Environment International 124 (2019) 16–24

Contents lists available at ScienceDirect

Environment International

journal homepage: www.elsevier.com/locate/envint

Exposure to traffic and mortality risk in the 1991–2011 Canadian Census Health and Environment Cohort (CanCHEC)

Sabit Cakmak[a,*], Chris Hebbern[b], Jennifer Vanos[c,d], Dan Lawson Crouse[e], Michael Tjepkema[f]

[a] Population Studies Division, Environmental Health Science & Research Bureau, Health Canada, 101 Tunney's Pasture Driveway, Ottawa, ON K1A 0K9, Canada
[b] Climate Change and Innovation Bureau, Health Canada, 269 Laurier Avenue, Ottawa, ON K1A 0K9, Canada
[c] Climate, Atmospheric Science & Physical Oceanography, Scripps Institution of Oceanography, University of California at San Diego, 9500 Gilman Drive #0206, USA
[d] Arizona State University, School of Sustainability, Tempe, AZ 85281, USA
[e] New Brunswick Institute for Research, Data, and Training (NB-IRDT) and Department of Sociology University of New Brunswick Fredericton, NB PO Box 4400, E3B 5A3, Canada
[f] Health Analysis, Statistics Canada/Government of Canada, Canada

ARTICLE INFO

Handling Editor: Martí Nadal

Keywords:
Air pollution
Traffic
Environmental health
Mortality
Respiratory health
Climate

ABSTRACT

There is evidence that local traffic density and living near major roads can adversely affect health outcomes. We aimed to assess the relationship between local road length, proximity to primary highways, and cause-specific mortality in the 1991 Canadian Census Health and Environment Cohort (CanCHEC). In this long-term study of 2.6 million people, based on completion of the long-form census in 1991 and followed until 2011, we used annual residential addresses to determine the total length of local roads within 200 m of postal code representative points and the postal code's distance to primary highways. The association between exposure to traffic and cause-specific non-accidental mortality was estimated using Cox proportional hazards models, adjusting for individual covariates and contextual factors, including census division-level proportion in high school, the percentage of recent immigrants, and neighborhood income. We performed sensitivity analyses, including adjustment for exposure to $PM_{2.5}$, NO_2, or O_3, restricting to subjects in core urban areas, and spatial variation by climatic zone. The hazard ratio (HR) for all non-accidental mortality associated with an interquartile increase in length of local roads was 1.05 (95% CI 1.04, 1.05), while for an interquartile range increase in proximity to primary highways, the HR was 1.03 (95% CI 1.02, 1.04). HRs by traffic quartile increased with increasing lengths of local roads, as well as with closer proximity to primary highways, for all mortality causes. The associations were stronger within subjects' resident in urban core areas, attenuated by adjustment for $PM_{2.5}$, and HRs showed limited spatial variation by climatic zone. In the CanCHEC cohort, exposure to higher road density and proximity to major traffic roads was associated with increased mortality risk from cerebrovascular and cardiovascular disease, ischemic heart disease, COPD, respiratory disease, and lung cancer, with unclear results for diabetes.

1. Introduction

A number of epidemiologic studies have documented the adverse effects of traffic exposure on different health outcomes, including asthma, lung function, and cardiovascular health (Cakmak et al., 2016a; Dales et al., 2009; Urman et al., 2014). Proximity to traffic increases exposure to a broad suite of ambient pollutants generated from vehicle exhaust, combustion, and friction with road surfaces, including oxides of nitrogen, heavy metals, volatile organic compounds, and polycyclic aromatic hydrocarbons (Bell et al., 2014). As a major source of ambient air pollution in urban environments, traffic generates fine particulate matter and various gaseous pollutants, which are associated with a wide range of systemic effects including oxidative stress (Zhang et al., 2016), elevated blood pressure (Fuks et al., 2014), lung inflammation (Holguin et al., 2007), reduced lung function (Cakmak et al., 2016a), and increased mortality from cancer, cardiovascular causes, and diabetes (Weinmayr et al., 2015). People may be additionally burdened by exposure to traffic noise (Beelen et al., 2009; Tzivian et al., 2016), as well as limited access to green space due to urban design (Crouse et al., 2017).

Previous studies using the Canadian Census Health and Environment Cohort (CanCHEC) have considered the effects of nitrogen

* Corresponding author.
E-mail address: sabit.cakmak@canada.ca (S. Cakmak).

https://doi.org/10.1016/j.envint.2018.12.045
Received 12 October 2018; Received in revised form 18 December 2018; Accepted 20 December 2018
Available online 09 January 2019

dioxide (NO_2), fine particulate matter < 2.5 μm in diameter ($PM_{2.5}$), and ozone (O_3) on a range of mortality outcomes (Cakmak et al., 2016b, 2018; Crouse et al., 2015). These studies used land use regression (LUR) models, which capture spatial variability in pollutants and incorporate traffic-related information, such as roadway cartographic data, to assign these pollutant exposures each year to cohort subjects based on their six-digit postal code. Using either seven year average exposure values (Cakmak et al., 2016b; Crouse et al., 2015) or assigning annual exposure values (Cakmak et al., 2018), the studies then determine the effect of long term exposures on health outcomes. In the current study, we complement these previous studies with an analysis of the effect of traffic exposure on disease mortality within the 1991 CanCHEC using national contrasts of interquartile ranges of traffic density. We hypothesize that long term exposure to greater traffic density contributes to exacerbations of health conditions and increased mortality risk.

As smoking status (Turner et al., 2011) and body mass index (BMI) have also been observed to modify the effect of PM exposure on mortality (Tamayo et al., 2014), we use an indirect adjustment technique to consider the influence of smoking and obesity on population health in our cohort, with an additional exploration of the spatial variation in this risk by climate zone.

2. Methods

2.1. The study cohort

The study cohort has been described in detail previously (Crouse et al., 2015). In brief, CanCHEC is a population-based cohort with the following inclusion criteria: subjects who were 25 years of age or older at baseline, a resident of Canada on the census date, 4 June 1991, and one of approximately 3.6 million respondents (20% of Canadian households) to the long-form census questionnaire. Mortality data were obtained by probalistically linking subjects to the Canadian Mortality Database, with follow-up to 2011 (Peters et al., 2013). Residential 6-digit postal codes were assigned to each subject for each year of follow-up from the mailing addresses reported on annual personal tax returns (Canada Revenue Agency) for the tax years 1984 to 2011 (which were previously linked to the Cohort). For the purposes of linking exposure data to postal codes, the exposure data were geocoded using the Postal Code Conversion File Plus (PCCF+) program, v.6D (Statistics Canada, 2016). This software assigns postal code representative points using either a population-weighted random allocation algorithm for rural areas and equal random allocation for urban areas, deriving the representative points from the centroid of a block face, dissemination block, or dissemination area (Statistics Canada, 2016). Cause of death groupings were obtained from the World Health Organization International Classification of Diseases (ICD)-9th Revision codes (for deaths that occurred before 2000), and ICD-10th Revision codes (for deaths recorded after 2000): COPD: ICD-9, 490–496, and ICD-10, J19–J46; ischemic heart disease: ICD-9, 410–414, and ICD-10: I20–I25; all non-accidental mortality: ICD-9, < 800, and ICD-10, A–R; lung cancer: ICD-9, 162, and ICD-10, C33–C34; diabetes: ICD-9, 250, and ICD-10, E10-E14; cardiovascular disease: ICD-9, 410–440, and ICD-10, I20–I25, I30–I51, I60–I69, I70; respiratory disease: ICD-9, 460–519, and ICD-10, J00–J98; and cerebrovascular disease causes: ICD-9, 430–438, and ICD-10, 160–169. Individual risk factors for mortality were obtained from the long form census at baseline.

As socioeconomic status at the neighborhood level is also associated with health risks, we derived three time-varying contextual variables from the closest census year (1991, 1996, 2001, or 2006) that were reassigned each year of follow-up: the proportion in the lowest income quintile, a measure of relative household income, accounting for household size and community; the percentage of the population aged 15 years or older with less than high school education; and the percentage of recent immigrants (within the previous five years).

2.2. Exposure data

Exposure values for $PM_{2.5}$, O_3, and NO_2, were assigned to the cohort each year by postal code as previously described (Cakmak et al., 2018). If no postal code was available, which may be the result of the subject not filing an income tax return or leaving Canada, then exposures were missing for that year. Estimates for $PM_{2.5}$ were obtained from satellite-derived concentrations for the period 1998–2011 on a 10 by 10 km grid surface(van Donkelaar et al., 2010), with inferred changes in $PM_{2.5}$ over the period between 1998 and 2006 using two radiometrically stable satellite instruments, MISR and SeaWiFS (Boys et al., 2014). For O_3, a surface representing average daily 8-hr maximum concentrations for the months of May to October 2002–2009, was developed with a resolution of 21 km using an optimal interpolation technique based on that by Robichaud and Ménard (2013). Residential NO_2 exposures were estimated using a national LUR model that incorporates five variables: satellite NO_2 estimates (Hystad et al., 2011) for 2005–2011, 2006 National Air Pollution Surveillance monitoring data, total road length within 10 km of residence, mean summer rainfall, and area of industrial land use within 2 km (see Crouse et al., 2015 for a complete description of the NO_2 LUR method).

2.3. Traffic variables

Traffic data were linked to the cohort using the six digit postal code. Using information from CanMap® Major Roads & Highways, developed by DMTI Spatial (DMTI Spatial Inc., Markham, Ontario), the total length of different categories of roadways within a 200 m radius around a subject's home address (based on the postal code centroid) was determined using GIS software (ArcMap 9.0,ESRI, Redlands, California). This standardized 'Cartographic Road Classification' presents five types of roadways: expressways, primary highways, secondary highways, major roadways, and local roadways. Roadways predominantly serve local traffic moving to and from destinations at lower speeds than do primary highways and expressways, where traffic flow is more important and may travel at speeds up to 100 km/h. These estimates are based on 2008 roadway data. These methods of estimating exposure to traffic (i.e., total length of roadways and distance to certain roadway types) has been previously used elsewhere (Gilbert et al., 2005; McConnell et al., 2006). Here, we used the length of local roads with a 200 m radius of each postal code centroid, and the distance to the nearest primary highway.

2.4. Climate zones

We explored sensitivity of the analysis to spatial variation in the long-term traffic-health association based on spatial synoptic classification (SSC) climate zones using methods previously described (Cakmak et al., 2016b, 2018). In brief, based on the frequency of occurrence of different SSC weather type days over 30 years, clustering analysis was used to delineate climate zones of similar SSC patterns across Canada. The analysis found seven distinct climate zones: 1: Polar; 2 East coast; 3: Great Lakes/St. Lawrence; 4: West Prairies; 5: West coast; 6: East Prairies; 7: West Central. By including the most populated zone, 3, as a reference category in the model, spatial variation in mortality risk by climate zone across Canada could be explored. The distinction of climate zones by SSC lends itself well to climate and health analysis and relative geographical comparisons, particularly with air pollution and temperature (Hondula et al., 2014; Vanos et al., 2015).

2.5. Statistical analysis

We estimated hazard ratios (HRs) for the relationship between long-term exposure to the total length of local roads within 200 m of each subject's residential address, as well as proximity of the residential

address to primary highways, using standard Cox proportional hazards models. We stratified the baseline hazard function by sex and by 5-year increments in age from 25 to 89 years, censoring at 90 years due to potential likelihood of inaccuracies in linking records for older subjects, and followed subjects from 4 June 1991 to 31 December 2011. HRs were calculated per interquartile range change in each traffic variable. The models included the following personal level covariates: aboriginal ancestry, visible minority status, marital status, education level, occupational level, immigrant status, and income quintile. Three contextual neighborhood covariates established at census division level: the proportion of recent immigrants, the proportion of individuals in the lowest income quintile, and the proportion of individuals that had not completed high school.

In order to define urbanized areas, we used an urban index based on Statistics Canada's census definitions, selecting the urban core postal codes only for analysis (Statistics Canada, 2011). Using the annual six digit postal code, this index identified residents as living in an urban core (a census metropolitan area or agglomeration with a population of at least 50,000 persons at the previous census), a secondary urban core (an urban core merged with a larger census metropolitan area), and an urban fringe (all small urban areas within a census metropolitan area or agglomeration that is not contiguous with the urban core). The sensitivity of the models was tested by restricting to the subjects coded as resident in urban core areas only. This restriction is used because the correspondence between a postal code and a residential address in an urban area is much stronger than in rural areas, where a postal code may encompass more than a single census tract or census subdivision (Khan et al., 2018). Thus, restricting the analysis to urban areas reduces this potential source of variability. Models were also tested with and without adjustment for $PM_{2.5}$; by traffic variable quartile; and by comparing HRs between climate zones developed from the time series of SSC days (described below). These tests allowed for the exploration of spatial differences in the relationship between traffic exposure and mortality.

As information on each subject's smoking and BMI status was not available, we used an empirical method to indirectly adjust for potential confounding due to smoking and BMI. Information on these variables, while not captured in CanCHEC, are however available from a secondary dataset, the Canadian Community Health Survey, which collects information regarding health status, health care utilisation, and the determinants of health, across Canada, every two years. This information is assumed to be representative of subjects in the CanCHEC cohort. The indirect adjustment method for missing variables adjusts our hazard ratios for potential confounding due to smoking and BMI while simultaneously controlling for other individual and contextual risk factors included in our original model, and is explained in detail elsewhere (Shin et al., 2012). This method of adjustment was only applied to estimates of the overall effect of each traffic variable on health. Models that estimated risk between traffic quartiles or between climate zones used a selected reference level set to 1.0; hence the application of an indirect adjustment to each HR would distort the scaling between the levels being compared.

3. Results

The cohort consisted of 2,644,370 subjects at baseline. By traffic quartile (Table 1), subjects with any aboriginal ancestry were more likely to be in the lowest quartile of local roads length (7.5%), than in the fourth quartile as a proportion of that segment (2.7%). The cohort population showed further differences by various individual risk factors: subjects with a university degree were more likely (15.0% versus 9.9%) to live in the fourth quartile for local road length, as were immigrants (13.3 to 21.6%). Visible minorities were twice as likely to live in the fourth quartile of local road length (from 4.0 to 8.1%). Differences between individual risk factors and distances to primary highways were less distinct, although visible minorities were less likely (from 10.6 to 4.5%) to live closest to this type of road.

Pollutant levels (Table 2) increased, although not significantly, with interquartile increases in the two measures of traffic density. By length of local roads, concentrations of $PM_{2.5}$ ranged from 6.01 μg/m$_3$ (SD ± 2.65) for < 475 m of total roadway to 8.1 μm/m^3 (SD ± 2.62) for over 1583 m. Ozone levels ranged from 38.18 ppb (SD ± 7.95) in the first quartile, to 39.07 ppb (SD 6.36) in the fourth quartile. Levels of NO_2 ranged from 6.74 ppb (SD ± 4.97) to 13.05 ppb (SD ± 6.18) from the first to fourth quartiles of the length of local roads. By distance to primary highway (Table 2), $PM_{2.5}$ exposures in the first quartile (< 686 m) were 6.61 μg/m^3 (SD ± 2.48), increasing to 8.34 μg/m^3 (SD ± 3.26) in the fourth quartile (> 6678 m). Ozone exposure levels ranged from 37.00 ppb (SD ± 6.24) in the first quartile to 42.31 ppb (SD ± 7.27) in the fourth quartile. NO_2 exposures ranged from 9.47 ppb (SD ± 5.97) to 12.10 ppb (SD ± 7.77) from the first to fourth quartiles.

Cause-specific mortality as a percentage of the overall population was similar in each traffic variable quartile (Table 3), ranging from 0.91% for cerebrovascular disease in the first quartile of local roads length (< 475 m) to 0.89% for the fourth quartile (> 1583 m). Similarly, for all non-accidental mortality, the percentage of overall mortality ranged from 13.30 to 13.90% by length of local road first and fourth quartile, respectively. Hence, there was minimal change in percent of all-cause or a specific-cause mortality due to living near a greater roadway length.

By distance to primary highways, cerebrovascular disease declined from 1.01 to 0.86%, and all non-accidental mortality, 14.50 to 12.70% from the first to the fourth quartile. Mortality due to COPD and diabetes were < 1% in all quartiles, respiratory cause mortality < 1.50%, and lung cancer was around 1.30%. Hence, there were no significant differences in percent of all-cause or a specific-cause mortality for people living closer to primary highway.

We estimated model sensitivity to adjustment for annual ambient pollutant exposures, with a stepwise inclusion of $PM_{2.5}$, NO_2, or O_3 in Cox models for cause-specific mortality, and at an interquartile range (IQR) (1108 m) change in local roads length around the subject's residential address (see Supplementary material, Table S1) as well as by IQR change in distance to primary highway (5991 m) (see Supplementary material, Table S2). The inclusion of any of the pollutant terms attenuated the HRs for all mortality causes. For example, all non-accidental mortality was decreased from HR 1.04 (95% CI 1.04, 1.05) to HR 1.02 (95% CI 1.01, 1.02) for models adjusted for $PM_{2.5}$, NO_2, or O_3. In general, similar decreases were seen across the various cause-specific mortality, thus the extent of change in HR was not highly different for either NO_2, $PM_{2.5}$, or O_3, indicating a high level of colinearity between these pollutants.

We further estimated mortality risk for an IQR change in local roads length (1108 m) for the cohort overall – that is, not separating by traffic variable quartile – before and after adjustment for $PM_{2.5}$ (Table 4). The highest risk was estimated for lung cancer mortality with an IQR change in length of local roads, with a significant HR of 1.07 (95% CI 1.06, 1.09) in models adjusted for $PM_{2.5}$.

Indirect adjustment for smoking and obesity had very little effect on HRs; as in previous research with this cohort (Crouse et al., 2015), the magnitude of change was around 1–2% (see Table 4). The largest change found in the current study was a decrease in the HR for lung cancer mortality from HR 1.10 (95% CI: 1.08, 1.11) in the main model to 1.06 (95% CI: 1.05, 1.09) after adjustment for obesity and smoking.

HRs for an IQR change in local roads length were estimated for each quartile, relative to the first quartile (the lowest length of local roads (< 475 m)), set to 1 (Table 5). Relative to the lowest quartile and length of local roads, each quartile showed an increase in HR for all causes of death except diabetes. Non-accidental mortality also increased as the length of local roads increased, where the fourth quartile resulted in a significant non-adjusted HR of 1.20 (95% CI: 1.18, 1.23), which was also significantly higher than in quartile 2. HRs for lung cancer and

Table 1
Percentage of cohort population by individual-level risk factors in each traffic variable quartile.

	Local roads length quartile				Distance to primary highway quartile			
	1st quartile	2nd	3rd	4th	1st quartile	2nd	3rd	4th
	(< 475 m)	(475–1152 m)	(1152–1583 m)	(> 1583 m)	(< 686 m)	(686–2109 m)	(2109–6678 m)	(> 6678 m)
Any aboriginal ancestry								
Yes	7.5	3.0	2.9	2.7	4.6	3.4	3.0	4.9
No	92.5	97.0	97.1	97.3	95.4	96.6	97.0	95.1
Education level								
Post-secondary, non-university degree	14.4	16.0	16.2	15.3	14.9	16.1	16.2	14.9
High School with or without certificate	36.6	37.0	36.4	35.4	36.5	37.3	37.0	34.6
Did not complete high school	39.1	33.3	32.2	34.3	37.5	33.4	32.7	34.8
University degree	9.9	13.7	15.2	15.0	11.1	13.2	14.2	15.8
Immigrant status								
Non-immigrant	86.7	78.0	75.5	78.4	86.1	97.9	77.6	71.1
Immigrant/permanent resident	13.3	22.0	24.5	21.6	13.9	2.1	22.4	28.9
Labour force status								
Unemployed	7.7	6.7	6.5	6.8	7.1	6.7	6.6	7.3
Not in labour force	27.6	28.7	27.9	29.5	30.9	28.2	27.0	27.1
Employed	64.6	64.7	65.6	63.7	62.0	65.1	66.4	65.6
Marital status								
Single	11.6	13.8	13.5	14.4	14.6	13.4	12.8	12.3
Separated/divorced/widowed	10.6	12.3	13.0	14.3	13.4	12.2	11.5	12.8
Married/common law	77.8	73.9	73.5	71.3	72.0	74.3	75.6	74.9
Occupational level								
Management	7.6	8.6	8.7	8.0	7.4	8.2	8.4	8.9
Professional (all)	26.4	23.4	23.1	22.4	23.8	24.0	24.1	23.4
Skilled/technical/supervisor	24.8	24.3	24.2	23.6	23.6	24.2	24.7	24.5
Semi-skilled	8.4	7.5	7.6	7.8	8.2	7.7	7.6	7.8
Unskilled	9.3	11.3	12.3	12.2	9.9	11.4	11.8	12.1
NA	23.6	24.9	24.1	25.9	27.1	24.5	23.4	23.2
Income adequacy quintile								
1-Lowest	16.5	17.0	17.0	17.9	18.4	16.5	16.0	17.3
2	18.8	18.9	19.4	20.3	19.4	19.3	19.2	19.5
3	20.3	20.3	21.0	21.1	20.5	21.0	20.9	20.3
4	21.3	21.3	21.5	21.0	20.8	21.6	21.7	21.0
5-Highest	23.1	22.4	21.1	19.7	20.8	21.6	22.2	21.8
Sex								
Male	51.0	48.9	49.0	49.3	49.7	49.1	49.6	49.8
Female	49.0	51.1	51.0	50.7	50.3	50.9	50.4	50.2
Visible minority								
Yes	4.0	8.0	9.3	8.1	4.5	6.4	8.4	10.6
No	96.0	92.0	90.7	91.9	95.5	93.6	91.6	89.4

Table 2
Mean pollutant concentrations (and standard deviation (SD)) by traffic variable quartile. Pollutant concentration units are as follows: $PM_{2.5}$ ($\mu g/m^{-3}$), O_3 (ppb), NO_2 (ppb).

	Local roads Length Traffic quartile	Concentration of pollutant	Distance to primary highway traffic quartile	Concentration of pollutant
$PM_{2.5}$	1 (< 475 m)	6.01 (2.65)	1 (< 686 m)	6.61 (2.48)
O_3		38.18 (7.95)		37.00 (6.24)
NO_2		6.74 (4.97)		9.47 (5.97)
$PM_{2.5}$	2 (475–1152 m)	7.49 (2.90)	2 (686–2109 m)	7.18 (2.59)
O_3		39.97 (7.24)		38.10 (6.83)
NO_2		10.98 (6.56)		10.80 (5.97)
$PM_{2.5}$	3	7.92 (2.76)	3 (2109–6678 m)	7.55 (2.81)
O_3	(1152–1583 m)	39.85 (7.03)		40.16 (7.29)
NO_2		12.38 (6.19)		11.03 (5.83)
$PM_{2.5}$	4 (> 1583 m)	8.10 (2.62)	4 (> 6678 m)	8.34 (3.26)
O_3		39.07 (6.36)		42.31 (7.27)
NO_2		13.05 (6.18)		12.10 (7.77)

respiratory-cause mortality also increased with the length of local roads, before and after adjustment for $PM_{2.5}$, with small attenuation after adjustment.

We further examined risk by traffic quartile and cause-specific mortality, restricting the analysis to urban core residential postal codes only (Table 5). In the urban areas only, the highest HR was reported for lung cancer mortality risk, HR 1.54 (95% CI 1.36, 1.75), but attenuated after adjustment for $PM_{2.5}$ to HR 1.07 (95% CI 1.05, 1.10). The HR values in the urban core were overall greater for cardiovascular, ischemic heart, lung cancer, and diabetes than when estimating for all residential types, with lung cancer showing the greatest increase in risk for urban core residents compared to the total, with HR 1.27 (95% CI: 1.18, 1.37) versus 1.54 (95% CI: 1.36, 1.75), respectively, for non-adjusted values in the fourth quartile.

In examining the effect of distance to primary highways, the overall mortality risk for an IQR change (5991 m) in distance (Table 6) was highest for diabetes, HR 1.08 (95% CI: 1.05, 1.10). After adjustment for $PM_{2.5}$, HRs were slightly reduced with little additional change from indirect adjustment for smoking and BMI.

Finally, HRs for an IQR change in distance to primary highways were estimated for each quartile, relative to the fourth quartile representing the greatest distance (> 6678 m), which was set to 1 (Table 7). All causes of death showed an increase in HR relative to the fourth quartile of distance to primary highway, with the greatest risk in the first quartile, < 686 m away. Diabetes mortality risk did not

Table 3
Mortality counts ('events') by cause and by traffic variable quartile as a percentage of total population. Percentages are calculated as the number of cause-specific deaths recorded in the population in 2011 divided by total population in 2011 for each traffic variable quartile and then multiplied by 100. Percentages calculated before counts were rounded to the nearest 5.

	Local roads length				Distance to primary highway			
Mortality cause	Traffic quartile	Events	Total	Percent	Traffic quartile	Events	Total	Percent
Cerebrovascular	1 (< 475 m)	5930	652,365	0.91	1 (< 686 m)	7390	732,210	1.01
Cardiovascular		28,820	652,365	4.42		35,315	732,210	4.82
Ischemic heart		17,755	652,365	2.72		21,275	732,210	2.91
Non-accidental		87,090	652,365	13.30		105,910	732,210	14.50
COPD		4160	652,365	0.64		5100	732,210	0.70
Respiratory		8180	636,800	1.28		10,450	723,745	1.44
Lung cancer		8080	652,365	1.24		9845	732,210	1.34
Diabetes		2740	652,365	0.42		3130	732,210	0.43
Cerebrovascular	2 (475–1152 m)	6015	675,005	0.89	2 (686–2109 m)	5685	649,635	0.88
Cardiovascular		28,590	675,005	4.24		27,570	649,635	4.24
Ischemic heart		17,300	675,005	2.56		16,840	649,635	2.59
Non-accidental		87,800	675,005	13.00		84,540	649,635	13.00
COPD		4025	675,005	0.60		4015	649,635	0.62
Respiratory		8460	661,185	1.28		8260	641,035	1.28
Lung cancer		7695	675,005	1.14		7695	649,635	1.18
Diabetes		2550	675,005	0.38		2380	649,635	0.37
Cerebrovascular	3 (1152–1583 m)	6125	671,180	0.91	3 (2109–6678 m)	5405	635,745	0.85
Cardiovascular		28,580	671,180	4.26		26,480	635,745	4.17
Ischemic heart		17,370	671,180	2.59		16,350	635,745	2.57
Non-accidental		87,150	671,180	13.00		81,175	635,745	12.8
COPD		3820	671,180	0.57		3620	635,745	0.57
Respiratory		8375	665,100	1.26		7700	625,855	1.23
Lung cancer		7815	671,180	1.16		7385	635,745	1.16
Diabetes		2445	671,180	0.36		2400	635,745	0.38
Cerebrovascular	4 (> 1583 m)	5780	645,820	0.89	4 (> 6678 m)	5380	626,775	0.86
Cardiovascular		29,000	645,820	4.49		25,625	626,775	4.09
Ischemic heart		17,945	645,820	2.78		15,900	626,775	2.54
Non-accidental		89,475	645,820	13.9		79,890	626,775	12.70
COPD		4065	645,820	0.63		3330	626,775	0.53
Respiratory		8615	647,665	1.33		7215	620,110	1.16
Lung cancer		8385	645,820	1.30		7050	626,775	1.12
Diabetes		2725	645,820	0.42		2550	626,775	0.41

increase with greater proximity to primary highways in these models. In models restricted to urban core postal codes, after adjustment for $PM_{2.5}$, the HRs often became much higher: in the first quartile, 7.79 (95% CI: 4.80, 12.65) for cerebrovascular disease; 3.24 (95% CI 2.12, 4.96) for lung cancer, and 1.20 (95% CI: 1.05, 1.36) for all non-accidental mortality.

To explore spatial variability within the cohort, we estimated cause-specific mortality risk in each of the seven climate zones, relative to zone 3, the most populated area (Supplementary material, Table S3). Risks were greatest in zone 1, the northern part of Canada, for all mortality causes except COPD and respiratory disease, but otherwise HRs in other climate zones were similar to zone 3. It should be noted that the north of Canada is in general far less populated and less urban than other areas, and a postal code is likely to encompass a much greater area, with potential for misclassification. Differences by climate zone in HRs for an IQR change in distance to a primary highway (Supplementary material, Table S4) were not as great as for increases in local road length. The largest difference was between zone 3 and zone 4 for non-accidental mortality, but the confidence limits cross 1.0 after adjustment for $PM_{2.5}$.

4. Discussion

In this Canadian population-based cohort, residing near greater road density and near primary highways was associated with increased mortality risk from cardiovascular- and respiratory- disease-related causes, with mixed results for mortality due to diabetes. The risk appeared to be greater for subjects living in urban core areas, and associations became stronger with increasing traffic density or proximity to primary highways. Hazard ratios were attenuated by adjustment for

Table 4
Hazard ratios (and 95% confidence intervals) for mortality with an IQR change (1108.6 m) in local roads length, adjusted for $PM_{2.5}$, and indirectly adjusted for obesity and smoking status. Models stratified by sex and age (5-year increments), and adjusted for personal[a] and contextual[b] covariates.

Mortality cause	Main model	Adjusted for $PM_{2.5}$	Adjusted for $PM_{2.5}$; Indirectly adjusted for obesity and smoking
Cerebrovascular	1.01 (0.99, 1.02)	1.00 (0.98, 1.02)	1.00 (0.97, 1.02)
Cardiovascular	1.04 (1.03, 1.04)	1.03 (1.02, 1.04)	1.02 (1.01, 1.04)
Ischemic heart	1.05 (1.04, 1.06)	1.04 (1.03, 1.05)	1.04 (1.03, 1.05)
Non-accidental	1.05 (1.04, 1.05)	1.02 (1.02, 1.03)	1.02 (1.01, 1.03)
COPD	1.02 (1.00, 1.04)	1.02 (0.99, 1.04)	1.01 (0.98, 1.04)
Respiratory	1.03 (1.01, 1.04)	1.02 (1.00, 1.04)	1.01 (0.99, 1.04)
Lung cancer	1.10 (1.08, 1.11)	1.07 (1.06, 1.09)	1.06 (1.05, 1.09)
Diabetes	1.04 (1.02, 1.06)	0.98 (0.96, 1.01)	0.97 (0.95, 1.02)

[a] Aboriginal ancestry, visible minority status, highest level of education, employment status, occupational class, immigrant status, marital status, income quintile.
[b] Census division % of immigrants, % of adults without high school diploma, % of subjects in lowest income quintile.

Table 5
Hazard ratios for an IQR change (1108 m) in local roads length, adjusted for $PM_{2.5}$, by traffic variable quartile; for all subjects; and for subjects resident in urban core areas only. Models stratified by sex and age (5-year increments), and adjusted for personal[a] and contextual[b] covariates.

Mortality cause	Traffic quartile	All		Urban core	
		Main model HR	HR adjusted $PM_{2.5}$	Main model HR	HR adjusted $PM_{2.5}$
Cerebrovascular	1 (< 475 m)	1	1	1	1
	2 (475–1152 m)	1.00 (0.93, 1.06)	0.95 (0.89, 1.02)	0.81 (0.74, 0.89)	1.03 (1.09, 0.98)
	3 (1152–1583 m)	1.09 (1.01, 1.17)	1.01 (0.93, 1.1)	0.88 (0.79, 0.97)	1.09 (1.06, 1.13)
	4 (> 1583 m)	1.07 (0.97, 1.17)	1.00 (0.9, 1.11)	0.82 (0.72, 0.93)	1.02 (1, 1.05)
Cardiovascular	1 (< 475 m)	1	1	1	1
	2 (475–1152 m)	1.14 (1.11, 1.18)	1.04 (1.01, 1.08)	1.23 (1.17, 1.29)	1.1 (1.07, 1.12)
	3 (1152–1583 m)	1.24 (1.2, 1.28)	1.11 (1.06, 1.15)	1.25 (1.18, 1.32)	1.06 (1.04, 1.07)
	4 (> 1583 m)	1.25 (1.19, 1.3)	1.1 (1.05, 1.16)	1.25 (1.17, 1.33)	1.05 (1.04, 1.06)
Ischemic heart	1 (< 475 m)	1	1	1	1
	2 (475–1152 m)	1.14 (1.1, 1.19)	1.02 (0.98, 1.06)	1.21 (1.13, 1.29)	1.04 (1.01, 1.08)
	3 (1152–1583 m)	1.23 (1.18, 1.29)	1.08 (1.03, 1.13)	1.25 (1.16, 1.34)	1.06 (1.04, 1.08)
	4 (> 1583 m)	1.23 (1.17, 1.3)	1.07 (1.01, 1.14)	1.26 (1.16, 1.37)	1.05 (1.04, 1.07)
Non-accidental	1 (< 475 m)	1	1	1	1
	2 (475–1152 m)	1.12 (1.1, 1.14)	1.04 (1.02, 1.06)	1.11 (1.08, 1.14)	1.02 (1.00, 1.03)
	3 (1152–1583 m)	1.19 (1.17, 1.21)	1.09 (1.07, 1.11)	1.18 (1.14, 1.21)	1.04 (1.04, 1.05)
	4 (> 1583 m)	1.20 (1.18, 1.23)	1.09 (1.06, 1.12)	1.18 (1.14, 1.22)	1.04 (1.03, 1.04)
COPD	1 (< 475 m)	1	1	1	1
	2 (475–1152 m)	1.17 (1.09, 1.25)	1.1 0(1.02, 1.19)	1.00 (0.88, 1.13)	0.94 (0.88, 1.00)
	3 (1152–1583 m)	1.24 (1.14, 1.34)	1.16 (1.06, 1.27)	1.09 (0.95, 1.24)	1.02 (0.98, 1.06)
	4 (> 1583 m)	1.25 (1.13, 1.39)	1.14 (1.01, 1.28)	1.11 (0.95, 1.3)	1.00 (0.97, 1.03)
Respiratory	1 (< 475 m)	1	1	1	1
	2 (475–1152 m)	1.12 (1.06, 1.18)	1.07 (1.00, 1.13)	0.90 (0.82, 0.97)	0.96 (0.92, 1.00)
	3 (1152–1583 m)	1.20 (1.12, 1.27)	1.13 (1.05, 1.21)	0.99 (0.9, 1.08)	1.04 (1.01, 1.07)
	4 (> 1583 m)	1.19 (1.10, 1.29)	1.12 (1.02, 1.22)	1.00 (0.90, 1.12)	1.06 (1.04, 1.08)
Lung cancer	1 (< 475 m)	1	1	1	1
	2 (475–1152 m)	1.20 (1.15, 1.27)	1.14 (1.08, 1.21)	1.38 (1.25, 1.52)	1.05 (1.01, 1.10)
	3 (1152–1583 m)	1.26 (1.2, 1.34)	1.18 (1.11, 1.26)	1.50 (1.34, 1.67)	1.07 (1.04, 1.10)
	4 (> 1583 m)	1.27 (1.18, 1.37)	1.18 (1.08, 1.27)	1.54 (1.36, 1.75)	1.07 (1.05, 1.10)
Diabetes	1 (< 475 m)	1	1	1	1
	2 (475–1152 m)	0.88 (0.81, 0.96)	0.88 (0.81, 0.97)	1.12 (0.96, 1.3)	1.07 (0.99, 1.16)
	3 (1152–1583 m)	0.92 (0.83, 1.01)	0.88 (0.79, 0.98)	1.12 (0.95, 1.33)	0.99 (0.94, 1.04)
	4 (> 1583 m)	0.96 (0.85, 1.09)	0.92 (0.8, 1.05)	1.18 (0.97, 1.43)	1.04 (1.00, 1.08)

[a] Aboriginal ancestry, visible minority status, highest level of education, employment status, occupational class, immigrant status, marital status, income quintile.
[b] Census division % of immigrants, % of adults without high school diploma, % of subjects in lowest income quintile.

$PM_{2.5}$, NO_2, and O_3, with a high degree of collinearity between the three pollutant exposures, yet the associations remained positive. The slight differences in risk observed by climate zone suggest limited spatial variation in the association between traffic density and health across Canada's climate zones.

Living near dense traffic increases exposure to traffic related air pollution (Agudelo-Castañeda et al., 2016, 2017), which includes black carbon, combustion products, nitrogen oxides, ultrafine particulate matter, as well as traffic noise. Associations between these components of the air pollution mixture with specific health outcomes have been widely investigated (McGregor, 1999; Pinault et al., 2016; Vanos et al., 2013, 2014). The biological mechanisms by which various pollutants affect health may include systemic inflammation, oxidative stress and immune responses, and development of cancers (Cohen et al., 2017; Hamra et al., 2015), and stress resulting from traffic noise (Study et al., 2013). Human health outcomes to traffic-related air pollution and noise are consequently broad, and include cardiovascular diseases, stroke, heart attack, cognitive impairment, dementia and Parkinson's disease (Korek et al., 2015; Sorensen et al., 2013; Tzivian et al., 2016). In the current study, lung cancer mortality was frequently significantly associated with greater roadway density and closer proximity to major highways.

Our results are similar to Chen et al. (2013), who found positive associations between living close to major roads and cardiovascular mortality in a three-city cohort of 205,440 adults in Ontario, Canada, a result they find to also be consistent with LUR model estimates of NO_2 exposures that incorporate roadway data. Also using LUR to predict NO_2 exposures in Toronto, Jerrett et al. (2009) found that a 4 ppb

Table 6
Hazard ratios for an IQR change (5991 m) in distance to primary highway, adjusted for $PM_{2.5}$, and indirectly adjusted for obesity and smoking status. Models stratified by sex and age (5-year increments), and adjusted for personal[a] and contextual[b] covariates.

Mortality cause	Main model	Adjusted for $PM_{2.5}$	Adjusted for $PM_{2.5}$; Indirectly adjusted for obesity and smoking
Cerebrovascular	0.99 (0.96, 1.03)	0.97 (0.93, 1.01)	0.96 (0.92, 1.01)
Cardiovascular	1.01 (0.99, 1.02)	0.98 (0.96, 1.00)	0.98 (0.96, 1.00)
Ischemic heart	1.01 (1.00, 1.03)	0.98 (0.96, 1.00)	0.97 (0.95, 1.00)
Non-accidental	1.03 (1.02, 1.04)	1.01 (1.00, 1.02)	1.01 (1.00, 1.02)
COPD	1.02 (0.98, 1.06)	1.00 (0.96, 1.04)	1.00 (0.96, 1.04)
Respiratory	1.04 (1.02, 1.06)	1.03 (1.00, 1.05)	1.02 (0.99, 1.05)
Lung cancer	0.99 (0.96, 1.02)	0.98 (0.95, 1.01)	0.96 (0.93, 1.00)
Diabetes	1.08 (1.05, 1.10)	1.05 (1.02, 1.08)	1.04 (1.01, 1.08)

[a] Aboriginal ancestry, visible minority status, highest level of education, employment status, occupational class, immigrant status, marital status, income quintile.
[b] Census division % of immigrants, % of adults without high school diploma, % of subjects in lowest income quintile.

Table 7
Hazard ratios for an IQR change (5991 m) in distance to primary highway, adjusted for $PM_{2.5}$, by traffic variable quartile, for (a) all subjects, and (b) for subjects with residence in urban core areas only. Models stratified by sex and age (5-year increments) and adjusted for personal[a] and contextual[b] covariates.

Mortality cause	Traffic quartile	(a) All		(b) Urban core	
		Main model	Adjusted $PM_{2.5}$	Main model	Adjusted $PM_{2.5}$
Cerebrovascular	1 (< 686 m)	2.94 (2.05, 4.23)	1.84 (1.20, 2.82)	5.88 (4.08, 8.49)	7.79 (4.80, 12.65)
	2 (686–2109 m)	1.27 (1.14, 1.41)	1.28 (1.14, 1.45)	1.38 (1.25, 1.54)	1.59 (1.39, 1.83)
	3 (2109–6678 m)	1.10 (1.06, 1.14)	1.11 (1.07, 1.16)	1.19 (1.15, 1.24)	1.27 (1.21, 1.33)
	4 (> 6678 m)	1	1	1	1
Cardiovascular	1 (< 686 m)	1.70 (1.44, 2.01)	1.34 (1.11, 1.63)	1.34 (1.13, 1.61)	0.52 (0.41, 0.65)
	2 (686–2109 m)	1.18 (1.12, 1.24)	1.13 (1.07, 1.19)	1 (0.96, 1.06)	0.77 (0.73, 0.82)
	3 (2109–6678 m)	1.07 (1.05, 1.09)	1.06 (1.04, 1.08)	1.08 (1.06, 1.1)	1.01 (0.99, 1.04)
	4 (> 6678 m)	1	1	1	1
Ischemic heart	1 (< 686 m)	1.08 (0.87, 1.34)	1.06 (0.83, 1.37)	1.15 (0.91, 1.46)	0.72 (0.53, 0.98)
	2 (686–2109 m)	1.10 (1.04, 1.17)	1.1 (1.03, 1.18)	1.19 (1.12, 1.27)	1.02 (0.94, 1.11)
	3 (2109–6678 m)	1.05 (1.03, 1.07)	1.05 (1.03, 1.08)	1.12 (1.09, 1.14)	1.07 (1.03, 1.10)
	4 (> 6678 m)	1	1	1	1
Non-accidental	1 (< 686 m)	1.57 (1.44, 1.72)	1.25 (1.12, 1.39)	1.86 (1.68, 2.05)	1.20 (1.05, 1.36)
	2 (686–2109 m)	1.1 (1.07, 1.13)	1.07 (1.04, 1.10)	1.1 (1.07, 1.13)	0.94 (0.91, 0.98)
	3 (2109–6678 m)	1.04 (1.03, 1.05)	1.04 (1.03, 1.05)	1.07 (1.06, 1.08)	1.04 (1.02, 1.05)
	4 (> 6678 m)	1	1	1	1
COPD	1 (< 686 m)	1.99 (1.32, 2.99)	2.27 (1.41, 3.65)	1.68 (1.06, 2.66)	1.71 (0.95, 3.09)
	2 (686–2109 m)	1.15 (1.02, 1.30)	1.20 (1.05, 1.38)	1.29 (1.13, 1.47)	1.35 (1.14, 1.59)
	3 (2109–6678 m)	1.06 (1.02, 1.11)	1.06 (1.01, 1.11)	1.08 (1.03, 1.14)	1.08 (1.02, 1.15)
	4 (> 6678 m)	1	1	1	1
Respiratory	1 (< 686 m)	1.03 (0.76, 1.40)	1.26 (0.88, 1.8)	0.56 (0.41, 0.78)	0.62 (0.40, 0.94)
	2 (686–2109 m)	1.07 (0.98, 1.16)	1.13 (1.02, 1.25)	0.99 (0.90, 1.08)	0.99 (0.88, 1.12)
	3 (2109–6678 m)	1.06 (1.03, 1.09)	1.08 (1.05, 1.12)	1.03 (1.00, 1.06)	1.05 (1.01, 1.09)
	4 (> 6678 m)	1	1	1	1
Lung cancer	1 (< 686 m)	2.30 (1.74, 3.03)	1.82 (1.31, 2.53)	5.27 (3.75, 7.4)	3.24 (2.12, 4.96)
	2 (686–2109 m)	1.11 (1.03, 1.21)	1.07 (0.97, 1.17)	1.50 (1.36, 1.65)	1.12 (1.00, 1.27)
	3 (2109–6678 m)	1.02 (0.99, 1.05)	1.01 (0.98, 1.04)	1.11 (1.07, 1.15)	1.02 (0.98, 1.07)
	4 (> 6678 m)	1	1	1	1
Diabetes	1 (< 686 m)	1.10 (0.67, 1.79)	0.64 (0.35, 1.16)	1.80 (1.03, 3.15)	1.26 (0.59, 2.69)
	2 (686–2109 m)	0.77 (0.66, 0.88)	0.76 (0.64, 0.90)	0.83 (0.71, 0.97)	0.88 (0.71, 1.09)
	3 (2109–6678 m)	0.98 (0.94, 1.03)	1 (0.95, 1.06)	1.07 (1.01, 1.13)	1.11 (1.04, 1.2)
	4 (> 6678 m)	1	1	1	1

[a] Aboriginal ancestry, visible minority status, highest level of education, employment status, occupational class, immigrant status, marital status, income quintile.
[b] Census division % of immigrants, % of adults without high school diploma, % of subjects in lowest income quintile.

increase in NO_2 resulted in a 40% increase in circulatory mortality in susceptible individuals that had attended a respiratory clinic.

We found mixed results for an association between diabetes-related mortality and road length as a proxy for traffic density. Previous studies have identified links between road traffic noise, stress, and sleep disturbances, as well as higher type-2 diabetes risk (Sorensen et al., 2013). Others have found associations between NO_2 exposures and diabetes mellitus diagnosis (Brook et al., 2008), and an increase in mortality from diabetes due to traffic density (Raaschou-Nielsen et al., 2013). In our models, the positive association observed with increasing traffic density was not present, and adjustment for air pollution exposure reduced risk estimates to below 1. In single- and multi-pollutant models for mortality risk in the 1991 CanCHEC, Crouse et al. (2015) found similarly low estimates (e.g. HR 1.039, 95% CI: 0.999, 1.080, for models adjusted for NO_2).

In sensitivity analyses, restricting to urban core areas increased many of the risk estimates, but the health association was generally not sensitive to geographic location, suggesting that spatial variability was captured more effectively using quartiles of traffic density.

4.1. Strengths and limitations

The large size of this study and the inclusion of subjects from across Canada is a key strength. The information available allowed us to adjust for both individual and neighborhood-level mortality risk factors, and to assign exposures by 6-digit postal code for each year of follow-up. This method captured subjects that could otherwise be lost to follow-up due to movement over the duration of the cohort. As people spend time away from their place of residence, however, it is not possible to fully capture subject's exposures to pollution and traffic on a daily or long-term basis. Traffic variables were assigned by postal code, but as a rural postal code could encompass a much larger area than an urban postal code, there may have been misclassification related to measurements of local road lengths and distance to major highways. Additionally, exposure to traffic may be more important for a subject that has commuted in from a suburban, low traffic area, to an urban, dense, high traffic area; relatedly, it is likely that variations in exposures due to traffic are likely to be greater in urban rather than rural areas, so a misclassification due to the larger rural postal code areas is less impactful. The use of a geocoding technique to assign a centroid to a postal code for each subject would also add spatial variability to the dataset (Khan et al., 2018), but this is likely to be minimized in urban compared to rural areas.

Methods are limited by the resolution and coverage of air pollutant monitoring, as well as difficulties in assigning exposures to individuals as they move from residential to working spaces; it is difficult to adequately capture patterns of personal exposure in a long term study based on exposure estimates using residence.

This study was also limited in that we did not have access to individual risk factors that affect disease cause mortality, such as smoking and BMI. The use of an indirect adjustment technique for these risk factors had very little effect on hazard ratios, consistent with previous work with the CanCHEC cohort (Crouse et al., 2015).

Future studies with Canadian national cohorts should consider additional stressors related to traffic density including noise, as it is increasingly linked to health outcomes including hypertension, myocardial infarction, and stroke (Gori et al., 2014), as well as diabetes (Sorensen et al., 2013). The potential for effect modification by

socioeconomic background should also be explored where data permits.

5. Conclusion

In this long-term population-based cohort study, increased exposure to local roads and proximity to major highways was associated with increased mortality risk from cardiovascular and respiratory disease causes, even after adjustment for air pollution concentrations. Given the high proportion of the Canadian population that lives in urban areas, living near dense traffic presents a long-term population health burden. Such information is vital for urban planning and decision making, and may also help site new air pollution sensors within urban areas to more accurately monitor ground-level exposures by time of day and year, as related to traffic density and negative health outcomes.

Acknowledgements

This work was funded under Government of Canada's Clear Air Regulatory Agenda (CARA). The authors would like to thank Statistics Canada for help accessing census and socioeconomic data. The authors would also like to thank the reviewers for their helpful feedback.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.envint.2018.12.045.

References

Agudelo-Castañeda, D.M., Teixeira, E.C., Schneider, I.L., Pereira, F.N., Oliveira, M.L.S., Taffarel, S.R., Sehn, J.L., Ramos, C.G., Silva, L.F.O., 2016. Potential utilization for the evaluation of particulate and gaseous pollutants at an urban site near a major highway. Sci. Total Environ. 543, 161–170.

Agudelo-Castañeda, D.M., Teixeira, E.C., Schneider, I.L., Lara, S.R., Silva, L.F.O., 2017. Exposure to polycyclic aromatic hydrocarbons in atmospheric PM1.0 of urban environments: carcinogenic and mutagenic respiratory health risk by age groups. Environ. Pollut. 224, 158–170.

Beelen, R., Hoek, G., Houthuijs, D., van den Brandt, P.A., Goldbohm, R.A., Fischer, P., Schouten, L.J., Armstrong, B., Brunekreef, B., 2009. The joint association of air pollution and noise from road traffic with cardiovascular mortality in a cohort study. Occup. Environ. Med. 66, 243–250.

Bell, M.L., Ebisu, K., Leaderer, B.P., Gent, J.F., Lee, H.J., Koutrakis, P., Wang, Y., Dominici, F., Peng, R.D., 2014. Associations of PM2.5 constituents and sources with hospital admissions: analysis of four counties in Connecticut and Massachusetts (USA) for persons ≥65 years of age. Environ. Health Perspect. 122, 138–144.

Boys, B.L., Martin, R.V., Van Donkelaar, A., MacDonell, R.J., Hsu, N.C., Cooper, M.J., Yantosca, R.M., Lu, Z., Streets, D.G., Zhang, Q., Wang, S.W., 2014. Fifteen-year global time series of satellite-derived fine particulate matter. Environ. Sci. Technol. 48, 11109–11118.

Brook, R.D., Jerrett, M., Brook, J.R., Bard, R.L., Finkelstein, M.M., 2008. The relationship between diabetes mellitus and traffic-related air pollution. J. Occup. Environ. Med. 50, 32–38.

Cakmak, S., Hebbern, C., Cakmak, J.D.J.D., Vanos, J., 2016a. The modifying effect of socioeconomic status on the relationship between traffic, air pollution and respiratory health in elementary schoolchildren. J. Environ. Manag. 177, 1–8.

Cakmak, S., Hebbern, C., Vanos, J., Crouse, D.L., Burnett, R., 2016b. Ozone exposure and cardiovascular-related mortality in the Canadian Census Health and Environment Cohort (CANCHEC) by spatial synoptic classification zone. Environ. Pollut. 214, 589–599.

Cakmak, S., Hebbern, C., Pinault, L., Lavigne, E., Vanos, J., Crouse, D.L., Tjepkema, M., 2018. Associations between long-term PM2.5and ozone exposure and mortality in the Canadian Census Health and Environment Cohort (CANCHEC), by spatial synoptic classification zone. Environ. Int. 111, 200–211.

Chen, H., Goldberg, M.S., Burnett, R.T., Jerrett, M., Wheeler, A.J., Villeneuve, P.J., 2013. Long-term exposure to traffic-related air pollution and cardiovascular mortality. Epidemiology 24, 35–43.

Cohen, G., Levy, I., Yuval, Kark, J.D., Levin, N., Broday, D.M., Steinberg, D.M., Gerber, Y., 2017. Long-term exposure to traffic-related air pollution and cancer among survivors of myocardial infarction: a 20-year follow-up study. Eur. J. Prev. Cardiol. 24, 92–102.

Crouse, D.L., Peters, P.A., Hystad, P., Brook, J.R., van Donkelaar, A., Martin, R.V., Villeneuve, P.J., Jerrett, M., Goldberg, M.S., Arden Pope, C., Brauer, M., Brook, R.D., Robichaud, A., Menard, R., Burnett, R.T., Pope, C.A., Brauer, M., Brook, R.D., Robichaud, A., Menard, R., Burnett, R.T., Arden Pope, C., Brauer, M., Brook, R.D., Robichaud, A., Menard, R., Burnett, R.T., 2015. Ambient PM2.5, O3, and NO2 exposures and associations with mortality over 16 years of follow-up in the Canadian census health and environment cohort (CanCHEC). Environ. Health Perspect. 123, 1180–1186.

Crouse, D.L., Pinault, L., Balram, A., Hystad, P., Peters, P.A., Chen, H., van Donkelaar, A., Martin, R.V., Ménard, R., Robichaud, A., Villeneuve, P.J., 2017. Urban greenness and mortality in Canada's largest cities: a national cohort study. Lancet Planet. Health 1, e289–e297.

Dales, R., Wheeler, A.J., Mahmud, M., Frescura, A.-M., Liu, L., 2009. The influence of neighborhood roadways on respiratory symptoms among elementary schoolchildren. J. Occup. Environ. Med. 51, 654–660.

Fuks, K.B., Weinmayr, G., Foraster, M., Dratva, J., Hampel, R., Houthuijs, D., Oftedal, B., Oudin, A., Panasevich, S., Penell, J., Sommar, J.N., Sørensen, M., Tiittanen, P., Wolf, K., Xun, W.W., Aguilera, I., Basagaña, X., Beelen, R., Bots, M.L., Brunekreef, B., Bueno-de-Mesquita, H.B., Caracciolo, B., Cirach, M., de Faire, U., de Nazelle, A., Eeftens, M., Elosua, R., Erbel, R., Forsberg, B., Fratiglioni, L., Gaspoz, J.M., Hilding, A., Jula, A., Korek, M., Krämer, U., Künzli, N., Lanki, T., Leander, K., Magnusson, P.K.E., Marrugat, J., Nieuwenhuijsen, M.J., Östenson, C.G., Pedersen, N.L., Pershagen, G., Phuleria, H.C., Probst-Hensch, N.M., Raaschou-Nielsen, O., Schaffner, E., Schikowski, T., Schindler, C., Schwarze, P.E., Søgaard, A.J., Sugiri, D., Swart, W.J.R., Tsai, M.Y., Turunen, A.W., Vineis, P., Peters, A., Hoffmann, B., 2014. Arterial blood pressure and long-term exposure to traffic-related air pollution: an analysis in the european study of cohorts for air pollution effects (ESCAPE). Environ. Health Perspect. 122 (9), 896–905.

Gilbert, N.L., Goldberg, M.S., Beckerman, B., Brook, J.R., Jerrett, M., 2005. Assessing spatial variability of ambient nitrogen dioxide in Montréal, Canada, with a land-use regression model. J. Air Waste Manage. Assoc. 55, 1059–1063.

Gori, T., Babisch, W., Basner, M., Mu, T., 2014. Cardiovascular effects of environmental noise exposure. Eur. Heart J. 829–836.

Hamra, G.B., Laden, F., Cohen, A.J., Raaschou-Nielsen, O., Brauer, M., Loomis, D., 2015. Lung cancer and exposure to nitrogen dioxide and traffic: a systematic review and meta-analysis. Environ. Health Perspect. 123, 1107–1112.

Holguin, F., Flores, S., Ross, Z., Cortez, M., Molina, M., Molina, L., Rincon, C., Jerrett, M., Berhane, K., Granados, A., Romieu, I., 2007. Traffic-related exposures, airway function, inflammation, and respiratory symptoms in children. Am. J. Respir. Crit. Care Med. 176, 1236–1242.

Hondula, D.M., Vanos, J.K., Gosling, S.N., 2014. The SSC: a decade of climate-health research and future directions. Int. J. Biometeorol. 58 (2), 109–120.

Hystad, P., Setton, E., Cervantes, A., Poplawski, K., Deschenes, S., Brauer, M., van Donkelaar, A., Lamsal, L., Martin, R., Jerrett, M., Demers, P., 2011. Creating national air pollution models for population exposure assessment in Canada. Environ. Health Perspect. 119, 1123–1129.

Jerrett, M., Finkelstein, M.M., Brook, J.R., Arain, M.A., Kanaroglou, P., Stieb, D.M., Gilbert, N.L., Verma, D., Finkelstein, N., Chapman, K.R., Sears, M.R., 2009. A cohort study of traffic-related air pollution and mortality in Toronto, Ontario, Canada. Environ. Health Perspect. 117, 772–777.

Khan, S., Pinault, L., Tjepkema, M., Wilkins, R., 2018. Positional accuracy of geocoding from residential postal codes versus full street addresses. Health Rep. 29.

Korek, M.J., Bellander, T.D., Lind, T., Bottai, M., Eneroth, K.M., Caracciolo, B., de Faire, U.H., Fratiglioni, L., Hilding, A., Leander, K., Magnusson, P.K.E., Pedersen, N.L., Östenson, C.-G., Pershagen, G., Penell, J.C., 2015. Traffic-related air pollution exposure and incidence of stroke in four cohorts from Stockholm. J. Expo. Sci. Environ. Epidemiol. 25, 517–523.

McConnell, R., Berhane, K., Yao, L., Jerrett, M., Lurmann, F., Gilliland, F., Künzli, N., Gauderman, J., Avol, E., Thomas, D., Peters, J., 2006. Traffic, susceptibility, and childhood asthma. Environ. Health Perspect. 114, 766–772.

McGregor, G., 1999. Winter ischaemic heart disease deaths in Birmingham, United Kingdom: a synoptic climatological analysis. Clim. Res. 13, 17–31.

Peters, P.A., Tjepkema, M., Wilkins, R., Fines, P., Crouse, D.L., Chan, P.C.W., Burnett, R.T., Ching, P., Chan, W., Burnett, R.T., 2013. Data resource profile: 1991 Canadian Census Cohort. Int. J. Epidemiol. 42, 1319–1326.

Pinault, L., Tjepkema, M., Crouse, D.L., Weichenthal, S., van Donkelaar, A., Martin, R.V., Brauer, M., Chen, H., Burnett, R.T., 2016. Risk estimates of mortality attributed to low concentrations of ambient fine particulate matter in the Canadian community health survey cohort. Environ. Health 15, 18.

Raaschou-Nielsen, O., Sørensen, M., Ketzel, M., Hertel, O., Loft, S., Tjønneland, A., Overvad, K., Andersen, Z.J., 2013. Long-term exposure to traffic-related air pollution and diabetes-associated mortality: a cohort study. Diabetologia 56, 36–46.

Robichaud, A., Ménard, R., 2013. Multi-year objective analyses of warm season ground-level ozone and $PM_{2.5}$ over North America using real-time observations and Canadian operational air quality models. Atmos. Chem. Phys. Discuss. 13, 13967–14035.

Shin, H.H., Stieb, D., Burnett, R., Takahara, G., Jessiman, B., 2012. Tracking national and regional spatial-temporal mortality risk associated with NO2 concentrations in Canada: a Bayesian hierarchical two-level model. Risk Anal. 32, 513–530.

Sorensen, M., Andersen, Z.J., Nordsborg, R.B., Becker, T., Tjonneland, A., Overvad, K., Raaschou-Nielsen, O., 2013. Long-term exposure to road traffic noise and incident diabetes: a cohort study. Environ. Health Perspect. 121, 217–222.

Statistics Canada, 2011. Census Dictionary.

Statistics Canada, 2016. Postal CodeOM Conversion File Plus (PCCF+) Version 6C, Reference Guide.

Study, A.C., Sørensen, M., Andersen, Z.J., Nordsborg, R.B., Becker, T., Tjønneland, A., Overvad, K., Raaschou-Nielsen, O., 2013. Long-term exposure to road traffic noise and incident diabetes: a cohort study. Environ. Health Perspect. 121, 217–222.

Tamayo, T., Rathmann, W., Krämer, U., Sugiri, D., Grabert, M., Holl, R.W., 2014. Is particle pollution in outdoor air associated with metabolic control in type 2 diabetes? PLoS One 9.

Turner, M.C., Krewski, D., Pope, C.A., Chen, Y., Gapstur, S.M., Thun, M.J., 2011. Long-term ambient fine particulate matter air pollution and lung cancer in a large cohort of never-smokers. Am. J. Respir. Crit. Care Med. 184, 1374–1381.

Tzivian, L., Dlugaj, M., Winkler, A., Weinmayr, G., Hennig, F., Fuks, K.B., Vossoughi, M.,

Schikowski, T., Weimar, C., Erbel, R., Jöckel, K.H., Moebus, S., Hoffmann, B., 2016. Long-term air pollution and traffic noise exposures and mild cognitive impairment in older adults: a cross-sectional analysis of the Heinz Nixdorf recall study. Environ. Health Perspect. 124, 1361–1368.

Urman, R., McConnell, R., Islam, T., Avol, E.L., Lurmann, F.W., Vora, H., Linn, W.S., Rappaport, E.B., Gilliland, F.D., Gauderman, W.J., 2014. Associations of children's lung function with ambient air pollution: joint effects of regional and near-roadway pollutants. Thorax 69, 540–547.

van Donkelaar, A., Martin, R.V., Brauer, M., Kahn, R., Levy, R., Verduzco, C., Villeneuve, P.J., 2010. Global estimates of ambient fine particulate matter concentrations from satellite-based aerosol optical depth: development and application. Environ. Health Perspect. 118, 847–855.

Vanos, J.K., Cakmak, S., Bristow, C., Brion, V., Tremblay, N., Martin, S.L., Sheridan, S.S., 2013. Synoptic weather typing applied to air pollution mortality among the elderly in 10 Canadian cities. Environ. Res. 126, 66–75.

Vanos, J.K., Hebbern, C., Cakmak, S., 2014. Risk assessment for cardiovascular and respiratory mortality due to air pollution and synoptic meteorology in 10 Canadian cities. Environ. Pollut. 185, 322–332.

Vanos, J.K., Cakmak, S., Kalkstein, L.S., Yagouti, A., 2015. Association of weather and air pollution interactions on daily mortality in 12 Canadian cities. Air Qual. Atmos. Health 174, 15–26.

Weinmayr, G., Hennig, F., Fuks, K., Nonnemacher, M., Jakobs, H., Möhlenkamp, S., Erbel, R., Jöckel, K.-H.H., Hoffmann, B., Moebus, S., 2015. Long-term exposure to fine particulate matter and incidence of type 2 diabetes mellitus in a cohort study: effects of total and traffic-specific air pollution. Environ. Health 14, 53.

Zhang, X., Staimer, N., Tjoa, T., Gillen, D.L., Schauer, J.J., Shafer, M.M., Hasheminassab, S., Pakbin, P., Longhurst, J., Sioutas, C., Delfino, R.J., 2016. Associations between microvascular function and short-term exposure to traffic-related air pollution and particulate matter oxidative potential. Environ. Health 15, 81.