AIR POLLUTION AND HEALTH: THE EFFECT OF SHORT RUN CHANGES IN AIR QUALITY ON RESPIRATORY HOSPITALIZATIONS

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Recent changes in standards for air pollution are highly contentious and represent stringent constraints on economic activity. This paper presents evidence that directly informs this debate. By linking daily pollution to hospital admissions for municipalities across Ontario, I study the impact of air pollution at levels below those historically considered. Results indicate that particulate matter has a significant effect on respiratory health of children but that ozone and carbon monoxide have little effect on respiratory hospitalizations for all age groups. I estimate that a one standard deviation increase in the average five-day levels of fine PM leads to a 2.7 percent increase in respiratory admissions of children under the age of 6 and a 4.4 percent increase in the respiratory admissions of children 6 to 19 years. Considering the decreases in fine PM that have occurred over the 2000-2006 period, these estimates imply a decrease of 1,805 child hospitalizations and an approximate savings of $16 million.

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The protection of human health is one of the main justifications for environmental control policy; and the general consensus that exposure to elevated levels of air pollution is harmful to health has led environmental agencies to set standards for air quality. Because the link between pollution and health is believed to be immediate, agencies actively monitor day-to-day levels of air pollution and will issue public warnings when high levels of pollution are expected to occur within a locale. Such actions impose strict constraints on industrial and other economic activity and because such restrictions can be costly, it is important to consider the magnitude of the health gain associated with pollution abatement.

These issues are apparent in the recent controversy over air quality standards for particulate matter pollution (PM). Recently the Environmental Protection Agency (EPA) has revoked air quality standards for coarse PM in favor standards on fine PM. These changes are motivated by the lack of evidence of negative health effects from coarse PM and rely on the premise that fine PM is more harmful to health because particles are small enough to be inhaled (EPA 2010a). Under this premise variability in coarse PM may mask underlying variation in harmful fine particles. On the other hand, changes to PM standards impose more stringent controls on source emissions since fine particulate matter more closely tracks emissions from industrial and other economic activity. For instance, areas with higher levels of source emissions but low levels of natural source dust would no longer compare as well to areas with high levels of predominately natural source particles. Not surprisingly, several industrial organizations and state governments contested the fine PM standards initially proposed in 1997. It was not until ten years later that the standards on fine PM were promulgated and replaced those of coarse PM.

Despite the potential implications for industrial and other economic activity, little is known about the impact of fine particulate matter and health. Fine PM is thought to have more serious implications for asthma and other respiratory conditions because fine particles are more likely to be inhaled deep into the lungs; however, there is little evidence to confirm this in a natural setting.

This study is the first to address this question on a large-scale. In order to assess the linkage between fine particulate matter and health, I compare daily changes in fine PM to daily changes in respiratory hospitalizations within locale, month and year for a large number of small geographic locales over seven years. This framework allows for control of potential confounders in the estimation of causal effects of fine PM. For instance, it is often difficult to disentangle the effects of pollution from other characteristics of highly polluted areas. Higher pollution levels tend to be observed in areas with higher population density, lower incomes, and poorer access to medical care and these factors can be associated with lower average health regardless of air pollution. By including year, month and small area fixed effects, I can flexibly control for these and other unobservable differences that change across area or that change across area, season and year.

This study contributes to the literature on the health impact of pollution in three respects. First, it uses a rich set of data on hourly measurements of several criteria air pollutants and the key pollutant, fine particulate matter. Recent literature has found very little health impact from the coarser measures of pollution (Neidell 2004; Currie and Neidell 2005; Currie, Neidell and Schmieder 2009; Lleras-Muney 2009). In contrast, earlier work by Chay and Greenstone (2003a) found a significant effect of coarse particulate matter on infant mortality. However, these results may, in fact, be driven mostly by changes in smaller particles from source emissions since variation in particulate matter was induced by the 1980-82 recession. If it is only fine particulate pollution that is harmful to health, then the health impact estimated from courser measures in other contexts may be subject to measurement error causing estimates to be attenuated downward. This premise should be considered before drawing the conclusion that particulate matter pollution has little impact on health. On the other hand, if results on fine PM are consistent with the more recent evidence on coarse PM, then regulatory standards for fine PM may impose unnecessary restrictions on economic activity. Evidence from the current setting will directly inform this debate.

A second contribution of this study is that it focuses on several types of air pollution (fine PM, ozone and carbon monoxide) that are low when compared to those historically considered and low when compared to national standards set by the EPA. Given that set standards tend to imply that pollution below certain thresholds has little effect on health, it is important to test this hypothesis in a natural setting. By studying pollution levels in the recent decade in the region of Ontario Canada, I examine a natural laboratory where individuals are exposed to much lower levels of pollution. For instance, recent average ozone (O3) and carbon monoxide (CO) levels in Ontario are at one-third the levels studied in previous decades and are well below current EPA standards (Neidell 2004; Currie and Neidell 2005; Currie, Neidell and Schmieder 2009; Moretti and Neidell 2009; Lleras-Muney 2010; EPA 2010a). By including pollutants such as O3 and CO in the analysis, I not only control for the separate effects of these pollutants, I can also test the effect of each pollutant at much lower levels than previously observed. Since environmental standards for these forms of air pollution have become increasingly stringent in recent years, evidence from this study is relevant to the current debate.

A further benefit of studying the region of Ontario is that most of the population is located in areas of depressed topography near the great lakes where pollution tends to settle. This causes this region to be a net importer of air pollution and combined with the fact that areas to the north and south of Ontario have large differences in average pollution, leads to highly variable pollution levels depending on wind direction and daily weather conditions. The fact that, on any given day, high or low levels of air pollution may be delivered to the area, provides ample variation in pollution within relatively short periods of time and aids in the identification of the immediate respiratory effects of air pollution.

A third contribution of this study is that it uses a rich and comprehensive set of data on health outcomes for the entire population, available from the Hospital Morbidity Database (HMDB). This allows for more objective measures of respiratory illness on a daily basis (compared to retrospective measures), but importantly, it also allows me to isolate admissions for chronic conditions where respiratory problems are indicated as a co-contributor. Previous evidence has focused on respiratory hospitalization counts where the respiratory diagnosis is the primary reason for hospitalization. However, almost half of all admissions listing a respiratory illness have an underlying chronic condition listed as the primary diagnosis. Without detailed patient diagnosis data, these admissions would appear in aggregate data as unrelated to respiratory health and analysis would miss the deleterious effect of air pollution on individuals with non-respiratory chronic conditions. Furthermore, all patients in the HMDB have universal hospital and physician coverage, which allows a limited role for differential access to hospital services in explaining pollution effects.

The estimates in this study show that, at the relatively low levels of pollution experienced in the recent decade, there is little effect of O3 and CO on the respiratory hospitalizations of all age groups. On the other hand, there is an important role for fine PM in the respiratory health of children. I estimate that a one standard deviation increase in the average five-day levels of fine PM leads to a 2.7 percent increase in respiratory admissions of children under 6 and a 4.4 percent increase in the respiratory admissions of children 6 to 19 years.

These results are robust to several sensitivity analyses. They do not appear to be explained by multi-collinearity in the different measures of pollution, due to mortality displacement outside versus inside the hospital, due to admission displacement in the timing of hospitalizations, or because hospital resource constraints bind on more polluted days. The results also do not appear to reflect avoidance behavior. If individuals take steps to avoid pollution at higher levels, then the interpretation of the pollution effect does not reflect *exposure* to pollution but rather the *behavioral* effect of high pollution, not including the cost of avoidance. To test whether this is the case, I include air quality alerts of high O3 or fine PM issued by the Ontario Ministry of the Environment and determine if the impact of air pollution differs when individuals are warned of poor air quality. Adjusting for this possibility does not change the results of this analysis.

This paper is laid out as follows: Section 2 provides background on pollution, its implications for health, and discusses previous literature. Section 3 describes the various data sources. Section 4 describes methodology. Section 5 discusses results. Section 6 contains conclusions.

1 Air Pollution, Health Implications and Previous Literature

Several types of air pollution are thought to have negative effects on health. In this section, I briefly describe different types of air pollution and their mechanisms for affecting health.[1] Particulate matter air pollution can take many forms and is made up of a mixture of extremely small particles and liquid droplets. Different measurements of particulate matter vary based on the size of particles measured. Early EPA regulation for particulate matter was based on total suspended particulates (TSP) but the standard was replaced in 1987 with PM10, which limits the measured particles under 10 micrometers. In 1997, the EPA revised standards to include fine PM 2.5 (particles under 2.5 micrometers). The difference between smaller and larger particles is not trivial.[2] For instance, PM10 is primarily formed by the break up of large solids or droplets while PM2.5 is more specifically formed by combustion processes and atmospheric reactions. This means that while PM10 is mainly composed of suspended soil or street dust, PM2.5 is mainly composed of chemicals, metals and other organic compounds. Not only are these smaller, more harmful particles more likely to be inhaled deep into the lungs, there are also other implications relevant to respiratory health. For example, the atmospheric half-life of PM2.5 is days and average travelling distance is in the range of 100 to 1000 kms. For PM10 the atmospheric half-life is much more brief at minutes or hours and average travelling distance is much shorter at 1 to 10 kms (EPA 2004). Furthermore, the health

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[1] The Environmental Protection Agency website is a good source for a complete description of air pollution: http://www.epa.gov/air/urbanair/

[2] The EPA states: "Fine and coarse particles differ not only in size but also in formation mechanisms, sources, and chemical, physical, and biological properties. They also differ in concentration-exposure relationships, dosimetry (deposition and retention in the respiratory system), toxicity, and health effects" (EPA 2004)

effect of PM2.5 has stronger implications for environmental policy since fine PM is more closely tied to production, vehicle emissions and industrial processes. Because of this, the implementation of EPA standards for PM2.5 were challenged by several industries, state governments and other organizations before they were finally promulgated at the end of 2006 and replaced those of PM10 (EPA 2010a). Current EPA standards for PM2.5 are 35 µg/m3.[3]

Ozone (O3) is a gas formed by chemical reactions between oxides of nitrogen and volatile organic compounds in the presence of heat and sunlight. Ozone is not usually emitted directly into the air (implying that policy implications of the negative effects of O3 are less straightforward than emission abatement) and since sunlight and hot weather cause O3 to form at a higher rate, it is known as a summertime air pollutant. Ozone inhaled into the lungs acts as an irritant and reduces lung function (EPA 2006). The EPA developed national standards for O3 in 1979. Until 1997, the standard was set at 0.12 parts per million (ppm). Standards were revised to 0.08 ppm in 1997 and revised downward to 0.075ppm in 2008 (EPA 2006).[4]

Carbon monoxide (CO) is a colorless, odorless gas formed primarily through hydrocarbon fuel combustion and industrial processes. The highest levels of CO generally occur during the colder months of the year when there is an increase in fuel combustion for heating and

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[3] For compliance, this means that average 24-hour concentrations of PM2.5 must not exceed 35 µg/m3. The former standards for PM10 were 150 µg/m3 (note that magnitudes of PM2.5 and PM10 are not directly comparable).

[4] For compliance, this means that the 8-hour average should not exceed 0.075 ppm.

when temperature inversions are more frequent (EPA 2010b).[5] Carbon monoxide reduces oxygen delivery to the body's organs and tissues. Because of this, the effects of CO at low levels are most serious for those who suffer from chronic conditions such as heart disease. High levels of CO can cause vision problems, reduced ability to work or learn, reduced manual dexterity, and difficulty performing complex tasks. At extremely high levels, CO is poisonous, causing immediate death (EPA 2010b). The current EPA standard for CO is 9 ppm.[6]

Several studies have demonstrated strong correlations between air pollution and mortality (Pope et al, 2002; Samet at al. 2000). Accounting for cross-sectional unobservables, two seminal studies have linked air pollution to infant health using exogenous time series variation in pollution in order to identify the effects on infant mortality (Chay and Greenstone 2003a; Currie and Neidell 2005). Chay and Greenstone (2003a) studied the effects of TSP in the early 1980s and found that a 1 percent decrease in TSP levels led to a .35 percent decrease in infant mortality. Currie and Neidell (2005) studied the impact of CO, O3 and PM10 in the 1990s and found that CO had a significant impact on infant mortality with little impact from PM10 and O3.

More recent studies have addressed the sorting or avoidance concerns that may be present in the Chay and Greenstone, and Currie and Neidell studies. For instance, families may take steps to move or avoid high levels of pollution, especially if they are in poor respiratory health. Lleras-Muney (2009) overcomes this concern by looking at the effect of

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5 During a temperature inversion, CO becomes trapped near the ground beneath a layer of warm air.

6 For compliance, this means that the 8-hour average should not exceed 9 ppm.

air pollution on military children. Since military relocations are largely out of the control of individual families, sorting based on local pollution levels is not a concern in this context. For military children during the early 1990s, O3 had the largest affect on respiratory hospitalizations (a 1 standard deviation increase in O3 levels increased respiratory admissions by 8 to 23 percent). CO and PM10 appeared to have no significant effect on respiratory admissions.

In addition to relocating based on high pollution levels, individuals may also engage in short run avoidance of outdoor pollution (Neidell 2009; Neidell and Moretti 2009). For instance, Neidell (2009) showed that individuals actively avoid going outdoors when public warnings of high O3 levels are issued. Accounting for this yields significantly larger effects of the impact of O3 on respiratory admissions (a 0.01ppm increase in O3 leads to a 3 percent increase in admissions versus 1 percent without controls for avoidance). Neidell and Moretti (2009) use an instrumental variable design based on boat traffic in the ports of Los Angeles and found that unexpected changes in O3 levels led to large effects on respiratory admissions. Neither of these studies evaluates CO, fine PM, or coarse PM.

All previous studies have analyzed populations subject to much higher levels of pollution than experienced in the recent decade. Furthermore, of the studies that evaluated particulate matter, only Chay and Greenstone (2003a) found that changes in TSP had any effect on health. The current analysis contributes to the field by addressing the question of whether fine particulate matter has a role in respiratory health. If it is true that only smaller more respirable particles impact health, then previous analysis on courser measures of PM could suffer attenuation bias. Given recent changes in restrictions for air pollution, the results presented have direct implications for environmental policy.

2 Data Sources

Data on ambient air pollution come from air quality monitors maintained by the Ontario Ministry of the Environment (OME) and are supplemented with data from National monitoring stations maintained by Environment Canada's National Air Pollution Surveillance System (NAPS).[7] Each monitoring station collects hourly measurements of air pollutants and I use these data to construct average daily measures of pollution for each municipality and pollutant type.[8] Data for CO, PM2.5 and O3 are available from 2000 to 2006. Since monitors may be discontinued or introduced to a given location over the sample period, I only use monitors that exist continuously throughout the period.[9]

To assign daily pollution measures to each municipality, I first match each monitor location to the central location of each municipality and calculate the distance between each pairing.[10] I then calculate the weighted average pollution level for each municipality using monitors within 30km of the municipality centre, and using the inverse distance to the monitor as the weight. With this method, I am able to construct a pollution level for each day and each municipality. A small number of municipalities are left unmatched under this definition, however unmatched municipalities comprise less than 8 percent of

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[7] The NAPS network is a partnership of provincially maintained pollution networks and nationally maintained monitors. In general, national monitors are located in more rural locales than provincial monitoring stations.

[8] While a subset of monitors collects information for five criteria pollutants (CO, PM2.5, O3, NO2, and SO2), the most comprehensive dataset can be constructed using CO, PM2.5 and O3. In more limited samples using all pollutant data, NO2 and SO2 were not found to have an impact on respiratory admissions.

[9] Results are qualitatively unchanged when I use all monitor data.

[10] Municipalities are a unit of geography within Ontario consistent with census subdivisions. Some municipalities have been combined corresponding to the definition of a census-consolidated subdivision. Under this definition, municipalities are combined if the population is under 100,000 and the border is surrounded on more than half its perimeter by another municipality. Geographic locations of municipalities are shown in Figure 1.

the population and lie primarily in the northern and rural regions of the province. Figure 1 shows the central location of each municipality and Figure 2 shows the locations of each pollution monitor. The municipality centroid is found by calculating the geographic centre of all postal code locations within each municipality. To the extent that postal code size and location are assigned based on population density, the calculated centroid will be weighted towards areas with higher density and will more accurately depict pollution levels for individuals within the municipality.

Summary statistics for each pollution variable are shown in Table 1. The first column of the table shows daily means for each pollutant. Average pollution over this time period is low compared to historical levels and national standards. For instance, while average O3 levels are 26 ppb here, previous studies have examined time periods and locations with ozone levels in the range of 40 to 60 ppb (Neidell 2004; Currie and Neidell 2005; Moretti and Neidell 2009; Lleras-Muney 2010). Lleras-Muney (2009) and Currie and Neidell (2005) studied average CO concentrations in the range of 1-2 ppm and average PM10 in the range of 27-40 ug/m3, while average levels in the current context are 0.5 ppm and 21 ug/m3, respectively.[11] Moreover, national ambient air quality standards set by the EPA are 80 ppb for ozone, 9 ppm for carbon monoxide and 150 ug/m3 for PM10 (replaced by standards of 35 ug/m3 for PM2.5 in 2006).[12] If pollution is harmful only above a certain threshold (as air quality standards tend to imply), then the current setting is a good place to

[11] PM10 averages are based on a limited sample in the years 2000-2002.

[12] For comparison, standards set by Ontario Ministry of the Environment are: O3=80ppb averaged over 1 hour, CO=13ppm averaged over 8 hours, PM2.5=30 ug/m3 based on 24 hour 98th percentile averaging over three years.

test this hypothesis.[13] Table 1 also indicates that pollution levels have either maintained or decreased over time. The most substantial decline is in CO, although PM2.5 also decreased over this period.

The pollutants display seasonal patterns as shown for the Toronto municipality in Figure 3. Since Ozone forms through chemical reactions of NOx and VOCs in the presence of heat and sunlight, higher concentrations of ozone are observed throughout the summer months. On the other hand, CO and PM2.5 are largely formed through direct emissions and are tied to seasonal patterns in fuel combustion and weather patterns that trap emissions through temperature inversions. Figure 3 indicates substantial variability within month as shown by the within year-month standard deviation around the monthly average for the Toronto municipality. For all pollutants studied, the within municipality-month variation exceeds the between municipality-month variation leaving ample variation to identify the effects of pollution (shown in column 3 and 4 in Table 1).

Table 1 also displays descriptive statistics for respiratory disease. Data on respiratory admissions are collected from the Hospital Morbidity Database (HMDB) held by the Canadian Institute for Health Information. The HMDB is a comprehensive, administrative database of acute hospital admissions in Canada. The database contains patient demographic information, residential postal location, diagnosis information, and admission-discharge dates for each patient record. Data are available from 2000 to 2006 and contain over 400,000 admissions per year. For each admission abstract, all

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[13] In 2001 the U.S. Supreme Court clarified that EPA cannot consider costs of reduction of emissions in setting standards and must only consider benefits of air quality (largely measured in terms of human health). This would imply set thresholds for pollution levels do not incorporate trade offs in production and other costs.

contributing diagnoses are coded using the International Disease Classification system. This allows me to capture admissions for individuals admitted with a primary respiratory problem or with any primary chronic condition exacerbated by a respiratory problem. The advantage of using the HMDB is that it is a comprehensive source of objective health data recording the residential location of patients and dates and times of respiratory problems. This allows me to merge key measures of respiratory health (asthma, pneumonia, bronchiolitis, etc.) to pollution levels for each municipality at each date.

Descriptive statistics for respiratory health are given in the bottom of Table 1. Over the sample period, and average of 2.3 patients were admitted with a primary respiratory diagnosis each day, per municipality. An average of 4.3 patients were admitted with either a primary respiratory diagnosis or a respiratory co-morbidity. Of these admissions an average of 0.5 end in death. Table 1 also shows admissions by acute and chronic diagnosis. I divide the respiratory admissions by type to explore the differential impact of pollution for different age groups.[14]

The table indicates that respiratory admissions are trending downwards and Figure 3 confirms this for the Toronto municipality. It is also clear that there is seasonality in respiratory admissions. This seasonality appears to match seasonality in CO quite closely, both in trend and in the timing of the peaks and troughs. At times, respiratory health follows the patterns of PM2.5, while O3 and respiratory admissions appear negatively

[14] Respiratory problems are listed under the category of J in the ICD 10 diagnosis classification system. I divide acute respiratory diagnosis (J00-22) into pneumonia counts (J12-18) and into other acute diagnosis (J0-6 and J20-22). Chronic admissions are listed under J30-47. In the analysis, I also highlight evidence for chronic asthma (J45) the lead chronic disease in children.

correlated. CO and PM2.5 tend to peak in the fall and winter months while O3 is highest in the summer.

There are several alternative explanations for seasonal variation in respiratory admissions. For instance, some infectious diseases flourish in cold weather contributing to increases in respiratory admissions. On the other hand, cold weather may also lead to increases in CO and PM2.5 through increases in fuel combustion for heating. To correctly assess the impact of pollution on respiratory health, the seasonal effect of pollution must be disentangled from alternative explanations. I deal with this problem in two ways. The first is to include municipality-month fixed effects to account directly for seasonality and for seasonal differences across municipalities (for instance, seasonal differences in the great lakes district versus the northern capital region). The second is to account for weather patterns, which are key determinants of air pollution and are also shown to have independent effects on respiratory health (Deschênes and Moretti 2009). [15] If weather is not independently related to respiratory health, then including weather controls will reduce the degree of independent variation in pollution, making it difficult to precisely estimate its effect. On the other hand, if weather has an independent effect on respiratory health as evidence suggests, then including weather in the set of control variables removes this potential source of bias.

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[15] A third way to deal with seasonal bias from infectious disease would be to separate infectious disease counts from other respiratory admissions. Three potential problems arise: (1) the separation is not always clear-cut (pneumonia, for example, may develop from infectious disease or other respiratory irritants); (2) the health impact of infectious disease may be exacerbated by air pollution (an effect I wish to detect); and (3) independent seasonality in infectious disease is not the only source of potential bias.

Weather data are collected from the National Climate Data and Information Archive (NCD). These data contain the official climate and weather observations maintained by Environment Canada. For each weather station, I collect the average daily precipitation, relative humidity, wind speed and extreme daily maximum and minimum temperatures. Sunshine is an important factor in the creation of photochemical ozone. To get a measure of average sun cover throughout the day, I use hourly reports of weather conditions and parse the report based on descriptions of either clear weather or alternative weather. I define average sun cover as the proportion of hours that clear weather is observed throughout the daytime. Weather station data are matched to municipalities in the same way as pollution monitor data.

The last set of information I include is public information on current air quality released to the public by Air Quality Ontario (AQO) from the Ontario Ministry of the Environment. The AQO program reports on current and expected air quality for 37 air quality alert regions (municipalities nest within the alert regions). The AQO assesses air quality by calculating an index of standardized pollutant levels for each alert region. The air quality index (AQI) takes on the maximum value of standardized pollutant levels (pollutants included are O3, PM2.5, CO, NO2, SO2, and total reduced sulphur compounds). Air quality is assessed based on the AQI scale (0 to 31 is good quality, 32 to 49 is moderate quality and 50+ is poor quality).[16] In addition to providing information on current air quality, AQO has a main function in alerting the public when air quality is expected to be poor in any particular alert region. Even though high levels of any one of the pollutants

[16] Complete information about the standardization of each pollutant and the calculation of the air quality index is available at: http://www.airqualityontario.com/press/faq.cfm#aqi_calc

could cause a poor AQI alert, as an empirical matter O3 or PM2.5 are the only contributory sources. Ozone levels expected above 50ppb or PM2.5 levels expected above 22ug/m3 lead to an air quality alert for the upcoming day. Information is distributed based on the Smog Advisory Notification Network, which is a predetermined chain of notification for various media, public works, school, health and transportation institutions (OME 2010). From the OME, I collect data on all alerts issued for each day and region over the years 2000 to 2006 and match them by date to each municipality.

Panel 1 of Table 2 displays mean daily admissions, pollution levels, alert status and weather ranked by average municipality pollution levels (here, municipalities are ranked for descriptive comparisons only). To get a common measure of pollution, I standardize all pollutant measures and take the average of the pollutant z-scores for each municipality. This provides a rough rank of the level of pollution in each municipality and allows me to compare municipalities with high and low levels of pollution along other dimensions. The first two columns show summary statistics for municipalities with average pollution levels below and above the median. For example, municipalities above the median have CO levels almost two times as large as those below the median. In addition to higher mean levels of pollutants, municipalities also have a higher incidence of extreme values in PM2.5 and O3 (not shown) and this difference is reflected in higher incidence of air quality alerts.

Figure 4 depicts municipalities spatially by pollution rank. The darkest circles represent pollution levels in the top quartile and lighter circles mark each successive quartile. The highest levels of pollution most often occur around the areas with the densest population and areas of depressed topography around the great lakes.[17]

The differences in lower and higher polluted areas are also reflected in respiratory health: less polluted areas have lower admissions under all categories of respiratory illness (as shown in Table 2) and these results are uniform across age groups (not shown). On the other hand, a simple comparison of the admission rate for external accidents shows that high pollution municipalities have higher admission rates for this arguably unrelated type of hospitalization.[18] The remainder of Table 2 shows that these differences are partly due to the fact that average pollution levels are correlated with other characteristics that are themselves related to health. Panel 2 depicts socioeconomic differences among high and low pollution municipalities by matching average municipal pollution levels to census variables in the 2001 and 2006 censuses. Municipalities with above median levels of pollution have larger average populations (almost double), higher population density per square kilometer, and higher dwelling values. This reflects the fact that above median municipalities tend to be more urban while below median municipalities tend to be more rural. Other differences are not as large. For example, the percent of 25 to 64 year olds with post secondary education is 56 percent in higher pollution municipalities and 54 percent in lower pollution municipalities. Lone parent families and children living under the low-income cutoff are more common in less polluted areas but the difference is small.

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17 Pollutants are often trapped in valleys and basins (called pollution "airsheds").

18 Traffic accidents are not included in the external accident rate. This type of accident is more likely to occur under conditions of high traffic congestion, a factor that is also related to pollution levels.

Trends in characteristics over census years are relatively similar for high and low pollution municipalities.

3 Methods

Extensive monitoring of fine particulate matter and other air pollutants along with rich data on hospital admissions over time and space make it appear straightforward to relate fine PM to respiratory health. However, the effect of particulate matter on respiratory health may be confounded by several factors. First, while PM levels vary across locale, several other factors (arguably related to health) also tend to vary across locale. For example, individuals with poor health may sort into less polluted areas. On the other hand, disadvantaged or low SES families may be more likely to live in more polluted areas while wealthier families can afford to live in less polluted areas.[19] A second concern is that seasonal variation in fine PM may map closely to seasonal variation in respiratory health regardless of the health effect of fine PM and these unrelated seasonal effects in respiratory health may also differ across locale. For instance, population density may be related to high winter PM levels through heating and fuel combustion and higher winter contagion probabilities for infectious respiratory disease. Both could explain higher respiratory admissions. A third concern in studying the effect of fine PM is that individuals may avoid pollution when pollution levels are high so that estimates do not reflect the biological effect of fine PM but instead the behavioral effect not accounting for the costs of avoidance.

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[19] The relationship between socioeconomic status and health has been well documented. Furthermore, work by Chay and Greenstone (2005) shows that PM is a public good capitalized into housing prices.

To estimate the effect of air pollutants on health and address these issues, I employ a model that accounts for unobserved differences between municipalities and seasonal differences across municipalities by including municipality-month fixed effects. Controlling for municipality-month effects eliminates some of the variation in pollution, but ample variation remains within municipality and month to identify the effect on respiratory health (as shown in Table 1). In addition to including municipality-month fixed effects, I also rule out unobserved year-to-year differences that are specific to municipalities by including municipality-year fixed effects. This will account for year-to-year differences in respiratory health across municipality. To address the third concern listed above, I control for the public announcement of poor air quality and test whether pollution estimates differ when adjusting for this type of avoidance behavior. Previous studies have demonstrated that pollution avoidance behavior has bearing on estimation of pollution effects. For instance, Neidell (2009) shows that accounting for avoidance behavior induced from public alerts of high O3, leads to significantly higher estimates of the biological effect of ozone. Following this line of reasoning, I allow the effect of fine PM to vary by whether the OME issues an alert for expected poor air quality in the upcoming days. If individuals are able to avoid poor air when they are warned, and poor air affects health, then the impact of air pollution should be less when there is a warning of poor air. To incorporate the above, I start with the following model for the impact of fine particulate matter:

$$h_{jt} = \sum_{l=0}^{4}[\alpha_l A_{jt-l} + \beta_l PM_{jt-l} + \delta_l PM_{jt-l} A_{jt-l} + Q'_{jt-l}\Gamma_{l0} + W'_{jt-l}\Gamma_{l1}] + H'_t\Gamma_2 + \eta_{jm} + \sigma_{jy} + \varepsilon_{jt}$$

where the j subscript refers to municipality, the t subscript refers to the day, the l subscript refers to the lag value, and e is the error term. h denotes the number of respiratory admissions; PM refers to fine PM; A is a dummy variable indicating whether an air quality alert is issued in the Air Quality Ontario alert region;[20] Q refers to other air quality measures (CO and O3) and interactions of CO and O3 with the alert indicator; W refers to weather control variables; H are day of the week, holiday and ICD classification dummies (from ICD 9 to 10); η are municipality-month fixed effects; and σ are municipality-year fixed effects.

The main coefficients of interest are the sets of estimates for fine PM. For ease of exposition, I report the average impact across pollution lags (i.e. the long run propensity), evaluated at the average value of A. I include a contemporaneous effect and four lags of pollution to maintain comparability with previous evidence suggesting effects up to four days after exposure (Neidell 2009; Moretti and Neidell 2009).[21] Identification of pollution effects comes from the comparison of changes in pollution and changes in respiratory admissions within municipality-month and within municipality-year. As shown in Table 2, there are other differences among high and low pollution municipalities that could also explain differences in respiratory health. Furthermore, as Figure 3 shows, there may be other seasonal or trend factors driving both respiratory health and pollution. By including the set of fixed effects for municipality, month and year, I can account for all factors that

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[20] Municipalities nest within 32 alert regions.

[21] I explore the lag structure further by varying the number of leads and lags and find that results support this assumption. Furthermore, there is no evidence of negative higher order lead/lag effects implying that results are likely not driven by admission displacement in the timing of hospitalization.

are held fixed within municipality-month and municipality-year. This will not account for factors that are correlated with pollution exposure and respiratory health and change within municipality in the shorter-term. Weather and avoidance behaviors are important examples of such factors. To test whether confounding from these factors is a likely concern, I control for a broad set of weather variables (precipitation, relative humidity, wind speed, maximum/minimum temperatures and sky clarity) and AQO air quality warnings (a factor shifting demand for pollution avoidance). In the next section, I first present results comparing estimates for fine and coarse PM and then I provide evidence on the effect of fine PM after controlling air quality warnings.

4 Results

4.1 Main Findings

I start by comparing the estimated effects of fine and coarse particulate matter. Only a limited sample of monitoring stations between 2000-2002 collected data on both types of PM. Table 3 reports results for coarse and fine PM for the limited sample, over each age group. The reported coefficients are interpreted as the change in admissions arising from a 1ug/m3 increase in 5-day average PM levels. I also report standardized coefficients (where changes are measured in standard deviations) to compare results across pollutant type.

Both coarse and fine PM appear to have little impact on the respiratory admissions for adults. This is true in the limited sample comparing both types of PM and is also true for fine PM using the full sample over all years and municipalities. This evidence fits with previous literature finding very little impact for adults at even higher levels of pollution (Chay, Dobkin and Greenstone 2003).

Because the respiratory system continues to develop in early childhood, there is reason to expect that children are the most vulnerable to pollution and recent work has focused primarily on this age group. In Panel 1, the point estimate for coarse PM (without controls for fine PM) appears to be largest for children under 6 but the effect statistically insignificant. Once controls for PM2.5 are included (see Panel 2) the point estimates on coarse PM remains small and insignificant while PM2.5 appears to have a large effect. This is consistent with Panel 3 results, which shows estimates for fine PM for the full sample. For older children (6-19), a one standard deviation increase in five-day average PM2.5 levels leads to a change in respiratory admissions of 1.6 percent of a standard deviation (a 4.9 percent increase from the mean).[22] The impact of PM2.5 on children under 6 is somewhat smaller at a 2.3 percent increase from the mean (imprecisely measured).

The results presented in Table 3 do not account for the possibility of pollution avoidance. To test whether avoidance is an important concern, I compare results for all three types of pollutants with and without controls for air quality alerts. Because air quality warnings attempt to limit exposure to pollution, the effect of pollution may be smaller when individuals are warned of poor air quality.

4.1.1 Estimates Without Alert Controls

Panel 1 of Table 4 displays the results for all pollutant variables for different age groups without the inclusion of alert variables. Results show very little impact of all three

[22] Given a standard deviation of 7.05 for PM2.5 and an average admission rate of 0.23, the percent change is calculated as 0.00161*7.05/0.23

pollutants on the respiratory admissions of adults. Coefficient estimates are small and in all cases, the hypothesis that pollutant impact is zero cannot be rejected at conventional levels.

Aside from fine PM, all estimates for children are imprecise and small in magnitude. Consistent with previous studies, the point estimate for CO is larger for 0-5 year olds than for 6-19 year olds (Neidell 2004). Ozone has a perverse sign in all specifications, a finding that is not uncommon (Lleras-Muney 2009, Neidell 2004, Currie and Neidell 2005).

4.1.2 Estimates With Alert Controls

Estimates of the biological effect of pollutants will be biased downward if individuals respond to information about air quality. In this scenario, not accounting for these changes in avoidance behavior could explain negative pollution estimates. For instance, consider the case where pollution has no biological effect on respiratory health but the response to alert information decreases respiratory admissions. For example, children may engage in less physical activity on alert days and are thus less likely to experience respiratory distress regardless of pollution levels. Under this scenario, air pollution would appear to have a negative effect on respiratory admissions when adjustment for the impact of alerts is not made. Panel 2 tests the possibility that the biological effects of air pollutants are under estimated by directly controlling for warnings of poor air quality to capture the effect of avoidance behavior on pollution estimates.

In all cases the level effect of air quality alert has a negative value (on average admissions decrease on alert days) but this effect is small and insignificant for all ages except 65 and older. The last row of Panel 2 assesses whether coefficient estimates for PM2.5, CO and O3 differ when accounting for alerts. In general, the point estimates are larger, but differences are small. A Hausman test for comparison of estimates between the two models yields no significant difference at conventional levels.

This evidence contrasts to the findings of Neidell (2009), who found that smog alerts in California are strongly related to avoidance behavior and that accounting for this behavioral effect results in much higher estimates of the biological effect of O3. However, results in Neidell (2009) were found at much higher average O3 levels (more than double the levels analyzed here). Additionally, alerts under the California alert system are triggered at 200ppb rather than 50ppb under the AQO system.

The evidence provided here shows very little impact of alert warnings at such low levels of pollution.[23] This may occur because alerts issued at this threshold do not affect behavior, because air pollution is not easily avoided, or because exposure to pollution at these levels has no impact on health.[24] The fact that estimates for PM2.5 are unchanged implies that the effect of fine PM, at least, has a significant impact of the respiratory health of children. For all that follows I include controls for alerts although point estimates are robust to their exclusion.

The results in Table 4 indicate that, while O3 and CO appear to have no effect on respiratory admissions; particulate matter has a significant and deleterious effect on

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[23] This does not imply that there is no impact on respiratory health more generally.

[24] Since high frequency data on avoidance behavior is unavailable, it is difficult to test directly whether air quality alerts are related to avoidance behaviors. To provide some evidence on this issue, I used Google Trends data to construct a weekly time series of search terms related to pollution or the Air Quality Ontario website. Using this measure, I find no differential impact on search terms during either (1) an alert episode or (2) higher levels of ambient pollution. This does not refute the hypotheses that avoidance behavior is related to alerts or pollution levels, but it does provide some evidence that this type of information seeking behavior is not related to alerts or pollution.

children (a one standard deviation change in fine PM increases respiratory admission by 2.7 percent for young children and 4.4 percent for older children). Previous estimates of particulate matter based on PM10 did not yield such conclusions. Chay and Greenstone (2003a) found a significant effect of TSP on infant health, but the TSP changes they used were induced by a recession and may be more representative of changes in small particles. If smaller particles are truly the most harmful, then measurement error in the rougher measures of particulate matter will attenuate coefficient estimates downwards.

4.2 Single Versus Multi Pollutant Models

To further understand the relationship between particulate matter, other pollutants and respiratory health, I decompose the model to include only one pollutant at a time. Pollutant levels are highly correlated and it is possible that this is reflected in the precision of the estimates for CO and O3.[25] Table 5 compares the main set of results from Table 4 to results from models that include only one pollutant at a time. Since results for adults are small and insignificant in all that follows, I focus solely on children. Table 5 indicates that estimates for each pollutant in the multi-pollutant model are similar to those in single-pollutant models.

For respiratory admissions of children under 6, PM2.5 has the largest effect and the single pollutant model yields a similar point estimate with a more precise standard error. For this age group, CO results are similar in magnitude but in either case they are measured imprecisely. The point estimate implies that a one standard deviation increase in CO

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[25] Table 9 shows the correlation matrix for pollution variables.

results in a 0.8 percent standard deviation increase in admissions (2 percent increase from the mean). In both specifications, O3 appears to have no impact on respiratory admissions.

For 6 to 19 year olds, the estimate for PM2.5 is positive, significant, and stable across specification. The results for CO and O3 are small and insignificant. The stability of the estimates across specification provides further evidence that at these pollution levels, CO and O3 have little impact. If they did have an independent effect on respiratory admissions, then the results in the single pollutant PM2.5 model should suffer omitted variable bias. However, the estimate for PM2.5 remains stable in either specification.

4.3 Seasonal Impact

Next I explore the possibility that there are differential effects of pollutants at different points during the year where average pollution levels are higher. The year is broken into three seasons (fall, winter, summer) each with different levels of average pollution. The full year model could mask a potential effect for O3 during summer when pollution levels are highest or when outdoor activity is more common.[26]

Table 6 reports estimates for each season throughout the year. In the fall (September to December) and winter (January to June) seasons, results are very similar to the patterns discussed in the previous section. Particulate matter appears to be the only pollutant that impacts respiratory admissions significantly. Moreover, estimates for all pollutants are similar in the multi versus one at a time models.

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[26] I have also compared estimates over pollution quartiles but I find that this may mask seasonal differences in other behaviors (such as the difference between the school term and summer vacation).

The summer season shows a different pattern. Here, O3 has a positive impact on the hospitalizations of children under 6 while PM2.5 has a significant impact only when it is the only included pollutant. In general, for those under 6, the impact of pollution on respiratory health is larger in seasonal periods when pollution levels are higher. For PM2.5 and O3 this period is during the summer and for CO the period is during the winter. For school age children 6 to 19, the impact of PM2.5 is largest and is stable across season. O3 does not appear to have an impact in any season, but surprisingly CO has a large impact during summer, the period where CO levels are lowest. Since automobile exhaust contributes heavily to both PM2.5 and CO and this type of pollution is lower to the ground, these results could reflect a difference in exposure to outdoor air during summer vacation.

4.4 Type of Illness

Next I explore the impact of pollution over different periods during childhood for chronic and acute respiratory illness. If acute respiratory events caused by exposure to particulate matter in early childhood lead to the development later chronic conditions, then even short run changes in particulate matter may cause chronic respiratory sensitivities throughout childhood. To explore this, I first divide respiratory admissions into chronic asthma (the most common chronic respiratory diagnosis in children) and acute diagnoses (separating acute pneumonia admissions from croup, bronchitis or bronchiolitis admissions). Results are reported in Table 7.

For children under the age of 6, particulate matter has the largest impact on acute diagnosis for croup, bronchitis or bronchiolitis. Bronchiolitis (an inflammation of the smallest air passages of the lungs) is the most commonly diagnosed disease for this age group and category. Older children, on the other hand, are much more likely to be diagnosed with a chronic asthma. For this age group, PM has the largest effect on admissions for asthma.

Early bronchiolitis is believed to be linked with later asthma but it is unknown whether this link exists because children predisposed to developing asthma in later life are more susceptible to bronchiolitis early on or because early episodes of bronchiolitis cause later asthma by inducing long term inflammation (American Academy of Pediatrics 2006). Under the latter case, short-term fluctuations in particulate matter in early life may have longer-term consequences for development of chronic conditions. The timing of the effects found here supports such conclusions but the case for a causal link cannot be resolved in the current context.

4.5 Mortality and Resource Constraints

Results support a link between particulate matter and respiratory health but small and insignificant effects for CO and O3. To confirm these conclusions, I explore two scenarios under which pollution effects may be underestimated. The first scenario is that high levels of pollution may induce excess mortality not reflected in hospital admissions. In this case, if high levels of pollution are more likely to cause out-of-hospital death, then this mortality displacement could bias estimates downward. Table 8 show results for in-hospital mortality from respiratory disease and shows that death is not responsive to air pollution at the levels observed in Ontario. Because most child mortality occurs in hospital and given that pollution does not appear to be related to death in hospital, it is unlikely that mortality displacement is a source of bias.

A second scenario under which pollution effects may be underestimated will occur if hospital resource constraints bind in times of high pollution. If hospital resources are constrained during periods of high pollution this may result in fewer hospital admissions. To assess this possibility, I look at the admission rates for external accidents, an arguably unrelated hospital admission.[27] As Table 8 shows, variation in all three pollutants appears to have no association with admission rates for external accidents. Furthermore, as the last column shows, pollution levels do not affect wait time in the emergency room. In general, pollution appears to have no alternative association with use of hospital resources or processing time.

Lastly, to assess whether the impact of PM is overstated due to admission displacement in the timing of hospitalization. I explore the effect of several leads and lags of fine PM levels. If higher particulate matter "moves forward" admissions that would have taken place regardless, then the estimated impact of fine PM overstates the savings to the cost of respiratory illness. Figure 5 shows that significant impacts of PM are apparent for up to four lags. The effects appear to be more immediate for younger children than for older children. The figure also indicates that leads and lags of PM outside of this range have no effect on admissions. The absence of a strong negative effect indicates that admission displacement is not an explanation for impact of fine PM.

[27] I exclude traffic accidents from the definition of external accidents since this type of accident is more likely to occur under conditions of high traffic congestion, a factor that is also related to pollution levels.

5 Discussion and Conclusions

Environmental regulation for air quality continues to be controversial and pollution standards have become increasingly stringent in recent years. For example, the EPA revised the standard for O3 from 0.08ppm to 0.075ppm in 2008. Meanwhile, the EPA has phased out standards on PM10 citing the lack of evidence on negative health impacts and replaced them with standards for PM2.5 signifying stricter regulation of source emissions. State governments, industry and other organizations challenged the change in policy in the U.S. Court of Appeals for the D.C. Circuit but the new standards were eventually upheld. Currently, standards for PM2.5 are at a maximum of 35 μg/m3 (effective December 17, 2006).

The results in this paper inform this controversy by examining the health impacts of three pollutants observed at average levels well below national standards and historical levels. This study benefits from the fact that a considerable amount of air pollution is imported depending on weather conditions and emissions from elsewhere. This generates a large degree of variability in local pollution levels that can be used to identify the impact on respiratory health. In the results presented, I control for many potential confounders by comparing variation in pollutants to variation in respiratory health within municipality, month and year and including air quality warnings to control for changes in avoidance behavior.

My main finding is that, contrary to previous studies on courser particulate matter; fine particulate matter has a significant effect on the respiratory health of children. Furthermore, in the summer months when O3 it at its highest, children under 6 experience more admissions for respiratory problems as ozone levels fluctuate. On the other hand, at the lower levels of O3 and CO observed in Ontario there appears to be no general impact for these pollutants on the respiratory health of children or adults. It should be noted that this does not imply that pollution at these levels is not related to respiratory health more generally (over the longer term or for less severe health outcomes).

Estimates from this analysis could underestimate the biological effect of pollution as individuals make efforts to avoid high levels of pollution. Since avoidance likely comes with a cost, it is important to account for such a possibility when assessing the benefits of pollution abatement. To test whether the estimated pollution effects are smaller because of avoidance behavior, I compared days with public warnings of poor air quality to days without such warnings. I find that estimates are similar with or without controls for this type of avoidance behavior. This result may occur because behavior is unchanged as a result of air quality alerts, or because air pollution is not easily avoided.[28] Given the similarity of estimates when including controls for alerts, I conclude that this is not an important source of bias. It should be noted that this does not rule out other unobserved sources of short-term avoidance behaviors.[29]

Estimates indicate that fine PM has little impact on mortality but a significant impact on respiratory admissions for children. Early estimates of the effect of TSP indicated a significant impact on infant mortality: a 1 standard deviation decrease in coarse PM led to

[28] Air pollution is not an outdoor phenomenon. Indoor air pollution (and in vehicle air pollution) is often similar to outdoor levels (http://www.epa.gov/iaq/index.html)

[29] Future work can test this premise by comparing the impact of pollution when there are unexpected changes in traffic congestion arising from traffic accidents.

a 8 percent decline in mortality (Chay and Greenstone 2003a). More recent estimates found little impact of PM10 on mortality or respiratory hospitalizations. This study finds that a 1 standard deviation increase in fine PM levels leads to a 2.7 percent increase in admissions for children under 6 and a 4.4 percent increase in the respiratory admission of children age 6 to 19. This represents a yearly increase of 1,181 hospitalizations for children in Ontario. At an average cost of $8,629 per respiratory admission, this adds up to a total annual bill of $10 million. These numbers can also be put into context with the decreases in fine PM that have occurred over the 2000-2006 period. Over this period, average fine PM levels decreased by 1.5 ug/m3. According to estimates, this change eliminated 1,805 child admissions at a savings of $16 million.

I note that these results reflect only the hospitalization cost associated with fine PM and likely underestimates the total cost of PM pollution. For instance, less severe respiratory illness may not be reflected in admission counts, but is a source of higher physician visits, use of other types of primary care, and may be associated with lost work time or school absence (Currie, et al. 2009). Furthermore, early exposure to fine PM may lead to the later development of chronic condition and could represent significant cost to individual health stock, longevity and other economic factors.

A final finding from this paper is that particulate matter impacts the respiratory health of children at different ages in different ways. It primarily affects the acute conditions of croup, bronchitis and bronchiolitis in young children and the chronic condition of asthma in older children. It is unclear whether this occurs because acute diagnosis in younger children is undiagnosed asthma or whether early acute respiratory events predispose children to the later development of asthma. Since the latter possibility has implication for the long-term health trajectory of children growing up in more polluted areas, evidence rectifying these two possibilities is an interesting topic for future research.

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Table 1
Summary statistics for air pollution and respiratory health

Variable	Mean	Std. dev.	Between municipality - month std. dev.	Within municipality - month std. dev.
PM2.5 (ug/m3)	8.108	7.051	2.695	6.515
Trend PM2.5	-0.221			
PM10 (ug/m3)	21.024	10.920	3.965	10.174
Trend PM10	-0.329			
CO (ppm)	0.459	0.337	0.197	0.274
Trend CO	-0.039			
O3 (ppb)	25.794	11.455	7.191	8.917
Trend O3	0.467			
Resp. admission	4.332	11.225	10.810	3.023
Trend resp. admission	0.042			
Resp. admission - MRD	2.332	5.987	5.706	1.811
Trend resp. admission - MRD	0.144			
Resp. death	0.508	1.662	1.488	0.739
Trend resp. death	0.022			
Respiratory (age <6)	0.587	1.631	1.397	0.840
Acute (age <6)	0.394	1.185	0.974	0.675
Chronic (age <6)	0.249	0.802	0.597	0.537
Population (age <6)	6,081	17,551		
Respiratory (age 6-19)	0.223	0.697	0.486	0.499
Acute (age 6-19)	0.088	0.355	0.185	0.303
Chronic (age 6-19)	0.133	0.497	0.308	0.390
Population (age 6-19)	16,811	44,882		
Respiratory (age 21-64)	1.073	2.826	2.554	1.210
Acute (age 21-64)	0.381	1.130	0.930	0.643
Chronic (age 21-64)	0.492	1.401	1.160	0.785
Population (age 21-64)	51,579	158,548		
Respiratory (age >64)	2.493	6.917	6.603	2.062
Acute (age >64)	1.050	3.141	2.893	1.222
Chronic (age >64)	1.230	3.353	3.078	1.332
Population (age >64)	10,579	33,484		

The unit ug/m3 refers to micrograms per cubic meter of air. ppb and ppm refer to parts per billion and parts per million respectively. Trends are calculated as yearly trends. All admission variables are daily averages per municipality and all population variables are averages per municipality as given by the 2006 census. MRD respiratory admissions refers to hospitalizations with respiratory illness labeled the most responsible diagnosis.

Table 2
Comparison of municipalities with the highest and lowest levels of pollution

	Mean		Year trend	
	Pollution level below median	Pollution level above median	Pollution level below median	Pollution level above median
Panel 1 - Summary statistics				
PM2.5 (ug/m3)	8.099	8.116	-0.121	-0.321
CO (ppm)	0.360	0.551	0.004	-0.100
O3 (ppb)	24.352	27.186	0.701	0.220
Respiratory rate	3.199	6.149	-0.194	-0.525
Acute respiratory rate	1.409	2.699	-0.047	-0.117
Chronic respiratory rate	1.652	3.023	-0.128	-0.282
Air quality alert	0.040	0.046	0.010	0.007
Air quality alert for PM2.5	0.014	0.016	0.007	0.007
Air quality alert for O3	0.026	0.029	0.003	0.001
Max. Temp. (Celsius)	12.977	12.835	-0.506	-0.662
Min. Temp. (Celsius)	3.603	3.040	-0.493	-0.540
Precipitation (mm)	2.347	2.290	-0.025	-0.002
Clear (proportion day time hrs)	0.307	0.296	-0.007	-0.001
Accident admission rate	1.935	4.130	0.002	-0.098
Panel 2 - Census variables				
Population	57,699	102,054	646	1,627
Population density (pop/km2)	267.676	357.042	3.906	8.420
Sustainable transit	0.102	0.105	0.002	0.000
Housing costs > 30% of income	0.202	0.222	0.003	0.004
Dwelling value	185,520	236,066	13,466	18,748
Pop. < 17 under LICO	0.141	0.132	-0.001	0.001
Median HH income	54,303	59,580	1,479	1,535
Lone parent families	0.149	0.136	0.001	0.001
High school education	0.814	0.826	0.014	0.014
Post-secondary education	0.542	0.562	0.012	0.011
Observations	122	124		

In Panel 1, the first two columns report daily averages for municipalities above or below the median standardized pollution level and the third and fourth columns report the yearly trend. All hospital admissions means are given as rates per 100,000. Panel 2 includes census information (2001 and 2006 censuses) for each municipality at the census year-municipality level. All values with leading zeros are measured in proportion. Sustainable transit refers to the proportion of the employed who walk, bicycle, or use public transit to get most of the way to work. LICO stands for low income cut off. Post secondary education includes any university, trade school or college degree.

Table 3
The effect of fine and coarse particulate matter on respiratory admissions for all age groups

	Age <6	Age 6-19	Age 20-64	Age 65+
Panel 1 - Coarse particulate matter (limited sample)				
PM10 (ug/m3)	0.00102	0.00014	-0.00232	-0.00753
	(0.00147)	(0.00027)	(0.00201)	(0.00478)
	{0.00445}	{0.00177}	{-0.00664}	{-0.00876}
Panel 2 - Coarse and fine particulate matter (limited sample)				
PM10 (ug/m3)	-0.00067	-0.00054	-0.00111	-0.00737
	(0.00391)	(0.00150)	(0.00241)	(0.00599)
	{-0.00337}	{-0.00442}	{-0.00202}	{-0.00852}
PM2.5 (ug/m3)	0.00185	0.00089 **	-0.00077	-0.00075
	(0.00101)	(0.00034)	(0.00169)	(0.00319)
	{0.00787}	{0.01064}	{-0.00188}	{-0.00075}
Panel 3 - Fine particulate matter (full sample)				
PM2.5 (ug/m3)	0.00196	0.00161 **	-0.00003	-0.00064
	(0.00134)	(0.00061)	(0.00145)	(0.00272)
	{0.00824}	{0.01590}	{-0.00008}	{-0.00064}
Alert controls	N	N	N	N
CO, O3 controls	Y	Y	Y	Y
Mean admissions	0.592	0.225	1.082	2.511

Reported coefficients are given as the average effect over five days. Standard errors are clustered by municipality and reported below coefficient estimates. Standardized beta coefficients are in brackets below standard errors. All columns include controls for weather variables, O3 and CO, ICD classification changes, day of week fixed effects, holiday fixed effects, municipality-month, and municipality-year fixed effects. * significant at 5% ** significant at 1% *** significant at 0.1%

Table 4

The effect of pollution and air quality alerts on respiratory admissions for all age groups

	Age <6	Age 6-19	Age 20-64	Age 65+
Panel 1 - Estimates by age group				
PM2.5 (ug/m3)	0.00196	0.00161 **	-0.00003	-0.00064
	(0.00134)	(0.00061)	(0.00145)	(0.00272)
	{0.00824}	{0.01590}	{-0.00008}	{-0.00064}
CO (ppm)	0.03998	-0.00131	-0.03873	0.05346
	(0.02093)	(0.01435)	(0.02705)	(0.05327)
	{0.00814}	{-0.00062}	{-0.00455}	{0.00257}
O3 (ppb)	-0.00048	-0.00061	-0.00004	-0.00118
	(0.00042)	(0.00056)	(0.00092)	(0.00211)
	{-0.00340}	{-0.01011}	{-0.00016}	{-0.00198}
Panel 2 - Estimates by age group controlling for warnings of poor air quality				
PM2.5 (ug/m3)	0.00231 *	0.00142 **	-0.00063	0.00078
	(0.00103)	(0.00052)	(0.00141)	(0.00350)
	{0.00974}	{0.01398}	{-0.00154}	{0.00077}
CO (ppm)	0.03897	-0.00081	-0.03746	0.04929
	(0.02038)	(0.01394)	(0.02723)	(0.05417)
	{0.00794}	{-0.00039}	{-0.00440}	{0.00237}
O3 (ppb)	-0.00040	-0.00059	-0.00038	-0.00018
	(0.00040)	(0.00047)	(0.00094)	(0.00172)
	{-0.00286}	{-0.00985}	{-0.00154}	{-0.00030}
Alerts (in levels)	-0.01900	-0.00607	-0.00420	-0.34526 *
	(0.03557)	(0.03560)	(0.04278)	(0.16025)
	{-0.00233}	{-0.00174}	{-0.00030}	{-0.01001}
F-test (all alert var=0)	1.413	1.166	0.758	1.869 **
P value	0.163	0.316	0.718	0.040
Hausman test	0.600	3.481	3.876	2.005
P-value	0.741	0.176	0.144	0.367
Mean admissions	0.592	0.225	1.082	2.511
Observations	107,108	107,108	107,108	107,108

See Table 3 notes. In panel 1, reported coefficients are given as the average effect over the last five days. The specifications in panel 2 include an alert indicator for days with an air quality alert and interactions of the alert indicator with the pollutant variables. In panel 2, reported coefficients are given as the average effect over the last five days evaluated at the average of the alert indicator variable. The F-test is a joint test for all alert variables and alert interactions. The Hausman test is for equivalence of the pollutant coefficients across specifications with and without alert controls. * significant at 5% ** significant at 1% *** significant at 0.1%

Table 5
Effect of pollution on respiratory admissions for children, multi-pollutant vs. single-pollutant models

	(1)	(2)	(3)	(4)
	Age <6		Age 6-19	
	Multi-pollutant	Single-pollutant	Multi-pollutant	Single-pollutant
PM2.5 (ug/m3)	0.00231 *	0.00232 ***	0.00142 **	0.0009 **
	(0.00103)	(0.00067)	(0.00052)	(0.00031)
	{0.00974}	{0.01194}	{0.01398}	{0.01062}
CO (ppm)	0.03897	0.03954	-0.00081	0.007
	(0.02038)	(0.02161)	(0.01394)	(0.00843)
	{0.00794}	{0.00852}	{-0.00039}	{0.00349}
O3 (ppb)	-0.0004	-0.00011	-0.00059	0.00003
	(0.00040)	(0.00031)	(0.00047)	(0.00015)
	{-0.00286}	{-0.00096}	{-0.00985}	{0.00064}

See notes from Table 3. Specifications in columns1 and 3 replicate results from Panel 2 of Table 4 (include all pollutants in a single regression).The coefficients in columns 2 and 4 are estimated using three separate regressions including one pollutant at a time.   * significant at 5% ** significant at 1% *** significant at 0.1%

Table 6

Effect of pollution on respiratory admissions for children throughout the season

	Fall		Winter		Summer	
	Multi	Single	Multi	Single	Multi	Single
Panel 1 - Age<6						
PM2.5 (ug/m3)	0.00450 *	0.00348 **	0.00268	0.00230 *	-0.00014	0.00194 *
	(0.00192)	(0.00124)	(0.00177)	(0.00091)	(0.00159)	(0.00092)
	{0.01651}	{0.01580}	{0.00955}	{0.01001}	{-0.00145}	{0.02372}
CO (ppm)	0.01525	0.02610	0.04802	0.04784	0.00091	0.00440
	(0.04672)	(0.04023)	(0.03502)	(0.03029)	(0.05282)	(0.05058)
	{0.00306}	{0.00554}	{0.00908}	{0.00958}	{0.00030}	{0.00158}
O3 (ppb)	-0.00028	0.00044	-0.00132	-0.00103	0.00396 ***	0.00345 ***
	(0.00128)	(0.00084)	(0.00101)	(0.00057)	(0.00107)	(0.00054)
	{-0.00156}	{0.00301}	{-0.00820}	{-0.00797}	{0.05313}	{0.04126}
Mean admissions		0.603		0.695		0.273
Panel 2 - Age 6-19						
PM2.5 (ug/m3)	0.00247 *	0.00104	0.00201 *	0.00176 **	0.00128	0.00135 *
	(0.00122)	(0.00077)	(0.00088)	(0.00055)	(0.00115)	(0.00058)
	{0.02005}	{0.01030}	{0.01847}	{0.01936}	{0.01956}	{0.02467}
CO (ppm)	-0.02815	-0.01804	0.00015	0.00879	0.07777 *	0.09568 **
	(0.02725)	(0.02391)	(0.01665)	(0.01430)	(0.03841)	(0.03465)
	{-0.01251}	{-0.00838}	{0.00007}	{0.00448}	{0.03916}	{0.05127}
O3 (ppb)	-0.00111	-0.00009	0.00021	0.00012	0.00021	0.00031
	(0.00077)	(0.00052)	(0.00053)	(0.00025)	(0.00082)	(0.00043)
	{-0.01379}	{-0.00127}	{0.00338}	{0.00227}	{0.00430}	{0.00756}
Mean admissions		0.245		0.230		0.170
Mean PM2.5		7.438		8.049		11.779
Mean CO		0.462		0.507		0.364
Mean O3		18.342		26.421		31.143

See notes from Table 3. Fall=September to December, Winter=January to June, Summer=July to August. Multi refers to a model with all pollutants included and Single refers to models with each pollutant included one at a time. * significant at 5% ** significant at 1% *** significant at 0.1%

Table 7
Effect of pollution on respiratory admissions for children, acute versus chronic

Panel 1 - Acute respiratory

	(1)	(2)	(3)	(4)
	Pneumonia		Croup/Bronchitis/Bronchiolitis	
	Age <6	Age 6-19	Age <6	Age 6-19
PM2.5 (ug/m3)	0.00030	0.00068 *	0.00188 *	0.00003
	(0.00027)	(0.00029)	(0.00085)	(0.00026)
	{0.00339}	{0.01824}	{0.01577}	{0.00112}
CO (ppm)	-0.00997	0.00178	0.03069 **	0.00104
	(0.01102)	(0.00638)	(0.01115)	(0.00417)
	{-0.00549}	{0.00230}	{0.01245}	{0.00164}
O3 (ppb)	-0.00013	-0.00029	-0.00027	0.00018
	(0.00031)	(0.00019)	(0.00035)	(0.00013)
	{-0.00243}	{-0.01309}	{-0.00380}	{0.01016}
Mean admissions	0.166	0.052	0.255	0.039

Panel 2 - Chronic respiratory

	(1)	(2)
	Asthma	
	Age <6	Age 6-19
PM2.5 (ug/m3)	0.00115 *	0.00077 *
	(0.00054)	(0.00032)
	{0.01175}	{0.01459}
CO (ppm)	0.00526	0.00135
	(0.01968)	(0.00846)
	{0.00260}	{0.00124}
O3 (ppb)	0.00008	-0.00045 *
	(0.00027)	(0.00030)
	{0.00134}	{-0.01422}
Mean admissions	0.196	0.081

See Table 3 notes. Panel 1 refers to acute respiratory admissions (ICD10 codes J00-J22). In panel 1, columns 1 and 2 show results for pneumonia and columns 3 and 4 show results for other acute upper/lower respiratory admissions (croup, common cold, acute bronchitis/bronchiolitis). Panel 2 refers to chronic respiratory admissions and columns 2 and 3 show results for asthma, the most common chronic respiratory problem in children. * significant at 5% ** significant at 1% *** significant at 0.1%

Table 8
Pollution, respiratory mortality and hospital resources

	Resp. death (age 0-19)	External accidents (all)	Wait time in ER (all)
PM2.5 (ug/m3)	0.00000	0.00097	0.00114
	(0.00005)	(0.00187)	(0.00354)
	{-0.00035}	{0.00085}	{0.00204}
CO (ppm)	-0.00172	-0.00549	0.00553
	(0.00188)	(0.05737)	(0.10781)
	{-0.00936}	{-0.00023}	{0.00048}
O3 (ppb)	-0.00002	0.00136	0.00403
	(0.00004)	(0.00190)	(0.00247)
	{-0.00413}	{0.00200}	{0.01219}
Mean of outcome	0.0037	3.0956	2.4335

See notes from Table 3. External accidents exclude traffic accidents. Wait time in ER is measured in hours. * significant at 5% ** significant at 1% *** significant at 0.1%

Table 9
Pollution correlation matrix

Overall

	PM2.5	CO	O3
PM2.5	1.000		
CO	0.088	1.000	
O3	0.383	-0.151	1.000

Between municipality-month

	PM2.5	CO	O3
PM2.5	1.000		
CO	-0.400	1.000	
O3	0.572	-0.198	1.000

Within municipality-month

	PM2.5	CO	O3
PM2.5	1.000		
CO	0.212	1.000	
O3	0.343	-0.110	1.000

Figure 1 - Centroids of matched municipalities

Figure 2 - Pollution monitors (OME, NAPS)

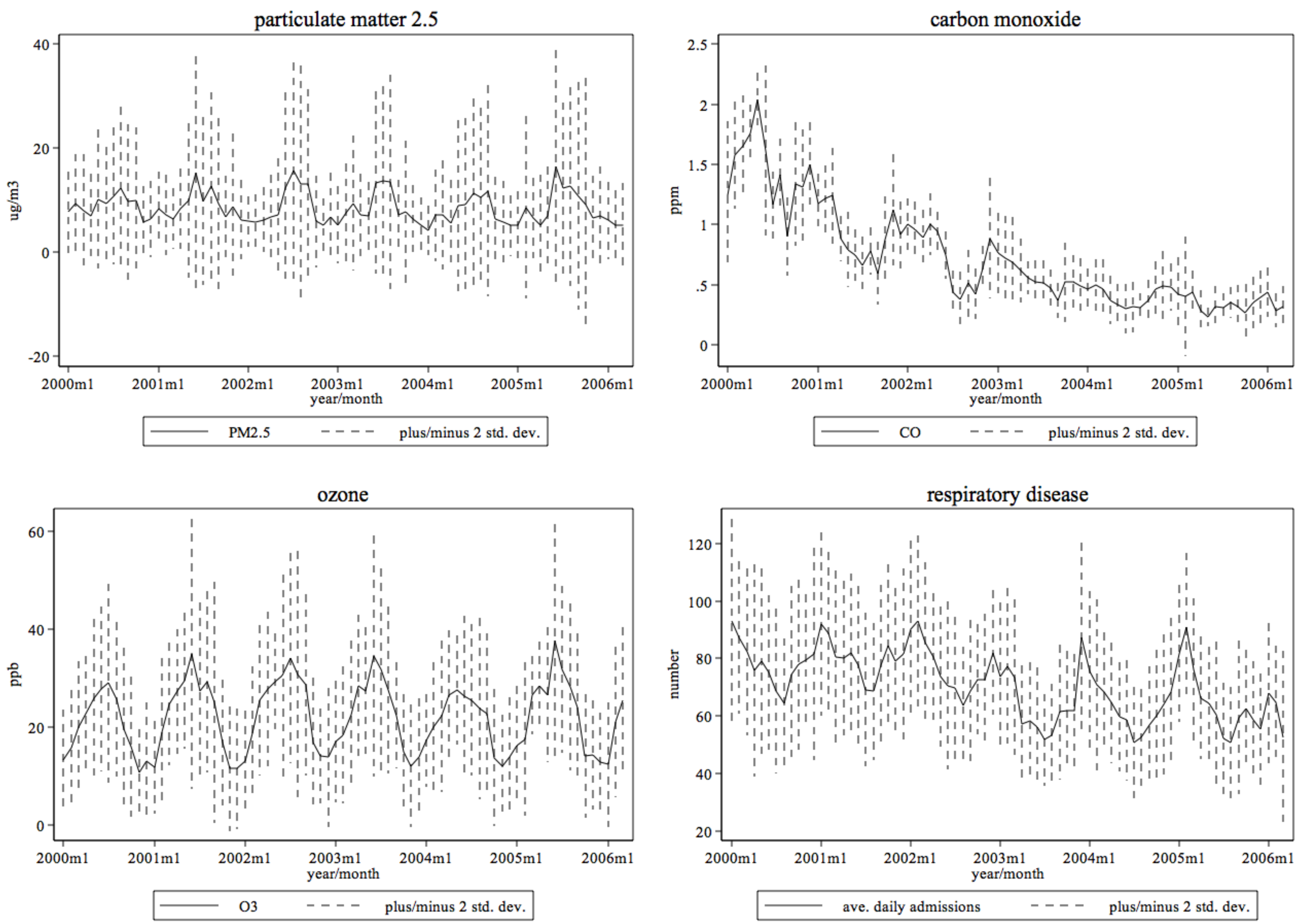

Figure 3 - Pollution and respiratory admissions in Toronto municipality
Note: each panel shows averages over year-month and within year-month standard deviations

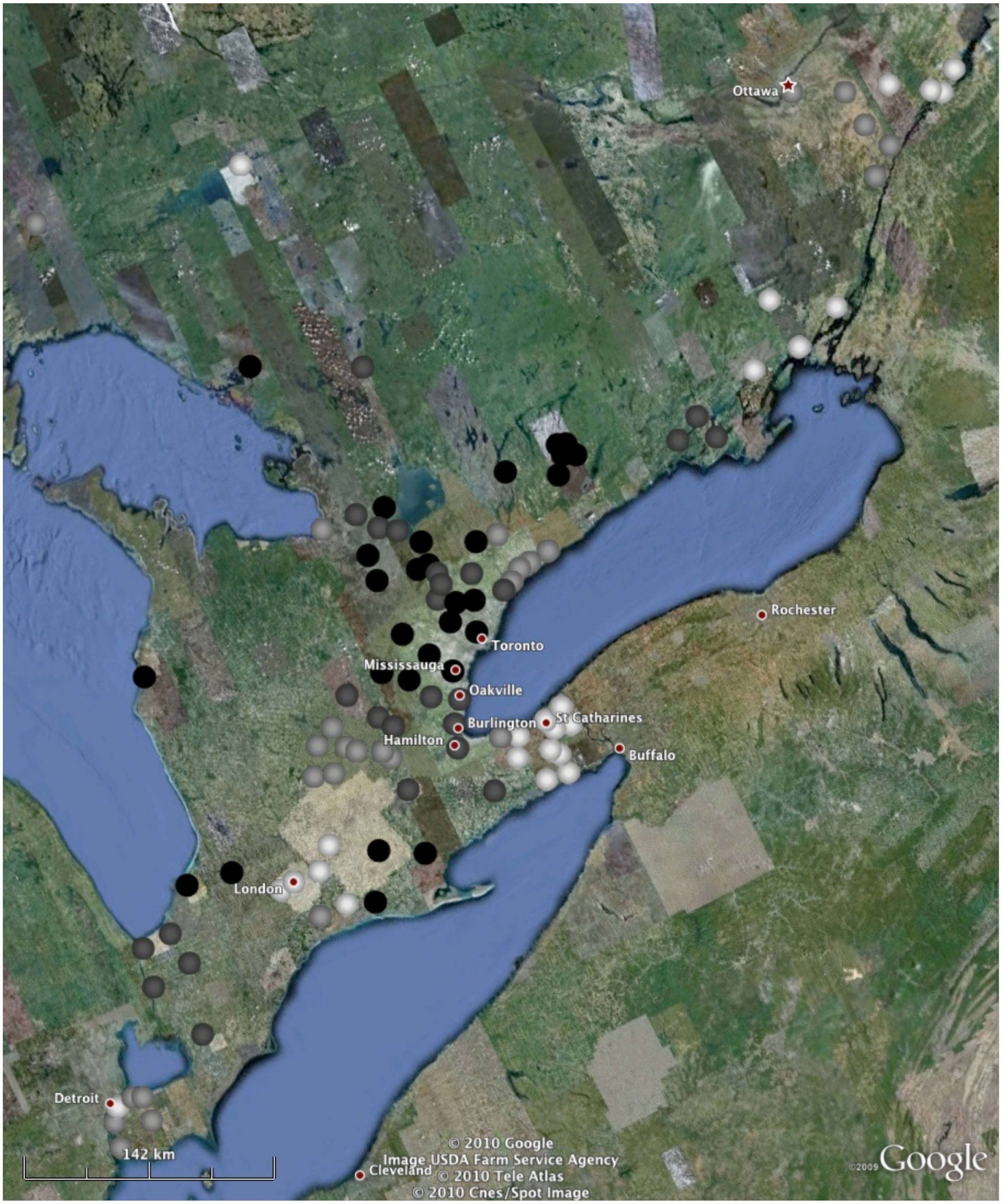

Figure 4 - Standardized pollution quartiles

1st quartile (lowest) 2nd quartile 3rd quartile 4th quartile (highest)

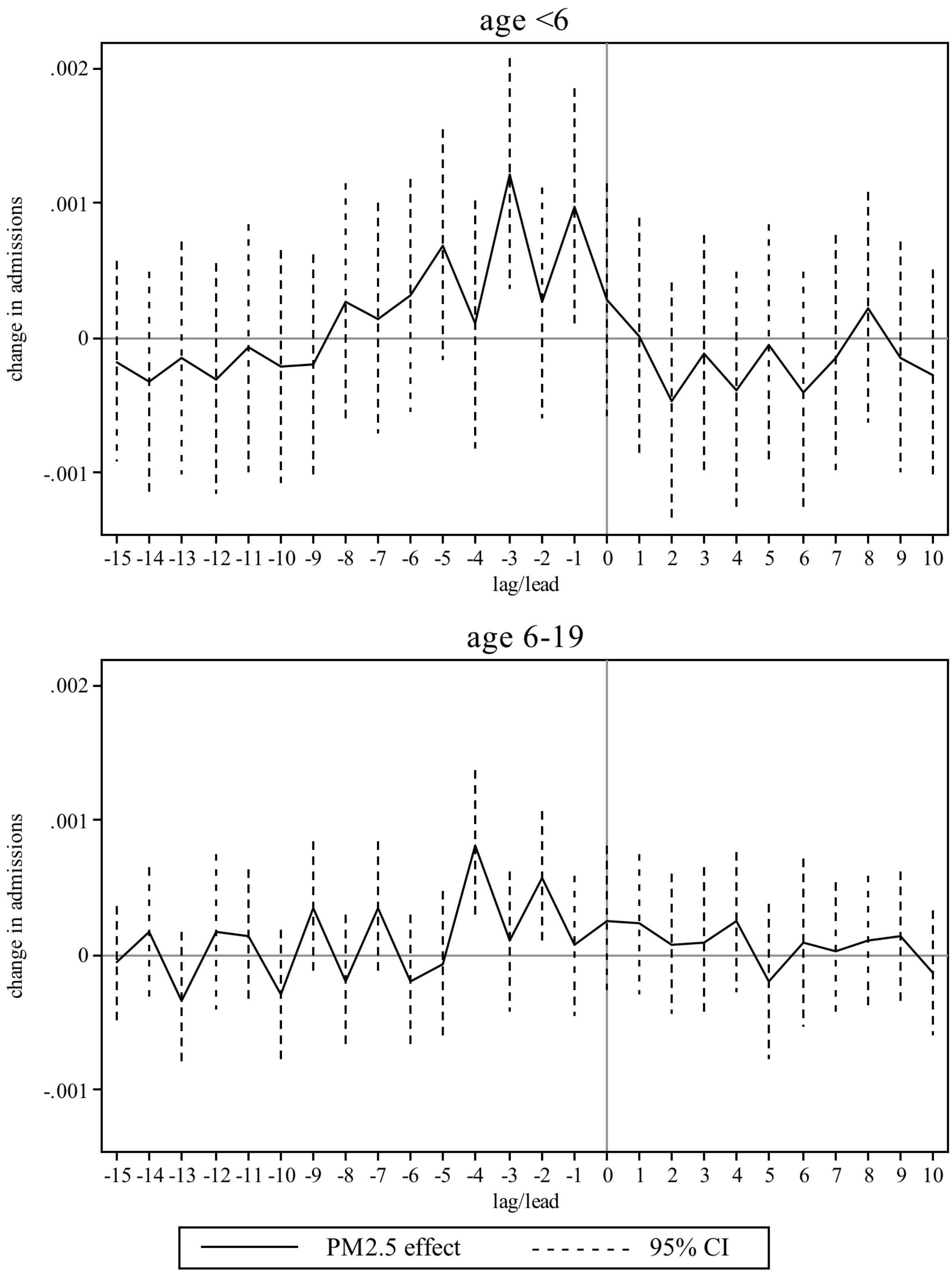

Figure 5 – Effect of fine particulate matter on respiratory admissions over lags/leads