AIR POLLUTION, ECONOMIC ACTIVITY AND RESPIRATORY ILLNESS: EVIDENCE FROM CANADIAN CITIES, 1974-1994

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ABSTRACT

Many studies have reported a relationship between urban air pollution levels and respiratory health problems. However, there are notable variations in results, depending on modeling approach, covariate selection, period of analysis, etc. To help clarify these factors we compare and apply two estimation approaches: model selection and Bayesian model averaging, to a new data base on 11 Canadian cities spanning 1974 to 1994. During this interval pollution levels were typically much higher than the present. Our data allow us to compare monthly hospital admission rates for all lung diagnostic categories to ambient levels of five common air contaminants, while controlling for income, smoking and meteorological covariates. In the most general specifications we find the here-observed health effects of air pollution are very small and insignificant, with signs that are typically opposite to conventional expectations. Smoking effects are robust across specifications. Considering the fact that we are examining an interval of comparatively high air pollution levels, and the contrast between our results and those that have been published previously, we conclude that extra caution should be applied to results estimated on short and/or recent data panels, and to those that do not control for model uncertainty and socioeconomic covariates.

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1. Introduction

Many epidemiological studies—numbering in the hundreds—have asserted a significant correlation between ambient air pollution and health in North American cities. Leading public health authorities in Canada (e.g. OMA 2000, Basrur 2000, Toronto Public Health 2004, 2007) have referred to some of this literature in issuing calls for tighter air pollution regulations, in the process making some rather dramatic numerical claims. For example, the City of Toronto claims that air pollution is responsible for 1,700 premature deaths and 6,000 hospitalizations each year in Toronto alone (Toronto Public Health 2004). Governments, too, have cited these concerns as the motivation for new regulatory measures (e.g. US EPA 2001, Government of Canada 2006) including more stringent motor vehicle emission standards and national reductions in industrial air pollution emissions.

Such policies impose costs on households and industries, therefore it is important to get accurate measures of the potential benefits of air pollution regulations, namely improved quality of life and reduced health care costs, in order to guide regulatory decision-making. In this paper we propose some methodological and data innovations to clarify a number of ongoing points of debate in the literature.

First, we control for both socioeconomic covariates and pollution levels. Addition of economic variables to epidemiological models is a relatively new feature in the air pollution-health literature, and has been shown to have important implications for the robustness of earlier findings. Second, we have developed a uniquely rich data base that covers substantial spatial and temporal variation. A limitation of many epidemiological studies is that they only examine short data samples drawn from the late-1980s or later. Many North American cities had high pollution levels in the 1960s and 1970s. But starting in the 1980s, steady reductions began, leading to the current situation in which air pollution levels in many urban areas are at historically low and relatively stable levels. Figure 1 illustrates this with carbon monoxide and sulphur dioxide data from Calgary, Toronto and Montreal over the interval of our data set. Studies that only examine recent data, or single-city samples, have less pollution variability with which to identify key

parameters, which may explain why the literature is filled with small and unstable parameter estimates. The validity of small positive coefficient estimates, implying a deleterious effect of pollution on a measure of ill-health, should be tested by using data back to the 1970s, since if the current pollution levels cause the kind of illness burdens alluded to above, we should expect to find correspondingly larger effects in past decades when air pollution was much higher.

Chay, Dobkin and Greenstone (2003) examined US county-level data over the 1969—1974 interval, when particulate levels were much higher than at present. Over a wide range of specifications they could find no evidence tying pollution to adult mortality, a surprising result since other studies looking at intervals with cleaner air have reported such a connection. For our study we have obtained a high-quality, consistent record of admissions to Canadian hospitals for all lung-related illnesses from 1974 to 1994. This start year (1974) coincides with the start date for the quality-controlled city-level air pollution archives maintained by the National Air Pollution Surveillance Network at Environment Canada (NAPS 2007). Consequently our study spans a time interval in which air pollution levels declined in many cities, increasing our confidence in the robustness of our findings. We have also been able to develop time series for eleven different Canadian cities, providing substantial cross-sectional variability in pollution and health as well as socioeconomic conditions.

Third, we explicitly address model uncertainty by using Bayesian Model Averaging. We demonstrate the sensitivity of results on our data from applying classical estimation methods by fitting a series of regressions representing plausible variations on model selection. The coefficients will be shown not only to vary in size but also in sign, yielding apparently contradictory implications about the relationship between air pollution and health. This leads us to conclude that uncertainty over model choice must be addressed as part of the empirical framework. The issue arises with particular force in regression-based epidemiological studies, where there is so much choice not only over dependent variables but also explanatory variable. 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. 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. However, the number of potential confounding variables implies that a huge number of models could be used to explain health effects. The number of potential models is on the order of 2^k where k is the number of potential explanatory variables, including lags. Since results can be sensitive to the particular regression specification, and since the number of potential models is so large, model uncertainty has been shown to be an important issue in this literature (Clyde 2000, Koop and Tole 2004).

In this paper, we consider the two major competing ways of dealing with model uncertainty: model selection (i.e. selecting a single model) and model averaging. Koop and Tole (2004; 2006) argued that there were many theoretical arguments in favour of model averaging for applications such as measuring the effect of air pollution on health. In an empirical exercise using daily time series data for Toronto, they found that the use of model averaging led to estimates of uncertainty about the magnitude of the health effects of air pollution that were larger than had emerged using model selection methods. In the present paper we note that we can present model specifications in which pollution apparently yields a significant negative effect on health. But we also show that this result is not robust, a finding confirmed by model averaging methods.

A potential contribution from using socioeconomic covariates is the ability to isolate the marginal effects on health of both air pollution and income. Air pollution is but one factor that affects public health: income and economic growth also matter (e.g. O'Neill and O'Neill 2007, Cutler, Deaton and Lleras-Muey 2006, Gerdtham and Johannesson 2004). Since policies to reduce air pollution can simultaneously reduce real income (e.g. Jorgensen and Wilcoxen 1990), it is important to assess the relative strength of these factors, not only to balance economic costs and benefits but to ascertain the net effect on health itself. A particular emissions control policy might have a net positive or negative effect on health, depending on the signs and relative strength of these factors.

One of our dependent variables in this study is local hospital admissions per 100,000 persons for lung-related illnesses, a standard health metric in this type of epidemiological studies (Bates 1983, Burnett et al. 1997a,b, Neidell 2004, etc.) For brevity we will use "admissions per capita" to denote admissions per 100,000 persons. Admissions per capita for Calgary, Toronto and Montreal are shown in Figure 2 along with average real income in the corresponding provinces (Alberta, Ontario and Quebec, respectively), where all data are indexed to begin at 100

in 1974 to facilitate comparison. Overall, our socioeconomic indicators—smoking, income and Gross Domestic Product (GDP)—have significant explanatory power, in accordance with prior expectations. But we find that, in most cases, the health effects associated with individual air pollutants are not significant. And where we do find significant health effects (particulates and ground-level ozone), the coefficients are negative, indicating, counter-intuitively, that these pollutants are associated with improved health. Negative pollution coefficients are not uncommon in the literature (see, e.g., Neidell 2006 and 2004, Chay et al. 2003, Domenici et al. 2002). In the last part of the paper we discuss possible explanations.

In this article, we are specifically looking for predictable, common physical effects from standardized exposure levels. By pooling across cities, rather than estimating each city separately, we identify the common marginal effect of air pollution, rather than looking for city-specific marginal effects, should they differ by location. By pooling across time we are also averaging over possible differences in marginal effects by month, whereby some groups may be more sensitive to certain pollutants in one season rather than another. Our main empirical conclusion, in line with a number of other recent studies that control for economic and pollution variables simultaneously, is that variations in smoking and income likely have a significant effect on lung-related health, but variations in pollution likely do not.

2. Literature Review

Some influential reviews, such as Stieb (2002) and Basrur (2000), conclude that air pollution has an impact on health. However the underlying epidemiological findings tend to be inconsistent. Burnett et al. (1998) 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) 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. (2000) studied 20 US cities and find PM_{10} and O_3 significant, but find CO, SO_2 and NO_2 insignificant. Chay et al. (2003) studied over 500 US counties and found no influence of Total Suspended Particulates (TSP) on adult mortality. Dominici et al. (2002) estimated dose-response curves relating PM_{10} exposure and mortality risk in 88 US cities. While the nationally-pooled results suggest a small

[1] $PM_{2.5}$ denotes particulate matter smaller than 2.5 microns; PM_{10} denotes PM smaller than 10 microns.

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 using risk of admission to hospital as the dependent variable are likewise conflicted. Bates (1983) studied 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 (in the present study we likewise find a negative coefficient on O_3). 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), by contrast, 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 further 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}. Neidell (2004) studied zip code-level data from California between 1992 and 1998 and found CO had a small positive effect on hospital asthma admissions, but O_3 had no effect (in some cases it was negative), nor did PM_{10}. Indeed the absence of effects for TSP and/or PM_{10} when entered simultaneously with other pollutants is common in admissions data studies (Thurston (1994), Burnett (1997c), Burnett (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 model uncertainty. With very few exceptions (e.g. Clyde (2000), Clyde and DeSimone-Sasinowska (1997) and Koop and Tole (2004; 2006)), previous studies on air pollution health effects have used model selection methods, i.e. choosing one or a few regression specifications and reporting point estimates and their associated variances conditional on that being the true model. However, the estimation exercise is inherently opportunistic. Many plausible covariates could be included, but the choice is not dictated by theory so much as by data availability. Hence there is not only uncertainty about regression slope coefficients conditional on the model selection, but about the model specification itself.

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, 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) than did the point estimators derived in Schwartz (1993, 1994). Koop and Tole (2004; 2006) analyzed daily Toronto data over the 1992 to 1997 interval using BMA, and found the data did not support the claim of a significant health effect for several different pollutants, but that it would be possible to conclude such a relationship exists by choosing a model favourable to such a conclusion.

The application of BMA in epidemiological studies as a means of resolving model uncertainty has been a source of some debate. Thomas et al. (2007) argue that insignificant results in multipollutant BMA models might be due to collinearity problems. This concern applies more generally, but is not an issue in the present data base, since the five pollutants are not highly correlated with one another. The highest correlation (between CO and NO2) is only 0.36 and some of the correlations between pollutants are actually negative. Although we have sufficient cross-sectional and time series variability to avoid collinearity problems, we nonetheless check our results in the multipollutant cases against models where pollutants are introduced one at a time.

Thomas et al. also point out that pollutant concentrations are only indirect measures of actual personal exposure, since people may go indoors on bad air days. This criticism applies to all epidemiological studies using ambient pollution data, which includes all the studies cited above. It does not apply to laboratory-based studies in which precisely controlled pollution doses are the treatment variable. However these studies, like our own, have raised questions about the robustness of correlation-based epidemiological results. Green and Armstrong (2003) review experimental evidence on the cardiopulmonary effects of particulates and 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 review by Health Canada (1997)[2] that provided background material for the development of Canadian air quality guidelines likewise 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[3] 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 in epidemiological modeling, which added an upward bias to many previously published risk estimates. It restated the problem that epidemiological findings are not well-supported by experimental results, but did not propose a resolution. A report by the Royal Society of Canada (Adamowicz et al. 2001), convened to review the adequacy of cost-benefit analysis associated with newly-proposed Canada-wide standards for ground-level ozone, also drew attention to the conflict between clinical studies and epidemiological findings.[4]

Thomas et al. criticize the idea of averaging coefficients across different models since the interpretation of the coefficients may change slightly from one specification to another. This criticism however applies just as much to model-selection techniques, since uncertainty about the meaning of a parameter will not be apparent if only one model is reported. Under Bayesian

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[2] See Health Canada (1997) http://www.hc-sc.gc.ca/hecs-sesc/air_quality/publications/particulate_matter_science_assessment/addendum/impacts.htm#7 .

[3] Available at http://www.ccme.ca/assets/pdf/prrvw_pm_fine_rvsd_es_e.pdf.

[4] "With the exception of heart-rate variability, controlled studies involving relatively brief exposures of healthy individuals to single agents or binary mixtures have yielded few measurable responses even when the exposures are at concentrations far higher than the ambient concentrations associated with measurable responses in populations." Adamowicz et al. (2001, p. 114).

methodology, each estimation is weighted by a posterior model probability score indicating how much support each model has in the data. Hence the weighted average will be an appropriate measure of the centre of the distribution of coefficient values, since the values that receive the most support from the data will have the greatest influence on the position of the distribution. In the present study, by comparing our results with model-selection results and one-at-a-time pollutant results, we show that our conclusions are not dependent on use of the BMA methodology, though we consider it the most reliable approach for the purpose.

Another commonly-cited concern is the possibility of missing confounders, i.e. socioeconomic covariates that may be excluded due to unavailability. A dramatic illustration of the importance of this is in Chay et al (2003), where a positive and significant effect of TSP on adult mortality vanishes when controls for income and employment are introduced. Likewise, Neidell (2004) reports an effect of CO on asthma admissions to hospital across ages 1—18, but when interactions with socioeconomic status are introduced, the effects disappear for all but the age 3—6 age group. In a study of pollution and infant mortality in California (Currie and Neidell 2004), statistically significant effects between pollution and short gestation periods are observed, but when fixed effects due to community socioeconomic characteristics are controlled for, the effects of pollution on gestational age 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, but a small but significant relative risk (0.014%) between increased carbon monoxide and infant mortality is found after socioeconomic covariates are introduced Currie and Neidell 2005).

3. Data and Methods

3.1 General points

As discussed in the introduction, this study attempts to shed new light on the issues raised in the previous section by way of three empirical innovations. First, our data panel spans eleven Canadian cities from 1974 to 1994. Unfortunately we were not able to use post-1994 admissions data in this study: see Section 4 for a discussion. Extending the data back to the 1970s yields both higher average levels and larger annual and between-city variance (see Figure 2). Second, the length of the time series allows us to control for a group of socioeconomic covariates, including income, local Gross Domestic Product (GDP), smoking rates and demographic changes, reducing the risk of omitted variable bias. It also allows us to include month and city

dummy variables, thus controlling for unobservable heterogeneity due to location and season. 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.

The price of these gains is that we are using monthly averages 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 daily admissions up to monthly frequency suffices to remove any correlation with average pollution, then the effects in studies of daily frequency data are likely spurious. Or, put another way, if there really is an effect in the daily data, it should remain when the data are smoothed over monthly intervals, given the length of our time series.

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 of other representative examples include Chay and Greenstone (2003) and Pope (1991).

3.2 Data collection and pre-processing

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 variables, plus and city and monthly dummy variables. A dummy variable takes the value 1 if the observation falls in that category and 0 otherwise.

Our data panel covers eleven Canadian cities from January 1974 through March 1994. Summary statistics are provided in Tables 1 and 2. Table 1 provides nationwide means, standard errors, and so forth. Table 2 breaks out some air pollution measures by city. Our pollutants are CO, TSP, SO_2, NO2 and O_3. Observations are monthly means of daily means. All data on

Canadian urban air pollution were 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. Monitoring site locations are mapped at http://www.etc-cte.ec.gc.ca/napsstations/main.aspx. For some years, additional data were obtained from a compilation supplied by Environment Canada to Brown et al. (2004). The NAPS archive is a quality-controlled subset of air pollution data collected by provincial government agencies across Canada, where admission to the NAPS archive requires checks for methodological consistency and verification of equipment reliability. NAPS prescribes equipment siting and operation parameters, and sets quality control objectives so that data generated by the network should have a calibration accuracy of ±15% compared to true values (full methodological details are available at http://www.etc-cte.ec.gc.ca/publications/naps/NAPSQAQC.pdf.

Our ambient pollution level data set likely provides a good measure of exposure in the North American context because we extend the sample back in time to cover periods of higher pollution levels, and because we use multiple cities. Cross-sectional variance helps capture effects due to long term exposure differences, independently of acute effects due to temporal variation in pollution. We discuss the issue of exposure modeling when discussing our results below.

The total sample size was limited by the extent of missing data in the air pollution records. Vancouver, Edmonton, Montreal, Saskatoon, Ottawa and Toronto typically had at least two monitoring stations available in any one month throughout the sample, whereas the other cities typically had one. Where cities had multiple monitoring sites a simple average was taken. Altogether the panels yielded sample sizes of between approximately 2500 and 3300 observations for each pollutant type. SO_2 had the highest rate of missing values, particularly in western cities. Calgary was missing SO_2 data for 26% of months, Edmonton for 14%, Saskatoon for 79% and Regina 62%. Other cities had complete or nearly complete SO_2 records. However, this likely overstates the sampling problem. Western cities had noticeably lower SO_2 levels throughout the sample compared to eastern cities. Maximum levels across the sample were 10 parts per billion (ppb) in Calgary, 6 ppb in Edmonton, and 2 ppb in Regina and Saskatoon, compared to about 60 ppb in Ottawa and Montreal. A missing observation in Regina or

Saskatoon likely indicates a value near zero, although this assumption was not imposed on the analysis.

We use two different dependent variables: hospital admissions per capita summed across all respiratory categories, and average patient days (i.e. days spent in hospital) for admissions due to all respiratory categories. Each variable represents an implicit threshold: the health impact must lead to hospital admission. Duration of stay is a further indicator of severity, conditional on the patient having crossed the admission threshold. All health admissions data were supplied by 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

The data extractions specified "acute care" admissions only, as opposed to, e.g., scheduled surgeries. Chronic conditions (such as chronic sinusitis) can require acute care in the event of flare-ups. Our sample includes all admitting hospitals in each city as recorded in Statistics Canada records, and the data extractions cover reporting years 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 March 1994 end-date of the Statistics Canada archive.

We use the admissions data in per capita (or, more precisely, per 100,000 persons). We noticed a number of months in which the rate of admissions fell anomalously low, dropping to fewer than 5 admissions. We confirmed that these observations were in the actual data as received from Statistics Canada, but they were so far removed from the general patterns in the data for those cities that we regard them as likely being inaccurate. We decided to omit any month with fewer than 5 admissions per 100,000, which removed 33 of approximately 2700 observations.

For the three biggest cities (Montreal, Toronto and Vancouver), we report the results with the city treated as a single region. However, in supplementary tables available from the authors have also separated the admissions data for these cities into inner city and suburbs, yielding 14 different municipal regions. However, no changes of any consequence were observed.

All weather data were supplied by 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 is measured using Statistics Canada estimates of average earnings. Monthly income series for each city up to 1985 were obtained from the Statistics Canada Average Weekly Earnings by city (All Employees) for the period 1973-1985, at which point the series ends.The series were adjusted for inflation using the Consumer Price Index (CPI). An extrapolation through 1994 was constructed by taking the Statistics Canada Average Weekly Earnings of Employees by Province, which extends to the present, deflating it using the monthly CPI, using the resulting series to construct monthly percentage changes by province, and then running the city-specific earnings forward to the end of the sample. The CPI for Toronto was used to estimate the values for London.[5]

Annual real GDP at the Provincial level was obtained from the Provincial Economic Accounts from Statistics Canada.

Information on smoking rates 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

[5] The CANSIM 2 income series numbers 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; and Tables 2810021 and 2810002.

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, 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.

Our data set also incorporates monthly and city dummy variables. Dummy variables take the value 0 or 1 depending on whether the observation is associated with the appropriate month or city. The latter controls for unobservable but fixed heterogeneity across cities, including geographical features, including different built skylines, different local terrain, and other factors affecting the urban microclimate, and fixed demographic differences, such as age composition. The former controls for monthly, or seasonal, effects.

Some variables (such as smoking and real GDP) do not vary within a province or within a year. In an ordinary least squares model, when data with different resolution across time and/or space are grouped together, an adjustment should be made to the OLS covariance matrix estimator to take into account the non-independence of the observations (Moulton 1990). This is called a 'clustering' adjustment, and is applied to the linear regression results presented in Section 4.1.

3.3 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. The most common and familiar approach is model selection, in which hypothesis testing procedures are used to select explanatory variables and then estimates are reported conditional on these choices. An alternative is to average across various plausible models (e.g. Clyde 2000, Clyde and DeSimone-Sasinowska 1997, Koop and Tole 2004; 2006). Model selection is the more familiar approach so we will limit the discussion to some brief comments in the results section. We will focus our explanation herein on the model selection method.

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). 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 worse 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 false hypothesis is rejected at the 5% level of significance is spurious and, potentially misleading, if the regression itself is selected on the basis of previous hypothesis tests. 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 estimates 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, $p(M_r \mid Data)$ 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 ordinary least squares (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 both arbitrary model selection and selection of 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.

Each regression model is a linear panel, pooling cross-sectional and time series information. Use of panel methods offers a couple of advantages over estimating each city

separately. First, the between-city information, which reflects long term exposure differences due to historical differences in pollution levels among cities, is retained and allowed to influence parameter estimates. Second, the sample size is much bigger, yielding more efficient parameter estimates. Third, it allows use of dummy variables (as discussed earlier) to control for fixed but unobservable heterogeneity. Note that we have used city dummy variables but we have not interacted them with the pollution variables. That is, we restrict the term that estimates the marginal effect of pollution on health to be constant across cities, but we allow cities to have independent intercepts. Certain kinds of differences may affect the slope itself. For instance, if people in one city have a relatively large propensity to be out of doors this may translate into a higher exposure level and a larger marginal effect of pollution compared to another city. However our goal is to identify the marginal effect of pollution common to all cities, which requires that we use a single slope coefficient.

4. Empirical Results

4.1 Linear Regression Variants

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 to ensure consistency of interpretation without affecting the empirical results.

To illustrate the issues that motivate the model averaging approach, we begin with an estimation of four linear regression models using ordinary least squares (OLS). Our dependent variable was total admissions per 100,000 persons; in subsequent estimations we will show results for length of stay as well. A positive coefficient on a pollutant indicates a negative effect on health, and vice-versa.

Table 3, column 1, reports the coefficients from a basic OLS regression of total admissions per capita on the five pollutants, three weather variables, smoking rates, income and real GDP. The coefficients appear to show positive but insignificant effects on admissions from CO and NO_2, coupled with a significant negative effect from O_3, TSP and SO_2. All the meteorological and socioeconomic variables matter. Smoking raises the admissions rate, as expected, and increased income reduces it.

The error clustering adjustment, discussed above, is applied in Columns 2-4. Comparing Columns 1 and 2 the coefficient magnitudes and signs are all preserved, but the variance estimates are wider, and hence the t-statistics are smaller. The effects associated with SO_2, Temperature and real GDP become insignificant. In Column 3 we introduce dummy variables for city and month to control for fixed effects across location and season (these coefficients are omitted to save space). This has a large effect on the results. The CO coefficient becomes negative and marginally significant while the other pollution effects and windspeed essentially disappear. Air pressure, income and real GDP also take on the opposite signs, while Smoking increases in size.

In Column 4 we add a linear time trend by defining an index that increases by one unit each month, and repeating the measure for all cities. Upon adding this to the model the pollution variables remain small, though CO becomes significant. The other variables retain their general size and significance. The admissions trend, after controlling for all other factors, is downwards over time.

The results in Table 3 illustrate two points. First, the basic effects are apparently small and not indicative of a positive effect of pollution on lung-related hospital admissions. To illustrate the scale, a one standard deviation increase in admissions corresponds to 11.5 persons per 100,000 population (see Table 1). A coefficient of 0.02 on NO_2 means that a one standard deviation increase in the monthly average concentration (8.2 parts per billion) would be associated with about one additional hospitalization per month for every half-million people in a city. Because of the combination of positive and negative coefficients in Column 4, if all pollutant types increased by one standard deviation—which would, in the present-day context, represent a very large proportionate increase in urban pollution loads—total hospital admissions per month would be expected to decline by 0.2 standard deviations, or 12 persons per month per half-million population. Or, to put it another way, suppose all current air pollution in Toronto fell

to zero. The Toronto Board of Public Health estimated that contemporary air pollution in Toronto causes 6,000 hospitalizations annually (see Introduction). Using the average hourly readings from the downtown Toronto air pollution monitoring site (see http://airqualityontario.com/history/station.cfm?stationid=31103) we can estimate the size of such a reduction in standard deviation units, and use the coefficients from column 4 of Table 3 to estimate the resulting change in per capita admissions. On this calculation, the result would be a monthly increase in admissions of 0.36 persons per 100,000, or 238 persons per year in a city of 5.5 million, Greater Toronto's current population. Ignoring the sign difference, this is an order of magnitude smaller than the official estimate published by the Toronto Board of Public Health.

The second point is that, across a few reasonably similar specifications, we can generate divergent results with conflicting implications. A researcher must choose which model to report, but doing so amounts to assigning full support to one model and zero to the others, whereas the data are not so categorical. The R^2 and Schwarz Information Criteria are reported in the final two rows. While they indicate the data give much greater support to the latter 3 models they also show that the data do not give *zero* support to the first two. We could introduce any number of variants of these four models, each of which might obtain greater or lesser support in the data. To the extent coefficient estimates change across models, we would be faced with the model selection problem described above. We therefore turn to the discussion of Model Averaging results.

4.2 Model Averaging and Model Selection

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 (current and lagged one month) as potential explanatory variables. All of them may have an effect on hospital admissions. However, most papers in the epidemiology literature use a single pollutant as an explanatory variable. Accordingly, we also present empirical results for

specifications that include only one pollutant at a time. The results below are therefore based on 24 different statistical exercises. 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).

Tables 4 through 7 summarize our most important empirical results, those relating air pollution and socioeconomic covariates to health outcomes. The regressions included numerous variables of secondary interest (such as daily temperature minima and maxima) not listed in those tables. Complete tables are in the Appendix. These tables presents the point estimates (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. The cumulative effect is the standard multiplier, i.e. for any pollutant, the sum of 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.

As was hinted at by the results in the previous section, there is a striking contrast between the pollutant and socioeconomic effects. In only one case is an increase in current air pollution associated with an increase in hospital admissions: in the multipollutant model a one standard deviation increase in NO_2 is associated with a 0.053 standard deviation increase in admissions per 100,000 people (just over 0.6 persons per 100,000), and all other pollutant terms are insignificant or their inclusion is not supported by the data. Using Model Selection, the one month lag of NO_2 is associated with increased admissions in the multipollutant case, while TSP, CO and SO_2 exhibit significant negative effects in the multipollutant or single-pollutant cases. A negative coefficient indicates—on the standard interpretation—that increases in pollution are associated with decreases in admission counts. A negative effect between CO and patient days is observed in both the BMA and model selection results. Otherwise the data do not provide support for inclusion of the pollution variables in the model.

By contrast, an increase in smoking rates has a significant positive effect in all four multipollutant models, indicating an association with higher admission counts and longer patient stays. Across all 24 models a positive coefficient is supported in 20 cases and it is significant in 19. Provincial GDP is positive in all four multipollutant models and significant in three, as well as positive and significant in two of the other 20 single-pollutant models. This may be an

indication that provinces with larger economies can afford more hospital entry points (making it easier to get admitted), and longer patient treatment regimens. The income coefficient is always positive or zero, except for SO_2 in the single-pollutant patient days regression when it is negative. It is never significant in the multipollutant case.

Overall we find that health effects of air pollution are numerically very small and in almost all cases are either insignificant or negative (i.e. they imply pollutants are beneficial for health). This conclusion holds regardless of whether we use BMA or model selection, and therefore we consider it a very robust implication of the data.

Negative coefficients for pollutants have been found in other studies. Such studies have speculated that they may reflect evidence of averting behaviour by sensitive individuals during smog episodes (e,g, Niedell 2006). It is reasoned that since smog primarily involves elevated O_3 and TSP levels, individuals facing a lung-related health risk protect themselves by staying indoors, thus facing reduced actual (indoor) exposure compared to days with moderate outdoor pollution. In this way, higher ozone levels may appear to correlate with lower health risks. In respect to these findings, we have no clinical or other basis for understanding the negative coefficients on TSP, SO_2 and CO. But it is noteworthy that our sample period runs prior to the introduction in Canada of public "smog warnings," which only started in the mid-1990s.

Hence we cannot speculate on whether sensitive individuals were engaging in averting behavior. In light of the difficulty of actually measuring averting behaviour itself we can only conclude that our results provide no evidence that air pollution causes acute respiratory disease in the Canadian cities we consider. Repeating the experiment of the previous section, if all air pollution in Toronto disappeared, using the coefficients from column 1 of Table 4 we would expect an annual decrease in hospital admissions of about 20 persons out of 5.5 million across Greater Toronto, one three-hundredth the number estimated by Toronto Public Health (2004).

Koop and Tole (2004) found that BMA 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 insignificance 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. If the data provide only weak support for the selected model relative to models with very different variable choices, the model selection estimates, by ignoring this information, will appear more precise than they actually are. Even when more sophisticated residual models are used (e.g. clustering) to better approximate the underlying population, the failure to consider model uncertainty yields standard deviations that are likely too small. We would argue that BMA is the preferred statistical methodology and, thus, that any researcher should report the BMA results when advising policymakers.

In the expanded tables, available in the Supplementary Information, many of the city dummies are consistently significant, indicating that including key variables to reflect city characteristics is insufficient to control for fixed differences across cities.

We mentioned above the concern that many recent studies use data that are sampled from the mid-1980s onward. We re-did the regression in Table 3 using only data from January 1985 through March 1994, leaving out the city and monthly dummies and the income and GDP measures. The results are not shown but are available on request. If we introduce pollutants one-at-a time we can find a positive and significant effect on admissions from CO, where the coefficient appears relatively large (0.22). When all five pollutants are included, the CO coefficient becomes insignificant and NO_2 and TSP are negative and significant. Consequently, this finding mainly serves as an example of how a positive and significant relationship between pollution and illness can be found in a data set with some digging, but may not be robust to a change in modeling technique nor an extension of the data back in time.

5. Conclusions

In this paper, we have addressed three deficiencies in epidemiological modeling of the air pollution-health relationship. Using a new, long-term multi-city Canadian data set we have been able to test the robustness of results estimated on shorter, more recent data sets. Using a richer suite of economic and social covariates, as well as other exogenous factors, we have addressed in part the concerns of omitted variable bias and missing confounders. Using Bayesian Model Averaging we have addressed the concern that results may be sensitive to model selection.

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 levels observed from 1974 to 1994 had a

detrimental effect on either excess hospital admissions or time spent in hospital, for the Canadian cities comprising our data set, but we find consistent evidence that lower smoking rates lead to fewer admissions and shorter stays. We also find evidence that, all else equal, regions with higher Gross Domestic Product tend to have higher admission rates. These findings add an important dimension to the existing literature, leading us to conclude that the methodological issues addressed herein are pertinent for characterizing the health impacts of ambient air pollution.

We also illustrated the danger that incomplete modeling efforts could yield apparent pollution-health correlations that are not robust to reasonable variations in estimation methods. Model selection methods applied to a subset of the data, or without use of the appropriate socioeconomic controls, can (for example) yield an apparently significant health effect from increased carbon monoxide levels, but such effects change sign and/or become insignificant upon application of more complete empirical methods. By contrast we showed that the effects of smoking rates remain robust across different methods,

Our examination of data back to the early 1970s was motivated in part by the observation that air pollution was much higher then compared to today. Even still we did not find evidence of health effects at the pollution levels observed in Canada in recent decades. Epidemiological and clinical evidence suggests, however, that health effects must be observable at some level of pollution inhalation, suggesting the possible existence of a nonlinearity or threshold effect. The existence of such thresholds could be investigated by expanding the sample to include some of the most heavily-polluted Third World cities, such as Beijing, Tianjin, Calcutta, Delhi, Mexico City and Rio di Janiero, in which pollution levels can be well over double the maximum levels observed in Canadian cities (World Bank 1998). The challenge is that it can be extremely difficult to get comparable data on health indicators and smoking rates in low-income countries, hence such a comparison would have to be limited to cities where reasonable hospital admission or mortality data are available.

The issues addressed in this paper are complex and will no doubt invite further examination. Directions for future work include: modeling hospital admissions using a count data framework, treatment of possible spatial autocorrelation in nearby cities, partitioning results by different age groups, and consideration of nonlinear effects using quadratic forms.

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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 $y = (y_1,.., y_T)'$. K is the number of potential explanatory variables (including lags and a linear time trend) and these 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 number k_r of the K potential explanatory variables (i.e. the columns of *X*), α is the regression constant and β_r is a k_r×1 vector of coefficients on these explanatory variables. 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:

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

where g_r is a scalar. 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 using 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.

Table 1: Summary Statistics

Variable	Obs	Mean	Std. Dev.	Min	Max
CO	2530	1.0	0.6	0.0	7.1
TSP	2599	59.8	33.5	0.0	439.0
SO2	2142	5.6	5.7	0.0	63.0
NO2	2287	22.4	8.2	0.0	81.0
O3	2321	17.0	7.5	0.7	46.0
Air Pressure	2639	97.5	3.8	88.2	102.6
Windspeed	2640	14.6	3.2	4.6	28.7
Mean Temperature	2640	5.2	10.8	-26.5	22.9
Max Temperature	2640	20.2	10.2	-8.3	40.6
Min Temperature	2640	-9.2	13.7	-45.6	12.8
Smokers	2640	0.4	0.1	0.2	0.6
Average Income	2640	$33,790	$3,322	$26,327	$42,929
Real GDP	2640	$126,074	$112,140	$12,555	$362,939
Admissions per 100,000	2640	27.2	11.5	5.1	107.3

Table 2a: Air Pollution sample means and max values by city

	Carbon monoxide		TSP		Sulphur dioxide		Ozone		NOx	
City	mean	max	mean	max	mean	max	mean	max	mean	max
Calgary	0.97	3.8	98.18	439	3.03	10	17.73	40	26.30	44
Edmonton	0.99	2.3	65.34	166	1.99	6	16.80	46	28.01	56
Halifax	1.17	4.4	30.40	61	7.31	27	20.42	36	14.15	48
London	0.65	2.4	68.93	199	5.63	20	20.28	42	22.40	39
Montreal	1.14	2.6	71.05	245	11.54	58	15.42	40	26.67	48
Ottawa	1.46	4.6	53.34	156	8.22	63	15.47	32	23.26	36
Regina	0.95	4.0	37.70	283	1.19	2	19.56	41	19.53	55
Saskatoon	0.54	2.2	57.03	247	1.08	2	18.32	30	14.87	27
Toronto	1.59	7.1	65.45	147	6.64	21	15.85	39	27.19	46
Vancouver	1.30	3.5	49.29	118	5.59	26	11.69	24	23.07	36
Winnipeg	0.76	2.1	58.06	231	1.54	9	17.16	43	16.57	81

Table 2b: Admissions and Stays sample means and max values by city

	Admissions per 100,000		Length of stay per 100,000	
City	mean	max	mean	max
Calgary	22.4	38.1	9.4	188.5
Edmonton	30.2	54.3	9.0	186.5
Halifax	33.5	60.8	7.7	86.5
London	33.4	107.3	9.0	131.0
Montreal	16.6	30.2	8.7	19.5
Ottawa	21.0	36.6	8.0	176.4
Regina	46.5	83.6	6.4	13.0
Saskatoon	30.6	72.2	6.7	17.9
Toronto	19.2	51.0	11.9	685.0
Vancouver	21.4	32.9	8.3	76.0
Winnipeg	21.3	40.5	7.9	23.4

Table 3: Coefficient Estimates Using Ordinary Least Squares. Dependent Variable is Admissions per capita (t-statistic in parentheses). Column headings refer to model specification: see text for explanation. **Bold** denotes *t* statistic > 1.96, * denotes *t* statistic greater than 1.

Variable	BasicOLS	Cluster	CityMonth	TimeTrend
CO	0.013	0.013	-0.073*	**-0.096**
	(0.64)	(0.31)	(-1.91)	(-2.26)
TSP	**-0.118**	**-0.118**	-0.022	**-0.056**
	(-5.42)	(-2.67)	(-0.82)	(-2.14)
SO2	**-0.054**	-0.054*	-0.032*	-0.035*
	(-2.32)	(-1.26)	(-1.18)	(-1.25)
NO2	0.033*	0.033	0.032*	0.019
	(1.32)	(0.73)	(1.21)	(0.71)
O3	**-0.093**	**-0.093**	-0.030	-0.039
	(-4.15)	(-2.05)	(-0.67)	(-0.93)
Air Pressure	**-0.308**	**-0.308**	**0.437**	**0.511**
	(-12.66)	(-6.50)	(2.10)	(2.46)
Windspeed	**0.122**	**0.122**	0.013	-0.009
	(5.79)	(2.81)	(0.52)	(-0.33)
Temperature	**0.075**	0.075*	**0.161**	**0.187**
	(2.76)	(1.67)	(3.28)	(4.08)
Smokers	**0.088**	**0.088**	**0.097**	**0.071**
	(5.12)	(2.30)	(4.27)	(2.93)
Income	**-0.310**	**-0.310**	**0.190**	0.131*
	(-12.26)	(-6.12)	(2.50)	(1.53)
Real GDP	**0.047**	0.047*	**-0.615**	**-0.358**
	(2.39)	(1.07)	(-4.46)	(-2.13)
Time Trend				**-1.769**
				(-2.38)
Const	**-0.104**	**-0.104**	**-0.755**	-1.238*
	(-5.74)	(-2.05)	(-4.57)	(-1.37)
N	1909	1909	1909	1909

R^2	0.21	0.21	0.66	0.67
BIC	4298.48	4298.48	2856.01	2832.81

Table 4: Point Estimates of Each Coefficient Using BMA. Dependent Variable is Admissions per capita (posterior standard deviations in parentheses) Point estimates denoted with * are one standard deviation from zero; **bold**-faced denotes two standard deviations from zero.

	Pollutants included as explanatory variables					
Pollutants	All	CO	TSP	SO2	NO2	O3
CO	-.058* (.048)	-.039* (.036)	--	--	--	--
CO-lag	-.037 (.047)	-.030 (.035)	--	--	--	--
TSP	-.041* (.036)	--	-.008* (.008)	--	--	--
TSP-lag	-.011 (.024)	--	-.001 (.007)	--	--	--
SO2	-.011* (.008)	--	--	-.004 (.053)	--	--
SO2-lag	-.012 (.025)	--	--	**-.070** (.029)**	--	--
NO2	**.053 (.017)**	--	--	--	-.010 (.006)	--
NO2 -lag	.051* (.036)	--	--	--	.000 (.004)	--
O3	-.001 (.019)	--	--	--	--	-.017 (.028)
O3-lag	-.008* (.007)	--	--	--	--	--
Income	.004 (.020)	**.110 (.050)**	**.114 (.047)**	.001 (.010)	**.233** (.041)**	**.170** (.037)**
GDP	**.151 (.057)**	.008 (.024)	.008 (.024)	.004 (.020)	.003 (.014)	.001 (.008)
Smoking	**.130 (.020)**	.060* (.049)	**.064 (.014)**	**.120 (.017)**	**.150 (.014)**	**.130 (.018)**
Sample Size	1889	2536	2617	2131	2295	2329

Table 5: Point Estimates of Each Coefficient Using Model Selection. Dependent Variable is Admissions per capita (posterior standard deviations in parentheses) Note: Entries of "----" for the Model selection case indicate that the corresponding pollutant was not included in the model selected as best.

	Pollutants included as explanatory variables					
Pollutants	All	CO	TSP	SO2	NO2	O3
CO	--	**-.068 (.016)**	--	--	--	--
CO-lag	**-.092 (.020)**	--	--	--	--	--
TSP	**-.066 (.019)**	--	--	--	--	--
TSP-lag	--	--	--	--	--	--
SO2	--	--	--	--	--	--
SO2-lag	--	--	--	**-.077 (.019)**	--	--
NO2	--	--	--	--	--	--
NO2 -lag	**.066 (.020)**	--	--	--	--	--
O3	--	--	--	--	--	--
O3-lag	--	--	--	--	--	--
Income	--	**.140 (.029)**	**.119 (.031)**	--	**.229 (.038)**	**.181 (.034)**
GDP	.014 (.028)	--	--	--	--	--
Smoking	**.138 (.017)**	**.058 (.014)**	**.067 (.014)**	**.116 (.016)**	**.150 (.014)**	**.127 (.015)**
Sample size	1889	2536	2617	2131	2295	2329

Table 6: Point Estimates of Each Coefficient Using BMA, Dependent Variable is Patient Days per capita (posterior standard deviations in parentheses)

	Pollutants included as explanatory variables					
Pollutants	All	CO	TSP	SO2	NO2	O3
CO	**-.027 (.013)**	-.003 (.015)	--	--	--	--
CO-lag	-.016 (.030)	**-.078 (.027)**	--	--	--	--
TSP	-.004 (.014)	--	.000 (.004)	--	--	--
TSP-lag	-.002 (.009)	--	-.000 (.003)	--	--	--
SO2	-.006 (.019)	--	--	-.004 (.014)	--	--
SO2-lag	-.021 (.035).	--	--	-.004 (.015)	--	--
NO2	.007 (.021)	--	--	--	.000 (.004)	--
NO2 -lag	.005 (.017)	--	--	--	-.000 (.004)	--
O3	-.004 (.015)	--	--	--	--	-.013 (.027)
O3-lag	-.001 (.009)	--	--	--	--	-.006 (.010)
Income	-.001 (.012)	-.011 (.031)	-.010 (.029)	**-.170 (.007)**	.002 (.013)	.000 (.006)
GDP	**.140 (.061)**	.006 (.021)	.011 (.027)	.008 (.031)	**.147 (.045)**	.040 (.053)
Smoking	**.120 (.022)**	.000 (.003)	.000 (.002)	**.080 (.027)**	**.012 (.002)**	**.096 (.020)**
Sample size	1889	2536	2617	2131	2295	2329

Table 7: Point Estimates of Each Coefficient Using Model Selection, Dependent Variable is Patient Days per capita (posterior standard deviations in parentheses) Note: Entries of "----" for the Model selection case indicate that the corresponding pollutant was not included in the model selected as best.

	Pollutants included as explanatory variables					
Pollutants	All	CO	TSP	SO2	NO2	O3
CO	--	--	--	--	--	--
CO-lag	--	**-.078 (.019)**	--	-	--	--
TSP	--	--	--	--	--	--
TSP-lag	--	--	--	--	--	--
SO2	--	--	--	--	--	--
SO2-lag	--	--	--	--	--	--
NO2	--	--	--	--	--	--
NO2 -lag	--	--	--	--	--	--
O3	--	--	--	--	--	--
O3-lag	--	--	--	--	--	--
Income	--	--	--	**-.206 (.021)**	--	--
GDP	**.125 (.032)**	--	--	--	**.151 (.037)**	--
Smoking	**.123 (.021)**	--	--	**.088 (.020)**	**.119 (.019)**	**.095 (.019)**
Sample size	1889	2536	2617	2131	2295	2329

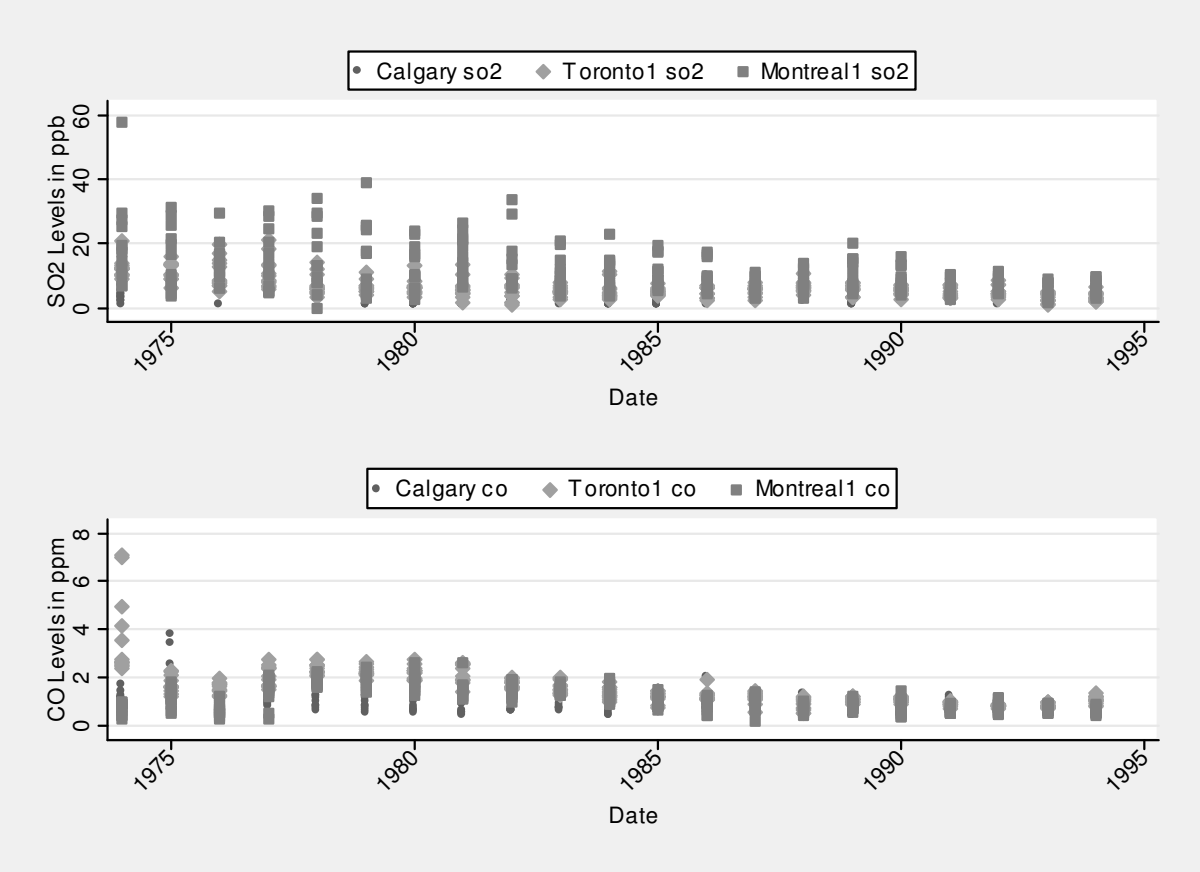

Figure 1. Air pollution levels January 1974 to December 1994, grouped by year. Legend: so2=sulphur dioxide, co=carbon monoxide;

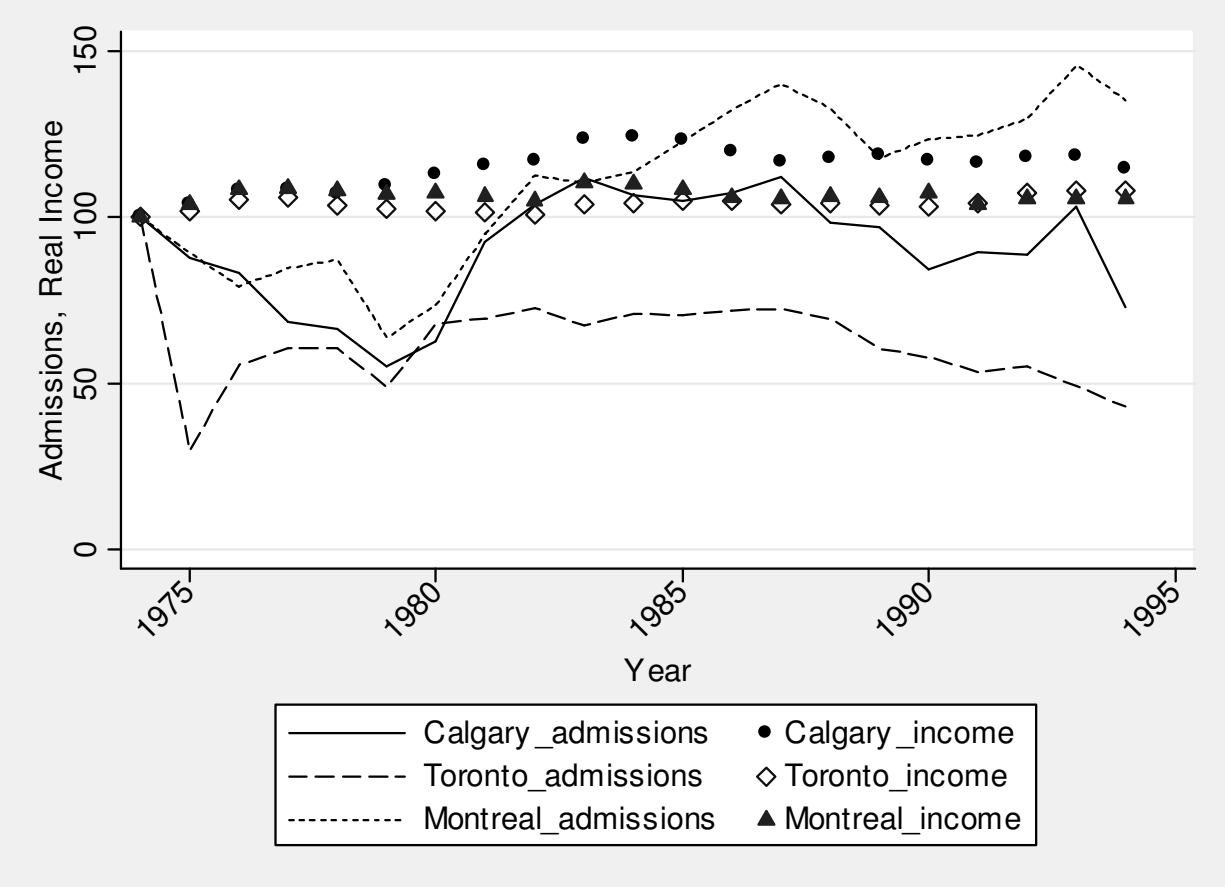

Figure 2. Hospital admissions per capita (lines) and real income (markers) for Calgary, Toronto and Montreal 1974 to 1994, annual averages. All data scaled to begin at 1974=100.

Supplementary Information: Detailed Empirical Results

This Supplement 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 (i.e. number of months for which data is available in each city) differs due to missing values; different pollutants have different numbers of missing values.

Table S1: Point Estimates of Each Coefficient Using BMA. Dependent Variable is Admissions per capita (posterior standard deviations in parentheses)						
	Pollutants included as explanatory variables					
Pollutants	All	CO	TSP	SO2	NOX	O3
CO	-.058* (.048)	-.039* (.036)	--	--	--	--
CO-lag	-.037 (.047)	-.030 (.035)	--	--	--	--
TSP	-.041* (.036)	--	-.008* (.008)	--	--	--
TSP-lag	-.011 (.024)	--	-.001 (.007)	--	--	--
SO2	-.011* (.008)	--	--	-.004 (.053)	--	--
SO2-lag	-.012 (.025)	--	--	**-.070** (.029)**	--	--
NOX	**.053** (.017)**	--	--	--	-.001 (.006)	--
NOX-lag	.051* (.036)	--	--	--	.000 (.004)	--
O3	-.001 (.019)	--	--	--	--	-.017 (.028)
O3-lag	-.008* (.007)	--	--	--	--	--
Weather Variables						
Pressure	.043 (.112)	**.181** (.077)**	.039 (.097)	-.051 (.126)	.081 (.117)	-.099 (.129)
Press-Lag	.082 (.151)	.066 (.128)	.096 (.140)	-.020 (.097)	.110 (.126)	.090 (.127)
Windspeed	-.008 (.021)	-.004 (.013)	**-.046** (.013)**	.000 (.005)	-.000 (.005)	-.000 (.005)
Wind-lag	-.007 (.019)	-.006 (.016)	.003 (.009)	-.000 (.004)	-.003 (.001)	-.002 (.009)
Temp(mean)	.004 (.030)	.024 (.063)	.016 (.053)	.001 (.002)	.017 (.054)	.002 (.002)
T(mean)-lag	.040	.002	.003	.008	.054	.017

	(.061)	(.002)	(.046)	(.003)	(.065)	(.039)
Temp(max)	-.001 (.001)	-.000 (.007)	-.000 (.007)	-.001 (.010)	-.000 (.008)	-.000 (.006)
T(max)-lag	.000 (.014)	-.000 (.005)	-.000 (.008)	.000 (.099)	-.000 (.014)	-.000 (.015)
Temp(min)	**.179**** **(.058)**	**.166**** **(.067)**	**.177**** **(.057)**	**.182**** **(.056)**	**.159**** **(.079)**	**.209**** **(.044)**
T(min)-lag	.024 (.047)	.002 (.009)	.001 (.008)	.006 (.020)	.009 (.027)	.003 (.015)
Other Variables						
Income	.004 (.020)	**0.11**** **(.050)**	**0.11**** **(.047)**	.001 (.010)	**.233**** **(.041)**	**.170**** **(.037)**
GDP	**.151**** **(.057)**	.008 (.024)	.008 (.024)	.004 (.020)	.003 (.014)	.001 (.008)
Smoking	**.130**** **(.020)**	.060* (.049)	**.064**** **(.014)**	**.120**** **(.017)**	**.150**** **(.014)**	**.130**** **(.018)**
Trend	**-.218**** **(.061)**	**-.304**** **(.028)**	**-.258**** **(.029)**	**-.147**** **(.035)**	-.000 (.007)	-.091* (.048)
January	**-.081**** **(.011)**	-.011 (.035)	-.052* (.041)	-.000 (.085)	-.012 (.042)	-.055 (.081)
February	**-.283**** **(.100)**	**-.209**** **(.053)**	**-.209**** **(.049)**	**-.225**** **(.084)**	**-.196**** **(.086)**	**-.234**** **(.070)**
March	-.071 (.106)	-.004 (.019)	-.001 (.011)	-.031 (.066)	-.023 (.059)	-.017 (.048)
April	-.006 (.048)	-.000 (.007)	.000 (.010)	.004 (.033)	.039 (.083)	.017 (.053)
May	**-.253**** **(.100)**	**-.239**** **(.054)**	**-.194**** **(.058)**	**-.220**** **(.092)**	**-.189**** **(.010)**	**-.174**** **(.097)**
June	**-.761**** **(.084)**	**-.684**** **(.056)**	**-.632**** **(.054)**	**-.687**** **(.097)**	**-.705**** **(.079)**	**-.676**** **(.076)**
July	**-1.273**** **(.091)**	**-1.101**** **(.059)**	**-1.069**** **(.058)**	**-1.134**** **(.106)**	**-1.230**** **(.080)**	**-1.18**** **(.076)**
August	**-1.291**** **(.089)**	**-1.065**** **(.057)**	**-1.023**** **(.056)**	**-1.154**** **(.103)**	**-1.120**** **(.080)**	**-1.146**** **(.068)**
September	-.002 (.041)	-.000 (.013)	.000 (.011)	.018 (.068)	-.002 (.002)	.000 (.013)
October	.011 (.045)	.003 (.017)	.003 (.016)	.0210 (.077)	.002 (.002)	.002 (.014)
November	.013 (.045)	.010 (.032)	.007 (.026)	.044 (.079)	.006 (.002)	.003 (.017)
Calgary	-.271 (.300)	-.577* (.394)	-.369 (.415)	-.204 (.387)	-.063 (.234)	-.112 (.273)
Edmonton	.074 (.182)	.161 (.249)	.296* (.276)	.392* (.235)	**.801**** **(.015)**	**.694**** **(.178)**

Halifax	**.973** (.187)	**.729** (.103)	**.655** (.103)	**.966** (.124)	**1.042** (.089)	**.894** (.104)
London	.357* (.202)	**.407** (.079)	**.477** (.088)	.891* (.991)	**.712** (.072)	**.611** (.098)
Montreal	**-1.240** (.284)	**-1.266** (.123)	**-1.260** (.126)	**-.727** (.160)	**-1.073** (.123)	**-1.191** (.164)
Ottawa	**-.693** (.273)	**-.603** (.090)	**-.644** (.092)	-.187* (.146)	**-.450** (.101)	**-.536** (.013)
Regina	**2.609** (.217)	**2.356** (.163)	**2.383** (.168)	**2.257** (.198)	**2.259** (.119)	**2.352** (.145)
Saskatoon	**.623** (.190)	**1.032** (.135)	**1.079** (.138)	**.547** (.225)	**.865** (.111)	**.852** (.137)
Toronto	**-.848** (.237)	**-.839** (.113)	**-.883** (.113)	**-.425** (.114)	**-.816** (.111)	**-.861** (.133)
Vancouver	**-.694** (.031)	**-.687** (.155)	**-.757** (.153)	**-.192** (.168)	**-.871** (.159)	**-.839** (.174)
Sample size	1889	2536	2617	2131	2295	2329

Table S2: Point Estimates of Each Coefficient Using Model Selection. Dependent Variable is Admissions per capita (posterior standard deviations in parentheses)						
	Pollutants included as explanatory variables					
Pollutants	All	CO	TSP	SO2	NOX	O3
CO	--	**-.068** (.016)	--	--	--	--
CO-lag	**-.092** (.020)	--	--	--	--	--
TSP	**-.066** (.019)	--	--	--	--	--
TSP-lag	--	--	--	--	--	--
SO2	--	--	--	--	--	--
SO2-lag	--	--	--	**-.077** (.019)	--	--
NOX	--	--	--	--	--	--
NOX-lag	**.066** (.020)	--	--	--	--	--
O3	--	--	--	--	--	--
O3-lag	--	--	--	--	--	--
Weather Variables						
Pressure	--	--	--	--	--	**.258** (.048)

Press-Lag	--	--	**.274** (.042)**	--	**.211** (.045)**	--
Windspeed	--	--	--	--	--	--
Wind-lag	--	--	--	--	--	--
Temp(mean)	--	--	--	--	--	--
T(mean)-lag	--	--	--	--	**.078** (.025)**	--
Temp(max)	--	--	--	--	--	--
T(max)-lag	--	--	--	--	--	--
Temp(min)	**.204** (.029)**	**.198** (.020)**	**.200** (.020)**	**.219** (.024)**	**.174** (.030)**	**.241** (.021)**
T(min)-lag	--	--	--	--	--	--
Other Variables						
Income	--	**.140** (.029)**	**.119** (.031)**	--	**.229** (.038)**	**.181** (.034)**
GDP	.014 (.028)	--	--	--	--	--
Smoking	**.138** (.017)**	**.058** (.014)**	**.067** (.014)**	**.116** (.016)**	**.150** (.014)**	**.127** (.015)**
Trend	**-.191** (.029)**	**-.311** (.024)**	**-.247** (.027)**	**-.151** (.024)**	--	**-.107** (.032)**
January	**-.233** (.064)**	--	--	--	--	--
February	**-.396** (.062)**	**-.207** (.047)**	**-.207** (.046)**	**-.221** (.054)**	**-.194** (.052)**	**-.232** (.049)**
March	**-.218** (.059)**	--	--	--	--	--
April	--	--	.--	--	--	--
May	**-.321** (.060)**	**-.251** (.050)**	**-.198** (.048)**	**-.256** (.057)**	**-.206** (.055)**	**-.232** (.052)**
June	**-.790** (.064)**	**-.695** (.053)**	**-.632** (.051)**	**-.724** (.061)**	**-.718** (.057)**	**-.716** (.055)**
July	**-1.273** (.068)**	**-1.108** (.056)**	**-1.062** (.054)**	**-1.177** (.065)**	**-1.245** (.060)**	**-1.227** (.058)**
August	**-1.263** (.066)**	**-1.060** (.054)**	**-1.016** (.053)**	**-1.191** (.063)**	**-1.216** (.059)**	**-1.730** (.056)**
September	--	--	--	--	--	--
October	--	--	--	--	--	--
November	--	--	--	--	--	--
Calgary	**-.473** (.067)**	**-.862** (.072)**	--	--	--	--

Edmonton	--	--	**.550** (.525)	**.501** (.063)	**.846** (.051)	**.757** (.055)
Halifax	**1.051** (.090)	**.767** (.066)	**.590** (.071)	**.905** (.061)	**.102** (.067)	**.833** (.076)
London	**.438** (.058)	**.362** (.051)	**.514** (.070)	**.885** (.058)	**.715** (.060)	**.573** (.076)
Montreal	**-1.094** (.074)	**-1.271d** * (.055)	**-1.328** (.109)	**-.786** (.065)	**-1.082** (.099)	**-1.278** (.119)
Ottawa	**-.570** (.059)	**-.607** (.053)	**-.668** (.088)	**-.243** (.059)	**-.451** (.086)	**-.595** (.097)
Regina	**2.441** (.104)	**2.239** (.062)	**2.522** (.061)	**2.338** (.091)	**2.277** (.069)	**2.424** (.078)
Saskatoon	**.507** (.012)	**.970** (.961)	**1.197** (.067)	**.638** (.117)	**.881** (.075)	**.924** (.090)
Toronto	**-.741** (.061)	**-.890** (.067)	**-.896** (.010)	**-.453** (.058)	**-.815** (.101)	**-.920** (.011)
Vancouver	**-.512** (.069)	**-.694** (.072)	**-.856** (.119)	**-.278** (.062)	**-.883** (.129)	**-.929** (.128)
Sample size	1889	2536	2617	2131	2295	2329

Table S3: Point Estimates of Each Coefficient Using BMA, Dependent Variable is Patient Days per capita (posterior standard deviations in parentheses)						
	Pollutants included as explanatory variables					
Pollutants	All	CO	TSP	SO2	NOX	O3
CO	**-.027** (.013)	-.003 (.015)	--	--	--	--
CO-lag	**-.016** (.030)	**-.078** (.027)	--	--	--	--
TSP	-.004 (.014)	--	.000 (.004)	--	--	--
TSP-lag	-.002 (.009)	--	-.000 (.003)	--	--	--
SO2	-.006 (.019)	--	--	-.004 (.014)	--	--
SO2-lag	-.021 (.035).	--	--	-.004 (.015)	--	--
NOX	.007 (.021)	--	--	--	-.000 (.004)	--
NOX-lag	.005 (.017)	--	--	--	-.000 (.004)	--
O3	-.004 (.015)	--	--	--	--	-.013 (.027)
O3-lag	-.001	--	--	--	--	-.006

	(.009)					(.010)
Weather Variables						
Pressure	.017 (.159)	-.060 (.127)	.050 (.128)	-.009 (.131)	**-.417**** **(.180)**	-.315* (.238)
Press-Lag	.300* (.188)	**.389**** **(.127)**	**.347**** **(.147)**	.026 (.080)	-.045 (.142)	-.046 (.201)
Windspeed	.000 (.007)	.000 (.003)	.000 (.003)	.001 (.008)	.000 (.003)	.000 (.004)
Wind-lag	.000 (.005)	.000 (.003)	.000 (.003)	.000 (.005)	.000 (.003)	.000 (.004)
Temp(mean)	.000 (.008)	.000 (.007)	.000 (.007)	.000 (.016)	.000 (.008)	.000 (.009)
T(mean)-lag	.000 (.012)	-.001 (.010)	-.001 (.010)	.000 (.008)	.000 (.007)	.000 (.006)
Temp(max)	-.003 (.016)	.000 (.006)	.000 (.006)	-.003 (.018)	.000 (.005)	.000 (.006)
T(max)-lag	-.003 (.016)	-.003 (.012)	-.003 (.012)	-.001 (.009)	.000 (.007)	-.002 (.010)
Temp(min)	.000 (.011)	.000 (.007)	.000 (.007)	.004 (.021)	.000 (.006)	.002 (.012)
T(min)-lag	-.005 (.028)	-.002 (.001)	-.002 (.011)	.000 (.006)	.000 (.007)	.000 (.008)
Other Variables						
Income	-.001 (.012)	-.011 (.031)	-.010 (.029)	**-.170**** **(.007)**	.002 (.013)	.000 (.006)
GDP	**.140**** **(.061)**	.006 (.021)	.011 (.027)	.008 (.031)	**.147**** **(.045)**	.040 (.053)
Smoking	**.120**** **(.022)**	.000 (.003)	.000 (.002)	**.080**** **(.027)**	**.012**** **(.002)**	**.096**** **(.020)**
Trend	**-.461**** **(.045)**	**-.757**** **(.030)**	**-.698**** **(.027)**	**-.462**** **(.050)**	**-.461**** **(.033)**	**-.504**** **(.035)**
January	.000 (.016)	.000 (.011)	.000 (.009)	-.001 (.013)	.000 (.013)	.000 (.001)
February	**-.135**** **(.120)**	**-.111**** **(.097)**	**-.155**** **(.088)**	**-.197**** **(.098)**	**-.158**** **(.102)**	**.149**** **(.105)**
March	.001 (.018)	.001 (.014)	.000 (.009)	.000 (.010)	.000 (.001)	.003 (.024)
April	.037 (.080)	.019 (.051)	.017 (.047)	.009 (.038)	.031 (.067)	.092 (.112)
May	-.009 (.040)	-.021 (.055)	-.001 (.012)	-.003 (.025)	-.015 (.047)	-.012 (.046)
June	**-.207****	**-.291****	**-.250****	**-.260****	**-.286****	**-.250****

	(.124)	**(.067)**	**(.006)**	**(.087)**	**(.070)**	**(.108)**
July	**-.479**** **(.103)**	**-.506**** **(.067)**	**-.489**** **(.060)**	**-.474**** **(.077)**	**-.559**** **(.070)**	**-.522**** **(.084)**
August	**-.503**** **(.108)**	**-.498**** **(.067)**	**-.469**** **(.060)**	**-.494**** **(.076)**	**-.512**** **(.070)**	**-.481**** **(.080)**
September	.005 (.053)	-.045 (.025)	-.009 (.015)	.000 (.020)	.000 (.015)	.000 (.017)
October	.152* (.130)	.059 (.086)	.015 (.045)	.009 (.105)	.177* (.108)	.110* (.107)
November	.004 (.032)	.000 (.008)	.000 (.011)	.000 (.012)	.000 (.010)	.000 (.011)
Calgary	-.215 (.540)	-.065 (.350)	**-.411**** **(.181)**	-.159 (.512)	**-2.150**** **(.324)**	**-1.73**** **(.632)**
Edmonton	-.127 (.321)	-.021 (.204)	-.016 (.100)	-.089 (.309)	**-1.047**** **(.213)**	**-.777**** **(.377)**
Halifax	.214 (.250)	.004 (.064)	-.006 (.003)	.060 (.147)	**.794**** **(.117)**	**.525**** **(.208)**
London	-.011 (.049)	-.013 (.054)	.000 (.015)	**.336**** **(.146)**	.002 (.024)	.006 (.038)
Montreal	**-1.551**** **(.216)**	**-1.81**** **(.117)**	**-1.772**** **(.094)**	**-1.131**** **(.183)**	**-.957**** **(.112)**	**-1.138**** **(.210)**
Ottawa	**-1.320**** **(.150)**	**-1.25**** **(.090)**	**-1.28**** **(.076)**	**-.833**** **(.150)**	**-.945**** **(.085)**	**-.949**** **(.143)**
Regina	**1.649**** **(.248)**	**2.132**** **(.166)**	**2.030*** **(.112)**	**1.269**** **(.194)**	**.718**** **(.142)**	.088 (.259)
Saskatoon	.212 (.260)	**1.519**** **(.146)**	**1.135**** **(.102)**	.067 (.175)	.010 (.080)	.068 (.190)
Toronto	**-1.391**** **(.126)**	**-1.310**** **(.095)**	**1.359**** **(.080)**	**-.842**** **(.194)**	**-1.152**** **(.076)**	**-1.153**** **(.110)**
Vancouver	**-.674**** **(.239)**	**-.690**** **(.138)**	**-.737** **(.106)**	**-.049**** **(.189)**	-.016 (.093)	-.085 (.226)
Sample size	1889	2536	2617	2131	2295	2329

Table S4: Point Estimates of Each Coefficient Using Model Selection, Dependent Variable is Patient Days per capita (posterior standard deviations in parentheses)

	Pollutants included as explanatory variables					
Pollutants	All	CO	TSP	SO2	NOX	O3
CO	--	--	--	--	--	--
CO-lag	--	**-.078**** **(.019)**	--	-	--	--
TSP	--	--	--	--	--	--
TSP-lag	--	--	--	--	--	--

SO2	--	--	--	--	--	--
SO2-lag	--	--	--	--	--	--
NOX	--	--	--	--	--	--
NOX-lag	--	--	--	--	--	--
O3	--	--	--	--	--	--
O3-lag	--	--	--	--	--	--
Weather Variables						
Pressure	--	--	--	--	**-.480** (.060)**	**-.446** (.060)**
Press-Lag	**.387** (.039)**	**.429** (.024)**	**.402** (.024)**	--	--	--
Windspeed	--	--	--	--	--	--
Wind-lag	--	--	--	--	--	--
Temp(mean)	--	--	--	--	--	--
T(mean)-lag	--	--	--	--	--	--
Temp(max)	--	--	--	--	--	--
T(max)-lag	--	--	--	--	--	--
Temp(min)	--	--	--	--	--	--
T(min)-lag	--	--	--	--	--	--
Other Variables						
Income	--	--	--	**-.206** (.021)**	--	--
GDP	**.125** (.032)**	--	--	--	**.151** (.037)**	--
Smoking	**.124** (.021)**	--	--	**.088** (.020)**	**.192** (.019)**	**.095** (.019)**
Trend	**.426** (.029)**	**-.755** (.029)**	**-.698** (.026)**	**-.430** (.027)**	**-.461** (.032)**	**-.507** (.030)**
January	-- --	-- --	-- --	-- --	-- --	-- --
February	**-.204** (.068)**	**-.166** (.056)**	**-1.852** (.051)**	**-.245** (.063)**	**-.196** (.061)**	**-.213** (.060)**
March	--	--	--	--	--	--
April	--	--	--	--	--	--
May	--	--	--	--	--	--
June	**-.251** (.069)**	**-.310** (.057)**	**-.259** (.056)**	**-.277** (.066)**	**-.292** (.062)**	**-.314** (.061)**
July	**-.497** (.069)**	**-.524** (.057)**	**-.495** (.056)**	**-.485** (.066)**	**-.561** (.063)**	**-.567** (.061)**
August	**-.524** (.069)**	**-.519** (.057)**	**-.476** (.056)**	**-.506** (.065)**	**-.514** (.062)**	**-.518** (.061)**
September	--	--	--	--	--	--

October	**.193** (.068)**	--	--	--	**.204** (.062)**	--
November	--	--	--	--	--	--
Calgary	--	--	--	--	**-2.199** (.194)**	**-1.871** (.182)**
Edmonton	--	--	--	--	**-1.080** (.143)**	**-.829** (.128)**
Halifax	--	--	--	--	**.811** (.087)**	**.518** (.068)**
London	--	--	--	**.403** (.059)**	--	--
Montreal	**-1.644** (.081)**	**-1.840** (.073)**	**-1.790** (.072)**	**-1.045** (.063)**	**-.941** (.080)**	**-1.034** (.071)**
Ottawa	**-1.384** (.079)**	**-1.259** (.064)**	**-1.280** (.062)**	**-.764** (.059)**	**.939** (.069)**	**-.886** (.065)**
Regina	**1.614** (.129)**	**2.154** (.078)**	**2.041** (.074)**	**1.214** (.104)**	**.703** (.101)**	**.783** (.096)**
Saskatoon	--	**1.181** (.075)**	**1.151** (.072)**	--	--	--
Toronto	**1.451** (.072)**	**-1.329** (.063)**	**-1.365** (.061)**	**-.764** (.061)**	**-1.148** (.066)**	**-1.105** (.063)**
Vancouver	**.796** (.080)**	**-.727** (.065)**	**-.757** (.064)**	--	--	--
Sample size	1889	2536	2617	2131	2295	2329

Table S5: Point Estimates of Each Coefficient Using Model Selection among Models which Include at Least one Pollutant. Dependent Variable is Admissions per capita (posterior standard deviations in parentheses) Note: Entries of "----" for the Model selection case indicate that the corresponding pollutant was not included in the model selected as best.

	Pollutants included as explanatory variables					
Pollutants	All	CO	TSP	SO2	NOX	O3
CO	--	**-.068** (.016)**	--	--	--	--
CO-lag	**-.092** (.020)**	--	--	--	--	-
TSP	**-.066** (.019)**	--	-.028* (.016)	--	--	--
TSP-lag	--	--	--	--	---	--
SO2	--	--	--	--	--	--
SO2-lag	--	--	--	**-.077** (.019)**	--	--
NOX	--	--	--	--	-.015 (.018)	--
NOX-lag	**.066** (.020)**	--	---	--	--	--
O3	--	--	--	--	--	**-.041** (.017)**
O3-lag	--	--	--	--	--	--
Income	--	**.140** (.029)**	**.139** (.028)**	--	**.231** (.038)**	**.175** (.034)**
GDP	**.139** (.028)**	--	--	--	--	--
Smoking	**.138** (.017)**	**.058** (.014)**	**.060** (.013)**	**.151** (.016)**	**.151** (.014)**	**.123** (.016)**
Sample size	1889	2536	2617	2131	2295	2329