Goldberg, M.S., Burnett, R.T., Bailar, J.C., Brook, J., Bonvalot, Y., Tamblyn, R., Singh, R., Valois, M-F. and R. Vincent. [2001b]. The Association Between Daily Mortality and Ambient Air Particle Pollution in Montreal, Quebec 2. Cause-Specific Mortality. *Environmental Research* 96(1): 26-36.

Green, L. and S. Armstrong [2003]. Particulate Matter in Ambient Air and Mortality:

Toxicologic Perspectives. *Regulatory Toxicology and Pharmacology* 38: 326-335.
Hodges, J. [1987]. Uncertainty, Policy Analysis and Statistics. *Statistical Science* 2: 259-291.
Hoeting, J., Madigan, D., Raftery, A. and Volinsky, C. [1999]. Bayesian Model Averaging: A Tutorial. S*tatistical Science* 14: 382-417.
Koop, G. [2003]. *Bayesian Econometrics*. New York: John Wiley and Sons.
Koop, G. and L. 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.
Madigan, D. and J. York. [1995]. Bayesian Graphical Models for Discrete Data. *International Statistical Review,* 63: 215-232.
Ontario Medical Association (OMA) [2000]. Illness Cost of Air Pollution. Prepared by DSS Management Consultants, June 2000.
Poirier, D. [1995]. *Intermediate Statistics and Econometrics: A Comparative Approach*. Cambridge: The MIT Press.
Pope, C.A. [1991]. Respiratory Hospital Admissions Associated with PM10 Pollution in Utah, Salt Lake, and Cache Valleys. *Archives of Environmental Health* March-April.
Samet, J.M., Dominici, F., Curriero, F.C., Coursac, I. and S.L. Zeger. [2000]. Fine Particulate Air Pollution and Mortality in 20 U.S. Cities, 1987-1994. *New England Journal of Medicine* 343(24): 1742-1749.
Schwartz J. [1993]. Air pollution and Daily Mortality in Birmingham Alabama. *American Journal of Epidemiology* 137:1136-1147.
Schwartz J. (1994) "Air pollution and hospital admissions for the elderly in Birmingham, Alabama." *American Journal of Epidemiology* 139:589-598.
Smith, R.L., Davis, J.M., Stacks, Speckman, P. and P. Styer [2000]. Regression Models for Air Pollution and Daily Mortality: Analysis of Data from Birmingham, Alabama. *Environmetrics* 11: 719–743.
Stieb, D.M., Judek, S. and R.T. Burnett [2002]. Meta-Analysis of Time-Series Studies of Air Pollution and Mortality: Effects of Gases and Particles and the Influence of Cause of Death, Age, and Season. *Journal of the Air & Waste Management Association* 52(4): 470-484.
Thurston, G.D., Ito, K., Hayes, C.G., Bates, D.V., and M. Lippmann. [1994]. Respiratory Hospital Admissions and Summertime Haze Air Pollution in Toronto, Ontario: Consideration of the Role of Acid Aerosols. *Environmental Research* 65: 271-290.

8. Data Appendix

Data is from January 1974 through March 1994 for the following urban areas: Calgary, Edmonton, Halifax, London, Montreal (inner city), Montreal (outer suburbs), Ottawa, Regina, Saskatoon, Toronto (inner city), Toronto (outer suburbs), Vancouver (inner city), Vancouver (outer suburbs) and Winnipeg, for the following variables.

Health Admissions:
All data were supplied by Richard Trudeau of the Health Statistics Division at Statistics Canada, and refer to the "most responsible diagnosis" at the time of admission. The six categories of admission were based on the ICD-9 codes as follows:

1 ICD-9 461.* (acute sinusitis)
2 ICD-9 465.* (acute upper respiratory infections of multiple or unspecified sites)
3 ICD-9 473.* (chronic sinusitis)
4 ICD-9 490 (bronchitis, not specified as acute or chronic), 491.* (chronic bronchitis), 492 (emphysema) and 494 (bronchiectasis)
5 ICD-9 493.* (asthma)
6 ICD-9 5190 (tracheostomy malfunction), 5194 (disorders of diaphragm) and 5199 (other unspecified chronic disease of respiratory system

A list of admitting hospitals in each city was compiled based on Statistics Canada listing, and the data extractions cover 1974-75 to 1993-94.

After 1994, Statistics Canada transferred the archiving of hospital admissions data to the Canadian Institute for Health Information (CIHI). We obtained admissions data for 1994 to 2003 from CIHI, including a 6-month overlap period. However the diagnostic coding was not identical in each system, so while we are able to match admissions records for certain individual disease codes, the totals across the above six categories do not match. In order to ensure we are using a consistent definition of disease categories we did not extend the admissions data past the end of the Statistics Canada archive, which was March 1994.

Meteorology
All data were supplied by Walter Dnes of the Meteorological Service of Canada. For each city we received elevation, average temperature, mean barometric pressure, mean windspeed, extreme maximum temperature and extreme minimum temperature.

Income
Monthly Real Average Earnings were estimated using the Statistics Canada series Average Weekly Earnings, All Employees, (SEPH), by Standard Industrial Classification, 1960 (SIC), monthly (Dollars) for the period 1961-1985; while the data for the period 1983-2000 was estimated using the Average Weekly Earnings of Employees, (SEPH), monthly (Dollars), obtained from CANSIM 2 Table # 2810021 and 2810002 respectively. These data were indexed to 2000 dollars using the monthly Consumer Price Index (CPI). The CPI for Toronto was used to estimate the values for London. The Monthly CANSIM 2 series #s by province and city from 1961-1985, 1983-2000 are: v76233, v78335, v76493, v78483, v76943, v79056, v78815, v79555,

v77638, v79814, v77772, v79866, v79882, v77888, v79909, v79958, v78082, v80028, v265027, v275763, v283107, v290329, v296195, v301891, v308359.

Gross Domestic Product
Annual real GDP is reported at the Provincial level in the Provincial Economic Accounts from Statistics Canada.

Smoking rates
This information was obtained from the Survey of Smoking Habits archive at the University of Guelph data Library for the years 1973, 1974, 1977, 1979, 1981, 1983 and 1986; from the Survey of smoking in Canada 1994, the National Alcohol and Drug Survey 1989, the Health Promotion Survey 1990, the Canadian Tobacco Use Monitoring Survey 1999, 2000, 2001, 2002, 2003 and 2004. The percentage of smokers in the population for the years 1985, 1989, 1991, 1994 and 1996 were estimated using the number of smokers in the population data from the Health Indicators-Health Statistics B2020 table and dividing each value by the corresponding total of Age group 15 and above in the population. These survey results are done on a provincial level basis. The observations for the years with no related survey were estimated by interpolation.

Pollutants
All data on Canadian urban air pollution was obtained from Environment Canada. Most of the data are available on-line at the National Air Pollution Surveillance System (NAPS) website: http://www.etc-cte.ec.gc.ca/NAPSData/Default.aspx. For some years, additional data were obtained from a compilation supplied by Environment Canada to Brown et al. (2004).

9. Technical Appendix

We implement Bayesian model averaging using the approach outlined in Fernandez, Ley and Steel (2001), using the MC^3 algorithm developed in Madigan and York (1995). The reader is referred to these papers (see also Hoeting et al, 1999) for details beyond those presented in this appendix.

We have data for t=1,..,T months and denote data on the dependent variable (a health outcome) by $y = (y_1,..,y_T)'$. All the potential explanatory variables (including lags) are stacked in a T×K matrix X. We have r=1,..,R models, denoted by M_r. These are all Normal linear regression models which differ in their explanatory variables,

$$y = \alpha \iota_T + X_r \beta_r + \varepsilon$$

where ι_T is a T×1 vector of ones, X_r is a T×k_r matrix containing some (or all) columns of X. The T-vector of errors, ε, is assumed to be $N(0_T, \sigma^2 I_T)$ where 0_T is a T-vector of zeros and I_T is the T×T identity matrix. Note that we are assuming all models contain an intercept.

The models are thus defined by their choice of explanatory variables (i.e. by the choice of X_r). The standard approach to Bayesian model averaging assumes different models are defined by the inclusion or exclusion of each variable. This leads to 2^K models. If K is at all large, the enormous number of potential models imposes commensurately enormous computational

demands. It is worth noting that these computational demands help motivate our choice of the Normal linear regression model.

We use a Normal-Gamma natural conjugate prior with hyperparameters chosen in the objective fashion described in Fernandez, Ley and Steel (2001). To be precise, for the error variance we use the standard noninformative prior:

$$p(\sigma) \propto \frac{1}{\sigma}.$$

We standardize all the explanatory variables by subtracting off their means and dividing by their standard deviations. Once this is done, it makes sense to use a flat prior for the intercept:

$$p(\alpha) \propto 1.$$

For the slope coefficients we assume a g-prior of the form:

$$\beta_r \sim N\left(0_{k_r}, \sigma^2\left[g_r X_r{}' X_r\right]^{-1}\right).$$

Following Fernandez, Ley and Steel (2001), who relate these choices to common information criteria, we choose

$$g_r = \begin{cases} \frac{1}{K^2} if T \le K^2 \\ \frac{1}{T} if T > K^2 \end{cases}.$$

The resulting posterior for β_r follows a multivariate t-distribution with mean:

$$E(\beta_r \mid Data, M_r) = \left[(1+g_r)X_r{}'' X_r\right]^{-1} X_r{}' y,$$

covariance matrix:

$$\text{var}(\beta_r \mid Data, M_r) = \frac{\bar{\nu}\bar{s}^2}{\bar{\nu}-2}\left[(1+g_r)X_r{}' X_r\right],$$

where $\bar{\nu} = T$ and

$$\bar{s}^2 = \frac{\frac{1}{g_r+1} y' P_{X_r} y + \frac{g_r}{g_r+1}(y-\bar{y}\iota_T)'(y-\bar{y}\iota_T)}{\bar{\nu}},$$

where

$$P_{X_r} = I_T - X_r\left(X_r{}' X_r\right)X_r{}'.$$

The posterior model probabilities are given by:

$$p(M_r \mid Data) = c\left(\frac{g_r}{g_r+1}\right)^{\frac{k_r}{2}}\left(\bar{\nu}\bar{s}^2\right)^{-\frac{T-1}{2}},$$

where c is a constant common to all models. If desired, the fact that $\sum_{r=1}^{R} p(M_r \mid Data) = 1$ can be used to evaluate c.

Our parameters of interest measure the cumulative effect of a pollutant on a health outcome and these are a linear function of the regression coefficients. Hence, the previous

equations are all that is required to carry out Bayesian model averaging or Bayesian model selection.

If the number of models, R, is relatively small, these equations can be evaluated for every possible model and Bayesian model averaging or selection can be implemented directly. In traditional applications of Bayesian model averaging, $R=2^K$ (i.e. every possible explanatory variable can either be included or excluded). For cases where K>20 direct implementation of Bayesian model averaging is computationally infeasible. Accordingly, we adopt the MC^3 algorithm described in Madigan and York (1995). This is a Metropolis algorithm which is very simple to implement. In particular, if the current model in the chain is M_s then a candidate model, M_j, which is randomly (with equal probability) selected from the set of models including M_s and all models containing one more or one less explanatory variable (i.e. the algorithm randomly either adds or subtract one column from X_s), is drawn. M_j is accepted with probability:

$$\min\left\{1, \frac{p(M_j \mid Data)}{p(M_s \mid Data)}\right\}.$$

To monitor convergence of the chain we calculate the probability of the ten most probable models drawn in two different ways. First, we calculate them analytically the equation above. Then we approximate this probability using output from the MC^3 algorithm. When these probabilities are the same to two decimal places, we deem convergence to have taken place. The number of draws required for the various models considered varied from 1,000,000 to 2,000,000.

10. Detailed Breakdown of Empirical Results Appendix

This Appendix provides empirical results for all coefficients in each of our 24 statistical exercises. We have two dependent variables, six choices of a set of potential explanatory variable and two statistical methodologies. Note that the sample size differs due to missing values; different pollutants have different numbers of missing values.

Table 3: Point Estimates of Each Coefficient Using BMA. Dependent Variable is Admissions (posterior standard deviations in parentheses)						
	Pollutants included as explanatory variables					
Pollutants	All	CO	TSP	SO2	NOX	O3
CO	0.000 (0.003)	-0.016 (0.016)	--	--	--	--
CO-lag	0.000 (0.003)	-0.003 (0.008)	--	--	--	--
TSP	-0.048 (0.015)	--	-0.043 (0.014)	--	--	--
TSP-lag	-0.054 (0.014)	--	-0.040 (0.015)	--	--	--
SO2	0.000 (0.002)	--	--	0.000 (0.002)	--	--
SO2-lag	0.000 (0.001)	--	--	0.000 (0.002)	--	--
NOX	0.000 (0.002)	--	--	--	-0.003 (0.009)	--
NOX-lag	0.000 (0.002)	--	--	--	-0.001 (0.005)	--
O3	-0.056 (0.023)	--	--	--	--	-0.064 (0.025)
O3-lag	0.005 (0.015)	--	--	--	--	0.013 (0.023)
Weather Variables						
Pressure	0.074 (0.148)	0.302 (0.127)	0.113 (0.149)	0.236 (0.164)	0.293 (0.088)	0.302 (0.164)
Press-Lag	0.400 (0.146)	0.402 (0.126)	0.453 (0.147)	0.388 (0.159)	0.368 (0.141)	0.348 (0.161)
Windspeed	0.000 (0.003)	0.000 (0.002)	0.000 (0.003)	0.000 (0.002)	0.000 (0.002)	0.000 (0.003)
Wind-lag	0.000 (0.002)	0.000 (0.002)	0.000 (0.003)	0.000 (0.002)	0.000 (0.002)	0.000 (0.002)
Temp(mean)	0.003 (0.018)	-0.004 (0.018)	0.000 (0.011)	-0.014 (0.040)	-0.004 (0.018)	-0.001 (0.015)
T(mean)-lag	0.002 (0.010)	0.001 (0.009)	0.008 (0.024)	0.001 (0.010)	0.001 (0.007)	0.001 (0.007)
Temp(max)	0.002 (0.010)	-0.001 (0.005)	0.000 (0.004)	-0.001 (0.007)	0.000 (0.004)	0.000 (0.003)
T(max)-lag	0.001	0.000	0.004	0.000	0.000	0.000

	(0.008)	(0.004)	(0.014)	(0.006)	(0.005)	(0.004)
Temp(min)	0.092 (0.030)	0.095 (0.024)	0.094 (0.028)	0.069 (0.054)	0.096 (0.027)	0.088 (0.024)
T(min)-lag	0.001 (0.008)	0.001 (0.008)	0.004 (0.014)	0.001 (0.008)	0.001 (0.009)	0.000 (0.005)
Other Variables						
Earnings	-0.138 (0.014)	-0.177 (0.014)	-0.144 (0.016)	-0.186 (0.013)	-0.203 (0.014)	-0.194 (0.013)
GDP	0.170 (0.036)	0.205 (0.019)	0.199 (0.025)	0.199 (0.023)	0.219 (0.029)	0.208 (0.024)
Smoking	0.049 (0.010)	0.046 (0.009)	0.048 (0.009)	0.041 (0.010)	0.049 (0.009)	0.043 (0.009)
January	-0.047 (0.014)	-0.046 (0.012)	-0.033 (0.018)	-0.046 (0.015)	-0.050 (0.012)	-0.048 (0.012)
February	-0.078 (0.013	-0.086 (0.011)	-0.071 (0.016)	-0.087 (0.014)	-0.090 (0.012)	-0.082 (0.011)
March	-0.028 (0.018)	-0.052 (0.012)	-0.032 (0.019)	-0.047 (0.018)	-0.056 (0.012)	-0.034 (0.015)
April	0.001 (0.007)	-0.029 (0.014)	-0.006 (0.012)	-0.016 (0.019)	-0.028 (0.015)	-0.001 (0.007)
May	-0.049 (0.064)	-0.214 (0.041)	-0.146 (0.052)	-0.130 (0.106)	-0.216 (0.049)	-0.084 (0.067)
June	-0.343 (0.052)	-0.429 (0.043)	-0.388 (0.047)	-0.353 (0.109)	-0.436 (0.051)	-0.330 (0.051)
July	-0.654 (0.053)	-0.693 (0.045)	-0.667 (0.049)	-0.622 (0.120)	-0.727 (0.055)	-0.617 (0.053)
August	-0.680 (0.049)	-0.687 (0.043)	-0.674 (0.047)	-0.638 (0.116)	-0.723 (0.052)	-0.644 (0.048)
September	0.002 (0.018)	-0.005 (0.021)	-0.004 (0.025)	0.065 (0.089)	-0.003 (0.029)	0.003 (0.019)
October	0.028 (0.048)	0.011 (0.030)	0.028 (0.047)	0.091 (0.100)	0.017 (0.042)	0.020 (0.041)
November	0.006 (0.025)	0.005 (0.022)	0.019 (0.041)	0.056 (0.072)	0.007 (0.028)	0.005 (0.022)
Calgary	1.778 (0.281)	2.129 (0.110)	1.803 (0.360)	2.127 (0.334)	2.043 (0.333)	1.969 (0.327)
Edmonton	1.379 (0.176)	1.773 (0.070)	1.539 (0.228)	1.685 (0.204)	1.696 (0.203)	1.669 (0.202)
Halifax	-0.789 (0.075)	-0.830 (0.051)	-0.827 (0.065)	-0.836 (0.081)	-0.838 (0.083)	-0.771 (0.076)
London	-0.434 (0.107)	-0.453 (0.052)	-0.463 (0.082)	-0.429 (0.068)	-0.497 (0.085)	-0.455 (0.077)
Montreal (Inner)	1.359 (0.059)	1.318 (0.041)	1.419 (0.065)	1.298 (0.074)	1.297 (0.079)	1.327 (0.070)
Montreal (Outer)	-0.699 (0.059)	-0.849 (0.041)	-0.763 (0.065)	-0.824 (0.074)	-0.825 (0.079)	-0.819 (0.070)
Ottawa	-0.305 (0.081)	-0.351 (0.049)	-0.348 (0.062)	-0.352 (0.058)	-0.351 (0.074)	-0.326 (0.063)
Regina	0.038 (0.108)	0.443 (0.051)	0.237 (0.173)	0.293 (0.148)	0.302 (0.138)	0.336 (0.142)

Saskatoon	-0.243 (0.129)	-0.002 (0.020)	-0.107 (0.140)	-0.052 (0.118)	-0.044 (0.106)	-0.046 (0.115)
Toronto (Inner)	0.015 (0.077)	-0.005 (0.033)	-0.013 (0.054)	-0.006 (0.118)	-0.017 (0.065)	-0.010 (0.051)
Toronto (Outer)	2.076 (0.087)	2.161 (0.050)	2.166 (0.065)	2.116 (0.055)	2.100 (0.099)	2.134 (0.062)
Vancouver (Inner)	-0.676 (0.071)	-0.649 (0.041)	-0.641 (0.071)	-0.600 (0.096)	-0.639 (0.099)	-0.621 (0.086)
Vancouver (Outer)	-0.014 (0.060)	0.000 (0.016)	0.012 (0.058)	0.031 (0.089)	0.036 (0.093)	0.024 (0.076)
Sample size	2,586	3,258	3,337	2,878	2,981	3,052

Table 4: Point Estimates of Each Coefficient Using Model Selection. Dependent Variable is Admissions (posterior standard deviations in parentheses)						
	Pollutants included as explanatory variables					
Pollutants	All	CO	TSP	SO2	NOX	O3
CO	--	-0.029 (0.010)	--	--	--	--
CO-lag	--	--	--	--	--	--
TSP	-0.048 (0.012)	--	-0.044 (0.011)	--	--	--
TSP-lag	-0.054 (0.012)	--	-0.037 (0.011)	--	--	--
SO2	--	--	--	--	--	--
SO2-lag	--	--	--	--	--	--
NOX	--	--	--	--	--	--
NOX-lag	--	--	--	--	--	--
O3	-0.048 (0.013)	--	--	--	--	-0.053 (0.012)
O3-lag	--	--	--	--	--	
Weather Variables						
Pressure	--	0.328 (0.089)	0.260 (0.088)	0.301 (0.090)	0.339 (0.092)	0.335 (0.093)
Press-Lag	0.439 (0.035)	0.383 (0.090)	0.396 (0.089)	0.354 (0.091)	0.374 (0.093)	0.355 (0.094)
Windspeed	--	--	--	--	--	--
Wind-lag	--	--	--	--	--	--
Temp(mean)	--	--	--	--	--	--
T(mean)-lag	--	--	--	--	--	--
Temp(max)	--	--	--	--	--	--
T(max)-lag	--	--	--	--	--	--
Temp(min)	0.105 (0.017)	0.091 (0.015)	0.095 (0.014)	0.085 (0.016)	0.097 (0.015)	0.095 (0.015)
T(min)-lag	--	--	--	--	--	--
Other Variables						
Earnings	-0.138 (0.013)	-0.173 (0.013)	-0.149 (0.013)	-0.187 (0.013)	-0.206 (0.013)	-0.195 (0.013)
GDP	0.177 (0.015)	0.204 (0.014)	0.199 (0.014)	0.192 (0.015)	0.207 (0.015)	0.202 (0.015)
Smoking	0.049 (0.009)	0.047 (0.009)	0.050 (0.008)	0.041 (0.009)	0.048 (0.009)	0.042 (0.009)
January	-0.051 (0.010)	-0.047 (0.009)	-0.044 (0.009)	-0.053 (0.010)	-0.052 (0.010)	-0.050 (0.010)
February	-0.083 (0.010)	-0.087 (0.009)	-0.080 (0.009)	-0.095 (0.010)	-0.093 (0.010)	-0.085 (0.010)
March	-0.039 (0.010)	-0.055 (0.009)	-0.041 (0.008)	-0.059 (0.009)	-0.059 (0.009)	-0.040 (0.009)

April	--	-0.034 (0.008)	--	-0.033 (0.009)	-0.033 (0.009)	---
May	-0.112 (0.039)	-0.225 (0.032)	-0.157 (0.031)	-0.220 (0.035)	-0.226 (0.034)	-0.120 (0.036)
June	-0.375 (0.040)	-0.440 (0.035)	-0.386 (0.033)	-0.443 (0.037)	-0.445 (0.036)	-0.343 (0.037)
July	-0.684 (0.042)	-0.704 (0.037)	-0.664 (0.035)	-0.722 (0.040)	-0.737 (0.038)	-0.632 (0.039)
August	-0.705 (0.039)	-0.706 (0.035)	-0.670 (0.034)	-0.736 (0.038)	-0.734 (0.037)	-0.656 (0.036)
September	--	--	--	--	--	--
October	--	--	--	--	--	--
November	--	--	--	--	--	--
Calgary	1.671 (0.111)	2.143 (0.092)	2.073 (0.089)	2.224 (0.127)	2.185 (0.106)	2.086 (0.109)
Edmonton	1.313 (0.072)	1.781 (0.059)	1.708 (0.058)	1.752 (0.082)	1.780 (0.068)	1.743 (0.070)
Halifax	-0.778 (0.054)	-0.826 (0.049)	-0.854 (0.046)	-0.862 (0.048)	-0.869 (0.047)	-0.791 (0.049)
London	-0.463 (0.043)	-0.455 (0.041)	-0.418 (0.040)	-0.413 (0.043)	-0.463 (0.042)	-0.431 (0.042)
Montreal (Inner)	1.369 (0.039)	1.316 (0.038)	1.390 (0.038)	1.285 (0.039)	1.275 (0.038)	1.313 (0.038)
Montreal (Outer)	-0.690 (0.039)	-0.850 (0.038)	-0.791 (0.038)	-0.837 (0.039)	-0.847 (0.038)	-0.833 (0.038)
Ottawa	-0.313 (0.042)	-0.344 (0.040)	-0.354 (0.039)	-0.346 (0.040)	-0.343 (0.041)	-0.833 (0.038)
Regina	--	0.451 (0.045)	0.368 (0.045)	0.340 (0.071)	0.357 (0.058)	-0.322 (0.041)
Saskatoon	-0.293 (0.078)	--	--	--	--	--
Toronto (Inner)	--	--	--	--	--	--
Toronto (Outer)	2.061 (0.039)	2.170 (0.039)	2.183 (0.039)	2.123 (0.040)	2.115 (0.039)	2.144 (0.039)
Vancouver (Inner)	-0.661 (0.039)	-0.650 (0.038)	-0.672 (0.037)	-0.638 (0.039)	-0.674 (0.041)	-0.650 (0.038)
Vancouver (Outer)	--	--	--	--	--	--
Sample size	2,586	3,258	3,337	2,878	2,981	3,052

Table 5: Point Estimates of Each Coefficient Using BMA, Dependent Variable is Total Patient Days (posterior standard deviations in parentheses)						
	Pollutants included as explanatory variables					
Pollutants	All	CO	TSP	SO2	NOX	O3
CO	0.002 (0.007)	0.000 (0.002)	--	--	--	--
CO-lag	0.001 (0.005)	0.000 (0.002)	--	--	--	--
TSP	-0.006 (0.014)	--	0.000 (0.002)	--	--	--
TSP-lag	-0.005 (0.013)	--	0.000 (0.003)	--	--	--
SO2	0.000 (0.002)	--	--	0.000 (0.003)	--	--
SO2-lag	0.000 (0.002)	--	--	0.001 (0.004)	--	--
NOX	0.000 (0.003)	--	--	--	0.000 (0.002)	--
NOX-lag	0.000 (0.052)	--	--	--	0.000 (0.002)	--
O3	-0.001 (0.006)	--	--	--	--	-0.053 (0.014)
O3-lag	0.000 (0.003)	--	--	--	--	0.001 (0.06)
Weather Variables						
Pressure	0.010 (0.052)	0.013 (0.065)	0.013 (0.064)	0.017 (0.072)	0.009 (0.048)	0.025 (0.084)
Press-Lag	0.590 (0.138)	0.350 (0.14)	0.349 (0.134)	0.434 (0.165)	0.124 (0.201)	0.262 (0.184)
Windspeed	0.001 (0.005)	0.003 (0.010)	0.003 (0.010)	0.003 (0.011)	0.001 (0.004)	0.002 (0.007)
Wind-lag	0.002 (0.009)	0.003 (0.009)	0.006 (0.014)	0.004 (0.012)	0.001 (0.004)	0.003 (0.010)
Temp(mean)	0.000 (0.005)	0.000 (0.004)	0.000 (0.004)	0.000 (0.005)	0.000 (0.005)	0.000 (0.005)
T(mean)-lag	0.000 (0.004)	0.000 (0.004)	0.000 (0.004)	0.000 (0.004)	0.000 (0.004)	0.000 (0.004)
Temp(max)	0.000 (0.004)	0.000 (0.003)	0.000 (0.004)	0.000 (0.004)	0.000 (0.003)	0.000 (0.005)
T(max)-lag	0.000 (0.003)	0.000 (0.003)	0.000 (0.003)	0.000 (0.003)	0.000 (0.003)	0.000 (0.004)
Temp(min)	0.000 (0.005)	0.000 (0.004)	0.000 (0.004)	0.000 (0.004)	0.000 (0.004)	0.000 (0.005)
T(min)-lag	0.000 (0.004)	0.000 (0.005)	0.000 (0.005)	0.000 (0.005)	0.000 (0.004)	0.000 (0.004)
Other Variables						
Earnings	-0.056	-0.089	-0.009	-0.089	-0.081	-0.091

	(0.026)	(0.018)	(0.018)	(0.021)	(0.020)	(0.020)
GDP	-0.348 (0.056)	-0.413 (0.044)	-0.414 (0.044)	-0.441 (0.050)	-0.320 (0.053)	-0.38 (0.051)
Smoking	0.049 (0.014)	0.052 (0.011)	0.048 (0.011)	0.048 (0.014)	0.057 (0.011)	0.055 (0.011)
January	0.004 (0.011)	0.006 (0.012)	0.005 (0.010)	0.003 (0.009)	0.005 (0.012)	0.005 (0.011)
February	-0.007 (0.013)	-0.011 (0.015)	-0.013 (0.016)	-0.016 (0.018)	-0.011 (0.015)	-0.012 (0.016)
March	0.000 (0.002)	0.000 (0.002)	0.000 (0.002)	0.000 (0.002)	0.000 (0.002)	0.000 (0.002)
April	0.000 (0.002)	0.000 (0.002)	0.000 (0.002)	0.000 (0.003)	0.000 (0.002)	0.000 (0.003)
May	-0.006 (0.024)	-0.020 (0.043)	-0/015 (0.036)	-0.008 (0.028)	-0.018 (0.039)	-0.016 (0.038)
June	-0.240 (0.043)	-0.232 (0.040)	-0.232 (0.040)	-0.239 (0.042)	-0.242 (0.041)	-0.230 (0.042)
July	-0.335 (0.045)	-0.318 ().043)	-0.325 (0.042)	-0.323 (0.044)	-0.355 (0.043)	-0.338 (0.044)
August	-0.384 (0.046)	-0.36 (0.043)	-0.361 (0.043)	-0.378 (0.046)	-0.376 (0.042)	-0.366 (0.044)
September	0.000 (0.008)	0.000 (0.006)	0.000 (0.008)	0.000 (0.009)	0.000 (0.007)	0.000 (0.007)
October	0.106 (0.066)	0.105 (0.057)	0.087 (0.061)	0.092 (0.066)	0.132 (0.052)	0.115 (0.057)
November	0.000 (0.006)	0.002 (0.006)	0.000 (0.005)	0.000 (0.006)	0.000 (0.006)	0.000 (0.006)
Calgary	2.156 (0.376)	1.480 (0.368)	1.486 (0.364)	1.893 (0.432)	0.757 (0.574)	1.248 (0.520)
Edmonton	1.831 (0.229)	1.603 (0.240)	1.606 (0.240)	1.755 (0.269)	1.105 (0.355)	1.440 (0.339)
Halifax	-0.827 (0.111)	-0.652 (0.080)	-0.648 (0.077)	-0.734 (0.111)	-0.550 (0.124)	-0.609 (0.091)
London	0.766 (0.164)	0.988 (0.143)	1.0069 (0.141)	1.074 (0.156)	0.644 (0.169)	0.861 (0.185)
Montreal (Inner)	2.191 (0.134)	2.609 (0.059)	2.621 (0.050)	2.521 (0.127)	2.541 (0.123)	2.584 (0.061)
Montreal (Outer)	-0.302 (0.135)	-0.003 (0.037)	-0.000 (0.020)	-0.050 (0.116)	-0.032 (0.112)	-0.005 (0.042)
Ottawa	0.660 (0.168)	1.006 (0.100)	1.013 (0.095)	1.016 (0.144)	0.795 (0.135)	0.945 (0.121)
Regina	0.016 (0.138)	-0.085 (0.162)	-0.075 (0.158)	-0.068 (0.169)	-0.483 (0.242)	-0.235 (0.238)
Saskatoon	-0.118 (0.193)	-0.380 (0.135)	-0.379 (0.133)	-0.285 (0.232)	-0.648 (0.204)	-0.502 (0.189)
Toronto (Inner)	1.031 (0.167)	1.336 (0.117)	1.348 (0.113)	1.362 (0.148)	1.090 (0.141)	1.260 (0.144)
Toronto (Outer)	2.553 (0.167)	2.923 (0.117)	2.942 (0.113)	2.923 (0.148)	2.650 (0.142)	2.833 (0.144)

Vancouver (Inner)	-0.470 (0.155)	-0.028 (0.072)	-0.024 (0.061)	-0.127 (0.172)	-0.051 (0.146)	-0.013 (0.061)
Vancouver (Outer)	0.052 (0.139)	0.415 (0.076)	0.420 (0.067)	0.314 (0.160)	0.489 (0.158)	0.443 (0.079)
Sample size	2,440	2,959	3,018	2,716	2,785	2,847

Table 6: Point Estimates of Each Coefficient Using Model Selection, Dependent Variable is Total Patient Days (posterior standard deviations in parentheses)						
	Pollutants included as explanatory variables					
Pollutants	All	CO	TSP	SO2	NOX	O3
CO	--	--	--	--	--	--
CO-lag	--	--	--	--	--	--
TSP	--	--	--	--	--	--
TSP-lag	--	--	--	--	--	--
SO2	--	--	--	--	--	--
SO2-lag	--	--	--	--	--	--
NOX	--	--	--	--	--	--
NOX-lag	--	--	--	--	--	--
O3	--	--	--	--	--	--
O3-lag	--	--	--	--	--	--
Weather Variables						
Pressure	--	--	--	--	--	--
Press-Lag	0.644 (0.045)	0.401 (0.035)	0.392 (0.034)	0.410 (0.043)	--	0.238 (0.075)
Windspeed	--	--	--	--	--	--
Wind-lag	--	--	--	--	--	--
Temp(mean)	--	--	--	--	--	--
T(mean)-lag	--	--	--	--	--	--
Temp(max)	--	--	--	--	--	--
T(max)-lag	--	--	--	--	--	--
Temp(min)	--	--	--	--	--	--
T(min)-lag	--	--	--	--	--	--
Other Variables						
Earnings	-0.061 (0.016)	-0.096 (0.015)	-0.095 (0.015)	-0.100 (0.016)	-0.075 (0.015)	-0.090 (0.016)
GDP	-0.339 (0.049)	-0.421 (0.033)	-0.421 (0.031)	-0.439 (0.035)	-0.295 (0.030)	-0.375 (0.037)
Smoking	0.049 (0.012)	0.054 (0.010)	0.050 (0.010)	0.054 (0.011)	0.057 (0.011)	0.055 (0.011)
January	--	--	--	--	--	--
February	--	--	--	--	--	--
March	--	--	--	--	--	--
April	--	--	--	--	--	--
May	--	--	--	--	--	--
June	-0.238 (0.040)	-0.224 (0.036)	-0.224 (0.036)	-0.231 (0.038)	-0.241 (0.037)	-0.232 (0.037)
July	-0.329 (0.041)	-0.312 (0.037)	-0.320 (0.036)	-0.319 (0.039)	-0.356 (0.038)	-0.341 (0.038)
August	-0.380 (0.041)	-0.358 (0.037)	-0.360 (0.037)	-0.375 (0.039)	-0.375 (0.038)	-0.367 (0.038)
September	--	--	--	--	--	--

October	0.133 (0.039)	0.125 (0.035)	0.116 (0.035)	0.128 (0.038)	0.147 (0.036)	0.136 (0.036)
November	--	--	--	--	--	--
Calgary	2.247 (0.155)	1.618 (0.112)	1.596 (0.110)	1.828 (0.148)	0.374 (0.040)	1.100 (0.238)
Edmonton	1.882 (0.103)	1.691 (0.075)	1.673 (0.074)	1.726 (0.098)	0.865 (0.041)	1.340 (0.157)
Halifax	-0.860 (0.069)	-0.660 (0.061)	-0.655 (0.059)	-0.685 (0.062)	-0.481 (0.051)	-0.600 (0.063)
London	0.733 (0.129)	1.042 (0.083)	1.051 (0.083)	1.114 (0.090)	0.548 (0.070)	0.822 (0.110)
Montreal (Inner)	2.134 (0.065)	2.619 (0.045)	2.629 (0.045)	2.587 (0.045)	2.577 (0.044)	2.50 (0.045)
Montreal (Outer)	-0.360 (0.065)	--	--	--	--	--
Ottawa	0.611 (0.117)	1.036 (0.072)	1.032 (0.072)	1.071 (0.075)	0.764 (0.068)	0.930 (0.084)
Regina	--	--	--	--	-0.647 (0.064)	-0.325 (0.112)
Saskatoon	--	-0.328 (0.059)	-0.337 (0.057)	-0.359 (0.104)	-0.774 (0.069)	-0.561 (0.104)
Toronto (Inner)	0.990 (0.120)	1.380 (0.078)	1.385 (0.077)	1.421 (0.083)	1.036 (0.068)	1.239 (0.093)
Toronto (Outer)	2.512 (0.120)	2.966 (0.078)	2.978 (0.077)	2.981 (0.083)	2.600 (0.068)	2.810 (0.093)
Vancouver (Inner)	-0.518 (0.056)	--	--	--	--	--
Vancouver (Outer)	--	0.416 (0.045)	0.417 (0.046)	0.400 (0.047)	0.558 (0.046)	0.456 (0.048)
Sample size	2,440	2,959	3,018	2,716	2,785	2,847

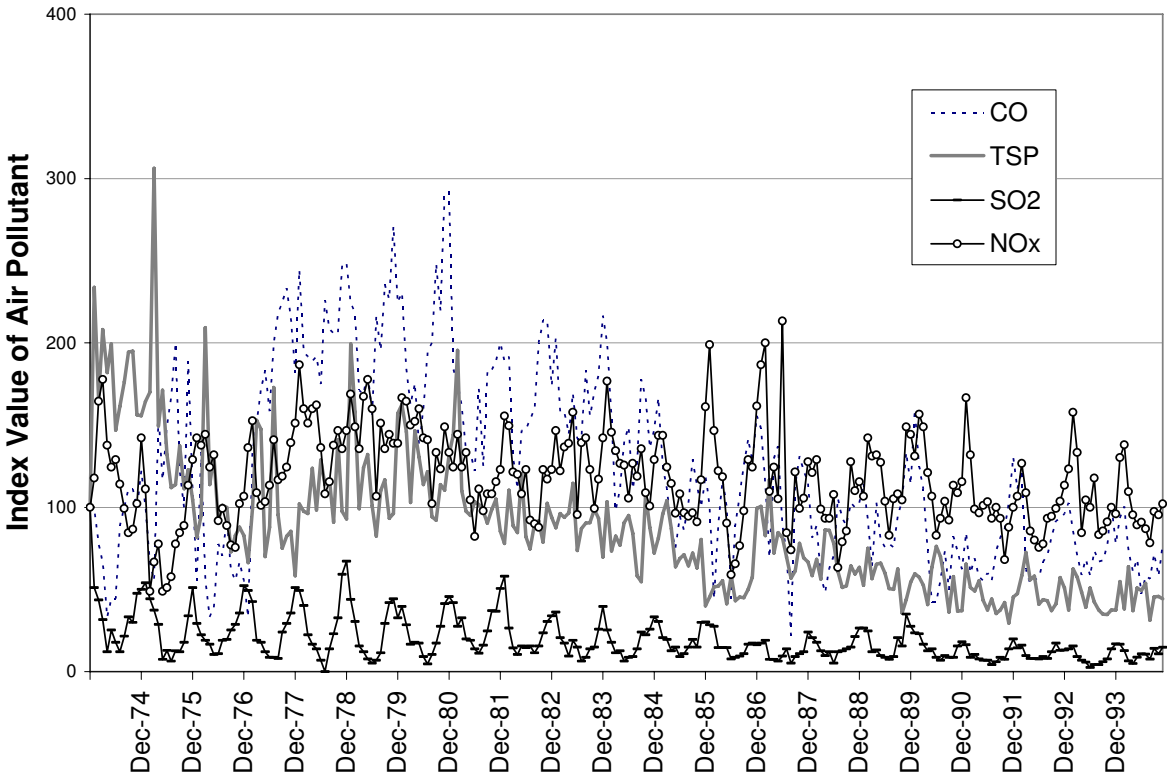

Figure 1. Montreal air pollution levels January 1974 to December 1994. Legend: CO=Carbon monoxide, TSP=Total Suspended Particulates, SO2 = Sulphur Dioxide, NOx = Nitrogen Oxides. All data scaled to January 1974=100 to permit comparison. Note general decline in mean and variability over time.

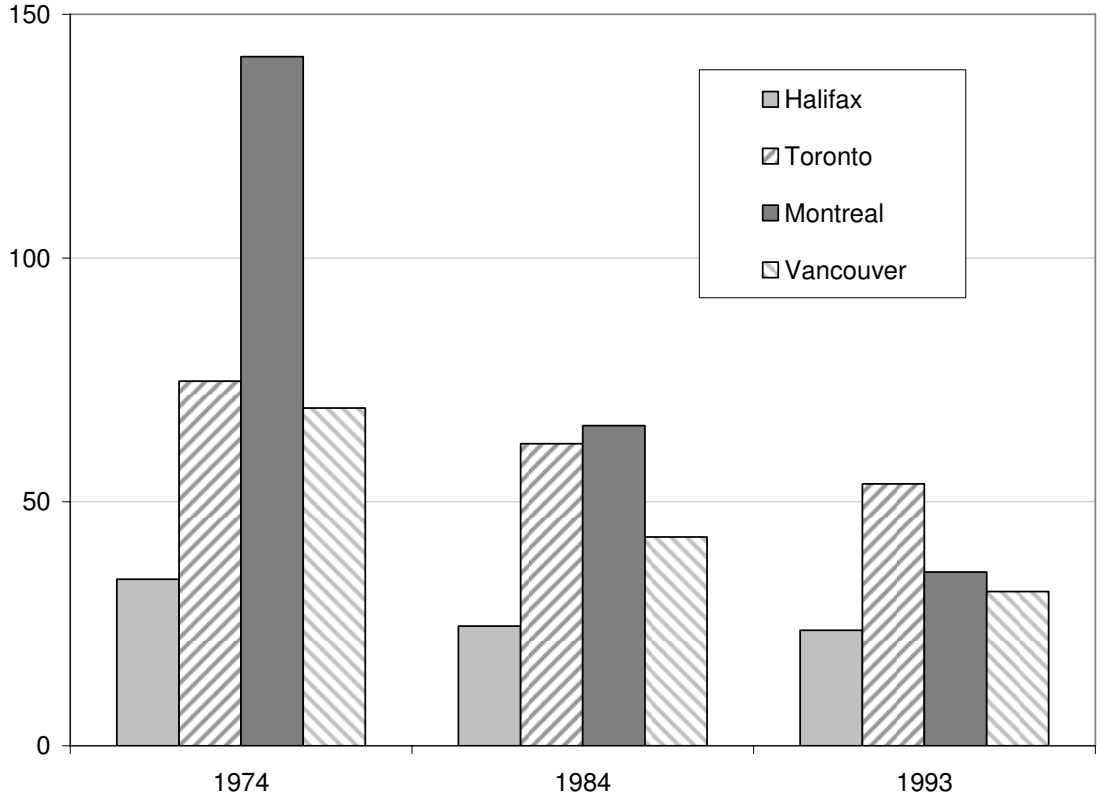

Figure 2. Annual average Total Suspended Particulate Levels (micrograms per cubic meter) for four Canadian Cities, 1974—1993. Note higher mean levels and wider variation in 1970s compared to 1980s and 1990s.