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Temporal-Spatial Case-Crossover Analysis of the Effect of Air Pollution on Myocardial Infarction

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Abstract:	Background: Studies demonstrating associations between air pollution and myocardial infarction have not adequately considered the inherent intra-urban spatial nature of air pollution. We examined the effects of temporal and spatial distribution of air pollution on myocardial infarction. Methods: We identified adults living in Calgary who had a myocardial infarction from 2004–2012 (n=6,142). We evaluated associations between acute exposure to air pollution (ozone [O3], nitrogen dioxide

	[NO2], sulfur dioxide [SO2], carbon monoxide [CO], particulate matter<10 microns in diameter [PM10], and particulate matter<2.5 microns in diameter [PM2.5]), and onset of myocardial infarction using a time-stratified, case-crossover study design. Air Quality Health Index (AQHI) values were calculated from a composition of O3, NO2, and PM2.5. Conditional logistic regression models were stratified by neighborhood exposure to NO2 concentrations derived from land use regression models. Results are provided as odds ratios (OR) with associated 95% confidence intervals (CI). Results: Individuals living in neighborhoods with higher exposure to air pollution were more susceptible to myocardial infarction following acute elevations in air pollution (e.g., five-day average NO2: OR:1.20; 95% CI:1.03, 1.40 per interquartile range (IQR)) as compared to regions with lower air pollution (e.g., five-day average NO2: OR:0.90; 95% CI:0.78, 1.04 per IQR). In high NO2 regions the AQHI was significantly associated with MI (e.g. five-day average OR:1.13; 95% CI:1.02, 1.24 per IQR; three-day average OR:1.13; 95% CI:1.04, 1.23 per IQR). Interpretation: Those who live in neighborhoods with chronically higher concentrations of NO2 are more susceptible to myocardial infarction with short-term increases of air pollution.

STROBE Statement—checklist of items that should be included in reports of observational studies

	Item No	Recommendation	Reported on Page #
Title and abstract	1	(*a*) Indicate the study's design with a commonly used term in the title or the abstract	1,3
		(*b*) Provide in the abstract an informative and balanced summary of what was done and what was found	3
Introduction			
Background/rationale	2	Explain the scientific background and rationale for the investigation being reported	4
Objectives	3	State specific objectives, including any prespecified hypotheses	4-5
Methods			
Study design	4	Present key elements of study design early in the paper	6
Setting	5	Describe the setting, locations, and relevant dates, including periods of recruitment, exposure, follow-up, and data collection	5,6
Participants	6	(*a*) *Cohort study*—Give the eligibility criteria, and the sources and methods of selection of participants. Describe methods of follow-up *Case-control study*—Give the eligibility criteria, and the sources and methods of case ascertainment and control selection. Give the rationale for the choice of cases and controls *Cross-sectional study*—Give the eligibility criteria, and the sources and methods of selection of participants	6,7
		(*b*) *Cohort study*—For matched studies, give matching criteria and number of exposed and unexposed *Case-control study*—For matched studies, give matching criteria and the number of controls per case	6,7
Variables	7	Clearly define all outcomes, exposures, predictors, potential confounders, and effect modifiers. Give diagnostic criteria, if applicable	5-7
Data sources/ measurement	8*	For each variable of interest, give sources of data and details of methods of assessment (measurement). Describe comparability of assessment methods if there is more than one group	5,6
Bias	9	Describe any efforts to address potential sources of	7

		bias	
Study size	10	Explain how the study size was arrived at	5
Quantitative variables	11	Explain how quantitative variables were handled in the analyses. If applicable, describe which groupings were chosen and why	7
Statistical methods	12	(*a*) Describe all statistical methods, including those used to control for confounding	7
		(*b*) Describe any methods used to examine subgroups and interactions	7
		(*c*) Explain how missing data were addressed	7
		(*d*) *Cohort study*—If applicable, explain how loss to follow-up was addressed *Case-control study*—If applicable, explain how matching of cases and controls was addressed *Cross-sectional study*—If applicable, describe analytical methods taking account of sampling strategy	6,7
		(e) Describe any sensitivity analyses	7
Results			
Participants	13*	(a) Report numbers of individuals at each stage of study—eg numbers potentially eligible, examined for eligibility, confirmed eligible, included in the study, completing follow-up, and analysed	8
		(b) Give reasons for non-participation at each stage	
		(c) Consider use of a flow diagram	
Descriptive data	14*	(a) Give characteristics of study participants (eg demographic, clinical, social) and information on exposures and potential confounders	8, Table 1
		(b) Indicate number of participants with missing data for each variable of interest	n/a
		(c) *Cohort study*—Summarise follow-up time (eg, average and total amount)	n/a
Outcome data	15*	*Cohort study*—Report numbers of outcome events or summary measures over time	8
		Case-control study—Report numbers in each exposure category, or summary measures of exposure	
		Cross-sectional study—Report numbers of outcome events or summary measures	
Main results	16	(*a*) Give unadjusted estimates and, if applicable, confounder-adjusted estimates and their precision (eg, 95% confidence interval). Make clear which confounders were adjusted for and why they were included	8, Table 2
		(*b*) Report category boundaries when continuous variables were categorized	8

		(*c*) If relevant, consider translating estimates of relative risk into absolute risk for a meaningful time period	n/a
Other analyses	17	Report other analyses done—eg analyses of subgroups and interactions, and sensitivity analyses	8, Table 2
Discussion			
Key results	18	Summarise key results with reference to study objectives	8,9
Limitations	19	Discuss limitations of the study, taking into account sources of potential bias or imprecision. Discuss both direction and magnitude of any potential bias	9, 10
Interpretation	20	Give a cautious overall interpretation of results considering objectives, limitations, multiplicity of analyses, results from similar studies, and other relevant evidence	9, 10
Generalisability	21	Discuss the generalisability (external validity) of the study results	9, 10
Other information			
Funding	22	Give the source of funding and the role of the funders for the present study and, if applicable, for the original study on which the present article is based	2

*Give information separately for cases and controls in case-control studies and, if applicable, for exposed and unexposed groups in cohort and cross-sectional studies.

Note: An Explanation and Elaboration article discusses each checklist item and gives methodological background and published examples of transparent reporting. The STROBE checklist is best used in conjunction with this article (freely available on the Web sites of PLoS Medicine at http://www.plosmedicine.org/, Annals of Internal Medicine at http://www.annals.org/, and Epidemiology at http://www.epidem.com/). Information on the STROBE Initiative is available at www.strobe-statement.org.

Temporal-Spatial Case-Crossover Analysis of the Effect of Air Pollution on Myocardial Infarction

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Keywords: myocardial infarction; air pollution; spatial variation; time-stratified; land use regression model; susceptible population.

Abstract

Background: Studies demonstrating associations between air pollution and myocardial infarction have not adequately considered the inherent intra-urban spatial nature of air pollution. We examined the effects of temporal and spatial distribution of air pollution on myocardial infarction.

Methods: We identified adults living in Calgary who had a myocardial infarction from 2004–2012 (*n*=6,142). We evaluated associations between acute exposure to air pollution (ozone [O_3], nitrogen dioxide [NO_2], sulfur dioxide [SO_2], carbon monoxide [CO], particulate matter<10 microns in diameter [PM_{10}], and particulate matter<2.5 microns in diameter [$PM_{2.5}$]), and onset of myocardial infarction using a time-stratified, case-crossover study design. Air Quality Health Index (AQHI) values were calculated from a composition of O_3, NO_2, and $PM_{2.5}$. Conditional logistic regression models were stratified by neighborhood exposure to NO_2 concentrations derived from land use regression models. Results are provided as odds ratios (OR) with associated 95% confidence intervals (CI).

Results: Individuals living in neighborhoods with higher exposure to air pollution were more susceptible to myocardial infarction following acute elevations in air pollution (e.g., five-day average NO_2: OR:1.20; 95% CI:1.03, 1.40 per interquartile range (IQR)) as compared to regions with lower air pollution (e.g., five-day average NO_2: OR:0.90; 95% CI:0.78, 1.04 per IQR). In high NO_2 regions the AQHI was significantly associated with MI (e.g. five-day average OR:1.13; 95% CI:1.02, 1.24 per IQR; three-day average OR:1.13; 95% CI:1.04, 1.23 per IQR).

Interpretation: Those who live in neighborhoods with chronically higher concentrations of NO_2 are more susceptible to myocardial infarction with short-term increases of air pollution.

Introduction

Atherosclerotic coronary artery disease remains a common cause of morbidity and mortality.[1] Despite improvements in risk factor burden[2] and management,[3] over 36,000 Canadians die annually from myocardial infarctions (MI).[4] MI is modified by several risk factors including smoking cigarettes, dyslipidemia, hypertension, diabetes, abdominal obesity, diet, socioeconomic status, and insufficient physical activity.[5-6] Studies have consistently demonstrated that short-term elevations in air pollution concentrations increase the risk of MI.[7-9] Improving our understanding of the effects of acute exposure to air pollution on MI may inform government policy and facilitating prevention by warning populations at risk.

In Calgary, the major contributor to air pollution is transportation for nitrogen dioxide (NO_2) and carbon monoxide (CO); construction for particulate matter < 10 microns in diameter (PM_{10}), and particulate matter < 2.5 microns in diameter ($PM_{2.5}$); and, cement and rock industries for sulfur dioxide (SO_2)[10]; the relatively higher air pollution regions are mainly distributed along major traffic corridors and close to industrial areas.[11-12] Air pollution exposure studies relying on the average of air pollution ignore the inherent spatial nature of air pollution.[13-14]

Historically, temporal analyses exploring the association between air pollution and health outcomes have assumed that pollutants are spatially homogeneous.[15-18] However, research has demonstrated that spatial distribution patterns differ by pollutant.[19] For example, it is widely recognized that ozone (O_3) is relatively spatially homogenous due to consistent concentration levels and temporal fluctuations, while NO_2 is spatially heterogeneous because it is attributable to traffic emissions. Using city-wide averages as air pollution estimates fails to consider the spatial variation within a city.[18]

The objective of this study is to evaluate if the spatial distribution of air pollution influences the temporal associations between air pollution and MI. By integrating spatial variation captured by an NO_2 land use regression (LUR) model with temporal analysis, our study aims to assess the association of short-term elevations in air pollution with the risk of

MI in regions with different air pollution levels and to identify populations that may be at increased susceptibility.

Methods

Clinical data

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 January 1, 1995 (see 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. Because the data collection is prospective, missing data on key variables are minimal. Our population was comprised of adults over the age of 18 years at the time of incidence, living in Calgary during the study period, January 1, 2004 to December 31, 2012. The population was extracted by first acute MI diagnosis, including ST elevation MI and non-ST elevation MI.

Air pollution and meteorological data from fixed monitoring sites

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.[20-22] The three stations were Calgary Central, Calgary East, and Calgary Northwest; they provided hourly concentrations of the six air pollutants investigated in this study: O_3, NO_2, SO_2, CO, PM_{10}, and $PM_{2.5}$. Daily air pollution levels were calculated from hourly records by averaging across the three fixed monitoring stations.[8,23] For all air pollutants, with the exception of ozone, daily mean exposure estimates were used. Ozone values were based on an eight-hour maximum value. Additionally, Air Quality Health Indices (AQHI) were calculated from a composition of three-hour average values of O_3, NO_2, and $PM_{2.5}$ based on the formula[24]:

$$\mathrm{AQHI} = 10/10.4 * (100 * (\exp(0.000871 * \mathrm{NO_2}) - 1 + \exp(0.000537 * \mathrm{O_3}) - 1 + \exp(0.000487 * \mathrm{PM_{2.5}}) - 1))$$

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 monitoring stations. These daily time series of meteorological data were linked with MI hospitalizations and used as adjustment factors in a multivariable conditional logistic regression model.

Spatial classification based on NO_2 estimates from an LUR model

LUR models have been widely used to assess the spatial variation of outdoor air pollution and to estimate fine scale pollution concentrations.[25-28] Significant intra-urban variation for NO_2, $PM_{2.5}$, and metals associated with $PM_{1.0}$ has been observed in previous analyses conducted on air pollution with LUR models in Calgary.[11,29] These previous studies suggest that the major contributors to the spatial variation of air pollution are emissions from motor vehicles and industrial sources,[30] resulting in relatively higher air pollution along major traffic corridors and the northeast industrial areas.[11-12] Temporal stability of LUR over time has been previously validated.[31] Further, the LUR model used in Calgary was shown to remain stable over a five year interval.[32] We used the NO_2 estimates from the air pollution study reported in Bertazzon et al. (2015) for the study period,[11] and divided the city into three levels based on ambient NO_2 concentrations: low NO_2 pollution (first tertile), medium NO_2 pollution (second tertile), and high NO_2 pollution (third tertile) (Figure 1). MI patients were assigned to each of the three areas based on the six-digit postal codes of their residential locations.

Study design

We used a time-stratified, case-crossover study design to evaluate associations between an acute exposure and the acute onset of a disease[33-34]; this is an adaptation of the case-control study in which cases serve as their own controls.[35] 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.[36] The case's exposure at the index time (*i.e.*, day of admission for MI) is compared to their exposure at control time intervals, which

are chosen using a time-stratified design.[37] The time-stratified selection of periods occurs as follows: i. The index period is measured before the event; ii. the control period is measured before and after the event.[38-40] 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.[40] 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.[36-37,41]

Statistical analysis

To examine the temporal relationship between outdoor air pollution levels (O_3, NO_2, SO_2, CO, PM_{10}, $PM_{2.5}$, and AQHI) and presentation to hospitals due to MI, we constructed several different metrics: same day exposure, one- and two-day lagged exposures, and cumulative three-day and five-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 (OR) with associated 95% confidence intervals (CI) were calculated to evaluate the association between MI hospitalizations 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.[8,20,42] Temperature and relative humidity were entered as linear terms in models. We verified the linearity of the relationship using natural cubic spline functions. The AQHI was also included in the 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) was stratified by an individual's neighborhood exposure to NO_2 concentrations (stratified as high, medium, and low), as derived from LUR models.

The study was approved by University of Calgary and Health Canada Research Ethics Boards.

Results

We identified 6,142 adult patients admitted to hospital due to MI during the study period (Table 1). Males were 73% of the MI population; patients aged 65 or under account for 52%; patients with diabetes were 24%; and, 59% were either a current or a former smoker. When stratified by residential location, 23% of MI patients reside in areas of relatively high NO_2 pollution. The distribution of air pollutants (e.g. median, IQR) and their correlation to each other are provided in Appendix 1.

Associations between air pollution and MI are shown in Table 2. For the overall city-wide study population, only SO_2, lag 1 day exhibited a statistically significant positive association with MI (OR: 1.049; 95% CI: 1.007, 1.093 per IQR). Associations between pollutants and MI were primarily observed for those residing in areas in the highest tertile of NO_2. Significant with the exception of O_3, all pollutants were associated with MI in high NO_2 areas with the ORs ranging from 1.06 to 1.20 per IQR. The strongest effect on MI was identified for five-day cumulative average of NO_2 (OR: 1.20; 95% CI: 1.03, 1.40 per IQR). In high NO_2 regions the AQHI was significantly associated with MI (five-day average OR: 1.13; 95% CI: 1.02, 1.24 per IQR; three-day average OR: 1.13; 95% CI: 1.04, 1.23 per IQR).

Interpretation

We evaluated the associations between air pollution and risk of MI with a time-stratified, case-crossover study design. Our analysis was consistent with Wang et al. (2015) who also explored the effects of air pollution on MI in Calgary.[23] Neither that study nor ours identified strong effects of air pollution on MI when assuming that the spatial distribution of air pollution was 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 fewer than 1% of days annually during our study period.[43] Environment and Climate Change Canada (2015) reported that air pollution (NO_2, SO_2, O_3, and CO) has dramatically improved, including in Calgary, from 1990 to 2015.[44] In part, improved air quality in Calgary may also explain the decrease incidence of MI in Calgary observed by Liu and Bertazzon (2017) between 2004 and 2013.[12]

However, unique to our study, we stratified our models by spatial distribution of NO_2. Individuals living in regions of high NO_2 exposure demonstrated significant associations for all individual pollutants and MI with the exception of O_3. Further, the AQHI was also associated with MI for patients living in areas with higher NO_2 concentrations. 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 et al. (2012) provide a systematic review concluding that SO_2 was positively associated with increased MI.[9] Our results also align with previous studies that report that O_3 has no association with MI hospitalizations.[9,23,45] As well, 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 $PM_{2.5}$); which is aligned with previous studies that find a positive association between MI and NO_2 and $PM_{2.5}$.[9,42]

The AQHI, 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.[46-47] The AQHI 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, while NO_2 and $PM_{2.5}$ exhibited significant associations in our spatial stratification.

A limitation of our study is in using fixed-site monitoring data rather than personal monitoring. Fixed-site monitoring is subject to misclassification of the exposure because fixed-site monitors do not account for individual mobility; this could result in non-differential exposure misclassification, which may underestimate the risk of air pollution.[40,48] Results of the current study support further investigation of whether living in a high pollution area increases vulnerability to temporal spikes in pollution concentrations. However, this should be interpreted with caution because high pollution areas may correspond with other risk factors for MI such as low socioeconomic status and obesity. Misclassification of timing of

onset of myocardial infarction may introduce bias into the results. Multiple comparison errors may account for some of the statistically significant associations observed and thus, replication studies are necessary.

We examined the effects of increased air pollution on the increased odds of MI by integrating spatial variation in air pollution derived from NO_2 LUR models. Our results showed that the effect of air pollution on MI was stronger in areas with higher NO concentrations than 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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Figure Legend

Figure 1. Area stratified by average NO_2 from LUR estimates. Darker shading represents higher air pollution and lighter shading represents lower air pollution. Stars denote the three continuous monitoring stations in Calgary.

Tables

Table 1. Demographics of MI population.

Characteristics	*n* (%)
Total Population	6,142 (100%)
Sex	
Male	4,482 (73%)
Female	1,660 (27%)
Age	
Age ≤ 65	3,209 (52%)
Age > 65	2,933 (48%)
Comorbidity	
No diabetes	4,649 (76%)
Diabetes	1,493 (24%)
Cigarette Smoking	
Never smoker	2,496 (41%)
Former smoker	1,798 (29%)
Current smoker	1,848 (30%)
Residential Location*	
Low NO_2 air pollution (1st tertile)	1,660 (27%)
Medium NO_2 air pollution (2nd tertile)	3,088 (50%)
High NO_2 air pollution (3rd tertile)	1,384 (23%)

*10 patients with missing data.

Table 2. Association between air pollution and MI with increases in the interquartile range of pollutants during various referent time intervals, in regions with differing NO_2 pollution levels. Acronyms: ozone (O3), nitrogen dioxide (NO2), sulfur dioxide (SO2), carbon monoxide (CO), particulate matter < 10 microns in diameter (PM10), and particulate matter < 2.5 microns in diameter (PM2.5). Significant associations are bolded. IQR: Interquartile Range.

		Odds Ratio[a] (95% Confidence Intervals)			
Pollutant (Median with Interquartile Range)	Lag (days)	Entire study population (city-wide NO_2) (*n* = 6,142)	Low NO_2 region (*n* = 1,660)	Medium NO_2 region (*n*= 3,088)	High NO_2 region (*n* = 1,384)
CO (0.35, IQR: 0.27, 0.47)	0 Index Day	0.972 (0.926, 1.022)	0.943 (0.881, 1.009)	0.970 (0.923, 1.019)	1.016 (0.947, 1.091)
	1 Day Lag	1.029 (0.980, 1.080)	0.984 (0.919, 1.054)	1.020 (0.972, 1.070)	**1.099 (1.022, 1.181)**
	2 Day Lag	1.008 (0.959, 1.060)	1.025 (0.957, 1.097)	0.985 (0.937, 1.036)	1.039 (0.966, 1.117)
	0-2 Day Average	1.006 (0.940, 1.076)	0.968 (0.881, 1.062)	0.985 (0.921, 1.054)	1.094 (0.993, 1.206)
	0-4 Day Average	0.974 (0.900, 1.054)	0.923 (0.826, 1.030)	0.956 (0.884, 1.034)	1.079 (0.961, 1.212)
NO_2 (18.22, IQR: 12.67, 25.00)	0 Index Day	1.003 (0.941, 1.070)	0.968 (0.887, 1.056)	1.008 (0.946, 1.075)	1.045 (0.951, 1.149)
	1 Day Lag	1.040 (0.974, 1.109)	0.998 (0.911, 1.093)	1.007 (0.944, 1.074)	**1.159 (1.054, 1.275)**
	2 Day Lag	1.030 (0.966, 1.099)	1.030 (0.943, 1.124)	0.996 (0.934, 1.062)	**1.109 (1.008, 1.220)**
	0-2 Day Average	1.045 (0.957, 1.141)	0.996 (0.883, 1.123)	1.007 (0.923, 1.100)	**1.197 (1.053, 1.361)**
	0-4 Day Average	0.975 (0.878, 1.082)	0.902 (0.781, 1.042)	0.927 (0.836, 1.029)	**1.200 (1.029, 1.400)**
O_3 max (39.00, IQR: 32.00, 47.00)	0 Index Day	1.003 (0.954, 1.056)	1.024 (0.956, 1.098)	0.980 (0.932, 1.031)	1.031 (0.956, 1.111)
	1 Day Lag	0.992 (0.943, 1.044)	0.963 (0.898, 1.031)	0.974 (0.926, 1.025)	1.068 (0.989, 1.153)
	2 Day Lag	0.989 (0.940, 1.041)	0.972 (0.907, 1.041)	0.986 (0.937, 1.037)	1.016 (0.941, 1.097)
	0-2 Day Average	0.992 (0.930, 1.058)	0.977 (0.895, 1.067)	0.968 (0.908, 1.033)	1.062 (0.964, 1.170)
	0-4 Day Average	1.003 (0.930, 1.080)	0.999 (0.903, 1.106)	0.967 (0.898, 1.042)	1.087 (0.972, 1.216)
SO_2 (1.00, IQR: 1.00, 2.00)	0 Index Day	1.002 (0.960, 1.045)	1.002 (0.947, 1.060)	0.969 (0.929, 1.011)	**1.081 (1.016, 1.150)**
	1 Day Lag	**1.049 (1.007, 1.093)**	1.033 (0.977, 1.092)	1.039 (0.997, 1.083)	**1.095 (1.031, 1.164)**
	2 Day Lag	1.035 (0.994, 1.079)	1.037 (0.979, 1.098)	1.028 (0.987, 1.071)	1.051 (0.989, 1.117)

	0-2 Day Average	1.059 (0.999, 1.122)	1.050 (0.970, 1.136)	1.025 (0.967, 1.087)	**1.151 (1.058, 1.252)**
	0-4 Day Average	1.045 (0.976, 1.119)	1.054 (0.961, 1.157)	1.020 (0.953, 1.092)	1.099 (0.994, 1.216)
PM_{10} 20.00, IQR: 14.00, 30.00)	0 Index Day	0.982 (0.947, 1.018)	**0.948 (0.901, 0.999)**	0.981 (0.947, 1.017)	1.026 (0.973, 1.082)
	1 Day Lag	1.010 (0.974, 1.047)	0.968 (0.921, 1.018)	1.005 (0.970, 1.042)	**1.064 (1.010, 1.120)**
	2 Day Lag	1.012 (0.976, 1.049)	0.992 (0.945, 1.041)	1.000 (0.964, 1.037)	**1.058 (1.004, 1.116)**
	0-2 Day Average	1.002 (0.957, 1.050)	0.950 (0.891, 1.014)	0.993 (0.948, 1.040)	**1.083 (1.013, 1.158)**
	0-4 Day Average	0.990 (0.938, 1.045)	0.945 (0.878, 1.018)	0.981 (0.930, 1.036)	1.066 (0.986, 1.153)
$PM_{2.5}$ (7.00, IQR: 4.33, 10.50)	0 Index Day	1.014 (0.977, 1.051)	0.994 (0.939, 1.053)	1.003 (0.968, 1.040)	**1.055 (1.003, 1.109)**
	1 Day Lag	1.024 (0.987, 1.062)	0.980 (0.932, 1.031)	**1.043 (1.005, 1.082)**	1.044 (0.989, 1.101)
	2 Day Lag	1.003 (0.964, 1.042)	0.955 (0.904, 1.010)	1.015 (0.974, 1.057)	1.030 (0.980, 1.083)
	0-2 Day Average	1.020 (0.974, 1.068)	0.963 (0.902, 1.029)	1.029 (0.982, 1.078)	1.065 (0.999, 1.134)
	0-4 Day Average	1.024 (0.969, 1.082)	0.982 (0.911, 1.059)	1.043 (0.986, 1.102)	1.037 (0.957, 1.123)
AQHI (4.01, IQR: 3.49, 4.65)	0 Index Day	1.007 (0.963, 1.053)	0.998 (0.938, 1.062)	0.991 (0.948, 1.036)	1.058 (0.991, 1.130)
	1 Day Lag	1.019 (0.974, 1.066)	0.968 (0.909, 1.030)	1.001 (0.957, 1.047)	**1.119 (1.048, 1.195)**
	2 Day Lag	1.010 (0.965, 1.057)	0.988 (0.930, 1.051)	0.995 (0.951, 1.042)	1.066 (0.998, 1.140)
	0-2 Day Average	1.020 (0.964, 1.080)	0.976 (0.903, 1.056)	0.996 (0.941, 1.054)	**1.130 (1.041, 1.227)**
	0-4 Day Average	1.007 (0.942, 1.076)	0.973 (0.888, 1.066)	0.974 (0.911, 1.041)	**1.127 (1.022, 1.243)**

[a] Odds ratios are adjusted for temperature and relative humidity.

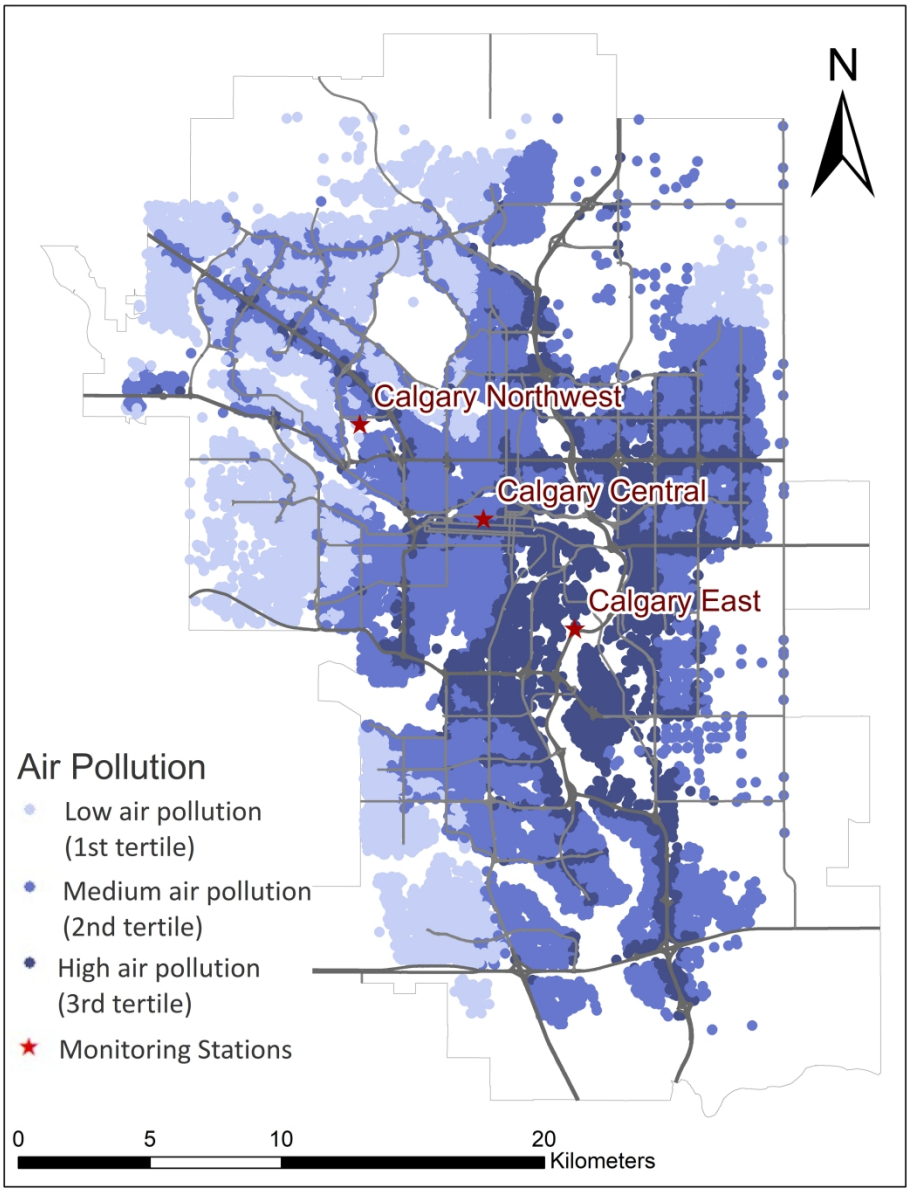

Figure 1. Area stratified by average NO2 from LUR estimates. Darker shading represents higher air pollution and lighter shading represents lower air pollution. Stars denote the three continuous monitoring stations in Calgary.

215x279mm (300 x 300 DPI)

Appendix 1: The distribution of air pollutants and their correlation to each other. Values for O3, NO2, CO, PM10, PM25, SO2, Temperature, and RH are 24-hour means. Values for ozone (O3 max) are daily maximum 8-hour average.

Descriptive statistics					Pearson Correlation Coefficients								
	Mean	**Std. Dev**	**Median**	**IQR**	**O3**	**O3 max**	**NO**	**CO**	**PM10**	**PM25**	**SO2_mean**	**Temperature**	**RH**
O_3	20.46	8.80	20.00	13.50	1.00								
O_3 max	39.48	10.94	39.00	15.00	0.81	1.00							
NO_2	17.90	8.95	18.33	12.33	-0.64	-0.32	1.00						
CO	0.33	0.16	0.35	0.20	-0.54	-0.26	0.80	1.00					
PM_{10}	21.94	12.78	20.00	16.00	-0.12	0.18	0.40	0.40	1.00				
PM_{25}	9.79	5.91	7.00	6.17	-0.02	0.08	0.08	0.07	0.43	1.00			
SO_2	1.78	1.29	1.00	1.00	-0.34	-0.15	0.53	0.63	0.24	0.07	1.00		
Temperature	4.43	10.20	5.08	14.89	0.41	0.49	-0.54	-0.29	0.16	0.31	-0.24	1.00	
RH	64.03	14.79	67.65	22.33	-0.36	-0.49	0.02	0.09	-0.23	0.02	-0.05	-0.31	1.00