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Research

Temporal and spatial effect of air pollution on hospital admissions for myocardial infarction: a case-crossover study

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Abstract

Background: In studies showing associations between ambient air pollution and myocardial infarction (MI), data have been lacking on the inherent spatial variability of air pollution. The aim of this study was to determine whether the long-term spatial distribution of air pollution influences short-term temporal associations between air pollution and admission to hospital for MI.

Methods: We identified adults living in Calgary who were admitted to hospital for an MI between 2004 and 2012. We evaluated associations between short-term exposure to air pollution (ozone [O_3], nitrogen dioxide [NO_2], sulfur dioxide [SO_2], carbon monoxide [CO], particulate matter < 10 μm in diameter [PM_{10}] and particulate matter < 2.5 μm in diameter [$PM_{2.5}$]), and hospital admissions for MI using a time-stratified, case-crossover study design. Air Quality Health Index (AQHI) scores were calculated from a composition of O_3, NO_2 and $PM_{2.5}$. Conditional logistic regression models were stratified by low, medium and high levels of neighbourhood NO_2 concentrations derived from land use regression models; results of these analyses are presented as odds ratios (ORs) with 95% confidence intervals (CIs).

Results: From 2004 to 2012, 6142 MIs were recorded in Calgary. Individuals living in neighbourhoods with higher long-term air pollution concentrations were more likely to be admitted to hospital for MI after short-term elevations in air pollution (e.g., 5-day average NO_2: OR 1.20, 95% CI 1.03–1.40, per interquartile range [IQR]) as compared with regions with lower air pollution (e.g., 5-day average NO_2: OR 0.90, 95% CI 0.78–1.04, per IQR). In high NO_2 tertiles, the AQHI score was associated with MI (e.g., 5-day average OR 1.13, 95% CI 1.02–1.24, per IQR; 3-day average OR 1.13, 95% CI 1.04–1.23, per IQR).

Interpretation: Our results show that the effect of air pollution on hospital admissions for MI was stronger in areas with higher NO_2 concentrations than that in areas with lower NO_2 concentrations. Individuals living in neighbourhoods with higher traffic-related pollution should be advised of the health risks and be attentive to special air quality warnings.

Studies have consistently shown that short-term elevations in air pollution concentrations increase the risk of myocardial infarction (MI).[1,2] Improving our understanding of the effects of short-term exposure to air pollution on MI may inform government policy and facilitate prevention by warning populations at risk. In Calgary, the major contributors to air pollution are transportation for nitrogen dioxide (NO_2) and carbon monoxide (CO); construction for particulate matter less than 10 μm in diameter (PM_{10}) and particulate matter less than 2.5 μm in diameter ($PM_{2.5}$); and industry for sulfur dioxide (SO_2).[3] The relatively higher air pollution regions, from a long-term exposure perspective, are mainly distributed along major traffic corridors and close to industrial areas.[4,5] Air pollution exposure studies that consider an average of air pollution levels overlook the inherent spatial nature of air pollution.[6,7]

Historically, temporal analyses exploring the association between short-term exposure to air pollution and health outcomes have assumed that pollutants are spatially homogeneous.[8–11] However, research has shown that spatial distribution patterns differ by pollutant.[12] For example, it is widely recognized that ozone (O_3) is relatively spatially homogenous because of consistent concentration levels and temporal fluctuations, whereas NO_2 is spatially heterogeneous

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because it is attributable to traffic emissions. If city-wide averages are used as air pollution estimates, they fail to consider the spatial variation within a city.[11] The aim of this study was to determine whether the long-term spatial distribution of air pollution influences the short-term temporal associations between air pollution and MI.

Methods

Study design

We used a time-stratified, case-crossover study design to evaluate associations between a short-term exposure and the acute onset of a disease;[13,14] this is an adaptation of the case–control study in which cases serve as their own controls (Figure 1).[15] This study design has been used extensively to characterize associations between day-to-day changes in air pollution and adverse health events. Because within-individual comparisons are being made, confounding from time-independent risk factors is controlled for by the design of the study. The case-crossover study design has been shown to effectively control for confounders that are relatively stable in time, such as obesity, diabetes, smoking and socioeconomic status.[16]

The case's exposure at the index time (i.e., day of admission for MI) is compared with its exposure at control time intervals, which are chosen using a time-stratified design.[17] The index period is measured before the event and the control period is measured before and after the event.[18–20] For example, if the MI occurs on the second Wednesday in the month of July of 2011, then the referent period will be the other Wednesdays in July of 2011. The time-stratified approach matches the exposure by day of the week and month to control for the influence of day-of-week effects. It also adjusts for seasonal trends in exposure levels.[21] The time-stratified approach is not subject to bias resulting from time trends, because there is no pattern in the placement of referents relative to the index time.[16,17,21]

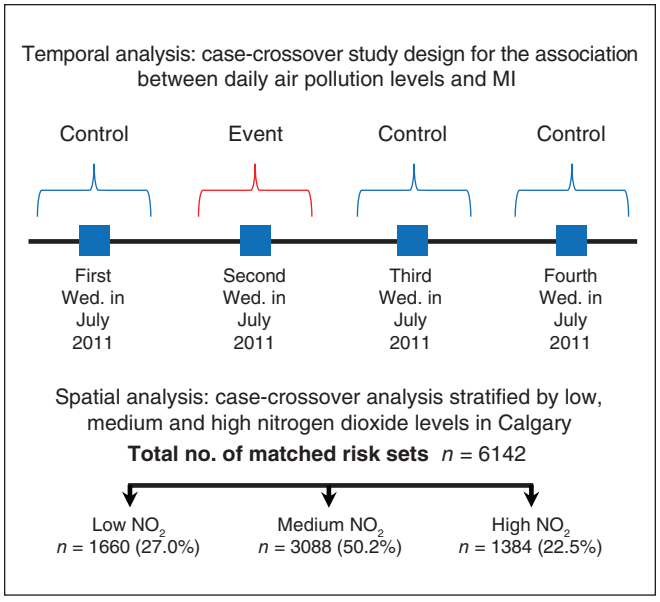

Figure 1: Study design for temporal and spatial analysis of air pollutants effect on hospital admission for myocardial infarction (MI). A time-stratified case-crossover study design that evaluates the short-term (temporal) effect of O_3, NO_2, SO_2, CO, PM_{10} and $PM_{2.5}$ on MI. Analyses are stratified by high, medium and low levels of NO_2 (spatial) as defined by land use regression estimates. Ten patients had missing data. Note: CO = carbon monoxide, NO_2 = nitrogen dioxide, O_3 = ozone, PM_{10} = particulate matter < 10 μm in diameter, $PM_{2.5}$ = particulate matter < 2.5 μm in diameter, SO_2 = sulfur dioxide.

Study population

Our population comprised adults over the age of 18 years at the time of incidence of MI, living in Calgary and admitted to hospital with a diagnosis of MI during the study period from Jan. 1, 2004, to Dec. 31, 2012. Patients who died before presentation to an emergency department were excluded. The population was extracted by first acute MI diagnosis, including ST elevation MI and non–ST elevation MI.

Data sources

The Alberta Provincial Project for Outcome Assessment in Coronary Heart Disease (APPROACH) is a registry that captures all patients undergoing cardiac catheterization in the province of Alberta since Jan. 1, 1995 (www.approach.org). In 2004, APPROACH expanded to include the Heart Alert initiative in southern Alberta, which enhances data collection by including detailed information on all patients admitted to cardiology services of acute care facilities in Calgary. The data collection is prospective and collected by trained clinical staff using standardized operating procedures and data definitions as part of the medical record in Alberta, and therefore, missing data on key variables are minimal.

Air pollution data were obtained from automated fixed-site continuous monitoring stations maintained by Environment and Climate Change Canada as part of the National Air Pollution Surveillance Network.[22–24] The 3 stations were Calgary Central, Calgary East and Calgary Northwest, which are positioned to be representative of the background air pollution concentrations across Calgary. Hourly data from each of the 3 fixed sites were averaged together to provide regional estimates of hourly concentrations of the 6 criteria air pollutants investigated in this study: O_3, NO_2, SO_2, CO, PM_{10} and $PM_{2.5}$. Daily air pollution levels for Calgary were calculated from hourly records by averaging across the 3 fixed-site monitoring stations.[25] For all air pollutants, with the exception of ozone, daily mean exposure estimates were used. Ozone values were based on an 8-hour maximum value. In addition, Air Quality Health Index (AQHI) scores were calculated from a composition of 3-hour average values of O_3, NO_2 and $PM_{2.5}$ based on the following formula:[26]

$$AQHI = 1000/10.4 \times [(e^{0.000871 \times NO_2} - 1) + (e^{0.000537 \times O_3} - 1) + (e^{0.000487 \times PM_{2.5}} - 1)]$$

Data for daily mean temperature and relative humidity were provided by Environment and Climate Change Canada, which averaged the hourly mean temperature and relative humidity across the 3 monitoring stations. These daily time

series of meteorological data were linked with MI hospital admissions and used as adjustment factors in a multivariable conditional logistic regression model.

Land use regression (LUR) models have been widely used to assess the spatial variation of outdoor air pollution and to estimate fine scale pollution concentrations.[27–30] Land use regression models capture longer-term measures of ambient air pollution rather than day-to-day fluctuations. Substantial intra-urban variation for NO_2, $PM_{2.5}$ and metals associated with PM_{10} has been observed in previous analyses conducted on air pollution with LUR models in Calgary.[4,31] These previous studies suggest that the major contributors to the spatial variation of air pollution are emissions from motor vehicles and industrial sources,[32] resulting in relatively higher air pollution along major traffic corridors and the Northeast Industrial area.[4,5] The stability of LUR models over time has been previously validated.[33] Further, the LUR model used in Calgary was shown to remain stable over a 5-year interval.[34] We used the NO_2 estimates from the air pollution study reported by Bertazzon and colleagues for the study period.[4] Land use regression estimates were assigned to each patient based on the 6-digit postal codes of their residential locations, which we defined as their neighbourhood. Patients who were admitted to hospital for MI were then divided into tertiles based on ambient NO_2 concentrations at their residential locations: low NO_2 pollution (first tertile), medium NO_2 pollution (second tertile) and high NO_2 pollution (third tertile) (Figure 2). Patients who were admitted to hospital for MI were assigned to only 1 of the 3 NO_2 concentrations based on the 6-digit postal codes of their residential locations.

Statistical analysis

To evaluate the temporal relation between outdoor air pollution levels (O_3, NO_2, SO_2, CO, PM_{10}, $PM_{2.5}$ and AQHI score) and presentation to hospitals because of MI, we constructed several different metrics: same-day exposure, 1-day and 2-day lagged exposures and cumulative 3-day and 5-day average exposure estimates. Correlation between pollutants was assessed using Pearson correlation coefficients. After matching the case period and referent periods, we used conditional logistic regression to produce risk estimates by comparing exposure data on case and control days. Odds ratios (ORs) with associated 95% confidence intervals (CIs) were calculated to describe the association between hospital admissions for MI and any increase in the interquartile range (IQR) of the daily concentrations of air pollutants during the different time intervals. We adjusted ORs for temperature and relative humidity.[22,35] Temperature and relative humidity were entered as linear terms in the models. We verified the linearity of the relation using natural cubic spline functions. The AQHI score was also included in a separate model to explore the composite effects of air pollution on MI. Finally, each pollutant model (O_3, NO_2, SO_2, CO, PM_{10}, $PM_{2.5}$ and the AQHI score) was stratified by an individual's residential exposure to NO_2 concentrations (stratified as high, medium and low, based on their 6-digit postal code), as derived from LUR models. Model stratification of high NO_2 was compared with low NO_2 concentration using a Cochran Q test.

Ethics approval

This study was approved by the University of Calgary's Conjoint Health Research Ethics Board (CHREB) and the Health Canada-Public Health Agency of Canada Research Ethics Board.

Results

We identified 6142 adult patients admitted to hospital for MI during the study period (Table 1). Of all patients who had an MI, 4482 (72.9%) were men, 3209 (52.2%) patients were aged 65 years or younger, 1493 (24.3%) patients had diabetes and 3646 (59.2%) patients were either a current or former smoker. When stratified by residential location, 1384 (22.5%) of patients who had an MI lived in neighbourhoods with the highest tertile of NO_2 pollution. The distribution of air pollutants and their correlations are provided in Appendix 1, available at www.cmajopen.ca/content/8/4/E619/suppl/DC1.

Associations between short-term air pollution and MI are shown in Table 2. For the overall city-wide study population, only 1-day lag for SO_2 exhibited a significant positive association with admissions for MI (OR 1.05, 95% CI 1.01–1.09 per IQR). Associations between short-term pollutant concentrations and MI were observed for those residing in neighbourhoods with the highest long-term concentrations of NO_2. With the exception of O_3, all pollutants were associated with MI in high NO_2 areas with ORs ranging from 1.06 to 1.20 per IQR. The strongest effect on hospital admissions for MI was identified for 5-day cumulative average of NO_2 (OR 1.20, 95% CI 1.03–1.40 per IQR) in high NO_2 areas, whereas the association was not significant in low NO_2 areas (5-day average NO_2: OR 0.90, 95% CI 0.78–1.04, per IQR; Table 2). In high NO_2 regions, the AQHI was significantly associated with MI (5-day average OR 1.13, 95% CI 1.02–1.24 per IQR; 3-day average OR 1.13, 95% CI 1.04–1.23 per IQR).

Interpretation

We evaluated the associations between air pollution and risk of hospital admissions for MI with a time-stratified, case-crossover study design. Our analysis was consistent with that of Wang and colleagues, who also explored the effects of air pollution on MI in Calgary.[25] Neither that study nor ours identified strong temporal effects of air pollution on MI when the spatial distribution of air pollution was assumed to be homogenous across the city of Calgary. The weak association between air pollution and MI in our non–spatially stratified analyses may partially be explained by the generally low air pollution concentrations in Calgary, where warning advisories were issued for less than 1% of days annually during our study period.[36] Environment and Climate Change Canada reported in 2015 that air pollution (NO_2, SO_2, O_3 and CO) had substantially improved, including in Calgary, from 1990 to 2015.[37] In a small part, improved air quality in

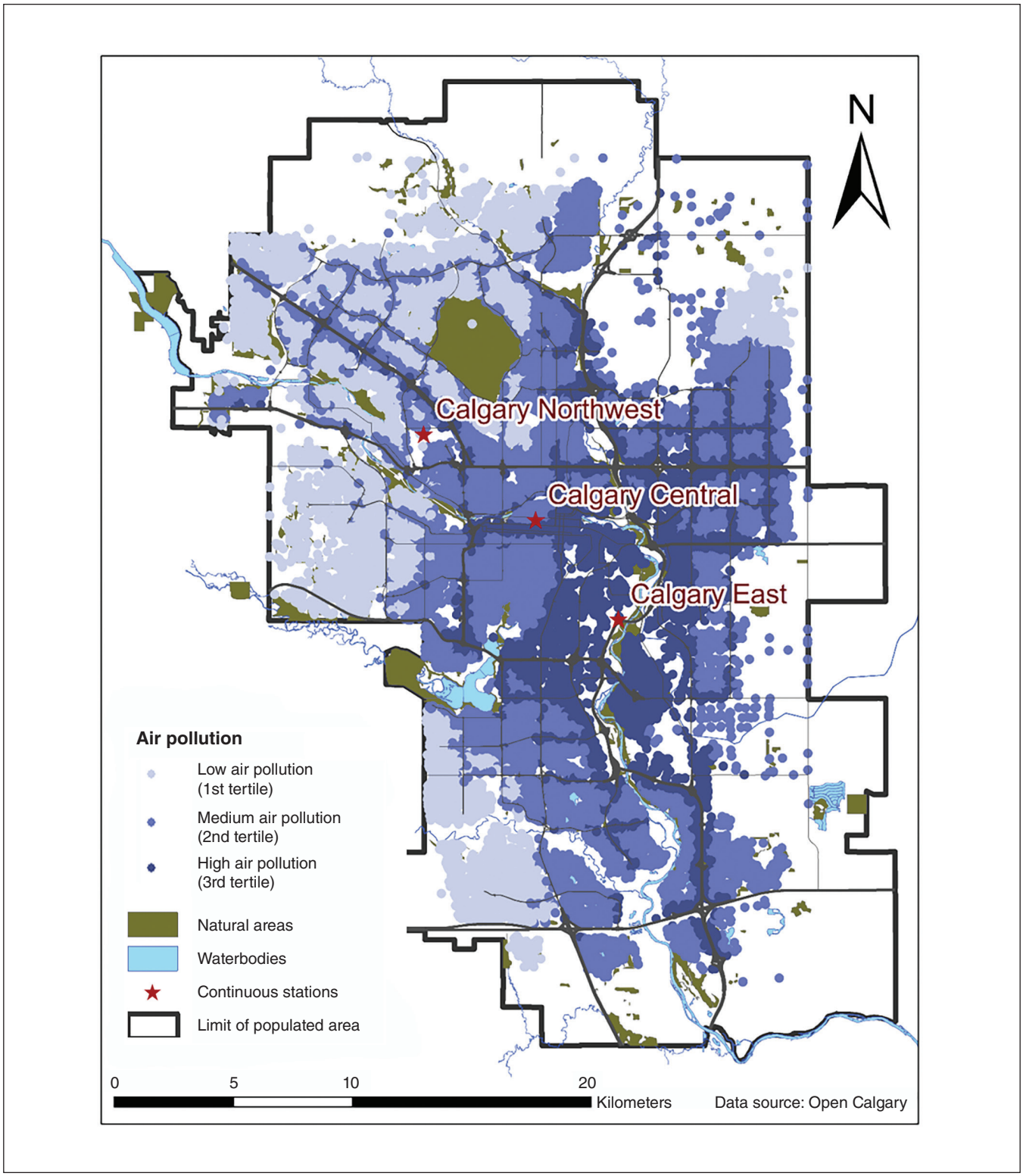

Figure 2: Spatial distribution of nitrogen dioxide in the city of Calgary derived from land use regression estimates. Each dot represents the centroid of a 6-digit postal code with darker shading representing higher air pollution and lighter shading representing lower air pollution. Stars denote the 3 continuous monitoring stations in Calgary.

Calgary may also explain the decreased incidence of hospital admissions for MI in Calgary, as observed by Liu and Bertazzon between 2004 and 2013.[38]

However, in our study, we also stratified our models by spatial distribution of NO_2. Individuals living in regions of high NO_2 exposure showed significant associations for all

Table 1: Demographic characteristics of patients who were admitted to hospital for myocardial infarction in Calgary (2004–2012)

Characteristic	No. (%) of patients *n* = 6142
Sex	
Male	4482 (72.9)
Female	1660 (27.0)
Age, yr	
≤ 65	3209 (52.2)
> 65	2933 (47.7)
Diabetes	
No diabetes	4649 (75.6)
Diabetes	1493 (24.3)
Cigarette smoking	
Never smoker	2496 (40.6)
Former smoker	1798 (29.2)
Current smoker	1848 (30.0)
Residential location*	
Low NO_2 air pollution (1st tertile)	1660 (27.0)
Medium NO_2 air pollution (2nd tertile)	3088 (50.2)
High NO_2 air pollution (3rd tertile)	1384 (22.5)

Note: NO_2 = nitrogen dioxide.
*10 patients with missing data.

individual pollutants and hospital admissions for MI with the exception of O_3. Further, the AQHI score was also associated with hospital admissions for MI for patients living in areas with higher NO_2 concentrations. These findings imply that living in a neighbourhood with elevated long-term exposure to NO_2 may predispose residents of these areas to the acute effects of air pollution that are associated with an increased risk of MI. Alternatively, acute temporal spikes in air pollution levels across the city may be amplified in areas with higher baseline NO_2. These results highlight the importance of accounting for spatial variation when studying the health effects of air pollution.

Associations between SO_2 and MI are consistently reported in the literature. Mustafic and colleagues provide a systematic review concluding that SO_2 was positively associated with increased incidence of MI.[2] Our results also align with previous studies that report that O_3 has no association with hospital admissions for MI.[2,25,39] In addition, our results suggest that NO_2 and $PM_{2.5}$ levels are associated with increased MI in areas of medium NO_2 ($PM_{2.5}$ only) and areas of high NO_2 (both NO_2 and $PM_{2.5}$); this is aligned with previous studies that find a positive association between MI and NO_2 and $PM_{2.5}$.[2,35]

The AQHI score, as a composite score indicating the overall air quality, did not exhibit a positive association with MI except in areas of high NO_2. However, most evidence to date indicates that the effects of air pollution are linear, particularly for O_3 and $PM_{2.5}$, such that detection of effects is not dependent on infrequent days with high pollutant concentrations.[40,41] The AQHI score is calculated based on the combination of NO_2, O_3 and $PM_{2.5}$, of which O_3 exhibited no significant associations with MI among either the entire study population or any subgroups, whereas NO_2 and $PM_{2.5}$ exhibited significant associations in our spatial stratification.

Limitations

A limitation of our study was the use of fixed-site monitoring data as opposed to personal monitoring. Air pollution studies that rely on outdoor air pollution monitoring are subject to misclassification of the exposure (i.e., measurement error). For example, averaging measurements from the 3 fixed monitoring sites into 1 daily value for the city of Calgary may lead to air pollution exposure misclassification at the individual level. In addition, air pollution monitoring occurred outdoors, which does not account for differences of indoor air pollution exposure. Furthermore, fixed-site monitors do not account for individual mobility, as an individual may not have been near their home when they experienced an MI. The Canadian Human Activity Pattern Survey 2 was a national survey that showed that most individuals spent the day indoors and that seniors spent more than 80% of their time at or near their home.[42] Typically, these errors in measurement result in nondifferential exposure misclassification, which serve to underestimate the risks of air pollution.[20,43]

The case-crossover study design of temporal associations controls for non–time dependent confounders; however, factors such as pollen that vary on a daily basis and may be correlated

Table 2: Association between air pollution and hospital admission for myocardial infarction with increases in the interquartile range of pollutants during various referent time intervals in regions with differing nitrogen dioxide pollution levels

Pollutant (median with IQR)	Lag (days)	OR* (95% CI)			
		Entire study population (citywide NO_2) n = 6142	Low NO_2 tertile n = 1660	Medium NO_2 tertile n = 3088	High NO_2 tertile† n = 1384
CO (0.35, IQR 0.27–0.47)	0 index day	0.97 (0.93–1.02)	0.94 (0.88–1.01)	0.97 (0.92–1.02)	1.02 (0.95–1.09)
	1-day lag	1.03 (0.98–1.08)	0.98 (0.92–1.05)	1.02 (0.97–1.07)	**1.10 (1.02–1.18)**
	2-day lag	1.01 (0.96–1.06)	1.03 (0.96–1.10)	0.99 (0.94–1.04)	1.04 (0.97–1.12)
	0- to 2-day average	1.01 (0.94–1.08)	0.97 (0.88–1.06)	0.99 (0.92–1.05)	1.09 (0.99–1.21)
	0- to 4-day average	0.97 (0.90–1.05)	0.92 (0.83–1.03)	0.96 (0.88–1.03)	1.08 (0.96–1.21)
NO_2 (18.22, IQR 12.67–25.00)	0 index day	1.00 (0.94–1.07)	0.97 (0.89–1.06)	1.01 (0.95–1.08)	1.05 (0.95–1.15)
	1-day lag	1.04 (0.97–1.11)	1.00 (0.91–1.09)	1.01 (0.94–1.07)	**1.16 (1.05–1.28)†**
	2-day lag	1.03 (0.97–1.10)	1.03 (0.94–1.12)	1.00 (0.93–1.06)	**1.11 (1.01–1.22)**
	0- to 2-day average	1.05 (0.96–1.14)	1.00 (0.88–1.12)	1.01 (0.92–1.10)	**1.20 (1.05–1.36)**
	0- to 4-day average	0.98 (0.88–1.08)	0.90 (0.78–1.04)	0.93 (0.84–1.03)	**1.20 (1.03–1.40)†**
O_3 max (39.00, IQR 32.00–47.00)	0 index day	1.00 (0.95–1.06)	1.02 (0.96–1.10)	0.98 (0.93–1.03)	1.03 (0.96–1.11)
	1-day lag	0.99 (0.94–1.04)	0.96 (0.90–1.03)	0.97 (0.93–1.03)	1.07 (0.99–1.15)
	2-day lag	0.99 (0.94–1.04)	0.97 (0.91–1.04)	0.99 (0.94–1.04)	1.02 (0.94–1.10)
	0- to 2-day average	0.99 (0.93–1.06)	0.98 (0.90–1.07)	0.97 (0.91–1.03)	1.06 (0.96–1.17)
	0- to 4-day average	1.00 (0.93–1.08)	1.00 (0.90–1.11)	0.97 (0.90–1.04)	1.09 (0.97–1.22)
SO_2 (1.00, IQR 1.00–2.00)	0 index day	1.00 (0.96–1.05)	1.00 (0.95–1.06)	0.97 (0.93–1.01)	**1.08 (1.02–1.15)†**
	1-day lag	**1.05 (1.01–1.09)**	1.03 (0.98–1.09)	1.04 (1.00–1.08)	**1.10 (1.03–1.16)**
	2-day lag	1.04 (0.99–1.08)	1.04 (0.98–1.10)	1.03 (0.99–1.07)	1.05 (0.99–1.12)
	0- to 2-day average	1.06 (1.00–1.12)	1.05 (0.97–1.14)	1.03 (0.97–1.09)	**1.15 (1.06–1.25)**
	0- to 4-day average	1.05 (0.98–1.12)	1.05 (0.96–1.16)	1.02 (0.95–1.09)	1.10 (0.99–1.22)
PM_{10} 20.00, IQR 14.00–30.00)	0 index day	0.98 (0.95–1.02)	0.95 (0.90–1.00)	0.98 (0.95–1.02)	1.03 (0.97–1.08)
	1-day lag	1.01 (0.97–1.05)	0.97 (0.92–1.02)	1.01 (0.97–1.04)	**1.06 (1.01–1.12)†**
	2-day lag	1.01 (0.98–1.05)	0.99 (0.95–1.04)	1.00 (0.96–1.04)	1.06 (1.00–1.12)
	0- to 2-day average	1.00 (0.96–1.05)	0.95 (0.89–1.01)	0.99 (0.95–1.04)	**1.08 (1.01–1.16)†**
	0- to 4-day average	0.99 (0.94–1.05)	0.95 (0.88–1.02)	0.98 (0.93–1.04)	1.07 (0.99–1.15)
$PM_{2.5}$ (7.00, IQR 4.33–10.50)	0 index day	1.01 (0.98–1.05)	0.99 (0.94–1.05)	1.00 (0.97–1.04)	**1.06 (1.00–1.11)**
	1-day lag	1.02 (0.99–1.06)	0.98 (0.93–1.03)	**1.04 (1.01–1.08)**	1.04 (0.99–1.10)
	2-day lag	1.00 (0.96–1.04)	0.96 (0.90–1.01)	1.02 (0.97–1.06)	1.03 (0.98–1.08)
	0- to 2-day average	1.02 (0.97–1.07)	0.96 (0.90–1.03)	1.03 (0.98–1.08)	1.07 (1.00–1.13)
	0- to 4-day average	1.02 (0.97–1.08)	0.98 (0.91–1.06)	1.04 (0.99–1.10)	1.04 (0.96–1.12)
AQHI (4.01, IQR 3.49–4.65)	0 index day	1.01 (0.96–1.05)	1.00 (0.94–1.06)	0.99 (0.95–1.04)	1.06 (0.99–1.13)
	1-day lag	1.02 (0.97–1.07)	0.97 (0.91–1.03)	1.00 (0.96–1.05)	**1.12 (1.05–1.20)†**
	2-day lag	1.01 (0.97–1.06)	0.99 (0.93–1.05)	1.00 (0.95–1.04)	1.07 (1.00–1.14)
	0- to 2-day average	1.02 (0.96–1.08)	0.98 (0.90–1.06)	1.00 (0.94–1.05)	**1.13 (1.04–1.23)†**
	0- to 4-day average	1.01 (0.94–1.08)	0.97 (0.89–1.07)	0.97 (0.91–1.04)	**1.13 (1.02–1.24)†**

Note: CI = confidence interval, CO = carbon monoxide, IQR = interquartile range, NO_2 = nitrogen dioxide, OR = odds ratio, O_3 = ozone, PM_{10} = particulate matter < 10 μm in diameter, $PM_{2.5}$ = particulate matter < 2.5 μm in diameter, SO_2 = sulfur dioxide.
*Odds ratios are adjusted for temperature and relative humidity.
†Significant difference comparing OR in the highest NO_2 tertile to the lowest NO_2 tertile using the Cochran Q test.
Significant associations are bolded.

to air pollution levels are not controlled in a case-crossover study design. Although temporally stable cardiac risk factors (e.g., hypertension, diabetes and dyslipidemia) are controlled, risk factors such as smoking that may vary from day to day (e.g., smoking only on the weekend) may introduce bias in the temporal analyses. In addition, our stratified spatial analysis based on residential NO_2 exposure was subject to confounding because high pollution areas may correspond with other risk factors for MI, such as low socioeconomic status and obesity. For example, an individual with low socioeconomic status may live in proximity to major traffic arteries (i.e., higher air pollution) because these neighbourhoods have lower property value. Thus, the results of the current study should be interpreted cautiously without inference to causality. Further investigation on whether living in a high pollution area increases vulnerability to temporal spikes in pollution concentrations is necessary.

Misclassification of the timing of onset of MI may introduce bias into the results. In addition, the study was restricted to individuals who were admitted to hospital with MI; the study did not account for individuals who died out of hospital. Multiple comparison errors may account for some of the significant associations observed and pollutants are often correlated, leading to lack of independence, and thus, replication studies are necessary.

Conclusion

We evaluated the effects of increased air pollution on the increased odds of hospital admissions for MI by integrating spatial variation in air pollution derived from NO_2 LUR models. Our results show that the effect of air pollution on MI was stronger in areas with higher NO_2 concentrations than that in areas with lower NO_2 concentrations. These results highlight the need for preventive strategies targeted specifically to populations living in residential areas with higher traffic-related pollution, who should be advised of the health risks and to pay particular attention to special air quality statements.

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Data sharing: Health data cannot be shared publicly because of restrictions regarding patient privacy. To request access to data from the APPROACH database, visit the website at https://www.approach.org/contact_pages/contact.html. Air pollution data from the National Air Pollution Surveillance (NAPS) program is open access and can be accessed at this site: https://open.canada.ca/data/en/dataset/1b36a356-defd-4813-acea-47bc3abd859b.

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