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Original Contribution

Exposure to Primary Air Pollutants Generated by Highway Traffic and Daily Mortality Risk in Near-Road Communities: A Case-Crossover Study

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Most epidemiologic studies fail to capture the impact of spatiotemporal fluctuations in traffic on exposure to traffic-related air pollutants in the near-road population. Using a case-crossover design and the Research LINE source (R-LINE) dispersion model with spatiotemporally resolved highway traffic data, we quantified associations between primary pollutants generated by highway traffic—particulate matter with an aerodynamic diameter less than or equal to 2.5 μm ($PM_{2.5}$), oxides of nitrogen (NO_x), and black carbon (BC)—and daily nonaccidental, respiratory, cardiovascular, and cerebrovascular mortality among persons who had resided within 1 km (0.6 mile) of major highways in the Puget Sound area of Washington State between 2009 and 2013. We estimated these associations using conditional logistic regression, adjusting for time-varying covariates. Although highly resolved modeled concentrations of $PM_{2.5}$, NO_x, and BC from highway traffic in the hours before death were used, we found no evidence of an association between mortality and the preceding 24-hour average $PM_{2.5}$ exposure (odds ratio = 0.99, 95% confidence interval: 0.96, 1.02) or exposure during shorter averaging periods. This work did not support the hypothesis that mortality risk was meaningfully higher with greater exposures to $PM_{2.5}$, NO_x, and BC from highways in near-road populations, though we did incorporate a novel approach to estimate exposure to traffic-generated air pollution based on detailed traffic congestion data.

cardiovascular disease; case-crossover study; cerebrovascular disease; mortality; respiratory disease; Research LINE source dispersion model; traffic air pollution

Abbreviations: AQS, Air Quality System; BC, black carbon; CI, confidence interval; ICD-10, *International Classification of Diseases, Tenth Revision*; NO_x, oxides of nitrogen; OR, odds ratio; $PM_{2.5}$, particulate matter with an aerodynamic diameter less than or equal to 2.5 μm; R-LINE, Research LINE source.

Traffic is a major contributor to poor air quality in urban areas due to emissions of pollutants, including oxides of nitrogen (NO_x), black carbon (BC), and fine particulate matter, defined as particulate matter with an aerodynamic diameter less than or equal to 2.5 μm ($PM_{2.5}$) (1). Despite policies designed to control vehicle emissions, exposures to these traffic-related air pollutants continue to be associated with a broad range of acute and chronic health outcomes such as ischemic heart disease, stroke, lower respiratory tract infection, chronic obstructive pulmonary disease, and lung cancer (1–10). These health effects are probably shouldered predominantly by persons living closest to major roadways, as concentrations of traffic-related air pollutants are often highest within several hundred meters of highly trafficked roadways (1, 11). In the United States, more than 11 million people live within 150 m (164 yards) of a major highway (12). These buffers contain a disproportionately high fraction of minority racial and ethnic groups and low-income populations (13–18), placing them at greater risk of chronic health outcomes (2–4, 19, 20).

Disruptions to free-flowing traffic due to accidents, construction, policing, or congestion can affect vehicle emissions and exposures of persons living in near-road communities. In spite of these sources of variability, most epidemiologic studies are unsuited to capture these short-term fluctuations in exposure to traffic-related air pollution, as they have largely relied on measurements from urban-scale Air Quality System (AQS) monitors (1, 21, 22). However, AQS monitors

are few in number and too sparse to capture the small-scale spatial variation of traffic-related air pollutants in the near-road environment. Similarly, a lack of temporal resolution in monitoring data can limit the ability to examine exposures in shorter time periods that precede an individual's exact time of death, which has been shown to result in associations that are biased toward the null (21). Furthermore, traffic-related air pollutants monitored at urban-scale stations are comingled with pollutants from other local and regional sources, which makes it yet more challenging to disentangle the contribution of vehicle emissions to near-road exposures.

To our knowledge, only 1 study has investigated the health impacts of short-term spatiotemporal variations in traffic patterns (23). In that work, we used traffic congestion measured each half-mile (0.8 km) and every 5 minutes on the major highways in Washington's Puget Sound area to examine associations with mortality. We found that greater levels of traffic congestion were associated with increased odds of cerebrovascular and respiratory mortality, but lower odds of cardiovascular mortality, for decedents who had resided adjacent to a highway. For that study, however, we only used an indicator for nearby traffic congestion and did not account for differences in the fleet mix across highways and the influence of meteorological conditions on the dispersion of traffic-related air pollutants in the nearby communities. Here, we extend our previous work by leveraging highly resolved traffic activity data with the Research LINE source (R-LINE) dispersion model (24) to estimate concentrations of primary air pollutants generated by highway traffic with high temporal and spatial resolution.

We investigated whether primary $PM_{2.5}$, NO_x, and BC exposures generated by highway traffic (herein referred to as primary traffic-generated $PM_{2.5}$, NO_x, and BC) in the hours before an individual's exact time of death were associated with increased odds of mortality among people living near major highways. In secondary analyses, we explored whether these associations differed across racial, ethnic, and socioeconomic groups.

METHODS

Study design

We conducted a time-stratified case-crossover study (25) to quantify the association between short-term variations in primary traffic-generated $PM_{2.5}$, NO_x, and BC and all nonaccidental, respiratory, cardiovascular, and cerebrovascular mortality. Our study population included all individuals living within 1 km (0.6 mile) of all highways in the Puget Sound region surrounding Seattle, Washington, during 2009–2013 (Figure 1).

In a case-crossover study, each individual acts as their own control, thus minimizing potential confounding by measured and unmeasured time-independent covariates at the individual level (25, 26). We compared exposures to primary traffic-generated $PM_{2.5}$, NO_x, and BC among decedents immediately prior to the time of death (case period) with their own exposures during control periods, which were selected from all days within the same month as the case period, matched by day of the week. This approach prevents bias due to long-term temporal trends, time-independent confounding, and overlap bias (25–27). This study was approved by the institutional review board of the University of Michigan.

Outcome ascertainment

We identified deaths occurring within 1 km of our highways of interest using death certificates for 2009–2013 from the Washington State Department of Health. Underlying causes of death were derived from the *International Classification of Diseases, Tenth Revision* (ICD-10) as mortality due to all nonaccidental causes (ICD-10 codes A00–R99), respiratory disease (ICD-10 codes J00–J98), cardiovascular disease (ICD-10 codes I01–I52, I60–I69), or cerebrovascular disease (ICD-10 codes I60–I69). As a negative control group, we evaluated deaths among persons who had resided within 1 km of our studied highways with underlying causes that were not expected to have been due to primary traffic-generated air pollutants (i.e., injuries, poisoning, and external causes of death other than traffic accidents (ICD-10 codes S00–T88)). For all analyses, we included death certificates from individuals with valid dates of death and residential addresses geocoded by the Washington Department of Health. Inpatient deaths, hospice deaths, and cases with an undefined place of death were excluded from this analysis because the residential address may not have accurately reflected the individual's exposure during both the case and control periods.

Exposure assessment

The exposure assessment approach used in this study has been detailed elsewhere (28). Briefly, we incorporated highly resolved spatiotemporal traffic volume and speed data available for each half-mile and every 5 minutes for all highways (Interstates 5, 405, and 90 and State Routes 167 and 520) in the Puget Sound area from the Transportation Center of the University of Washington into R-LINE (version 1.2). R-LINE is a line-source dispersion model developed by the Environmental Protection Agency (24). The concentrations of primary, chemically inert air pollutants originating from roadways are found by approximating the line source as a series of point sources, where their contribution is computed with a modified Gaussian steady-state plume formulation (28). In performance evaluations, R-LINE model estimates have compared well with measured concentrations of most traffic-related air pollutants (24, 29, 30), and R-LINE has been used previously to assess exposure to air pollutants from traffic in epidemiologic studies (31, 32).

Using the numerical integration of R-LINE and the algorithm for roadside noise barriers, we predicted hourly concentrations of primary traffic-generated $PM_{2.5}$, NO_x, and BC originating from all half-mile road segments at the residential addresses of all decedents. These estimates incorporated fine-scale spatial and temporal variation in traffic congestion that are not typically reflected in other studies that have relied either on sparse measurements or on modeling based on aggregated traffic statistics for a limited number of places across a year. Using these fine-scale hourly-modeled concentrations, we calculated 1-, 6-, 12-, 24-, and 48-hour

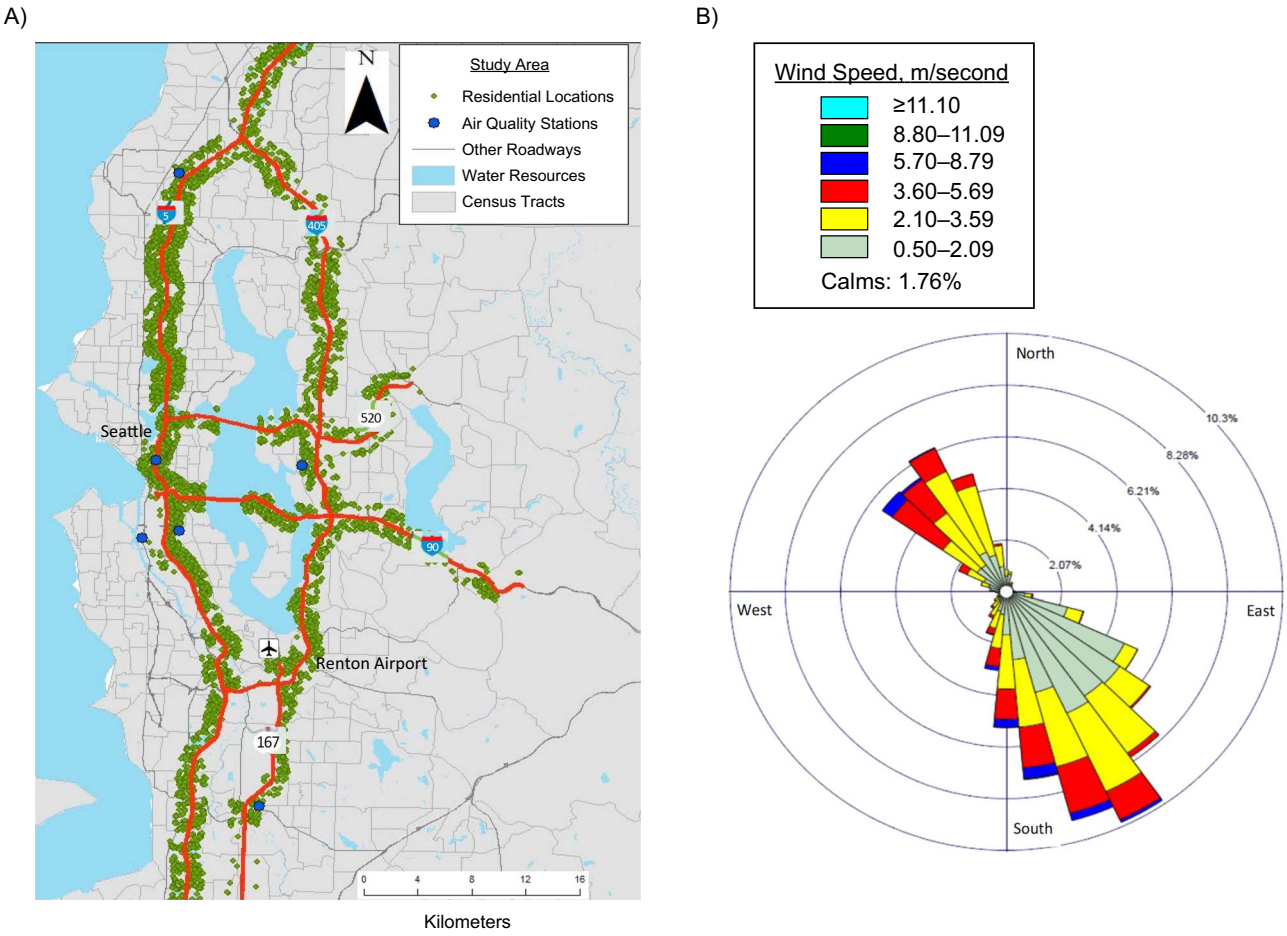

Figure 1. Study area (A) and wind speed (B) for decedents living near major highways in the Puget Sound area, Washington State, 2009–2013. A) Map of the study area. The 5 major highways included are shown as red lines and other larger roads as gray lines. Decedent residential locations within 1 km (0.6 mile) of the 5 study highways are shown as green dots, and the air quality monitoring stations where data on background $PM_{2.5}$ concentrations were obtained are shown as blue dots. B) Average wind speed during the study period. The prevailing wind came from the southeast and south-southeast. $PM_{2.5}$, particulate matter with an aerodynamic diameter less than or equal to 2.5 μm.

moving averages of pollutant concentrations matched to each individual's exact time of death. Based on past literature that found peak associations of 24-hour average concentrations of pollutants with mortality, we used this time frame before each case and control period as our primary exposure, but we explored other averaging periods in secondary analyses. The calculation of the 24-hour moving average accounts for the high temporal variability of both hourly traffic volume and speed and meteorological conditions. To compare our new exposures with those in our previously published work, we estimated Spearman correlation coefficients for the correlations between 24-hour average predicted concentrations and traffic congestion, expressed as the number of congested minute-kilometers near a decedent's home within the previous 24 hours.

Covariates

We obtained hourly temperature, relative humidity, dew point, and rain data from the weather station at Renton Municipal Airport (Renton, Washington) to control for confounding by meteorological conditions (33). We also obtained weekly counts of influenza cases validated by laboratory tests from outpatient surveillance data from the state of Washington (34). To control for potential confounding by holidays, our models included a dummy variable for New Year's Day, Memorial Day, Independence Day, Labor Day, Thanksgiving, or Christmas, as well as the 1 and 2 days before and after each holiday. In secondary analyses, we explored other notable days, such as Super Bowl Sunday (National Football League, New York, New York). To account for confounding by other nontraffic sources of air pollution, we used $PM_{2.5}$ concentrations measured hourly at 5 Environmental Protection Agency AQS monitors in the study area and daily 8-hour maximum ozone concentrations at 1 station in the area (Figure 1). In an attempt to isolate $PM_{2.5}$ pollution from regional sources from $PM_{2.5}$ emissions from the studied highways, we calculated the residuals from a regression of predicted $PM_{2.5}$ concentrations from the highways of interest at each

AQS station and measured levels. We then averaged these hourly residuals across all AQS monitors and estimated 24-hour moving averages of $PM_{2.5}$ background concentrations for each case and control period. In sensitivity analyses, we explored using unadjusted averages from the AQS monitors as an indicator of background levels of pollution.

We evaluated interaction with covariates at both the individual and neighborhood levels. We extracted information on age, race/ethnicity, and education from the death certificates. Following the approach of Diez-Roux et al. (35), we created a composite score of neighborhood disadvantage for each decedent address by combining census variables from the American Community Survey (2009–2013) representing domains of wealth and race/ethnicity for each census tract (i.e., median annual household income, log median value of occupied housing units, percentage of adults who completed college, percentage of persons in executive or managerial occupations, percentage of non-Hispanic Whites, percentage of non-Hispanic Blacks, etc.). We created an indicator of comorbidity for individuals who had diabetes mellitus (ICD-10 codes E10 and E11), overweight/obesity (ICD-10 code E66), hypertension (ICD-10 codes I10–I16), or respiratory disease (ICD-10 codes J00–J99) listed as contributory (but not underlying) causes of death.

Data analysis

We calculated summary statistics to describe the distribution of recorded causes of death, individual and neighborhood-level sociodemographic characteristics, and within-person variations in meteorological conditions, $PM_{2.5}$ background concentrations, ozone concentrations, and concentrations of primary traffic-generated air pollutants. We performed conditional logistic regression according to each cause of death to quantify the associations of daily and hourly variations in primary traffic-generated $PM_{2.5}$, NO_x, and BC at the residential address, matched to time of death, with mortality. We calculated odds ratios (ORs) and 95% confidence intervals (CIs) for all nonaccidental, respiratory, cardiovascular, and cerebrovascular mortality per interquartile-range increase in the moving average concentration of each pollutant. In all analyses, we controlled for the 24-hour moving average of temperature and relative humidity prior to each case and control period using natural cubic splines with knots at the 90th percentile of temperature and relative humidity. This specification was selected over models with up to 6 degrees of freedom and knots at the 10th percentile because it minimized the Akaike information criterion. Since we observed no departures from linearity using natural cubic splines, we controlled for the 24-hour moving average before death of $PM_{2.5}$ background concentrations and the daily 8-hour maximum concentration of ozone in all models as linear variables, while weekly influenza counts and rain levels were modeled using natural cubic splines with 2 degrees of freedom. Public holidays were entered into the models as dichotomous variables.

In secondary analyses, we assessed interaction by including terms for the interactions of age, race/ethnicity, individual-level socioeconomic status, comorbidity, and neighborhood disadvantage score with each pollutant in our regression models. We categorized age as <75 years and ≥ 75 years, and because of small sample sizes for some racial/ethnic groups, we dichotomized race/ethnicity as non-Hispanic White and non-White (i.e., African-American, Hispanic, Asian, Native American, or Hawaiian or other Pacific Islander). Individual-level socioeconomic status was based on educational attainment and was categorized as college, some college, or technical school; high school diploma; and less than high school. The neighborhood disadvantage score was categorized according to tertiles as a high (-20.50 to -2.48), middle (-2.47 to 3.06), or low (3.05 to 19.20) level of disadvantage.

In sensitivity analysis, we first examined associations with our negative control group to evaluate the potential influence of unmeasured confounders. We then assessed the robustness of our estimated associations to different approaches to obtaining $PM_{2.5}$ background concentrations as well as to different timing of the weather variables. We also evaluated interactions between each pollutant and year, rain, and season by including interaction terms in our regression models. Finally, since R-LINE performs better for flat roadways without large elevation differences or surrounding buildings, we also assessed whether excluding the 24% of decedents living at different elevations from the highway influenced our estimates.

Data management was performed with Stata statistical software, version 14.1 (StataCorp LLC, College Station, Texas) and ArcGIS, version 10.1 (ESRI, Redlands, California). Statistical analyses were performed with R software, version 3.1.1 (R Foundation for Statistical Computing, Vienna, Austria).

RESULTS

Between 2009 and 2013, there were 15,659 deaths due to nonaccidental causes in the Puget Sound area among persons living within 1 km of our 5 highways of interest. After exclusion of 5,780 deaths (37%) that occurred among hospital inpatients, in hospice care, or at an undefined location and 50 deaths (0.3%) with an inaccurate residential address, 9,829 deaths (63%) met our inclusion criteria (Table 1). Of those, 8%, 33%, and 5% had respiratory, cardiovascular, or cerebrovascular disease listed as the underlying cause of death, respectively. Decedents were predominantly non-Hispanic White (82%) and female (54%), with a mean age of 79 (standard deviation, 15) years, and had some college or technical school as their maximum level of education (50%). On average, decedents had resided in neighborhoods where 27% of adults had completed college, 46% were employed in an executive or managerial occupation, the median annual household income was $66,000 (interquartile range, 48,000–83,000), and fewer than 6% of persons aged ≥ 16 years were unemployed. Neighborhoods were also predominantly White (59%) (Table 1).

Table 2 shows the distributions of exposure to 1-, 6-, 12-, 24-, and 48-hour moving averages of primary traffic-generated air pollutants between case and control periods by cause of death, as well as the 24-hour moving average of time-varying covariates. Notably, the concentrations of the predicted primary traffic-generated pollutants from major

Table 1. Causes of Death and Individual- and Neighborhood-Level Sociodemographic Characteristics for Decedents in the Puget Sound Region Who Lived Within 1 km (0.6 mile) of a Major Highway ($n = 9,829$), Washington State, 2009–2013

Cause of Death or Sociodemographic Characteristic	No.	%	Mean (SD)	Median (IQR)
Cause of death				
All nonaccidental causes	9,829	100.0		
Respiratory disease	737	7.5		
Cardiovascular disease	3,235	32.9		
Cerebrovascular disease	479	4.9		
Individual-level sociodemographic characteristics				
Age, years			78.9 (14.9)	
Sex				
Female	5,286	53.7		
Male	4,543	46.2		
Race/ethnicity				
Non-Hispanic White	8,043	81.8		
Non-Hispanic Black	530	5.4		
Hispanic	141	1.4		
Asian	1,000	10.2		
Other	97	0.9		
Unknown	18	0.2		
Education				
Technical school or at least some college	4,877	49.6		
High school diploma	3,436	34.9		
Less than high school	1,255	12.8		
Unknown	261	2.7		
Neighborhood-level sociodemographic characteristics				
% of adult residents who completed high school			17.8 (8.3)	
% of adult residents who completed college			27.4 (10.3)	
% of employed residents in an executive or managerial occupation			45.7 (16.6)	
Median value of housing units (in thousands of dollars)				377 (266–436)
Median annual household income (in thousands of dollars)				66 (48–83)
% of households with interest, dividend, or rental income			29.4 (11.7)	
% non-Hispanic White residents			59.2 (16.9)	
% non-Hispanic Black residents			7.4 (7.8)	
% Hispanic residents			9.3 (6.7)	
% of residents aged ≥16 years unemployed			5.7 (2.4)	
% living below poverty level			13.7 (9.3)	

Abbreviations: IQR, interquartile range; SD, standard deviation.

roadways were all highly correlated (Spearman correlation coefficient = 0.99), though these exposure estimates differed (Spearman correlation coefficient = 0.24) from previous crude measures in the study area of proximity to the number of congested minute-kilometers (23) and were moderately correlated with measured daily AQS data (see Web Table 1, available at https://doi.org/10.1093/aje/kwab215). Among all decedents residing within 1 km of a major highway, there were no differences between case and control periods in mean exposures to primary traffic-generated $PM_{2.5}$, NO_x,

Table 2. Concentrations of Primary Traffic-Generated Air Pollutants From Highways Within 1 km (0.6 mile) of the Residence During Case and Control Periods for Decedents in the Puget Sound Region, by Cause of Death, Moving Average Time Window of Exposure, and Time-Varying Covariates, Washington State, 2009–2013

Variable	Moving Average of Primary Traffic-Generated Air Pollutant Exposure														
	24-Hour Average			1-Hour Average			6-Hour Average			12-Hour Average			48-Hour Average		
	Case Period	Control Period	Within-Person SD[a]	Case Period	Control Period	Within-Person SD	Case Period	Control Period	Within-Person SD	Case Period	Control Period	Within-Person SD	Case Period	Control Period	Within-Person SD
Pollutant and cause of death															
$PM_{2.5}$, $\mu g/m^3$															
All nonaccidental causes	1.7	1.7	1.1	1.7	1.8	1.9	1.6	1.6	1.4	1.7	1.7	1.3	1.7	1.7	0.9
Respiratory disease	1.7	1.6	1.1	2.0	1.9	2.1	1.5	1.5	1.4	1.5	1.6	1.3	1.5	1.6	0.9
Cardiovascular disease	1.7	1.7	1.1	1.7	1.9	2.0	1.6	1.7	1.5	1.7	1.7	1.3	1.6	1.7	0.9
Cerebrovascular disease	1.6	1.6	1.1	1.5	1.9	2.0	1.4	1.7	1.6	1.6	1.7	1.4	1.7	1.6	0.9
Negative control group	2.1	2.1	1.2	1.9	2.1	2.2	2.2	2.2	1.8	1.9	2.1	1.5	2.1	2.1	0.9
NO_x, ppb															
All nonaccidental causes	26.8	26.6	17.5	26.4	27.9	29.9	25.4	25.4	22.5	26.4	26.2	20.7	26.5	26.6	14.1
Respiratory disease	25.8	24.7	16.6	30.9	28.8	32.0	23.9	24.5	21.7	24.2	24.6	19.6	24.6	25.2	13.5
Cardiovascular disease	26.1	26.6	17.6	26.9	29.2	31.1	24.7	25.9	22.9	26.2	26.6	20.8	26.3	26.6	14.2
Cerebrovascular disease	25.6	25.6	17.3	24.0	29.3	31.1	25.2	22.8	24.5	25.1	25.8	21.6	26.9	26.2	14.4
Negative control group	32.0	33.4	18.8	31.5	33.9	35.9	35.1	35.1	28.9	30.7	32.5	22.7	32.5	33.1	15.0
Black carbon, $\mu g/m^3$															
All nonaccidental causes	0.7	0.7	0.5	0.7	0.7	0.8	0.7	0.7	0.6	0.7	0.7	0.5	0.7	0.7	0.4
Respiratory disease	0.7	0.6	0.4	0.8	0.8	0.9	0.6	0.6	0.6	0.6	0.6	0.5	0.7	0.7	0.3
Cardiovascular disease	0.7	0.7	0.5	0.7	0.8	0.8	0.6	0.7	0.6	0.7	0.7	0.5	0.7	0.7	0.4
Cerebrovascular disease	0.7	0.7	0.5	0.6	0.8	0.8	0.6	0.7	0.6	0.7	0.7	0.6	0.7	0.7	0.4
Negative control group	0.8	0.9	0.5	0.8	0.8	0.9	0.9	0.9	0.7	0.8	0.8	0.6	0.9	0.9	0.4
Time-varying covariates															
Background $PM_{2.5}$ level[b], $\mu g/m^3$	6.4	6.4	2.8												
Temperature[b], °F	52.3	52.2	4.4												
Relative humidity[b], %	68.7	68.9	9.2												
Weekly no. of influenza cases[b]	79.6	79.1	28.6												
Ozone level[c], ppm	0.02	0.02	0.07												

Abbreviation: BC, black carbon; NO_x, oxides of nitrogen; $PM_{2.5}$, particulate matter with an aerodynamic diameter less than or equal to 2.5 μm; ppb, parts per billion; ppm, parts per million; SD, standard deviation.

[a] All SDs presented in the table were calculated as the within-person variability and averaged across all subjects.

[b] 24-hour moving average.

[c] Daily maximum 8-hour concentration.

Table 3. Associations Between an Interquartile-Range Increase[a] in the 24-Hour Moving Average Concentration of Traffic-Generated Air Pollutants From Nearby Highways and Cause-Specific Mortality for Decedents in the Puget Sound Region, Washington State, 2009–2013

Cause of Death	Air Pollutant					
	$PM_{2.5}$		NO_x		BC	
	OR[b]	95% CI	OR[b]	95% CI	OR[b]	95% CI
All nonaccidental causes	0.99	0.96, 1.02	0.99	0.96, 1.02	0.98	0.96, 1.01
Respiratory disease	0.97	0.87, 1.09	0.98	0.86, 1.11	0.97	0.86, 1.09
Cardiovascular disease	0.96	0.91, 1.01	0.96	0.91, 1.01	0.96	0.91, 1.01
Cerebrovascular disease	0.91	0.78, 1.05	0.90	0.77, 1.05	0.91	0.78, 1.05
Negative control group	0.97	0.86, 1.09	0.96	0.84, 1.09	0.94	0.84, 1.06

Abbreviations: BC, black carbon; CI, confidence interval; IQR, interquartile range; NO_x, oxides of nitrogen; OR, odds ratio; $PM_{2.5}$, particulate matter with an aerodynamic diameter less than or equal to 2.5 μm.

[a] Interquartile range: $PM_{2.5}$, 0.42–2.09 μg/m^3; NO_x, 6.90–33.40 parts per billion; BC, 0.17–0.86 μg/m^3.

[b] Adjusted for temperature, relative humidity, influenza, holidays, rain, background $PM_{2.5}$ concentration, and ozone concentration.

and BC from neighboring highways in the hours before the time of death, except for the 1- and 6-hour pollutant averages for cardiovascular and cerebrovascular disease mortality. Similarly, no differences were found for time-varying covariates.

After adjusting for time-varying covariates, background $PM_{2.5}$ concentrations, and ozone concentrations, we found no evidence of an association between all nonaccidental mortality and exposure to primary traffic-generated $PM_{2.5}$, NO_x, or BC from nearby highways in the 24 hours before the time of death ($PM_{2.5}$: OR = 0.99 (95% CI: 0.96, 1.02); NO_x: OR = 0.99 (95% CI: 0.96, 1.02); BC: OR = 0.98 (95% CI: 0.96, 1.01)). These results were consistent across different causes of death (Table 3). Similar results were found for exposures to primary traffic-generated $PM_{2.5}$, NO_x, and BC in the 1, 6, 12, and 48 hours before death (Table 4 and Web Table 2). One exception was the presence of inverse associations of cardiovascular and cerebrovascular disease mortality with the 1- and 6-hour predeath averages of primary traffic-generated air pollutant levels, with lower odds of these causes of death for each interquartile-range increase in pollutant concentration (Table 4 and Web Table 2). No associations were found for the negative control group for any of the time averaging periods (Tables 3 and 4 and Web Table 2).

All results were robust with respect to alternate approaches for adjusting for $PM_{2.5}$ background concentrations and to the exclusion of decedents residing near road segments that were elevated relative to the land surface. We also did not find an association between our main outcomes of interest and ambient concentrations of $PM_{2.5}$ at AQS monitors in the region (all nonaccidental causes: OR = 0.99 (95% CI: 0.97, 1.02); respiratory disease: OR = 0.97 (95% CI: 0.89, 1.07); cardiovascular disease: OR = 0.99 (95% CI: 0.95, 1.04); cerebrovascular disease: OR = 1.01 (95% CI: 0.89, 1.13)). Similarly, we found no evidence of interaction with time, rain, or season (Web Table 3), nor did we find evidence of interaction with comorbidity or individual-level or neighborhood-level sociodemographic characteristics (Table 5).

DISCUSSION

In this study, we incorporated highly resolved spatiotemporal traffic data into a dispersion model to investigate associations between mortality and daily and hourly exposures to primary traffic-generated air pollutant concentrations in communities near highways in the Puget Sound area. Despite the use of highly resolved exposure estimates, we found no evidence of increased odds of all nonaccidental, respiratory, cardiovascular, or cerebrovascular mortality associated with daily or hourly exposures to primary $PM_{2.5}$, NO_x, and BC generated by highway traffic during the 1, 6, 12, 24, or 48 hours before death. This study added to the literature by newly evaluating the short-term fluctuations in concentrations of primary air pollutants from highway traffic in surrounding near-road communities using finely resolved traffic data.

The primary novelty of this work was our exposure assessment approach. Instead of using regional monitoring stations or models based on aggregated traffic data (e.g., annual average daily traffic) as most studies have done, we used highly localized information on traffic congestion to predict concentrations of primary traffic-generated air pollutants in near-road populations. Our use of local fleet mix data along with fine-scale traffic activity data allowed us to generate a spatiotemporally resolved emissions inventory at half-mile and hourly resolutions, which probably reduced measurement error due to improved accuracy of modeled NO_x concentrations (29) and more accurate matching of our exposure data to the time of death (21, 36, 37). Additionally, our detailed vehicle speed data allowed us to capture the influence of complex traffic patterns—for example, stop-and-go congestion—that is neglected by more conventional, aggregated metrics of exposure.

Table 4. Association Between an Interquartile-Range Increase[a] in Levels of Traffic-Generated Fine Particulate Matter ($PM_{2.5}$) From Nearby Highways and Cause-Specific Mortality in the Puget Sound Region, by Time Window of Exposure, Washington State, 2009–2013

Cause of Death	Moving Average $PM_{2.5}$ Concentration							
	1-Hour Average		6-Hour Average		12-Hour Average		48-Hour Average	
	OR[b]	95% CI	OR[b]	95% CI	OR[b]	95% CI	OR[b]	95% CI
All nonaccidental causes	0.99	0.99, 1.00	1.00	0.98, 1.02	0.99	0.97, 1.02	0.97	0.94, 1.01
Respiratory disease	1.00	0.98, 1.02	1.03	0.96, 1.10	1.02	0.92, 1.13	0.89	0.77, 1.03
Cardiovascular disease	0.99	0.98, 1.00	0.98	0.94, 0.99	0.97	0.93, 1.01	0.94	0.88, 1.00
Cerebrovascular disease	0.96	0.92, 0.99	0.92	0.84, 1.00	0.94	0.85, 1.05	0.96	0.81, 1.14
Negative control group	0.98	0.95, 1.02	1.01	0.94, 1.08	0.97	0.88, 1.07	0.96	0.82, 1.14

Abbreviations: CI, confidence interval; OR, odds ratio; $PM_{2.5}$, particulate matter with an aerodynamic diameter less than or equal to 2.5 μm.
[a] Interquartile range: 1-hour average, 0.07–1.23 μg/m^3; 6-hour average, 0.14–1.69 μg/m^3; 12-hour average, 0.25–2.00 μg/m^3; 48-hour average, 0.52–2.10 μg/m^3.
[b] Adjusted for temperature, relative humidity, influenza, holidays, rain, background $PM_{2.5}$ concentration, and ozone concentration.

Our approach in this study resulted in different exposure estimates than we used in our earlier work (23), which used a crude measure of exposure based on the time and spatial extent of traffic congestion near a person's home. In that study, while cerebrovascular and respiratory deaths were positively associated with the number of congested minute-kilometers near a decedent's home, there was no association with overall mortality and there were inverse associations with cardiovascular mortality (23). Incorporating the influences of meteorological conditions, differences in the fleet mix across highways, and other physical factors in the dispersion of traffic-generated air pollutants resulted in low correlations between the original metric of congested minute-kilometers and our dispersion-model–based estimates. While the current analysis did not illuminate any new associations, we did find some differences, including null results where positive associations were previously observed.

More generally, this was not the first study to find limited associations between short-term exposures to ambient air pollutants and mortality in the Seattle area. Sullivan et al. (38) found no association between $PM_{2.5}$ concentrations at 3 AQS monitors in the region and primary cardiac arrest. Zhou et al. (39) found no association with all-cause, cardiovascular, or respiratory mortality during the warm season (April–September) using $PM_{2.5}$ concentrations at 1 AQS monitor in Seattle. While reasons for largely null associations in the Seattle area as compared with strong and positive associations in many other settings are unknown, they might be explained by generally low pollutant levels in this region. With low levels, there is often a greater noise-to-signal ratio, which can reduce statistical power to detect associations. Alternatively, but perhaps more implausibly, it may be that there is something characteristic of Seattle (i.e., the meteorological conditions) or its population (i.e., high fish consumption) that confers protection (40–44). In addition to overall null findings, we found associations counter to our hypotheses between cardiovascular and cerebrovascular causes of mortality and higher levels of these primary traffic-generated air pollutants measured 1 and 6 hours before death. These findings differ from most previous research, which has consistently linked short-term exposure to traffic-related air pollution and cardiovascular and cerebrovascular mortality (8, 20, 45), as well as subclinical cardiovascular outcomes (46–49). A few other investigations have found similar results, showing a slightly reduced risk of mortality associated with exposure to higher levels of specific markers of air pollution from traffic (5, 50, 51). Similarly, we did not observe any evidence of interaction with sociodemographic factors and comorbid conditions, unlike some (52) but not all (53–56) other studies.

Although we considered many confounders in our analysis and restricted our inferences to within-person variation, it is possible that our null and counterintuitive findings for cardiovascular and cerebrovascular mortality resulted from residual confounding. Our negative control group did not seem to indicate that this was a strong factor, but this would only be informative if the confounding factors were also related to injuries and poisoning. This would be reasonable for factors such as meteorological conditions and stress, but perhaps not all possible confounders such as noise. In fact, traffic noise is a plausible source of confounding, since noise decreases but air pollution levels increase at low vehicle speeds (57). This hypothesis is consistent with our findings, given that we would expect negative confounding for cardiovascular and cerebrovascular disease mortality but not for respiratory disease mortality, which has not been linked to traffic noise. While several studies have demonstrated that confounding between noise and air pollution is often not of great significance (58), data on time-varying noise levels were not available to be included in our analysis. Interestingly, cardiovascular mortality was also inversely associated with higher congested minute-kilometers in our previous work, especially among residents

Table 5. Association Between an Interquartile-Range Increase[a] in the 24-Hour Moving Average Concentration of Traffic-Generated Fine Particulate Matter ($PM_{2.5}$) From Nearby Highways and Cause-Specific Mortality in the Puget Sound Region, by Race/Ethnicity and Socioeconomic Subgroup, Washington State, 2009–2013

Subgroup	Cause of Death											
	All Nonaccidental Causes			Respiratory Disease			Cardiovascular Disease			Cerebrovascular Disease		
	OR[b]	95% CI	*P* Value	OR[b]	95% CI	*P* Value	OR[b]	95% CI	*P* Value	OR[b]	95% CI	*P* Value
Age, years			0.49			0.04			0.29			0.15
<75	0.97	0.93, 1.02		1.14	0.94, 1.38		0.92	0.83, 1,02		0.63	0.36, 1.09	
≥75	0.99	0.96, 1.03		0.89	0.77, 1.04		0.97	0.92, 1.03		0.94	0.82, 1.08	
Comorbidity[c]			0.50			0.69			0.56			0.64
Yes	1.00	0.95, 1.05		1.00	0.82, 1.23		0.99	0.89, 1.09		0.95	0.75, 1.19	
No	0.98	0.95, 1.01		0.96	0.84, 1.10		0.95	0.90, 1.01		0.88	0.74, 1.06	
Education			0.74			0.56			0.11			0.80
Technical school or at least some college	0.98	0.94, 1.02		0.99	0.84, 1.16		0.94	0.87, 1.01		0.88	0.72, 1.08	
High school diploma	1.00	0.96, 1.05		0.98	0.82, 1.16		1.03	0.95, 1.11		0.97	0.75, 1.26	
Less than high school	0.99	0.92, 1.06		0.79	0.53, 1.20		0.90	0.77, 1.05		0.88	0.64, 1.21	
Race/ethnicity			0.09			0.21			0.37			0.82
Non-Hispanic White	0.97	0.94, 1.01		1.00	0.88, 1.13		0.95	0.89, 1.01		0.91	0.78, 1.08	
Non-White	1.03	0.97, 1.08		0.83	0.61, 1.12		0.99	0.91, 1.09		0.89	0.66, 1.18	
Neighborhood disadvantage score[d]			0.39			0.39			0.51			0.65
Low	1.03	0.96, 1.11		1.18	0.87, 1.59		1.02	0.90, 1.15		0.84	0.51, 1.16	
Middle	0.98	0.94, 1.02		0.93	0.78, 1.12		0.96	0.89, 1.04		0.98	0.79, 1.21	
High	0.98	0.94, 1.02		0.96	0.82, 1.13		0.94	0.87, 1.01		0.87	0.70, 1.08	

Abbreviations: CI, confidence interval; ICD-10, *International Classification of Diseases, Tenth Revision*; OR, odds ratio; $PM_{2.5}$, particulate matter with an aerodynamic diameter less than or equal to 2.5 μm.

[a] Interquartile range: 0.42–2.09 $\mu g/m^3$.

[b] Adjusted for temperature, relative humidity, influenza, holidays, $PM_{2.5}$ background concentration, and ozone concentration.

[c] An indicator of comorbidity was created for individuals who had diabetes mellitus (ICD-10 codes E10 and E11), overweight/obesity (ICD-10 code E66), hypertension (ICD-10 codes I10–I16), or respiratory disease (ICD-10 codes J00–J99) listed as contributory (but not underlying) causes of death.

[d] A composite score of neighborhood disadvantage was created for each decedent address by combining census variables from the American Community Survey (2009–2013) representing domains of wealth and race/ethnicity for each census tract (i.e., median annual household income, log median value of occupied housing units, percentage of adults who completed college, percentage of persons in executive or managerial occupations, percentage of non-Hispanic Whites, percentage of non-Hispanic Blacks, etc.). The neighborhood disadvantage score was categorized according to tertiles as a high (−20.50 to −2.48), middle (−2.47 to 3.06), or low (3.05 to 19.20) level of disadvantage.

adjacent to a highway. Similar to noise, ozone concentrations decrease with increased NO_x concentrations, which could also have confounded our associations. However, adjustment for ozone concentrations in our models did not change the magnitude or direction of our associations, so this seems to be a less likely source of confounding.

Although we estimated exposure to primary traffic-generated air pollution with high spatiotemporal resolution, all models inherently have errors. Specifically, the performance of R-LINE is sensitive to the accuracy of the input data, such as meteorological data, traffic activity, and emissions inventories (30). While R-LINE is a well-validated dispersion model (24, 29, 59, 60), a known limitation of R-LINE is that it does not model the influence of terrain on the dispersion of air pollutants, which may be important in Seattle. Exclusion of decedents who resided near road segments that were elevated relative to the road did not change the observed associations. Nevertheless, we cannot completely conclude that failure to capture differences in elevation did not influence our findings. Similarly, there is vegetation along some segments of our 5 highways, and several studies have shown a significant reduction in levels of traffic-generated air pollutants behind certain roadside vegetation barriers (61–64). Our modeling accounted for all existing solid noise barriers, but there was no feature with which to model the effect of near-road vegetation on the dispersion of air pollutants. It may be that the presence of barriers reduces the impacts of traffic-generated exposures on health, especially among residents nearest to the highways. In all cases, we would expect that any exposure measurement error from these causes or others should have been nondifferential based on the case-crossover design, such that the result would likely have biased any true association toward the null (36, 37).

Another limitation of our study is that we did not have information on each participant's exact location before death or on the control days. Several conditions of this study, however, may strengthen our confidence in the assumption that our modeled concentrations of air pollutants may reflect relevant exposure levels before death. First, most adults in the United States spend a great majority of their time (69%) in a residence (65), and those who are near death probably spend more time at home than others. We excluded hospital and hospice deaths, for which residential address may not have accurately reflected the decedents' exposures prior to death. Although hospitalized decedents were slightly younger, with a greater proportion of males and a lower proportion of non-Hispanic Whites, given our case-crossover design, where each decedent acted as their own control, we do not anticipate that this decision would have introduced selection bias. In a sensitivity analysis that included these decedents, we found similar results. In addition, because of small sample sizes for the cause-specific outcomes, we dichotomized race/ethnicity for our interaction analysis; however, this does not explain the lack of interaction, since similar null associations were found for all nonaccidental mortality by race/ethnicity (results not shown). We inherently focused this analysis on traffic emissions from highways, where we had strong data, but excluded traffic-generated air pollution from roadways beyond the 5 selected highways. We assume that these associations are reflective of exposures for populations that may be proximate to surface roads with appreciable traffic but distant from major highways; however, it remains possible that highway vehicles have emissions of different toxicities than vehicles on other roadways because of differences in fleet composition and/or driving conditions. Finally, although we had enough statistical power (i.e., 80%) to detect a minimum OR of 1.025 for total nonaccidental mortality, our analysis was not fully powered to detect associations with our cause-specific outcomes, which might have resulted in imprecise rather than biased estimations.

In conclusion, this study found no evidence of increased odds of mortality associated with exposure to primary $PM_{2.5}$, NO_x, or BC generated by highway traffic in near-road populations. Nonetheless, this study highlights the use of a novel approach to estimate near-road exposure to primary traffic-generated air pollution in epidemiology.

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