The impact of 9/11 on the association of ambient air pollution with daily respiratory hospital admissions in a Canada-US border city, Windsor, Ontario

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Abstract

The 11 September 2001 (9/11) terrorist attacks in the United States resulted in long lines of trucks at the border crossing in Windsor, Ontario. Public concern about the potential impact of these trucks spewing toxic pollutants into the air drew attention to the need to investigate the impact of 9/11 on the daily levels of air pollutants and respiratory hospitalization. In this study, significant increases in respiratory admissions were found one month and 6 months post-9/11. Mean daily respiratory admission was also significantly higher than the same period one year earlier and one year later. SO₂ and CO concentration levels were found to be generally higher after 9/11 than one year before and immediately before. Relative risk estimates of respiratory hospitalization after 9/11 showed that SO₂ ($RR = 1.15$ for two-day, $RR = 1.18$ for three-day, and $RR = 1.21$ for five-day averages), NO₂ ($RR = 1.10$ for current day), and COH ($RR = 1.09$ for current day, $RR = 1.10$ for two-day average) had the most significant effects after 9/11. These results suggest the need for more stringent regulatory efforts in air quality in the region in response to the changing transportation dynamics at this Canada-US border crossing.

Keywords

Air pollution; Hospital admissions; 11 September 2001; Windsor; Ontario

1. Introduction

Research using large-scale data sets has shown a fairly consistent relationship between air pollutant levels and health outcomes in a variety of communities in the industrialized world [1–3]. In Canada, southwestern Ontario has been identified as one of the most polluted regions. The region is heavily industrialized. In addition, there is the outstanding problem of transboundary air and water pollution from Ohio, Illinois and Michigan [4]. One of the

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conspicuous cities in this region in terms of health effects of environmental exposure is Windsor, Ontario [4].

The city of Windsor (42°18'N, 83° 01'W) is located in southwestern Ontario, with an estimated population of 208,000 [5] and an approximate area of 120.63 km². The local and regional industrial sources make the city and its surrounding region a high pollution zone. In addition to occupational exposures of the workforce to a variety of products during manufacturing, the city is under heavy influence of air pollution from vehicular sources. The Ambassador Bridge, which is linked to the Highway 401 through Huron Church Road at Windsor-Detroit border crossing, is known to be the busiest trading route in the world. The Ambassador Bridge consists of both Canadian and US Customs Inspection Stations, Bridge toll booth plazas, and duty-free shops on either side of the border. This crossing-point alone handles approximately 25% of all Canada-US trade [6]. The volume of the booming trade and international commerce especially following the North American Free Trade Agreement (NAFTA) has resulted in the movement of an estimated 10,000 trucks to cross the Bridge on a daily basis [6].

Furthermore, the city is immediately downwind of major steel mills with associated coking operations in Detroit; the wastewater treatment plant of the city of Detroit and associated sludge incineration facilities, and a major power plant which until recently was coal fired. The combined effect of these factors, plus traffic congestion at the Windsor-Detroit Tunnel located in downtown Windsor and equally carrying significant Canada-US traffic, may have led to the view that the area is a high pollution area as compared to other Canadian cities [7]. A recent community health profile by Gilbertson and Brophy indicated mortality and morbidity rates from various cancers, circulatory and respiratory disorders were higher in Windsor than the rest of the province of Ontario [4]. This work aroused great concern among the local people, public health professionals and government agencies. This in turn led to a number of other studies that examined the association between ambient air quality and health effects in Windsor [8,9]. These studies reported, among other things, short-term effects of air pollutants on daily cardiac and respiratory hospital admissions among the people in Windsor.

The events of 11 September 2001 reinforced concerns about the effects of ambient air pollution, especially traffic pollution in the city and surrounding areas. Immediately following 11 September 2001, there were delays resulting in long lines of trucks at the Ambassador Bridge border crossing point. The idling trucks spewed toxic pollutants from their exhaust systems into the air. Meanwhile, traffic-related pollution has been shown to be associated with a number of acute respiratory illnesses, including upper respiratory diseases such as sinusitis, otitis, bronchitis; lower respiratory diseases such as pneumonia and influenza, and airway diseases such as asthma and chronic obstructive pulmonary disease [10–12]. The United States Environmental Protection Agency (EPA) concluded that long-term inhalation exposure to diesel exhaust particles is likely to pose a lung damage threat, including a risk for cancer, to humans [13]. The study further noted that short-term exposures can cause irritation and inflammatory symptoms of a transient nature [13]. Diesel exhaust particles have been shown to worsen respiratory symptoms and to lead to deterioration in lung function, especially among individuals with pre-existing chronic

conditions such as asthma [14]. Increased health problems resulting from traffic-related pollution around the US-Canada border crossing point have been a major focus of previous studies [11,15]. These studies have provided evidence supporting the hypothesis that there is a high respiratory burden among residents living in close proximity to the NAFTA corridor. In order to address this issue in the border areas, Canada and the USA have recently unveiled a joint strategy, formally known as the Border Air Quality Strategy to deal with air quality issues in border areas. This strategy aims to improve the border air quality and to address human health concerns [16].

The main focus of this paper is to investigate the impact of 9/11 on daily respiratory =hospital admissions in Windsor, Ontario. This research will provide policy-makers as well as the public with estimates of current risks of respiratory hospitalization as a result of poor ambient air quality due to 9/11. The results will be useful for policy decisions in response to the changing air quality and transportation dynamics in the region.

2. Materials and methods

Data acquisition

We used the most recent hospitalization data available from 2000 to 2003 to examine short-term (one month), medium-term (six months) and long-term (one year) differences before and after 9/11. The study population consisted of all people who were admitted into one of the four hospitals in Windsor with primary diagnoses of respiratory disease (ICD9 codes 460-519, ICD-10-CA codes J00-J99) [17] during the period 11 September 2000 to 12 March 2003 and who were registered with the Ontario Health Insurance Plan. We could not extend our study time period because of extensive missing values (two or more months in a row) of the pollution data after 12 March 2003 due to a labor strike. Daily hospital admission records for Ontario Health Insurance Plan (OHIP) patients were obtained from the Canadian Institute for Health Information (CIHI) Discharge Abstract Database [18]. The data included date of admission, age and gender. Since our analysis was focused on finding the impact of 9/11 on daily respiratory hospitalizations, all elective admissions were removed from the analysis.

The hourly air pollution data from the two fixed monitoring stations in Windsor were obtained from the Ontario Ministry of the Environment. Monitoring station 12008 is located in downtown Windsor, approximately 2.4 km east of the Ambassador Bridge, while station 12016 is about 1 km west of the Ambassador Bridge. The pollutants were: sulphur dioxide (SO₂), nitrogen dioxide (NO₂), carbon monoxide (CO), coefficient of haze (COH) and inhalable particles (PM₁₀). CO readings were available from one station (12008) only. PM₁₀ values were available from station 12016 only and they were missing for the whole period 11 January 2002 to 12 March 2002. In order to capture the effects of exposure, the daily means of pollutants were used. Where we had data from both stations, the average of the daily means was calculated. We included coefficient of haze (COH) in our analysis following the recommendation by Goldberg *et al.* [19], who advocated that it is a reliable measure of the concentration of ambient carbon particles (generally from internal combustion, motor vehicle emissions, road dust, and smoke), with only limited contributions from other pollutants, such as sulphates, nitrates, or particle mass. Respirable particle

(PM_{2.5}) data were not available. Daily means of weather data including temperature, humidity and mean change in barometric pressure from the previous day were obtained from the Ontario Climate Centre [20] and were included in our analyses.

Statistical analysis

First, we linked together records from several databases comprising pollutants, temperature, humidity and pressure, and number of hospital admissions. Data from CIHI were given to us in a ready-to-use format.

To examine the differences before and after 9/11, we divided the data into three study periods: 1) one month before and after 9/11 (11 August 2001 to 10 October 2001), 2) approximately six months before and after 9/11 (1 March 2001 to 12 March 2002), 3) six-month period one year before, during, and one year after 9/11 (i.e. 11 September 2000 to 12 March 2001; 11 September 2001 to 12 March 2002; 11 September 2002 to 12 March 2003). For comparing group means before and after 9/11, we used SPSS software [21] to perform the *t*-test without assuming equal variances if the group variances were tested to be significantly different by Levene's test. *P*-values corresponding to two-sided tests were obtained. To relate the association of air pollution to respiratory hospitalizations, we used the time-series method. Relative risk estimates for respiratory hospitalization were calculated based on the data from 1 March 2001 to 12 March 2002, with temperature, humidity, and change in barometric pressure as covariates.

Time-series analysis has been used extensively to analyze this type of data [e.g. 19]. In time-series analysis, the standard practice is first to remove temporal trends, including seasonal and day-of-the-week effects, to obtain a time-series of the logarithm of the number of daily events that is as close to white noise as possible, as determined by Bartlett's test [22]. Smoothing techniques such as natural splines (ns) or LOESS may be chosen to smooth time and other continuous covariates. The number of knots per year that produces the largest *p*-value in Bartlett's test is often used in practice with natural spline smoothing. Daily air pollutant concentrations and other covariates are then added to the model. Model parameters can be estimated using the GLM (ns) or GAM (LOESS) function in S-Plus [23]. For details of these procedures, see [24–26].

For the time-series analysis in this paper, daily concentrations of each pollutant and covariates were related to respiratory admissions, *y*, by the model

$$E(y) = \exp\{\beta_0 + \beta_1 \text{pollutant} + \beta_2 \text{cohort} + \beta_3 (\text{pollutant} \times \text{cohort}) + \beta_4 \text{tempetature} + \beta_5 \text{humidity} + \beta_6 = \beta_7 \text{DOW} + z' \gamma\}, \quad (1)$$

where *E*(*y*) is the mean of *y*, DOW is the day-of-the-week effect which takes on values 1 to 7. Cohort takes on two possible values: cohort = 0 for before 9/11 and cohort = 1 for after 9/11. The weather variables used here are all daily mean values. The vector $z = (z_1, z_2, \dots, z_{dt})$

' represents the covariates produced by the smoothing natural spline technique $ns(time, df)$, and $\gamma = (\gamma_1, \gamma_2, \dots, \gamma_{df})'$ is the corresponding vector of regression coefficients. 'Pollutant $\times$ cohort' is the interaction term of the two main effects. This model allows us to estimate the relative risk (RR) and its confidence interval in the two cohorts separately.

We used the software S-Plus for the fitting of the model. The estimate of the RR for an interquartile range increase of a certain pollutant for cohort 1 (after 9/11) is given by

$$RR_1 = \exp\{(\hat{\beta}_1 \times IQR) + (\hat{\beta}_3 \times IQR)\}, \quad (2)$$

and for cohort 0 (before 9/11)

$$RR_0 = \exp\{(\hat{\beta}_1 \times IQR)\}. \quad (3)$$

Let $I_1 = (\beta_1 \times IQR) + (\beta_3 \times IQR)$. The 95% confidence interval for I_1 is given by

$$\hat{l}_1 \pm 1.96 \sqrt{\hat{V}(\hat{l}_1)}$$

where $\hat{V}(\hat{l}_1) = IQR^2 \hat{V}(\hat{\beta}_1) + IQR^2 \hat{V}(\hat{\beta}_3) + 2 \times IQR^2 COV(\hat{\beta}_1, \hat{\beta}_3)$.

Hence, the 95% confidence interval for RR_1 is

$$\exp\{\hat{l}_1 \pm 1.96 \sqrt{\hat{V}(\hat{l}_1)}\}$$

and the 95% confidence interval for RR_0 is

$$\exp\{\hat{l}_0 \pm 1.96 \sqrt{\hat{V}(\hat{l}_0)}\} \quad (5)$$

where $\hat{V}(\hat{l}_0) = IQR^2 \hat{V}(\hat{\beta}_1)$.

3. Results

The weekly trend of total respiratory admissions is shown in figure 1 for the period 1 March 2001 to 12 March 2002 with a line indicating 9/11. Table 1 gives the summary statistics of daily respiratory admissions for the three study periods. We found significant increases in the number of respiratory admissions in the one month ($p = 0.03$) and six months ($p = 0.001$) post-9/11.

In order to account for seasonal variations in respiratory admissions (since these are usually higher in the winter months), we also compared the data six months post-9/11 (11 September 2001 to 12 March 2002) with data in exactly the same period one year previously (11 September 2000 to 12 March 2001) and one year later (11 September 2002 to 12 March 2003). Results in table 1 show that the mean of daily respiratory admissions peaked right after 9/11 and was significantly higher than one year previously ($p = 0.019$) and one year later ($p = 0.012$). This shows that when there was no more line-up of trucks along the streets one year later, the number of respiratory admissions returned to before the 9/11 level.

An analysis of the Windsor yearly air pollution data for the period 1990 to 2003 showed an overall decreasing trend in ambient air pollutants (NO_2 , SO_2 , CO , COH), likely due to regulatory measures implemented by the government in the last 10 years [27]. The trends of the weekly pollutant levels from 1 March 2001 to 12 March 2002 are provided in figure 1. The trends suggest slight elevations in most of the pollutants immediately following 9/11, but these elevations, for the most part, were not sustained. Table 2 provides the summary statistics for daily mean concentrations of SO_2 , NO_2 , COH , CO and PM_{10} for the one-month and six-month study periods. For the one month before and after 9/11, there were increases in the levels of SO_2 , NO_2 and CO after 9/11, but only NO_2 was found to be significant at the 0.10 level. On the other hand, mean pollutant levels of COH and PM_{10} decreased. For the six-month period before (1 March 2001 to 10 September 2001) and after 9/11 (11 September 2001 to 12 March 2002), all pollutant levels increased except for PM_{10} . The increases in the ambient levels of SO_2 ($p < 0.05$) and CO ($p < 0.001$) post-9/11 were significant. The mean level of PM_{10} six months after 9/11 was significantly lower ($p < 0.001$). Comparing the six-month pollutant levels after 9/11 with the levels a year earlier (11 September 2000 to 12 March 2001), we also found CO and SO_2 to be higher. The increase in CO after 9/11 was significant ($p < 0.01$) but not for SO_2 . Reversely, NO_2 and PM_{10} levels were lower than one year ago. COH was not available to us before 2001.

The correlation coefficients for the air pollutants and weather variables based on the data from 1 March 2001 to 12 March 2002 are shown in table 3. All the pollutants were positively correlated with each other. Daily temperature was positively correlated with SO_2 , COH , and PM_{10} but negatively with NO_2 and CO . Relative humidity was negatively correlated with all pollutants except COH and CO . Mean change in barometric pressures from the previous day were negatively correlated with all pollutants and weather variables.

We also examined the air quality index (AQI) which is based on the pollutants ozone (O_3), fine particulate matter ($\text{PM}_{2.5}$), nitrogen dioxide (NO_2), carbon monoxide (CO), sulphur dioxide (SO_2), and total reduced sulphur (TRS) compounds. If the AQI index is 50 and over, it indicates poor air quality that may have adverse effects on human health. We found that the number of days AQI was 50 or more in the Windsor West location (station 12016, which is nearer to the Ambassador Bridge) was 19 for 2001 and 47 for 2002. In the downtown Windsor location (station 12008), the numbers were 15 and 30 for 2001 and 2002 respectively. Due to the lack of daily information, we were unable to determine the exact number of very good, good, moderate, poor and very poor days during the two years. The results show, however, a doubling of poor air quality days in 2002 as compared to 2001.

The relative risk (RR) estimates and 95% confidence intervals (CI) for respiratory hospitalizations for the two cohorts in relation to air pollutant concentrations were calculated according to formulae (2) to (5) and presented in table 4 for current day, two-day, three-day and five-day lagged moving averages of each pollutant (e.g. three-day average is the average of current day and the two days before). The study period here is 1 March 2001 to 12 March 2002, with cohort 0 being six months prior to 9/11 and cohort 1 being six months after 9/11. Temperature and other weather variables were incorporated into the model to account for seasonal effects. Because of the non-availability of PM₁₀ data for the whole period, this was not included in the time-series analysis. Twelve knots per year produced the largest *p*-value in Bartlett's test so we used 14 degrees of freedom to smooth time.

The results show that the effects of SO₂ on respiratory admission post-9/11 for two-day ($\hat{RR} = 1.15$, 95% confidence interval (CI) = 1.039, 1.264), three-day ($\hat{RR} = 1.18$, 95% CI = 1.046, 1.335) and five-day ($\hat{RR} = 1.21$, 95% CI = 1.016, 1.447) lagged averages were significant. In other words, the effect of SO₂ was mostly delayed. There was no significant effect with the current day exposure level of SO₂. However, results for current day exposure levels of NO₂ and COH showed relative risks that were significantly higher post-9/11 than pre-9/11, with $\hat{RR}$ of 1.101 (95% CI = 1.006, 1.205) and $\hat{RR}$ of 1.092 (95% CI = 1.013, 1.176) for NO₂ and COH respectively. This indicates that after 9/11, the percentage increase in the mean number of daily respiratory hospitalizations is 10.1% for an increase of current day exposure of 7.35 units of NO₂, and 9.2% for an increase of current day exposure of 0.119 unit of COH. Relative risk estimate for cohort 1 (post-9/11) was also significant for two-day lagged average for COH ($\hat{RR} = 1.104$, 95% CI = 1.008, 1.210).

4. Discussion

The 9/11 terrorist attack brought with it intense concerns about the potential effects of air pollution in the NAFTA corridor, especially Canada-US border cities. In a response, Lwebuga-Mukasa *et al.* [11] examined the relationship between traffic volumes and respiratory health care use among residents in close proximity to the Peace Bridge in Buffalo, New York following 11 September 2001. They reported a dramatic decrease in commercial and automobile traffic crossing the US-Canada border through the Peace Bridge and a decrease in health care use for respiratory disease. In this study, we were not able to obtain traffic numbers across the border. But, instead of using traffic as a surrogate for pollution level, we had the actual pollutant measurements from the monitoring stations in the city. Our main concern was about the stagnation of traffic which lined up for several kilometers in the major roads leading to the Ambassador Bridge and Windsor-Detroit Tunnel in the days and weeks immediately after 9/11. The results showed an association between ambient air pollution levels and respiratory hospitalizations following 9/11.

The effect of SO₂ on respiratory hospitalization varies considerably, especially at low levels of exposure. For example, some studies have reported a lack of association between SO₂ and hospitalization [e.g. 28]. On the other hand others found SO₂ levels influenced hospital admissions for all respiratory diseases [e.g. 1,29]. Bates and Sizto found an association between SO₂ (two-day lag) and respiratory admissions in southern Ontario [30]. Luginaah *et al.* working with data from Windsor, Ontario, 1995–2000, found a significant relationship

between SO₂ and respiratory admissions for females aged 0–14 with a RR of 1.11 [9]. In the current study, the relative risk estimate for respiratory admissions of all ages post-9/11 was not significant for the current day exposure, but significant for two, three, five-day averages at the 5% level. Although the ambient concentrations of SO₂ in Ontario have decreased by more than 86% over recent decades [27], there is still a need for continuous attention because of the effect of delayed exposure and the existence of high risk groups.

We found the relative risk of current day NO₂ to be significant (1.101; 95% CI: 1.006–1.205). NO₂ has been known to result from traffic pollution [31], and can increase the susceptibility to respiratory infections [32]. Yet, results of different studies that examined the link between NO₂ and respiratory outcomes continue to vary, possibly due to differences between various study contexts. Wong *et al.* reported significant associations between NO₂ and respiratory admissions for age groups 0–4, 5–64 and 65+ in Hong Kong [33]. But, Atkinson *et al.* working in London (UK), reported no significant associations between NO₂ and respiratory admissions [1]. As part of the Air Pollution and Health: A European Approach (APHEA) project, Spix *et al.* also reported no significant association between NO₂ and respiratory admissions for 15–64 and 65+ age groups [34].

Compared to other pollutants, the effect of COH on respiratory admissions has not been frequently examined [19]. One study, however, found that COH was the strongest predictor of hospitalizations for respiratory diseases among particle-related pollutants examined in both single and multiple pollutant regression models [35]. Also, Luginaah *et al.* found COH to be associated with respiratory admissions among females in Windsor, Ontario using data from 1995 to 2000 [9]. As indicated earlier, COH is a reliable measure of the concentration of ambient carbon particles from motor vehicle emissions, road dust, smoke, among others [19]. Hence, the significance of current and two-day average of COH in this analysis suggests the possible role of the 9/11 traffic stagnation and the increases in respiratory hospitalizations.

The literature on the effects of CO on respiratory illness has also been mixed at best [36]. For instance, Atkinson *et al.* found no association between CO and respiratory admissions either overall or by age group [1]. But, in Korea, Cho *et al.* after controlling for seasonal and temperature effects, found an association between CO and hospital admissions for respiratory disease with relative risks ranging from 1.21 to 3.55 depending on whether the area is rural or urban [37]. In this study, we found an elevated association of CO with respiratory admissions after 9/11, but none was significant. It is important to note that significant reduction in CO had already been achieved in Windsor in the years preceding 9/11 (mean = 1.0 ppm in 1991 to 0.3 in 2000) due to more stringent regulatory effort in air quality [38]. Thus, the elevation of CO observed in this study is likely due to the traffic pollution, resulting from the stagnation following 9/11.

Other possibilities that may influence the rise in respiratory hospitalization include the decrease of travelers (e.g. snowbirds – Canadians who spend a large portion of winter in the sunbelt of the United States) to the southern US because of border difficulties. Since these people are mostly retirees who have a high likelihood of respiratory diseases, they may be the ones that had to be hospitalized locally, thus influencing the increase in the number of

hospital admission. Another explanation may be the increase in smoking post-9/11 due to stress. Since there are no hard data on the number of people smoking before and after 9/11, we cannot confirm or deny this hypothesis. Furthermore, the impact of the confusion and congestion of traffic that occurred immediately after 9/11 may have led people to be sensitized, perceiving themselves as being sick and ending up in hospital [see 39].

Limitations of this study include the adequacy of covariate control and the impact of measurement error in the exposure and outcome variables. But, for most of the risk factors such as the presence of chronic conditions, there is no reason to believe that the individual risk factors are correlated with the daily changes in air pollution pre- and post-9/11; hence, they are not likely to be confounders in this study. Furthermore, the limitations of using fixed-monitors to represent the same exposure for the entire population, in environmental exposure studies have been frequently discussed [19]. In this situation, the concentration of the truck traffic and pollutants resulting in high levels of NO₂ around the Ambassador Bridge is a case in point [40]. From an epidemiological perspective, the measurement error that intrudes in to these analyses is very likely to be non-differential (i.e. similar before and after 9/11). Hence, the effect of such measurement error suggests that our observed relative risk estimates are probably underestimates of the reality. Moreover, because of unavailability of the air quality data due to a labor strike, we were limited by the length of the time-series and the number of admissions. As such, we could not delineate cardiac admissions in this study which are frequently associated with ambient concentrations of the pollutants [8]. For example, in the same context, Fung *et al.* found SO₂ to be associated with cardiac hospitalizations to a maximum of 5.6% increase in daily admissions for a three-day mean level with an increase in interquartile range of 19.3 ppb [8]. The effect was even stronger among people 65 years or older. Hence, these results must be interpreted with caution. Nevertheless, the findings still have implications for public health policy.

Although the overall risks of respiratory disease due to ambient air pollution post-11 September 2001 in the general population may seem low, it is reasonable to assume that the risks are much higher among susceptible groups such as those between ages 0–14 and 65 years or older. It is also reasonable to assume that there were more doctors' visits post-9/11 as some illnesses might not be severe enough to warrant hospitalization.

5. Conclusion

This study examines the impact of ambient air pollution on respiratory hospitalization of Windsorites before and after 9/11. Results show that respiratory hospitalizations significantly increased for the one-month period after 9/11 and peaked for the six-month period after 9/11 compared to a year before and a year later. NO₂ levels were significantly increased during the one month post-9/11. During the six months after 9/11, the levels of both SO₂ and CO increased significantly. Compared to the same period a year ago, the mean levels of CO was also significantly higher after 9/11. The time-series analysis revealed increased relative risk estimates for all the pollutants after 9/11, in the six months following the 11 September 2001 event. The relative risks of hospitalization showed that COH (one and two day average), NO₂ (current day) and SO₂ (two to five-day averages) had the most significant effects.

Even though the traffic stagnation since 9/11 seems to have decreased considerably in part due to the recent opening of more traffic check points on the US side which facilitated traffic movement, there is still occasional stagnation that is related to the raising or lowering of security levels in the US. These occasional back-ups may still continue to pose some health problems or persistent negative perceptions of environmental quality. Hence, there is a need to look for ways to minimize these problems, including the proposed opening of other entry routes to reduce the length of time that the trucks, in particular, take to cross in both directions. We recommend that meeting the intense public concerns about the health impacts of environmental quality in this area must not only include upholding stricter guidelines (which will be beneficial), but also communicating facts about environmental health risk, to improve public perception of risk due to poor air quality.

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Figure 1.

Time series plot of weekly levels of pollutants and respiratory hospital admissions data in Windsor.

*Period: 1 March 2001–10 January 2002 for PM_{10} and 1 March 2001–12 March 2002 for all other pollutants and admission data.

Table 1

Summary statistics of respiratory daily admissions in Windsor for different study periods

Statistics	One month before and after the 9/11 incident		Six months before and after the 9/11 incident		One year before, during, and one year after the 9/11 incident			
	11/8/01–10/9/01	11/9/01–10/10/01	1/3/01–10/9/01	11/9/01–12/3/02	11/9/00–12/3/01	11/9/01–12/3/02	11/9/02–12/3/03	
Minimum	0	2	0	1	1	1	1	
Maximum	9	15	15	16	14	16	15	
Mean	4.613	6.067	5.469	6.443	5.760	6.443	5.765	
Std Dev.	2.124	2.912	2.667	2.727	2.836	2.721	2.510	
SE ^a of mean	0.381	0.532	0.191	0.201	0.210	0.201	0.186	
Test statistic ^b (<i>p</i> -value)	2.233 (<i>p</i> = 0.029 ^{**})		3.504 (<i>p</i> = 0.001 ^{***})		2.351 (<i>p</i> = 0.019 ^{**})		2.526 (<i>p</i> = 0.012 ^{**})	

^aSE: standard error of mean.^bTwo-sided *t*-test.^{*}Significant at 10% level;^{**}significant at 5% level;^{***}significant at 1% level.

Table 2

Summary statistics of the pollutants in Windsor in different study periods

Time period	Statistics	SO ₂ (mean of 2 stations) ^c	NO ₂ (mean of 2 stations)	CoH (mean of 2 stations)	CO (daily mean) ^d	PM ₁₀ (daily mean) ^e
One month						
11/8/01–10/9/01	Minimum	1.440	8.500	0.060	0.058	9.292
	Maximum	13.770	32.380	0.400	0.827	36.917
	Mean	5.825	16.846	0.193	0.249	20.840
	Std dev.	3.623	5.658	0.079	0.169	7.669
	Minimum	0.830	10.440	0.070	0.000	4.208
11/9/01–10/10/01	Maximum	18.330	34.420	0.500	0.880	52.609
	Mean	6.438	19.887	0.185	0.319	19.522
	Std. dev.	4.639	6.433	0.103	0.243	11.186
	Test statistic ^{c,d} (2 sided <i>p</i> -value)	0.573 (<i>p</i> = 0.569)	1.899 (<i>p</i> = 0.063 [*])	–0.338 (<i>p</i> = 0.736)	1.293 (<i>p</i> = 0.202)	–0.538 (<i>p</i> = 0.592)
			Six-month periods			
One year before 11/9/00–12/3/01	Minimum	0.170	6.000		0.000	5.130
	Maximum	28.060	41.500		0.900	52.040
	Mean	7.920	22.623	N/A	0.262	21.379
	Std dev.	5.194	7.386		0.195	9.709
	Test statistic ^{c,d} (<i>p</i> -value)	0.674 (<i>p</i> = 0.503)	–4.258 (<i>p</i> < 0.001 ^{***})		2.67 (<i>p</i> = 0.008 ^{***})	–0.509 (<i>p</i> = 0.61)
During 11/9/01–12/3/02	Minimum	0.830	8.770	0.030	0.000	4.208
	Maximum	8.770	34.420	0.550	0.880	53.875
	Mean	8.268	19.766	0.186	0.313	20.855
	Std dev.	4.673	5.288	0.106	0.176	9.970
	Minimum	0.930	8.500	0.030	0.000	7.833
Six months before 1/3/01–10/9/01	Maximum	17.920	33.290	0.460	0.827	72.833
	Mean	7.160	19.579	0.181	0.211	25.625
	Std dev.	3.643	5.898	0.089	0.129	12.421
	Test statistic ^b (<i>p</i> -value)	2.535 (<i>p</i> = 0.012 ^{**})	0.322 (<i>p</i> = 0.748)	0.4000 (<i>p</i> = 0.689)	6.377 (<i>p</i> < 0.001 ^{***})	–3.759 (<i>p</i> < 0.001 ^{***})

^aTwo-sided *t*-test comparing means one year before with during 9/11.

- ^bTwo-sided *t*-test comparing means six months before with during 9/11.
- ^cPollutant values from two stations were available (12008 and 12016). The daily means from each station were first obtained, and then the means of the daily means were taken.
- ^dCO values were available only from station 12008. The daily means were considered.
- ^ePM₁₀ values were available only from station 12016, 11 September 2001 to 10 January 2002. The daily means were considered.
- ^{*} Significant at 10% level;
- ^{**} significant at 5% level,
- ^{***} significant at 1% level.

Table 3

Correlation coefficients between air pollutants and weather variables from 1 March 2001 to 12 March 2002

	SO ₂ (mean of 2 stations) ^a	NO ₂ (mean of 2 stations) ^a	COH (mean of 2 stations) ^a	CO (one station) ^b	PM ₁₀ (one station) ^{c, d}	Temp (daily mean)	RH (daily mean)	Mean Change in pressure
SO ₂ (mean of 2 stations) ^a	1.000							
NO ₂ (mean of 2 stations) ^a	0.507 ***	1.000						
COH (mean of 2 stations) ^a	0.468 ***	0.516 ***	1.000					
CO (one station) ^b	0.484 ***	0.555 ***	0.493 ***	1.000				
PM ₁₀ (one station) ^{c, d}	0.526 ***	0.391 ***	0.612 ***	0.276 ***	1.000			
Temp (daily mean)	0.010	-0.122 **	0.353 ***	-0.146 ***	0.434 ***	1.000		
RH (daily mean)	-0.153 ***	-0.032	0.031	0.144 ***	-0.227 ***	-0.119 **	1.000	
Mean change in pressure	-0.233 ***	-0.119 **	-0.172 ***	-0.173 ***	-0.282 ***	-0.160 ***	-0.224 ***	1.000

*** Significant at the 0.01 level;

** significant at the 0.05 level;

* significant at the 0.10 level; all two-tailed tests.

^a Pollutant values from two stations were available (12008 and 12016). The daily means from each station were first obtained from the 24 hours recordings, and then the means of the daily means were taken.

^b CO values were available only from station 12008, and the daily means were used.

^c PM₁₀ values were available only from station 12016, and the daily means were used.

^d We had PM₁₀ values from 1 March 2001 to 10 January 2002 ($N = 316$ days). The correlations between PM₁₀ and other pollutants and weather variables were calculated on the basis of these 316 observations

Table 4

Relative risks and 95% confidence interval for respiratory hospitalizations among the people in Windsor, Ontario before 9/11 (cohort 0) and after 9/11 (cohort 1) from 1 March 2001 to 12 March 2002

Pollutant (units) (Interquartile range ^a)	Lag exposure	$RR_{pre-9/11cohort}^b$ (95% confidence interval)	$RR_{post-9/11cohort}^b$ (95% confidence interval)
SO ₂ (ppb) (IQR = 0.5455)	Current day	1.005 (0.902, 1.120)	1.061 (0.981, 1.142)
	2-day average	0.996 (0.867, 1.144)	1.146^c (1.039, 1.264)
	3-day average	0.991 (0.839, 1.170)	1.182 (1.046, 1.335)
	5-day average	0.988 (0.795, 1.228)	1.213 (1.016, 1.447)
NO ₂ (ppb) (IQR = 7.3542)	Current day	0.948 (0.868, 1.036)	1.101 (1.006, 1.205)
	2-day average	0.951 (0.833, 1.077)	1.093 (0.996, 1.288)
	3-day average	0.947 (0.833, 1.077)	1.133 (0.996, 1.288)
	5-day average	0.943 (0.792, 1.124)	1.145 (0.969, 1.353)
COH (IQR = 0.1188)	Current day	0.877 (0.783, 0.982)	1.092 (1.013, 1.176)
	2-day average	0.874 (0.765, 0.998)	1.104 (1.008, 1.210)
	3-day average	0.861 (0.741, 1.000)	1.071 (0.963, 1.191)
	5-day average	0.843 (0.701, 1.014)	1.042 (0.914, 1.188)
CO (ppm) (IQR = 0.2178)	Current day	1.042 (0.935, 1.161)	1.056 (0.974, 1.146)
	2-day average	1.049 (0.911, 1.208)	1.097 (0.995, 1.210)
	3-day average	1.056 (0.886, 1.259)	1.099 (0.983, 1.229)
	5-day average	0.942 (0.737, 1.205)	1.076 (0.941, 1.230)

^aInter-quartile ranges (IQR) of the pollutant in the study period 1 March 2001 to 12 March 2002.

^bRelative risks and 95% C.I. were computed for an increase in pollutant equal to the interquartile range of that pollutant for cohort 0 (before 9/11) or 1 (after 9/11) according to formulae 2–5.

^cNumbers in bold indicate significance at the 5% level.