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Air pollution and daily hospitalization rates for cardiovascular and respiratory diseases in London, Ontario

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Abstract

In this paper, we examine the role that ambient air pollution plays in exacerbating cardiovascular and respiratory disease hospitalization in London, Ontario from 1 November 1995 to 31 December 2000. The number of daily cardiac and respiratory admissions was linked to concentrations of air pollutants (sulphur dioxide, nitrogen dioxide, ozone, carbon monoxide, coefficient of haze, PM_{10}) and weather variables (maximum and minimum of temperature and humidity). Results showed that current day carbon monoxide and coefficient of haze produced significant percentage increase in daily cardiac admissions of 8.0% (95% CI: 1.5–11.5%) and 5.7% (95% CI: 0.9–10.8%) for people < 65 years old. PM_{10} was found to be significantly associated with asthma admission in the > 65 group, with percentage increase in cardiac admission of 25% (95% CI: 2.8–52.3%) and 26.0% (95% CI: 5.3–50.9%) for current day and 2-day means, respectively.

Keywords

Air pollution; Cardiac disease; Respiratory disease; London, Ontario; Hospital admissions

1. Introduction

The link between air pollution and health has been an important public health issue in the last few decades. Recent research using large-scale datasets has shown a fairly consistent relationship between air pollutant levels and cardiovascular and/or respiratory diseases in a variety of communities in the industrialized world [1–7].

In Canada, several reports have been published linking air pollution to adverse population health in cities based on data that were collected in the 1980s and early 1990s [8–15]. Positive associations between short-term air pollutants and daily hospital admissions for cardiac and respiratory diseases using more current data (1995–2000) in Windsor were obtained by Fung *et al.* [16] and Luginaah *et al.* [17]. These reports have consistently shown that Southwestern Ontario is second to none as a ‘hot spot’ in terms of air pollution in

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Canada. Communities in this region are exposed to repeated episodes of long-range transport of air pollutants [12,18,19]. In this area, transported air pollution is characterized by low levels of primary gaseous pollutants (SO_2 and NO_2) and moderately elevated concentrations of particles and ozone [12]. According to Stern *et al.*, episodes of elevated sulphate and ozone concentrations occur frequently throughout the region, especially during the summer and early fall. Many pollutants in the region are generated both locally and regionally, and can be carried a long way by winds, affecting areas far removed from the source of the pollution. Also, data analysis strongly indicates that neighboring US states (Ohio, Illinois and Michigan) are significant contributors to elevated levels of ozones and inhalable particles in Southwest Ontario [20].

The terrorist attack of September 11, 2001 (9/11) has brought with it additional concerns about the effects of air pollution in the region, especially along the Highway 401 transportation corridor. With more vigorous security policies across the US/Canada border-crossing points in Windsor and Sarnia, there have been increasing delays resulting in long lines of trucks on the streets and highways. The idling trucks are spewing toxic pollutants from their archaic exhaust systems into the air. Since London is situated along the main highway east of these cities and the neighboring industrial US states, prevailing wind carries these pollutants to the London region, further subjecting the entire region to more ambient pollution. Consequently, we have embarked upon a comprehensive study aimed at assessing the potential burden of hospital admissions due to ambient air pollution in the region. As part of a larger study, this paper focuses on the association between ambient particulates and gaseous air pollutants and daily hospital admissions for respiratory and cardiovascular diseases in London, Ontario before 9/11. These results will establish a baseline with which we can compare findings from a future follow-up study of data being collected after 9/11.

The city of London (42° 59′ 00″ N; 81° 14′ 00″ W) is located along the Highway 401 in Southwestern Ontario and has a population of 432,451 in the metropolitan area (2001 Census of Canada). The city is noted for its economic diversity and is the home of many branches of industry, corporate offices, medical and educational facilities. It is also a manufacturing, distribution and financial centre. While London is usually referred to as a clean city, Burnett *et al.* [19], using 1981 to 1991 data, found the city to have the third highest increased risk of death (10.1%) attributable of change in mean air pollution among 11 Canadian cities. This current study will examine the time series relationship of daily hospitalization due to cardiovascular and respiratory diseases to ambient air pollutants using more recent data from 1 November 1995 to 31 December 2000.

2. Methodology

The study population consisted of all admissions in London hospitals during the given study period with primary diagnosis of asthma (ICD-9 code 493), all other respiratory diseases (460–519) and cardiovascular diseases (410–414, 427–428). All admissions for pre-arranged elective procedures and those that were transferred from other institutions were excluded. Daily hospital admission records for Ontario Health Insurance Plan patients were obtained from the Canadian Institute for Health's Discharge Abstract Database.

The hourly air pollution data from all monitoring stations in London were obtained from the Ministry of Environment for the same period. For each day, the highest hourly reading was taken for each city. These include gas data: sulphur dioxide (SO_2), carbon monoxide (CO), nitrogen oxide (NO_2), ozone (O_3), COH (coefficient of haze, a measure of organic and inorganic carbon), and inhalable particulates (PM_{10}). Daily weather data including maximum and minimum temperature and humidity were obtained from Environment Canada [20].

First, we linked together 1888 days of records from several databases comprising pollutants, temperature, humidity and pressure, and number of cardiovascular or respiratory admissions in the city hospitals. Data from CIHI were given to us in a ready-to-use format. Since we used the maximum of daily hourly pollutant values from four stations, there were not many missing values (less than 1%). If missing values were sporadic, we replaced the missing values by the mean of nearby points (3 days before and 3 days after). If missing values occurred for a series of days, we substituted the linear trend value for those points using other pollutants and covariates as predictors. In very few cases, if the highest hourly max was deemed extreme, it was replaced by the next highest value.

To relate short-term effects of air pollution to the number of hospital admissions, we used the time series procedure. In the past, the usual smoothing method that was used for producing residuals with no seasonality was locally weighted regression smoothers (LOESS) within the generalized additive model (GAM) [21]. It was later discovered [22–24] that the default settings of the GAM function in the computer software package S-Plus [25] do not assure convergence of its iterative estimation procedure and can provide biased estimates of regression coefficients and standard errors, especially when the concurvity is high. Dominici *et al.* [26] re-analyzed the National Morbidity, Mortality and Air Pollution Study data with the default implementation and found that the estimates were biased upward (i.e. higher than they should be). Since then, either the default option was set to a smaller number like 10^{-8} (S-Plus has already done that in their new release) or another smoother called natural splines is used in the generalized linear model (GLM) function.

We first removed the seasonal cycles, secular trends and day-of-the-week to produce a time series of logarithm of hospital admissions that is as close to white noise as possible, as determined by Bartlett's test [26]. Natural splines were used here to smooth the effects of all continuous covariates. We then extended the model by incorporating the smoothed weather variables (temperature and humidity) that yielded the lowest Akaike Information Criterion [27]. Last, we added the air pollution variable(s) into the model.

Relative risk of cardiovascular and respiratory hospital admissions attributable to each single pollutant using current day exposure level (1-day mean), average of today and yesterday's level (2-day mean), and average of current day and previous two day's level (3-day mean), were estimated for an increase in interquartile range (IQR = 75th percentile–25th percentile). Results are expressed as percentage changes in daily admissions associated with the pollutant, $[\exp(\beta \times IQR) - 1] \times 100\%$, where β is the estimated regression coefficient for pollutant. Ninety-five percent confidence intervals (CI) of the percentage change were

obtained on the assumption that the estimated regression coefficient was distributed normally.

3. Results

A total of 12,947 cardiac hospitalizations and 5574 respiratory admissions from 1 November 1995 to 31 December 2000 were analyzed in our study. Table 1 presents the summary statistics (mean, standard deviation, minimum and maximum) of maximum daily pollutant levels, weather, and number of respiratory and cardiac hospitalizations in this study period. Table 2 gives the correlation coefficients for the air pollutants and weather variables. All of the pollutants are positively correlated with each other, with O_3 and PM_{10} having the highest correlation of 0.53 and NO_2 and CO the next highest ($r = 0.45$).

The percentage changes in daily hospital admissions and 95% CI for different pollutants are given in table 3 for single pollutant models (current day to 3-day mean) for people < 65-years old and 65+. Results showed that the percentage increase in cardiac admissions, evaluated at the current day CO for an increase in IQR, was significant at 8.0% (95% CI: 1.5–11.5%) for people < 65-years old. For current day COH, the increase of hospital admissions was also significant at 5.7% (95% CI: 0.9–10.8%).

Although NO_2, O_3, COH and PM_{10} (current day to 3-day average) were all positively associated to respiratory hospitalizations for the elderly, none of these pollutants showed a significant positive relationship. Current day CO was found to have significant negative association with respiratory disease for people < 65-years old (decrease in admission of 7.8%, 95% CI: −14.8% to −0.1%). When we split all respiratory diseases into asthma and non-asthma respiratory diseases, an IQR increase in current-day PM_{10} produced a statistically significant increase of 25% in hospital admissions (95% CI: 2.8–52.3%) for people < 65-years old. Using the 2-day mean of PM_{10}, the increase in admissions was 26.0% (95% CI: 5.3%–50.9%), which was also significant (entire table of results not shown).

4. Discussion

In this paper, we related daily fluctuations in cardiac and respiratory hospital admissions for people < 65-years old and 65+ to daily variations in ambient levels of particulates and gaseous air pollutants in London, Ontario using data from 1995–2000.

The findings show that CO and COH contribute significantly to cardiovascular hospitalization. These results are quite consistent with previous findings in the literature. Using daily 1-h maximum concentration of CO data from 1981–1991, Burnett *et al.* [13] found positive associations with congestive heart failure hospitalization among the elderly in London, Ontario. In London, UK, Atkinson *et al.* [28] found a positive association between CO and emergency admission for cardiovascular disease. Lin *et al.* [29] also found an association between CO and ischaemic cardiac diseases in São Paulo, Brazil albeit for ages 45 to 80 years. Furthermore, Morris *et al.*[30] found that among the gaseous pollutants, CO was the only consistent pollutant for congestive heart failure admissions in seven large American cities. Similarly, Schwartz and Morris [31] found that CO and PM_{10} were

significant predictors of ischaemic heart disease hospitalizations in Detroit, Michigan using a two-pollutant model.

Our results also showed COH to be a significant predictor of cardiac admissions of people < 65 years of age in the single pollutant model. This is in agreement with recent results of Goldberg *et al.*[10] who found positive association between daily mortality for congestive heart failure and COH in Montreal, Quebec. The associations between cardiovascular diseases and some of the measures of NO_2 and PM_{10} were positive although not significant in this analysis.

While epidemiological findings linking air pollution to heart disease maybe consistent, the issue of biological plausibility remains uncertain since the underlying mechanisms of an acute heart effect of air pollution are still unknown [32]. Physiopathological pathways have been proposed for the relationship between particulate air pollution and cardiovascular health [33,34]. One of the major hypotheses is that particles induce activation of some mediators/indicators of alterations in blood coagulability [35]. Another mechanism that has been examined relates to cardiac autonomic control, such as heart rate and various indices of its variability [36]. Recently, Pope *et al.*[34] hypothesized that the link between fine particulates to cardiovascular mortality is via mechanisms that likely include pulmonary and systematic inflammation, accelerated atherosclerosis and altered cardiac autonomic function.

The significant association between PM_{10} and asthma admissions among the elderly in the city of London is consistent with those of Luginaah *et al.* [17] who found a significant increase in daily asthma admissions among the elderly women in Windsor, Ontario with elevated levels of PM_{10}. Schwartz [5] also found PM_{10} to be significantly associated with respiratory hospital admissions for elderly women in Spokane, Washington. Epidemiological studies [37–39] have demonstrated that acute increase in PM_{10} results in a greater use of asthma medication, more consultations of GPs and increased hospital admissions for asthma. However, the role of PM_{10} in raising the chances of initial sensitization and induction of disease has not been proved [40].

Our analysis showed a 2–3% increase in respiratory admissions among the elderly for ozone but it is not statistically significant. Yang *et al.*[41] found a positive association between O_3 and respiratory hospital admission among the elderly in Vancouver. In the 16 cities study across Canada, Burnett *et al.* [14] using data from 1981 to 1991, found positive associations between ozone and admission to hospital for respiratory diseases. However, Atkinson *et al.* [38] working in London, UK, did not find any association between O_3 and respiratory diseases.

Although the risks of health effects due to air pollution observed in these analyses are generally low, the implications for public health are important. Despite improvements in ambient air quality as a result of tighter regulations in the past few years [20], the event of 9/11 has brought with it renewed concerns about the effects of air pollution in Southwestern Ontario. The increasing delays resulting in long lines of trucks at the border crossing points, and the idling trucks spewing toxic pollutants from their archaic exhaust systems into the air is of grave concern. With London located downwind of Windsor and Detroit area, (which

together is a major source of industrial pollutants), the combined effect of these factors is that the conditions here may deteriorate. Increasing concerns about poor environmental quality in this region resulted in joint Canada and US border air strategy aimed at mapping the Southwestern Ontario air shed, and exploring its health impacts [42]. The plausible next step is to conduct more frequent air quality studies to continue to monitor any changes in hospitalizations due to ambient air pollution that may be taking place in London and the entire region.

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References

1. Dockery DW, Pope AC III, Xu X, Spengler JD, Ware JH, et al. An association between air pollution and mortality in six US cities. N Engl J Med. 1993; 329:1753–1759. [PubMed: 8179653]
2. Pope AC III, Dockery DW, Schwartz J. Review of epidemiological evidence of health effects of particulate air pollution. Inhalation Toxicology. 1995; 7:1–18.
3. Schwartz J. Air pollution and hospital admissions for cardiovascular disease in Tucson. Epidemiology. 1997; 8:371–377. [PubMed: 9209849]
4. Schwartz J. Air pollution and hospital admissions for respiratory disease. Epidemiology. 1996; 7(1): 20–28. [PubMed: 8664396]
5. Yang W, Jennison BL, Omaye ST. Cardiovascular disease hospitalization and ambient levels of carbon monoxide. J Toxicol Environ Health A. 1998; 55:185–196. [PubMed: 9772102]
6. Wong TW, Lau TS, Yu TS, Neller A, Wong SL, et al. Air pollution and hospital admissions for respiratory and cardiovascular diseases in Hong Kong. Occupational Environmental Medicine. 1999; 56(10):679–683. [PubMed: 10658547]
7. Anderson HR, Bremner SA, Atkinson RW, Harrison RM, Walters S. Particulate matter and daily mortality and hospital admissions in the west midlands conurbation of the United Kingdom: associations with fine and coarse particles, black smoke and sulphate. Occupational and Environmental Medicine. 2001; 58(8):504–510. [PubMed: 11452044]
8. Goldberg MS, Burnett R, Bailar JC III, Brook J, Bonvalot Y, et al. The association between daily mortality and short-term effects of ambient air particle pollution in Montreal, Quebec: 1. Nonaccidental mortality. Environ Res. 2001; A86:12–25.
9. Goldberg MS, Burnett R, Bailar JC III, Brook J, Bonvalot Y, et al. The association between daily mortality and short-term effects of ambient air particle pollution in Montreal, Quebec: 2. Cause-specific mortality. Environ Res. 2001; A86:26–36.
10. Goldberg MS, Burnett R, Valois M, Flegel K, Bailar JC, et al. Associations between ambient air pollution and daily mortality among persons with congestive heart failure. Environmental Research. 2003; 91:8–20. [PubMed: 12550083]
11. Lin M, Chen Y, Burnett RT, Villeneuve P, Krewski D. Effect of short-term exposure to gaseous pollution on asthma hospitalization in children: a bi-directional case-crossover analysis. J Epidemiol Community Health. 2002; 57:50–55.
12. Stern B, Jones L, Raizenne M, Burnett R, Meranger JC, et al. Respiratory health effects associated with ambient sulfates and ozone in two rural Canadian communities. Environmental Research. 1989; 49:20–39. [PubMed: 2721475]
13. Burnett RT, Dales RE, Brook JR, et al. Association between ambient carbon monoxide levels and hospitalizations for congestive heart failure in the elderly in 10 Canadian cities. Epidemiology. 1997; 8:162–167. [PubMed: 9229208]

14. Burnett RT, Brook JR, Yung WT, Dales RE, Krewski D. Association between ozone and hospitalization for respiratory diseases in 16 Canadian cities. Environmental Research. 1997; 72:24–31. [PubMed: 9012369]
15. Burnett RT, Smith-Doiron M, Stieb D, Cakmak S, Brook SR. Effects of particulate and gaseous air pollution on cardiorespiratory hospitalizations. Arch Environ Health. 1999; 54(2):130–139. [PubMed: 10094292]
16. Fung KY, Luginaah I, Gorey KM, Webster G. Air pollution and daily admissions for cardiovascular diseases in Windsor, Ontario. Canadian J of Public Health. 2005; 96:29–33.
17. Luginaah I, Fung KY, Gorey KM, Webster G. Association of ambient air pollution levels with asthma hospitalization in Windsor, Ontario. Environ Health Perspect. 2005; 113:290–296. [PubMed: 15743717]
18. Whelpdale DM. Large-scale atmospheric sulfur studies in Canada. Atmospheric Environment. 1978; 12:661.
19. Burnett RT, Cakmak S, Brook JR. The effect of the urban ambient air pollution mix on daily mortality rates in 11 Canadian cities. Canadian Journal of Public Health. 1998; 89:152–156. [PubMed: 9654797]
20. Ontario Ministry of the Environment. The Air Quality in Ontario. Toronto: Ontario Ministry of the Environment; 2000. Available at: http://www.ene.gov.on.ca/envision/air/AirQuality/2000report.htm [accessed 9 August 2002]
21. Hastie, T., Tibshirani, R. Generalized Additive Models. London: Chapman & Hall; 1990.
22. Samet JM, Dominici F, McDermott A, Zeger SL. New problems for an old design: time-series analyses of air pollution and health. Epidemiology. 2003; 14(1):11–12. [PubMed: 12500038]
23. Ramsay T, Burnett RT, Krewski D. The effect of concurvity in generalized additive models linking mortality to ambient particulate matter. Epidemiology. 2003; 14:18–23. [PubMed: 12500041]
24. Dominici F, McDermott A, Zeger SL, Samet JM. Airborne particulate matter and mortality: time-scale effects in four US cities (with invited commentary). Am J Epidemiol. 2003; 157:1055–1065. [PubMed: 12796040]
25. Insightful Corporation. S-PLUS 6 for Windows. Seattle, WA: Insightful Corporation; 2001.
26. Priestly, MB. Spectral Analysis of Time Series. San Diego, CA: Academic Press; 1981.
27. Akaike, H. Information theory and an extension of the maximum likelihood principle. In: Petrov, BN., Csaki, F., editors. Second International Symposium on Information Theory. Budapest: Academiai Kiado; 1973. p. 267-281.
28. Atkinson RW, Bremner SA, Anderson HR, Strachan DP, Bland JM, et al. Short-term associations between emergency hospital admissions for respiratory and cardiovascular disease and outdoor air pollution in London. Archives of Environmental Health. 1999; 54(6):398–411. [PubMed: 10634229]
29. Lin CA, Pereira LAA, Conceic GMD, Kishi HS, Milani R Jr, et al. Association between air pollution and ischemic cardiovascular emergency room visits. Environmental Research. 2003; 92:57–63. [PubMed: 12706756]
30. Morris R, Naumova R, Munasinghe R. Ambient air pollution and hospitalization for congestive heart failure among elderly people in seven large US cities. Am J Public Health. 1995; 85(10): 1361–1366. [PubMed: 7573618]
31. Schwartz J, Morris R. Air pollution and hospital admissions for cardiovascular disease in Detroit, Michigan. Am J Epidmiol. 1995; 142(1):23–35.
32. Sunyer J, Ballester F, Le Tertre A, Atkinson R, Ayres JG, et al. The association of daily sulfur dioxide air pollution levels with hospital admissions for cardiovascular diseases in Europe (The Aphea-II study). European Heart Journal. 2003; 24:752–760. [PubMed: 12713769]
33. Tunnicliffe WS, Mark D, Harrison RM, et al. Effect of particle and sulphur dioxide challenge on heart rate variability in normal and asthmatic subjects. European Respiratory Journal. 2001; 17:604–608. [PubMed: 11401052]
34. Pope CA III, Burnett RT, Thurston AD, Thun MK, Calle ME, et al. Cardiovascular mortality and long-term exposure to particulate air pollution. Circulation. 2004; 109:71–77. [PubMed: 14676145]

35. Seaton A, MacNee W, Donaldson K, et al. Particulate air pollution and acute health effects. Lancet. 1995; 345(8943):176–178. [PubMed: 7741860]

36. Gold DR, Litonjua A, Schwartz J, Lovett E, Larson A, et al. Ambient pollution and heart rate variability. Circulation. 2000; 101(11):1267–1273. [PubMed: 10725286]

37. Lipsett M, Hurley S, Ostro B. Air pollution and emergency room visits for asthma in Santa Clara county, California. Environ Health Perspect. 1997; 105:216–222. [PubMed: 9105797]

38. Atkinson RW, Anderson HR, Strachan DP, Bland JM, Bremmer SA, et al. Short-term associations between outdoor air pollution and visits to accident and emergency departments in London for respiratory complaints. Eur Respir J. 1999; 13:257–265. [PubMed: 10065665]

39. Anderson HR, Ponce de Leon L, Bland JM, Bower JS, Emberlin J, et al. Air pollution, pollens, and daily admissions for asthma in London 1987–92. Thorax. 1998; 53:842–848. [PubMed: 10193370]

40. Donaldson K, Gilmour MI, MacNee W. Asthma and PM_{10}. Respir Res. 2000; 1:12–15. [PubMed: 11667958]

41. Yang Q, Chen Y, Shi Y, Burnett RT, McGrail KM, Krewski D. Association between ozone and respiratory admissions among children and the elderly in Vancouver, Canada. Inhalation Toxicology. 2003; 15(13):1297–1308. [PubMed: 14569494]

42. Anderson, D, Hon. Minister for the Environment. Speaking notes: Border Air Quality Strategy Pilot Project Announcement. Washington, DC: 2003 Jun 23. Strengthening our cooperation for clean air: Canada-US air quality cooperation.

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Table 1

Mean and standard deviations of maximum daily pollutant levels, weather and number of cardiac and respiratory hospitalizations in London, Ontario, 1 November 1995–31 December 2000.

Variables	Mean	SD	Minimum	Maximum (AAQC[a])
Air pollutants				
NO_2 (ppb)	32.7	12.8	0	104(100/24 h)
SO_2 (ppb)	20.0	20.8	0	216(100/24 h)
CO (ppm)	0.5	0.6	0	4(3/h)
O_3 (ppb)	50.6	19.9	14	146(80/h)
COH	1.2	0.6	0.2	4.8(1.0/24h)
PM_{10} (μg/m^3)	38.0	23.5	5	248(30/24h)
Weather				
Max. temp.(°C)	12.6	11.2	−16.7	35
Min. temp. (°C)	3.0	9.8	−24.8	25
Max. humidity	91.6	8.5	49	100
Min humidity	58.4	15.7	17	100
Daily respiratory admissions				
Age< 65 (n = 2436)	1.29	1.26	0	7
Age 65+ (n = 3138)	1.66	1.48	0	12
Daily cardiac admissions				
Age < 65 (n = 3744)	2.0	1.5	0	8
Age 65+ (n = 9203)	4.9	2.2	0	13

Note:

[a]Ambient Air Quality Criteria [20].

Table 2

Correlation coefficients between air pollutants and weather variables.

	PM_{10}	NO_2	SO_2	CO	O_3	COH	Mint	Minh	Maxt	Maxh
PM_{10}	1.00									
NO_2	0.30	1.00								
SO_2	0.24	0.21	1.00							
CO	0.21	0.45	0.11	1.00						
O_3	0.53	0.13	0.28	0.05	1.00					
COH	0.29	0.31	0.15	0.21	0.26	1.00				
Mint	0.32	−0.12	0.17	−0.08	0.60	0.20	1.00			
Minh	−0.32	−0.18	−0.13	−0.12	−0.36	−0.11	−0.17	1.00		
Maxt	0.43	0.00	0.25	0.02	0.67	0.27	0.94	−0.36	1.00	
Maxh	−0.16	−0.04	−0.03	0.07	−0.09	0.03	0.12	0.50	0.07	1.00

Notes: Mint = minimum temperature; Maxt = maximum temperature; Minh = minimum humidity; Maxh = maximum humidity.

Table 3

Percentage change in daily admission and 95% confidence interval (CI) for different air pollutants in single pollutant models for an increase in interquartile range (IQR)[a].

		Cardiac diseases		Respiratory diseases	
Pollutants	**IQR**	**< 65 (*n* = 3744)**	**65+ (*n* = 9203)**	**<65 (*n* = 2436)**	**65+ (*n* = 3138)**
NO_2					
Current day	17	4.2 (−1.2,9.7)	−0.9 (−4.2,2.5)	−4.8 (−10.7,1.5)	2.2 (−3.3,8)
2-day mean		4.2 (−2.3,11.2)	−1.2 (−5.3,3.1)	−2.3 (−9.7,5.8)	1 (−5.7,8.1)
3-day mean		3.7 (−3.8,11.8)	−2.4 (−7.1,2.6)	−3.1 (−11.8,6.3)	0.4 (−7.4,8.8)
SO_2					
Current day	17	1.6 (−1.5,4.7)	1.2 (−0.9,3.3)	0.2 (−3.5,4.1)	−0.3 (−3.6,3.1)
2-day mean		0.7 (−3.3,4.9)	2.5 (−0.2,5.2)	2.6 (−2.3,7.8)	−1.7 (−6,2.8)
3-day mean		0.2 (−4.4,5.1)	2.8 (−0.3,6.1)	0.8 (−5.1,7.1)	−2.5 (−7.7,3)
CO					
Current day	1	8.0 (1.5,14.9)	0.3 (−3.9,4.8)	−7.8 (−14.8,-0.1)	−0.7 (−7.7,6.7)
2-day mean		7.4 (−0.6,16)	0.2 (−5.1,5.7)	−8.3 (−17,1.3)	−0.7 (−9.4,8.8)
3-day mean		6.5 (−2.5,16.3)	−2.5 (−8.3,3.7)	−7.7 (−17.7,3.5)	−4.1 (−13.8,6.6)
O_3					
Current day	22	−3.4 (−10.8,4.6)	4.4 (−0.8,10)	−3.5 (−13.0,7.1)	3.2 (−3.8,10.8)
2-day mean		−2 (−10.4,7.1)	2.8 (−3.1,9)	−0.2 (−11.3,12.3)	3.1 (−6,12.9)
3-day mean		−3.3 (−12.1,6.4)	3 (−3.2,9.7)	0.5 (−11.4,14)	2.6 (−7.8,14.1)
COH					
Current day	0.7	5.7 (0.9,10.8)	2.1 (−1.1,5.3)	−1.8 (−7.5,4.2)	3.2 (−1.7,8.4)
2-day mean		4.6 (−1.8,11.3)	1.9 (−2.2,6.2)	−1.2 (−8.8,6.9)	2.3 (−4.2,9.3)
3-day mean		5.8 (−1.7,13.9)	1.1 (−3.7,6.1)	1 (−8.1,11)	2.9 (−5.1,11.5)
PM_{10}					
Current day	26	2.6 (−2.3,7.7)	0.9 (−2.3,4.2)	−0.9 (−6.8,5.4)	3.3 (−1.7,8.6)
2-day mean		−1.2 (−7.2,5.1)	−0.9 (−4.8,3.2)	−1.3 (−8.5,6.6)	5 (−1.5,11.9)
3-day mean		−3 (−9.6,4)	−0.1 (−4.4,4.5)	1.9 (−6.5,11)	1.2 (−6.1,9.1)

Note:

[a]IQR = 75th percentile–25th percentile.