The Environmental and Population Health Impacts of Road Urban Transportation in the Puget Sound Region, WA

by

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A dissertation submitted in partial fulfillment
of the requirements for the degree of
Doctor of Philosophy
(Epidemiological Science)
in the University of Michigan
2019

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Dedication

A mi amado esposo, amor sin tú apoyo no hubiera sido posible. Te amo infinito.

A mi padres por sus enormes sacrificios y por guiarme para a ser quién soy hoy.

Acknowledgements

I would like to express my sincere gratitude to my advisor, Sara Adar, who gave me the opportunity to come to Michigan and join her research group to become an independent and rigorous researcher. Sara's permanent support, guidance and mentorship along these years have been essential to reach my educational goals and overcome the challenges of being in a doctoral program. I am very grateful for her determined support, patience and for giving me the opportunity to explore and develop my research interests. It was an honor working with you and learning from you all these years.

I also would like to thank the other members of my committee. I thank Dr. Batterman for your continuous guidance and insightful comments to this research. I am very grateful for giving me the opportunity to work with people in your laboratory that made important contributions to this work. I also want to thank Dr. Levy from Boston University for being willing to support and guide a student outside your university. Dr. Levy's guidance and insightful recommendations strengthen this dissertation. I also would like to thank Dr. Mukherjee who is an excellent professor from whom I learnt to be rigorous in scientific research and statistical analysis.

I am also grateful to the Colombian Administrative Department of Science, Technology and Innovation (COLCIENCIAS) for granting me a scholarship to develop my doctoral program. Without COLCIENCIAS resolute interest in strengthening Colombia's research capacity I would have not been able to achieve my dream of obtaining a PhD degree. I am also thankful to COLFUTURO for managing the resources of this scholarship and providing me the administrative support along these years. I also want to thank Rackham Graduate School for

providing me several grants to fund some components of the third aim of this dissertation as well as grants for professional development.

I also would like to thank Mark Hallenbeck and his team at the University of Washington Transportation Center for providing the core dataset I used to conduct aims 1 and 2 of this dissertation. I also thank Mark for his commitment to offer me guidance and answer my multiple questions about the nature of the data and the characteristics of traffic in the Puget Sound Region. I also would like to thank the Washington State Departments of Transportation, Ecology and Health as well as the Puget Sound Clean Air Agency and the Seattle State Department of Transportation for providing the data to conduct this dissertation.

I am also grateful to Chad Milando who was open to sharing with me his knowledge and gave me important recommendations to implement a core component of the dispersion-based air quality modeling that I executed throughout this dissertation. Chad was key to implementing the dispersion model more efficiently. I also would like to thank Alex Cao, consultant at the Consulting for Statistics, Computing and Analytics Research (CSCAR). His help was key to reducing the computational time of my programs and improving the efficiency of managing my big datasets.

I also greatly appreciate the Adar Research Group team, Meredith Pedde and Jen D'Souza, who were always willing to share their knowledge with me and gave me feedback on many components of this dissertation. Your helpful advice and feedback were key to strengthening this research. I am also grateful for Josette Gauthier, who helped me a great deal with the many pieces of the third aim of this dissertation. Your help and enthusiasm were crucial to successfully finishing that aim.

I am also grateful with the people of my cohort (2013) because I learned a great deal from you in our classes together. It was an honor sharing this time of graduate school with you all.

I am very grateful for the friends that I made during these years in Ann Arbor. Natalia Blanco, Lisbeth Iglesias and Constanza Fernandez, you are my Latin-American family in the US. I love the time that we spent together having happy hours, eating delicious food, and laughing or simply talking about our concerns, sorrows, expectations and plans. My experience and life in Ann Arbor would not have been the same without you being a part of it and bringing happiness to my life. In addition, I am grateful for my Colombian friends, you have brought joy to my life. Spending time with you guys was like having a piece of Colombia and my family close to me. Thank you all for the good moments and I hope we can keep enjoying our friendship.

Finally, I am extremely grateful to my husband Leonardo. Amor sabes que sin tú ayuda alcanzar este sueño no hubiera sido posible. Tú incansable amor, comprensión y apoyo a lo largo de estos años me mantuvo en pie y me dio la fuerza para seguir adelante, aún cuando mis propias dudas e inseguridades limitaban mi avance. Gracias por tus consejos, siempre muy sabios y acertados. Gracias por tú amor e infinita ternura, pero sobre todo gracias por atreverte a sacrificar parte de tú vida para ayudarme a cumplir este sueño. Deseo que podamos seguir creciendo y avanzando juntos. A mis padres Luz Mary y Armando gracias por su apoyo e infinitos sacrificios, por enseñarnos el valor de la vida, la honestidad, el trabajo responsable y porque su guía nos ha permitido a mi hermano y a mí ser buenos seres humanos. A mi hermano Nilson gracias por ayudarme a crecer y madurar. Espero podamos ser siempre un apoyo el uno para el otro. Al resto de mi familia materna y paterna gracias por su apoyo y por hacer parte de mis recuerdos felices

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Abstract

The preference for private vehicles over public transportation and physically active modes of transportation in urban areas poses important environmental and public health challenges. On-road motor vehicles are a major source of urban air pollution such as particulate matter less than 2.5 micrometers ($PM_{2.5}$), oxides of nitrogen (NO_x), carbon monoxide (CO), volatile organic compounds (VOC) and black carbon (BC), with important impacts on population health due to increased risks of mortality and adverse cardiovascular and respiratory endpoints. A reliance on on-road motor vehicles also impacts population health through noise, greenhouse gas emissions and reduced physical activity. Thus, there is a need for further epidemiologic research to more accurately characterize the links between on-road urban transportation and public health and to evaluate the public health benefits of transportation policies to support decision-making.

To this end, this dissertation proposed an integrated approach. First, we characterized the influence of highway traffic emissions on primary $PM_{2.5}$ and NO_x concentrations generated by highway traffic in communities near highways using a dispersion model with fine-scale spatiotemporal variations of traffic volume and flow and compared these results to those of a model with more aggregated traffic data. Second, we evaluated the association between primary $PM_{2.5}$, NO_x and BC generated by highway traffic and daily mortality in near-road communities using a time-stratified case-crossover design. Third, we conducted a health impact assessment to quantify the air pollution and health benefits of urban transportation policies promoting electric vehicle use and replacement of short car trips with walking and bicycling.

In the first aim, we found that spatiotemporal variations between and within highways in

traffic volume and flow are complex and not completely captured by traditional aggregated traffic metrics. For both sets of data, we observed highly variable concentrations over space and time of primary $PM_{2.5}$ and NO_x generated by highway traffic in near-road communities. While modeled concentrations of these pollutants from the fine-scale data had largely similar spatial and temporal distributions as the aggregated data, we observed some areas with larger differences between the two sources of traffic data, especially in communities closest to highly congested highways. In our second aim, we found no conclusive evidence of increased mortality with higher daily concentrations of $PM_{2.5}$, NO_x and BC generated by highway traffic. However, there was suggestive evidence that greater short-term exposures to these air pollutants were associated with greater odds of respiratory mortality. Contrary to our hypothesis, we found reduced odds of cardiovascular and cerebrovascular mortality with higher levels of these air pollutants. For both outcomes, observed associations were stronger among those living closest to the highways. In the third aim, we found that transportation policy scenarios promoting cleaner vehicles and replacing car trips with walking and bicycling reduce NO_x and $PM_{2.5}$ concentrations as well as CO_2 emissions as compared with a business as usual scenario. These policy interventions also reduce burden of mortality with fewer premature deaths in adults of the general population.

From an exposure assessment and environmental epidemiology standpoint, this dissertation provides evidence that improvements are needed over standard exposure assessment approaches when characterizing near-road exposures to air pollution. Furthermore, from a decision-making perspective, this dissertation provides evidence that transportation policies may mitigate the population health burdens of motorized transportation.

Chapter 1

Introduction

Background

Urban Transportation is a Major Source of Air Pollution in Communities

Around the world, the number of motor vehicles is steadily increasing with population growth, increased urbanization, economic improvement and rapid urban sprawl. According to the Special Report by the Health Effect Institute, (1) the worldwide fleet exceeded 1 billion vehicles in 2002 and it has been predicted that the number of motor vehicles will approach 50 million per year by 2020. The predominance of private vehicles as our primary mode of transportation over public transportation and physically active modes of transportation poses important challenges to public health through a variety of mechanisms including physical inactivity and traffic injuries as well as environmental exposures to traffic-related pollutants and noise. (2)

On-road traffic has been recognized as a major source of air pollution and is an increasingly important contributor to primary pollutants such as CO, NOx, VOC, BC and $PM_{2.5}$. (1) The contribution of traffic to these pollutants is greater in major metropolitan areas, where it accounts for 47%, 33%, 20% and 12% of total CO, NOx, VOC and $PM_{2.5}$ emissions in the U.S, respectively. (1) In addition, on-road traffic is also a major contributor to Green House Gases (GHG), approximately 28% of all emissions in the United States (3) and 23% worldwide. (1,2).

In recent years, concerns regarding near-road exposure to poor air quality has increased since there are substantially higher concentrations of particles and other toxic pollutants within

hundreds of meters of major roads than is found at greater distances. (4) This has important implications given the high proportion of the population worldwide and in the United States that live, attend school or work near major roadways. According to data from the American Housing Survey, more than 11 million people in United States live within 150 meters of a major highway and up to 45% of people live within 300 meters from major highways in large urban areas, resulting in an increased risk of exposure to vehicle emissions and greater risk of health effects. (5,6) Moreover, environmental justice issues have been raised since studies in the United States have documented that minority racial and ethnic groups and low socioeconomic position population are more likely than white and high income populations to live near major roadways. (7–13)

Links Between Traffic-Related Air Pollution and Health

A growing body of scientific evidence has shown that both long (1,14–20) and short-term (21–24) exposures to traffic-related air pollutants are associated with adverse health. Documented health endpoints associated with traffic-related air pollutants include respiratory outcomes such as nonmalignant respiratory mortality, (17,22,24,25) diagnoses of asthma and respiratory symptoms, (26–33) lung function impairment, (34,35) and chronic obstructive pulmonary disease (COPD) (36,37) as well as adverse cardiovascular outcomes such as coronary heart disease, (CHD) (38) cardiovascular mortality, (15–20,39) higher left or right ventricular mass (40,41), acute myocardial infarction (42) and progression of subclinical atherosclerosis. (38,43–45) These associations are hypothesized to occur via pulmonary and systemic oxidative stress and inflammation; autonomic nervous system imbalance; vascular changes and translocation of particles and their constituents into the circulation.

Epidemiologic studies looking at the specific role of short-term exposures to on-road traffic emissions suggest that traffic exposure is an important trigger for clinical and subclinical disease. For example, acute non-fatal myocardial infarction have been linked with time spent in traffic, with higher population attributable fraction than conventional triggers such as physical exertion, alcohol and coffee. (46) Several small in-vehicle panel studies suggest that exposures can have very rapid effects on the human body. In one of these studies, Adar and colleagues found that changes in 5-minutes $PM_{2.5}$ concentrations aboard a diesel bus were independently and statistically significant associated with decreased heart rate variability in nonsmoking seniors. (47) Similarly, studies measuring the health effects of commuting in private vehicles or public transportation found associations between traffic-related $PM_{2.5}$, black carbon (BC) and time spent commuting with reduced heart rate variability (HRV), increased levels of markers of systematic and pulmonary inflammation (48,49) and increased risks of myocardial infarction. (50) Collectively, these findings suggest that traffic emissions may influence biological pathways that act at very short time scales yet large-scale population studies have generally lacked detailed exposure data with which to capture such short-term changes.

Epidemiological and environmental justice studies also suggest that population exposures to traffic-generate air pollution and health outcomes are differentially distributed in the population. It has been recognized that socially disadvantaged population and minority ethnicity are more exposed to traffic-related pollution. Studies have further suggested that they may also be more susceptible to the health effects of this exposure. Both conditions might contribute to the race, ethnicity and socioeconomic disparities in premature mortality yet more research is needed to better understand these issues. (9)

Challenges to the Study of Traffic-Related Air Pollution

Despite a large literature suggesting an association between exposure to traffic-related air pollutants and health outcomes, there are some limitations to our current understanding of these associations. Many of these limitations result from challenges in accurately capturing the full spatial and temporal variability of individual-level exposures to traffic-generated pollutants.

One widely used approach is to investigate individual pollutants as tracers or markers of traffic (e.g., NO_x, NO_2, BC). These markers are not entirely unique to on-road traffic emissions, however, and are often correlated with pollutants from other sources over time due to the effect of meteorology. In addition, measurements of these indicator species are most often collected at fixed-site air quality monitors (AQS). This fails to account for any small-scale variations in traffic pollution because the small number of monitoring locations is typically insufficient to capture local spatial or spatiotemporal patterns.

To overcome the issue of insufficient monitors, proximity to major roads has been frequently used as an indicator of long-term exposures to traffic. Inherent to this approach is an assumption that roadways of a certain classification all have the same emission patterns and impacts on the community. Additionally, it is assumed that the same roadway has the same traffic at all locations. Investigators typically fail to capture any local differences between roadways or within a given roadway due to small-scale variations in vehicle fleet and traffic volume that impact emissions. Similarly, the meteorological factors and topography that influence the dispersion of pollutants from roadways are also often ignored. Instead it is assumed that emission is the same at all locations and dispersion is the same in all directions. (51,52) As such, this approach may result in important exposure measurement error. (1,48,51,53)

A more sophisticated approach as compared with those described above is the use of

spatial prediction models, including land use regression (LUR) and other interpolation models (universal kriging, inverse distance weighting, etc). (54–56) These approaches have been widely used in long-term studies and they account well for the small-scale spatial variation in traffic-related air pollution. However, LUR and interpolation models also have some limitations. First, they do not typically incorporate temporal variations in traffic pollution useful for studying short-term health effects. Second, these models are empirical so their ability to accurately capture high local variations in traffic-related air pollution concentrations (at short distances such as tens of meters) is based on the richness of the underlying monitoring campaigns and precision of the geographic data used to support the predictions. Finally, since these models are designed to predict the total concentration of a pollutant, the output will only be source-specific if there are no other sources of the predicted components. (54)

Dispersion-based air quality models combine emissions data and chemical and physical processes in the atmosphere to predict concentration of pollutants from specific sources. These models allow for the estimation of traffic-generated air pollution with a high spatial resolution at locations within hundreds of meters where the concentration gradient is the steepest. In addition, pollutants may be estimated with high temporal resolution such as hourly, daily, weekly as well as long-term. However, to accomplish predictions with high temporal and spatial resolution, input data with high spatial and temporal resolution is required but often not available. (52,57)

In summary, gaps remain in our existing characterization of the exposure and health implications of on-road traffic emissions. Given that most epidemiological studies have been unable to account for the small-scale spatial and temporal variability of traffic-generated air pollution, exposure measurement error may be a problem for existing estimates of the health impacts of traffic.

Policies to Reduce Motorized Transportation

In spite of the outstanding challenges to exposure assessment, there is an increased understanding that traffic-related air pollution likely impacts health. Thus, there is a growing interest in transportation policies that promote cleaner vehicles and alternatives to private motor vehicles. Greater use of active transportation could translate to substantial environmental and health benefits given that almost 28% and 41% of all car trips in the United States are less than 1 mile (1.6 km) and 2 miles (3.2 km), respectively. These are distances that can be readily walked or bicycled by healthy individuals. (58)

Health impact assessment (HIA) is a method to estimate the potential health consequences of non-health care interventions as a means to help decision-makers to understand the health implications of policies. (59,60) Most quantitative HIAs of the potential health impacts of transportation policies have found potential health benefits from policies to promote active transportation and reduce car trips, (58,67–69) but more research is needed to fully understand the impact of transportation policies on a range of different contexts and policy scenarios. Furthermore, most studies are needed to examine the impact of transportation scenarios on pollutants other than just the concentrations of $PM_{2.5}$ and its consequent health effects since motor vehicle emissions contribute in a higher extent to concentrations of incomplete fuel combustion pollutants such as NO_x. (60) Uncertainties also remain in the differences on the health impacts across groups in the population such age groups (younger and older adults) and sex. Thus, more scientific evidence is needed to guide the design of urban transportation policies to obtain the greatest public health benefits.

Dissertation Aims

Taking into consideration the literature above, this dissertation proposes to add the current knowledge by leveraging a unique source of traffic data that is highly spatiotemporal resolved (every 5 minutes each ½ mile) to characterize the influence of highway traffic emissions on the health and air pollution exposures to near-road communities under current and possible future conditions. Specifically, we have the following three specific aims:

Specific Aim 1

To characterize the influence of highway traffic emissions on concentrations of primary NO_x and $PM_{2.5}$ in communities near highways using a dispersion model with fine-scale spatiotemporal variations of traffic volume and flow, and compare these concentrations with those generated using more aggregated traffic data.

Specific Aim 2

To investigate associations between exposure to primary $PM_{2.5}$, NO_x and black carbon generated by highway traffic and daily all non-accidental, respiratory cardiovascular and cerebrovascular mortality in near-road populations using an air quality model with highly-resolved traffic data.

> **Hypothesis 2a:** Short-term elevations in primary $PM_{2.5}$, NO_x and black carbon generated by highway traffic are associated with higher risk of all non-accidental, respiratory, cardiovascular and cerebrovascular mortality.
>
> **Hypothesis 2b:** Racial minorities, populations of low socioeconomic status and populations with comorbidities have a greater risk of all non-accidental, respiratory cardiovascular and cerebrovascular mortality associated with short-term variations of primary air pollutants generated by highway traffic as compared to non-Hispanic white,

populations of high socioeconomic status, and populations without comorbidities.

Specific Aim 3

To quantify the air pollution and health benefits of urban transportation policies promoting electric vehicle use and replacement of short car trips with walking and bicycling in Seattle, Washington.

Hypothesis 3a: Transportation scenarios that promote electric vehicle use and increase walking and bicycling will reduce community levels of traffic-generated NO_x and $PM_{2.5}$ as compared with a business as usual scenario.

Hypothesis 3b: Transportation scenarios that promote electric vehicle use and increase walking and bicycling will reduce the population burden of mortality as compared with a business as usual scenario.

The following chapters describe the methods and results for each dissertation aim. Chapter 2 characterizes the influence of highway traffic emissions on concentrations of primary NO_x and $PM_{2.5}$ in communities near highways using a dispersion model with fine-scale spatiotemporal variations of traffic volume and flow, as compared with more aggregated traffic data (Aim 1). Chapter 3 investigates the association between exposures to primary $PM_{2.5}$, NO_x and black carbon generated by highway traffic and the risk of daily mortality in near-road populations. (Aim 2) Chapter 4 quantifies the air pollution and health benefits of urban transportation policies promoting electric vehicle use and replacement of short car trips with walking and bicycling (Aim 3). Chapter 5 presents a summary of the overall findings of this dissertation and discusses the implications of this work. I also highlight the strengths and limitations of this dissertation in Chapter 5 and discuss directions for future research.

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Chapter 2

Fine-Scale Spatiotemporal Variations in Traffic Volume and Flow and their Influence on Air Pollution Levels in Communities Near Highways

Introduction

Exposure to traffic-related air pollution remains a public health concern due to associations with increased risks of mortality and adverse cardiovascular and respiratory endpoints. (HEI, 2010) Concerns are especially great for communities living near to major highways since there are substantially higher concentrations of pollutants near roads (within hundreds of meters) than at greater distances. (1,2) This has important implications given that almost 45% of the United States population live within 300 meters from major highways in large urban areas. (2,3) Near-road populations also include larger proportions of low income households and minority ethnic groups. (3–9)

Accurately estimating exposures to traffic-related air pollutants is challenging due to the multiple factors determining both vehicle emissions and dispersion. (10–12) One of these factors is the variation of traffic by time and place, within and between highways. These variations likely influence the dispersion of traffic-related pollutants in nearby communities yet they have generally not been well studied and are typically not considered in epidemiologic studies due to the lack of spatiotemporally resolved traffic data. In fact, most epidemiologic studies of the health effects of short-term exposure to traffic-related air pollution have used the variation in measurements from air quality monitoring stations (AQS) to inform fluctuations in exposure to traffic-related air pollution. (13–17) However, existing monitoring networks are typically too

scarce and spatially disperse to capture any variation of traffic-related air pollution in the near-road environment.

Physically-based air quality models such as dispersion models have been recently considered as methods to overcome the challenges of assessing population exposures to traffic-related air pollutants. (18–22) These models, such as the Research Line source dispersion model (RLINE), can predict air pollutants from traffic over a large area with high spatiotemporal resolution by combining emissions from on-road traffic volumes and flow with physical processes in the atmosphere due to meteorology. In fact, this approach has been recently used in environmental health studies to estimate exposure to traffic-generated air pollution in near road environments. (18–20,23–26) However, due to a paucity of spatiotemporally resolved traffic data and higher computational demands, researchers who have employed dispersion models in the past have generally used traffic data aggregated over time and space as inputs to these models. For example, they have used annual average daily traffic (AADT) estimated at a few permanent traffic recorders (PTR) on roadways throughout a study area along with temporal allocation factors (TAF) and national vehicle speeds to estimate traffic activity. (18,19,23,24,27–29) Yet, these aggregated traffic metrics might not completely capture differences in traffic emissions due to localized differences in traffic patterns by time and place, within and between highways.

In this study, we aimed to characterize the concentrations of oxides of nitrogen (NO_x) and fine particulate matter ($PM_{2.5}$) generated as primary pollutants by highway traffic (herein referred to as traffic-generated NO_x and $PM_{2.5}$) in communities near the most trafficked highways in the Central Puget Sound Region of Washington State using a line-source dispersion model. We further aimed to compare the concentrations predicted using a unique source of traffic data with fine-scale spatiotemporal variations of traffic volume and flow as compared with more

traditionally available traffic data aggregated over space and time. We conducted this research in the urban area of the Puget Sound in Washington State because it is one of the few regions in the United States that has highly spatiotemporally-resolved traffic data.

Methods

Figure 2.1 outlines our basic approach for predicting concentrations of traffic generated NO_x and $PM_{2.5}$ from highways in near-road communities in the Puget Sound (Figure 2.1). Briefly, we leveraged two sources of traffic activity data for the main highways in the Puget Sound (i.e., Interstates 5, 405 and 90, and State Routes 167 and 520) during the year of 2013: one with high spatiotemporally resolved traffic volume and vehicle speed data and a second with aggregated traffic volume and vehicle speed (e.g., AADT, TAF, and national vehicle speeds). We incorporated these data with the Motor Vehicle Emissions Simulator (MOVES, version 2014) (30) to generate hourly emission factors (grams/vehicle-mile) of NO_x and $PM_{2.5}$ from the Puget Sound highways. We then used the Research Line source dispersion model (RLINE v1.2) (20) to predict hourly concentrations of traffic-generated NO_x and $PM_{2.5}$ at 3,784 population-based receptors within 1 km from a highway. Finally, we compared the spatial and temporal distribution of the traffic inputs and predicted concentrations between the two data sources. Below, we describe in more detail the three major components of this modeling framework.

Receptor Locations:

In preparation for our related epidemiology study, we estimated concentrations of traffic-generated NO_x and $PM_{2.5}$ at the residential locations of all non-accidental mortalities that occurred within 1 kilometer of our targeted highways between 2009 and 2013. We also

generated concentration estimates at one and five regulatory monitoring stations in the Air Quality System (AQS) in the Central Puget Sound that sampled for reactive oxides of nitrogen (NO_y) and $PM_{2.5}$, respectively. Figure 2.2 displays the locations of all of our receptors.

Traffic Data:

We obtained fine-scale spatiotemporally resolved traffic volume and vehicle speeds available for each half-mile and every 5-minutes for each direction (i.e., North, South, East or West bounds) of the highways in the region from the Washington State Transportation Center at the University of Washington (TRAC-UW). These five highways are classified as National Functional Class (NFC) 11 (Urban Interstate) and 12 (Urban Other Freeway or State Routes). Induction loop detectors embedded in the pavement of these highways measure real-time traffic volume and vehicle speeds. The data undergo routine quality assurance screening by the TRAC-UW to ensure high data quality.

For the aggregated traffic data, we obtained vehicle volumes from the Washington State Department of Transportation (WSDOT) as AADTs for each direction of the highways and collected at 20 PTRs distributed along the five highways (i.e., I-5: six, I-405: five, I-90: three, SR-167: three and SR-520: four). We obtained local TAFs estimated by month, day of week (weekday and weekend) and hour of day from the Washington State Department of Ecology. Due to the absence of aggregated local data for vehicle speeds, we obtained these data from the National Speed Survey for five time periods of the day (i.e., Off-peak-1 12:00 am-6:59 am; morning peak 7:00 am-8:59 am; mid-day 9:00 am-3:59 pm; afternoon peak 4:00 pm-6:59 pm and off-peak-2 7:00 pm-11:59 pm) (31)

For both sources of traffic data, we used fractions of vehicle class from the WSDOT collected at the PTRs. We mapped these vehicle classes to the six grouped Highway Performance Monitoring System classes (i.e., motorcycles, passenger cars, light-duty trucks, buses, single-unit trucks, multi-trailer trucks). (32) For the fine-scale traffic data, we calculated hourly traffic volume by vehicle class for each half-mile road segment and direction for all 8,670 hours of 2013 by weighting our spatiotemporal traffic volumes by the vehicle class fractions. For the aggregated data, we estimated hourly traffic volume by vehicle class using the AADT and monthly, day-of-week and hour-of-day TAFs, and fleet mix for each roadway link between PTRs, both directions, and hours of 2013.

We used data from the Tiger products of the US Census Bureau for the road network of our five targeted highways.

On-Road Mobile Vehicle Emissions:

For both fine-scale and aggregated traffic data, we generated hourly-resolved vehicle emissions factors (grams/vehicle-mile) of NO_x and $PM_{2.5}$ for each road segment using MOVES-2014. (30) In addition to the traffic information described above, we obtained MOVES input data for 2013 including vehicle class and age distributions as well as the fuel formulation for the main county in the Central Puget Sound Region (i.e., King county). Additional MOVES inputs included monthly average local temperature and relative humidity, which we obtained from the Renton airport station operated by the National Oceanic and Atmospheric Administration. (NOAA: ftp://ftp.ncdc.noaa.gov/pub/data/noaa)

Following the approach proposed by Cook et al. (33) and Snyder et al. (19) we ran MOVES at the county scale using rate-per-distance calculations of emission rates with hourly

aggregation. The emission rates for NO_x and $PM_{2.5}$ were generated for the running exhaust and evaporative emission processes for the unique combinations of 6 vehicle classes (MOVES vehicle classes were mapped to the six grouped HPMS classes), 16 vehicle speeds bins (ranging from 2.5 to 75+ mph), local ambient temperature and relative humidity (every 5 °F), and month. Since non-running emissions during extended idling are not related with highway driving we did not include this emission process in our calculations.(19,33) We did, however, include non-exhaust emissions of $PM_{2.5}$ such as tire wear and brake wear in our models.

To obtain emissions by pollutant, road segment, highway direction, and hour of the year, we multiplied the hourly emission rates from MOVES for each pollutant by vehicle class, speed bin, month, and temperature with the hourly traffic volume by vehicle class, road segment, and direction. We performed these calculations for each of the two sources of traffic data described above, resulting in hourly, segment-by-segment emission factors for NO_x and $PM_{2.5}$ for the spatiotemporal and aggregated traffic datasets that accounted for local weather and traffic activity in each road segment and direction.

Dispersion Modeling:

We ran a modified version of the RLINE dispersion model (v1.2) to predict hourly concentrations of primary NO_x and $PM_{2.5}$ originating from vehicle emissions on the major highways at our 3,748 receptor locations. RLINE simulates primary, chemically inert pollutants for near-road dispersion without accounting for chemical transformations or deposition. (20) We implemented RLINE using the numerical integration method, an iteration limit of 1,000, an error limit of 0.001 and the beta algorithms for roadside noise barriers. To avoid running RLINE for each air pollutant, we used the unit emission rate (1 g/m/s) input approach (19) and then scaled

the hourly RLINE outputs for the pollutant-specific emission factors from MOVES at each road segment.

We obtained the hourly meteorological parameters needed for dispersion modeling from the Renton airport station. These data were processed through the AERMET program by the Puget Sound Clean Air Agency. AERMET estimates boundary layer parameters required for RLINE to model dispersion such as friction velocity (u*), convective velocity (w*), surface roughness height (z_0), Monin-Obukhov length (L), moisture, albedo, cloud cover, and temperature. We selected the Renton airport as our primary source of data based on its location in our study region and because comparisons of wind profiles measured at other meteorological stations in the region showed similar behavior as that found at the Renton airport.

Concentration Estimation:

We aggregated the hourly-modeled concentrations of NO_x and $PM_{2.5}$ contributed by every road segment for each receptor location to obtain 24-hour and annual average concentrations.

Data Analysis:

We characterized the spatial and temporal distribution of traffic volumes and speeds as well as predicted concentrations of NO_x and $PM_{2.5}$ from highway traffic for the two sources of traffic data using descriptive statistics, Spearman correlation coefficients, box plots, cumulative density function graphics, and heat maps. We also compared the daily modeled concentration of NO_x and $PM_{2.5}$ with the observed concentrations at AQS monitoring sites. We made comparisons across the data sources for the entire period of study and by weekdays/weekends and distance to roadway. We also split the variation into a spatial component by evaluating only the annual

average concentrations at different locations and a temporal component by focusing on differences in daily concentrations from an annual average. All analyses we conducted using Stata statistical software version 14.1 (Stata Corp) and ArcGIS version 10.1 (ESRI).

Results

Variation in Traffic Volume and Speed on Major Highways

The section of the highways included in this study corresponds to 250 miles and 500 half-mile road segments with 8,760 hours of data (Figure 2.2). We found some differences in the daily average traffic volumes between the two sources of traffic data with higher traffic volumes in the fine-scale traffic data as compared with the aggregated data for all highways except for I-405. Volumes were also substantially more variable (1.5-3.5 times) for the fine-scale data than the aggregated data. (Table 2.1) For both sources of data, traffic volumes showed 20% higher annual average daily traffic volumes for weekdays as compared with weekends (Table 2.1) and 60% higher hourly average traffic volumes during morning and afternoon rush hours as compared to off-peak periods (Supplemental Table S2.1).

As shown in the heat maps of Figure 2.3, traffic volumes differed by highway in both sources of data. Variations between-highways were evident even among roads classified within the same functional classification (i.e., Interstates or State Routes). For example, in the fine-scale data, I-5 had 40% higher weekday traffic volumes than I-405 and I-90 while, SR-167 had 16% higher volumes than SR-520. (Table 2.1) Similarly, the boxplots of hourly variation for each road segment along I-5 (Figure 2.3) show that volumes also varied by location within a highway. As expected, within-highway variations were more evident for the fine-scale traffic data with volumes every half-mile than for the aggregated volumes that are limited to the PTRs available on each highway (e.g., six PTRs on I-5). Coefficients of variation of daily traffic volumes across

road segments within a highway ranged from 22 to 32% for the fine-scale data and were slightly higher than the aggregated traffic data, which ranged from 12 to 27%. The fine-scale data captured more highly congested (> 300,000 daily vehicles) half-mile road segments, especially near the highly populated area of downtown Seattle. (Figure 2.3 and 2.4)

Figure 2.3 also illustrates that vehicle speeds were also highly variable over time and space for the fine-scale traffic data. Temporal patterns of vehicle speed for the fine-scale traffic data showed the expected pattern of stop-and-go congestion during morning and afternoon peak hours. (Supplemental Figure S2.1) In addition, consistent with volume, speed was especially impacted in the half-mile road segments near the Seattle downtown area (mile posts 162-176 in Figure 2.5) as well as near the Seatac Airport in Renton south of the I-5/I-405 junction. For the aggregated traffic data there was no spatial or temporal variation in speed. The average speed for the two scenarios were similar, however, with the aggregated data at 70 mph for both weekdays and weekends and the fine-scale data at 63 mph and 68 mph for weekdays and weekends, respectively.

Comparison of Modeled Traffic-Generated NO_x and $PM_{2.5}$ in Near-Road Communities and Measurements

Table 2.2 compares modeled 24-hour average concentrations of traffic-generated NO_x and $PM_{2.5}$ using the two sources of traffic data to the observed NO_y and $PM_{2.5}$ concentrations at the AQS monitoring sites. The modeled 24-hour average concentrations of traffic-generated NO_x derived using both the fine-scale data and aggregated data were slightly higher than the observed concentrations of NO_y. Modeled concentrations of traffic-generated $PM_{2.5}$ obtained using the fine-scale data (range: 0.8 to 2.9 $\mu g/m^3$) and aggregated data (range: 0.7 to 2.1 $\mu g/m^3$) were

approximately 10 to 40% of concentrations of measured $PM_{2.5}$, which represents contributions from both traffic and non-traffic sources (range: 4.9±2.9 to 9.5±5.7 μg/m^3). Concentrations of traffic-generated $PM_{2.5}$ predicted from RLINE were similar to the BC concentrations at the AQS monitors (observed: 0.96±0.8 and 1.2±0.9 μg/m^3, modeled aggregate data: 0.7±1.0 and 1.2±1.3, modeled fine-scale 0.8±1.2 and 1.5±1.7), which is an indicator of particulate matter from traffic in the region. Even though concentrations of air pollutants measured at the AQS are not a perfect validation of our modeled contributions from highway traffic to concentrations of NO_x and $PM_{2.5}$, our modeled concentrations showed moderate (R_{sp} from 0.5 to 0.6) and low (R_{sp}: 0.3) to moderate (R_{sp}: 0.5) correlations with observed concentrations at the AQS, respectively.

Spatial Variation of Traffic-Generated NO_x and $PM_{2.5}$ in Near-Road Communities

At our receptor locations, mean modeled concentrations of traffic-generated NO_x and $PM_{2.5}$ were slightly higher for the fine-scale traffic data than the aggregated traffic data (Table 2.2) As shown in Figure 2.6 most (90%) of the receptor locations within 1,000 meters had concentrations of NO_x and $PM_{2.5}$ below 50 ppb and 2.5 μg/m^3, respectively. Both models showed higher mean concentrations for receptors closer to highways, with 90% of receptors within 150 meters showing concentrations below 75 ppb for NO_x and 5 μg/m^3 for $PM_{2.5}$. (Figure 2.6) For both input datasets, the highest concentrations were found for receptors within 50 m with significant reductions (i.e., ≥ 50%) in the concentrations after 300 meters. (Supplemental Figure S2.2)

Our modeled NO_x and $PM_{2.5}$ concentrations showed generally similar spatial distributions at the receptor locations on the annual average scale and tended to be greater for areas with more traffic and downwind of the highways for both sources of traffic input data (Figure 2.7). In fact, we observed high overall correlations between the modeled concentrations of NO_x and $PM_{2.5}$

obtained by each source of traffic input data at the receptor locations (R_{sp} NO_x: 0.96 and $PM_{2.5}$: 0.96). (Table 2.2) However, there were some areas showing larger differences between the two sources of data. We found that 25% of our receptors had differences in concentrations between the two models that were larger than 5 ppb for NO_x and 0.4 μg/m^3 for $PM_{2.5}$ (Figure 2.8). These differences were greater among receptors closer to the highways (i.e., < 150 m), with 25% of these near-road receptors having differences larger than 10 ppb and 0.8 μg/m^3 for NO_x and $PM_{2.5}$, respectively.

Temporal Variation of Traffic-Generated NO_x and $PM_{2.5}$ in Near-Road Communities

We observed important spatial patterns in the temporal variability in NO_x and $PM_{2.5}$ concentrations predicted using both sources of traffic input data. Figure 2.9 shows evidence of larger between-day standard deviations (SD) of NO_x and $PM_{2.5}$ for receptors within 150 m to highways and near to road segments with high traffic variation as compared to receptors further from highways or near lower trafficked road segments. These same areas also showed the greatest differences in estimated temporal variability between the two models with 25% of receptors within 150 m with differences on the between-day SD greater than 6 ppb of NO_x and 0.7 μg/m³ for $PM_{2.5}$. (Figure 2.8) Both sources of traffic data capture well the temporal variability in concentrations.

Discussion

In this study, we characterized the influence of highway traffic emissions on traffic-generated NO_x and $PM_{2.5}$ concentrations in near-road communities using dispersion modeling and two sources of traffic data. A key finding of our work is that traffic volume and speed, and thus

traffic-generated air pollution concentrations in near road communities, vary significantly over across space and time. These variations in traffic volume, speed, and concentrations of traffic-generated air pollution differ between different highways as well as within individual highways. Although traditional traffic input data that aggregates some of this information over space and time captures much of this variability, fine-scale spatiotemporally resolved traffic data add to both temporal and spatial variability of air pollutants concentrations, especially in communities near highly congested highways.

Our results showed that the variability in traffic patterns between and within highways was as much as 40% and 30% the mean levels of traffic, respectively, with some highways showing 40% greater traffic volume than highways within the same functional classification. In addition, traffic volumes in the fine-scale traffic data were between 1.5 and 3.5 times more variable than the aggregated traffic data. These results are consistent with a previous research showing that indicators of traffic flow differed between roadway types and showed different patterns between different sections of the same highway. (34,35) Some of the underlying factors that may explain differences in traffic between and within highways are the number of lanes and road intersections as well as population density and accessibility to employment centers. (36)

This work adds to the literature by modeling traffic-generated air pollutants using spatiotemporally fine-scale detailed traffic data, which has been uncommon in the environmental health science literature to date due to the lack of available detailed traffic information. This allowed us to assess how predicted traffic-generated NO_x and $PM_{2.5}$ concentrations in near-road communities depend on the traffic input data source. Our results suggest that even though both the aggregated and fine-scale input traffic data produced similar spatial and temporal variability in concentrations of traffic-generated NO_x and $PM_{2.5}$ across receptors, there are often large

spatial and temporal differences between the two sources of traffic data. Specifically, 25% of our receptors had differences in concentrations between the two sets of data (i.e., greater concentrations for the fine-scale data than the aggregated traffic data) greater than 5 ppb for NO_x and 0.4 $\mu g/m^3$ for $PM_{2.5}$. Within 150 m from the highways these differences were twice as high with 25% of receptors with differences greater than 10 ppb and 0.8 $\mu g/m^3$ for NO_x and $PM_{2.5}$, respectively. Receptors nearest highways also showed greater temporal differences in concentration between the two sources of traffic data (between-day SD: NO_x >6 ppb and $PM_{2.5}$ > 0.7 $\mu g/m^3$).

Unsurprisingly, we found most of the differences in concentrations of both air pollutants between the two models in regions where there were highly variable vehicle density and stop-and-go conditions during rush hours. Regions with highly variable traffic flow, such as near the highly-populated area of downtown Seattle, are presumably better captured by the fine-scale traffic data that capture variations over very short distances. In addition, we observed greater differences between the models downwind from roadways where RLINE is known to better predict concentrations. (37) Finally, the availability of variable vehicle speeds with our fine-scale data allowed us to capture the influence on $PM_{2.5}$ concentrations of non-exhaust emissions such as brake and tire wear emissions characteristic of stop-and-go traffic conditions. This differs from the national speed data, which showed no temporal variation. (2,38,39) Although variations in traffic speed data allowed us to model non-exhaust emissions, the estimation of brake and tire wear emissions is highly complex due to the multiple factors influencing them (e.g., break and tire materials, break pad size, mass and temperatures, vehicle load, road conditions, driving behavior, etc.). Despite the fact that most of these factors are considered in MOVES, our modeled non-exhaust emissions might be still underestimated. Thus, we anticipate that the

differences found between the two sources of data due to stop-and-go traffic conditions, especially on $PM_{2.5}$ concentrations could be larger than those found in this study

Most previous studies using RLINE to model ambient air pollutant concentrations from on-road vehicle emissions have relied on traffic volume aggregated over space (i.e., AADT at a limited number of PTRs along a road) with federal or local TAF to account for the temporal variability of traffic volume across days and hours within a day as well as vehicle speeds derived from federal data or transportation demand models (TDM). (18,19,23,24,28,29) Those studies, like this one, showed the ability of RLINE to capture the spatial and temporal variability of air pollutants due to traffic emissions in the near-road environment. (23–25) Previous studies have argued, however, that aggregated traffic data may capture well the spatial variability of traffic activity and thus its influence on vehicle emissions and dispersion of traffic-generated air pollutants in communities near roadways. (19,24,25) However, our results suggest that this may not always be the case, especially among receptors living very close to roadways. Our results further show that the inclusion of local data may improve predictions of concentrations of traffic-generated air pollutants in near road communities. This is supported by work by Batterman and colleagues who have shown that incorporating local TAF in air quality dispersion modeling explains almost all variation in traffic activity observed in an urban setting in the Detroit, Michigan metropolitan area. (12)

Importantly, the incorporation of finer detailed traffic data did not significantly increase the computational demands in our air quality modeling. For the same number of receptors, there was only a 17% increase in the time of RLINE implementation and post-processing of modeled concentrations with the fine-scale traffic data. This suggests that the inclusion of detailed spatiotemporal traffic data, when available, may have benefits for both regulatory purposes and

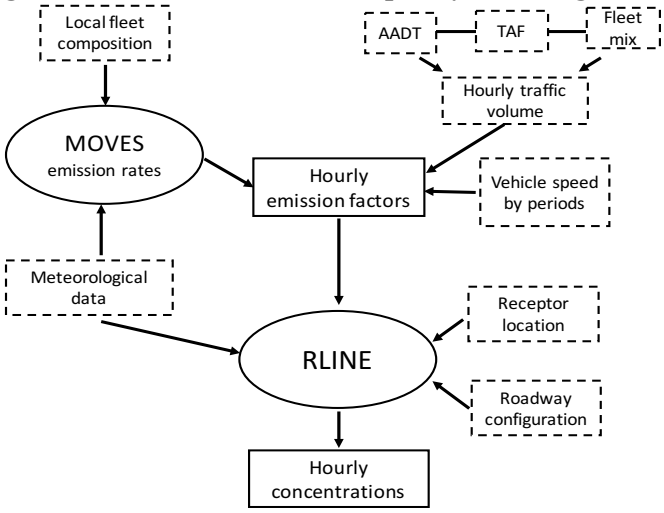
epidemiologic studies. This may be especially true for areas with high variations in traffic patterns. From a regulatory standpoint, state implementation plans could identify areas where incorporating additional PTRs would allow to further capture high variations in traffic pattern to contribute and inform traffic regulations. For exposure assessment in epidemiologic studies of traffic-related air pollution, it may be that using more detailed traffic data will increase the variability of exposures and thus the power to detect associations. They may also reduce bias due to exposure measurement error. (40,41) While we did not quantify the impacts of the differences in exposure on health estimates in this paper, future analyses are underway using these data and a simulation study to assess the added benefit to health models estimates in terms of power and bias.

In spite of the strengths of our approach, there are some limitations of this study. First, we focused this work on highway traffic and its contribution to concentrations of primary traffic-generated NO_x and $PM_{2.5}$ in nearby communities. The trade-off of using finer resolved traffic data to capture greater spatiotemporal variability is that this is only plausible on a subset of the true roadway system. While this is likely not highly problematic for the very near road receptors that are the focus of this work, this may be an important omission for receptors further from major roads. Thus, future work would be needed to explore the community impacts of vehicles on non-highway roads (i.e., principal and minor arterials). In addition, having traffic recorders for each half-mile road segment is challenging for the quality assurance of the data. Despite, the fine-scale traffic data undergo quality assessment processes some weaknesses may remain that explain the differences in traffic volume between the two sources of data, especially for the State Route 520.

Second, RLINE's performance is sensitive to many factors such as the model input data and assumptions regarding the impact of the local terrain. Unfortunately, there is no perfect validation dataset to confirm that our estimates of traffic-generated pollution were accurate. Nonetheless, the similarity in the magnitude of our modeled estimates of NO_x to measured levels of NO_y as well as moderate correlations (0.5 to 0.6) with these observed concentrations provide us with some reasonable reassurance that the model is working correctly even given the complex terrain of the Seattle area. Moreover, these correlations are within the range found in previous studies. (28) Although our predicted concentrations of $PM_{2.5}$ were lower and less correlated with observed $PM_{2.5}$, this is not unexpected since traffic is only one source of total $PM_{2.5}$ in the urban environment. In fact, our modeled concentrations are at the expected fractions of the contribution from traffic to total $PM_{2.5}$ ambient concentrations (~12%). (2) Theoretically, we anticipate strong performance of the model since we focus on concentrations within 1 km of the main highways as it has been shown that RLINE performs best for near-road receptors as opposed to further distances where atmospheric transformations and particles deposition, which are not captured by RLINE, play a more important role. (28,29)

In conclusion, this study showed that spatiotemporal variations between and within highways in traffic patterns are complex and result in similarly complex spatiotemporal variations of air pollutant concentrations in near road communities. The use of dispersion modeling, even with aggregated input data, will capture a great deal of this variation. However, the use of fine-scale spatiotemporally resolved traffic input data adds to both the temporal and spatial variability of air pollutants concentrations in certain communities, especially those closest to highly congested highways and under certain applications would reduce exposure misclassification in epidemiologic studies.

Figure 2.1 Flow chart of the air quality modeling framework



The dashed boxes represent input data, the ovals represent computational software and the rectangular elements represent output data. The input data represented in this flow chart corresponds to the aggregated traffic data. The main difference with the fine-scale traffic data is that we used measured hourly traffic volume and vehicle speeds as opposed to the AADT, TAF and national vehicle speed used for the aggregated traffic data.

Figure 2.2 Study area including major roadways, receptor locations, and prevailing wind direction in the Central Puget Sound, WA

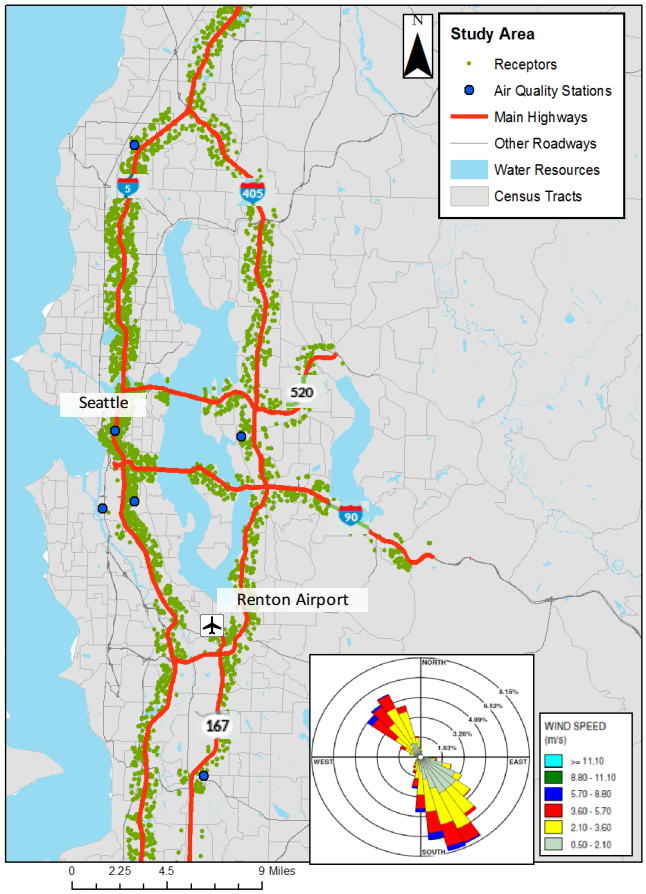
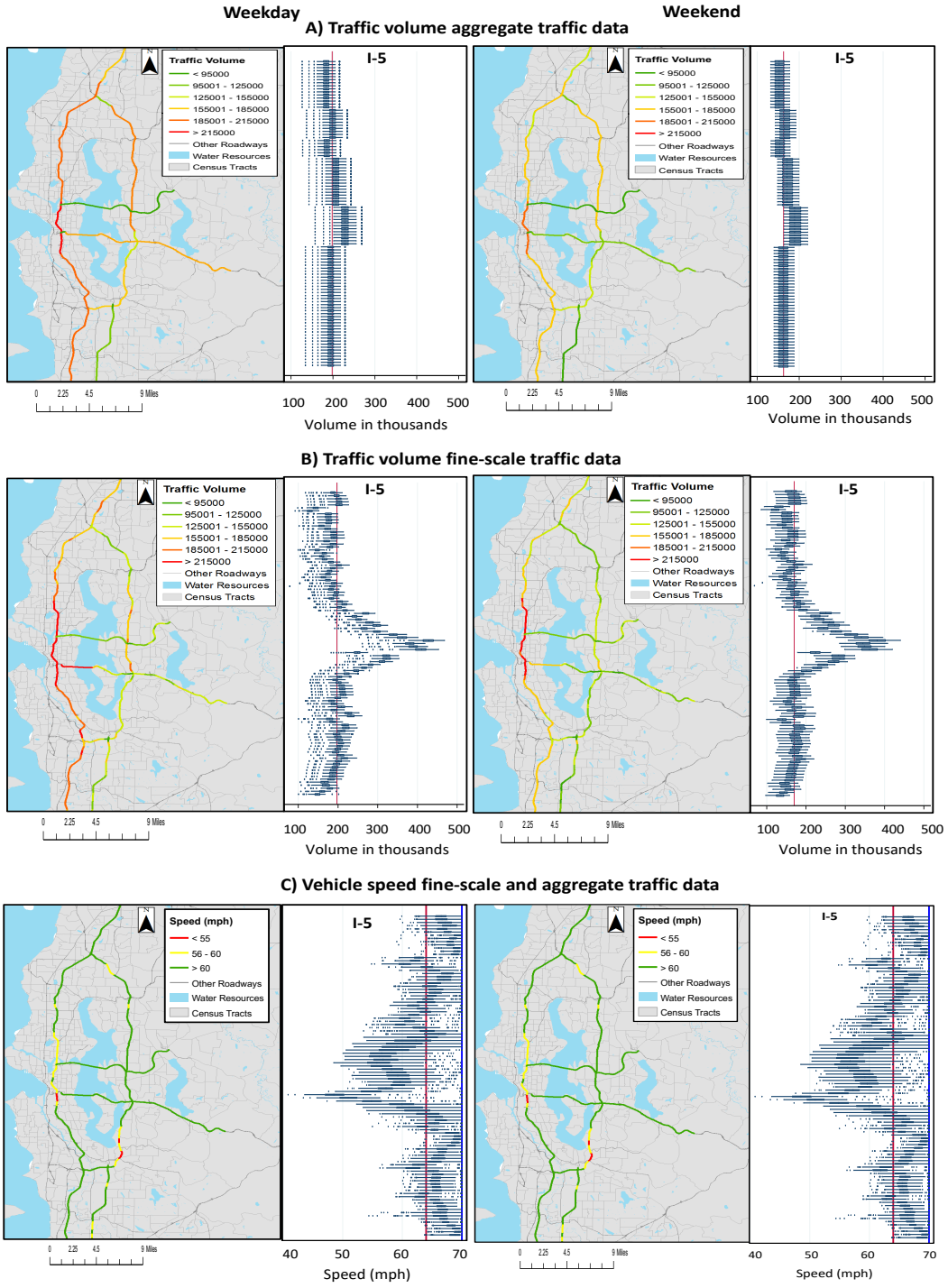


Table 2.1 Annual average (SD) daily traffic volumes by traffic input data, highway and day of week

Highway	Aggregate traffic data			Spatiotemporal traffic data		
	Weekdays	Saturdays	Sundays	Weekdays	Saturdays	Sundays
I-5	200,000 (±19,000)	176,000 (±18,000)	152,000 (±12,000)	210,000 (±62,000)	186,000 (±71,000)	171,000 (±58,000)
I-405	175,000 (±26,000)	154,000 (±22,000)	133,000 (±19,000)	148,000 (±32,000)	132,000 (±40,000)	114,000 (±26,000)
I-90	133,000 (±17,000)	117,000 (±14,000)	100,000 (±12,000)	150,000 (±42,000)	125,000 (±44,000)	115,000 (±33,000)
SR-167	97,000 (±10,000)	86,000 (±8,000)	74,000 (±7,000)	133,000 (±26,000)	121,000 (±23,000)	96,000 (±23,000)
SR-520	66,000 (±18,000)	54,000 (±16,000)	50,000 (±14,000)	114,000 (±25,000)	76,000 (±28,000)	66,000 (±18,000)

Note: For the aggregated traffic data the standard deviation (SD) was obtained by applying the TAFs by month, day of week and hour a day to the AADT reported at the PTRs throughout the highways.

Figure 2.3. Spatiotemporal variation of traffic volume and vehicle speed by day of week and traffic input data (vehicle speed for the aggregate data shown blue line) for all road and for I-5



Note: The map illustrates the mean volume and speed by location on all roads for weekdays and weekends separately. The box plots illustrate the within-road segment variability of traffic volumes and speed for all segments along I-5 (north and southbound combined). Mean speed for the aggregate traffic data is represented as blue line (mean weekday and weekends: 70 mph). Mean speed for fine-scale traffic data is represented as red line (mean weekday: 63 mph and mean weekend: 68 mph)

Figure 2.4 Distribution of the annual average daily traffic on weekdays by location on I-5

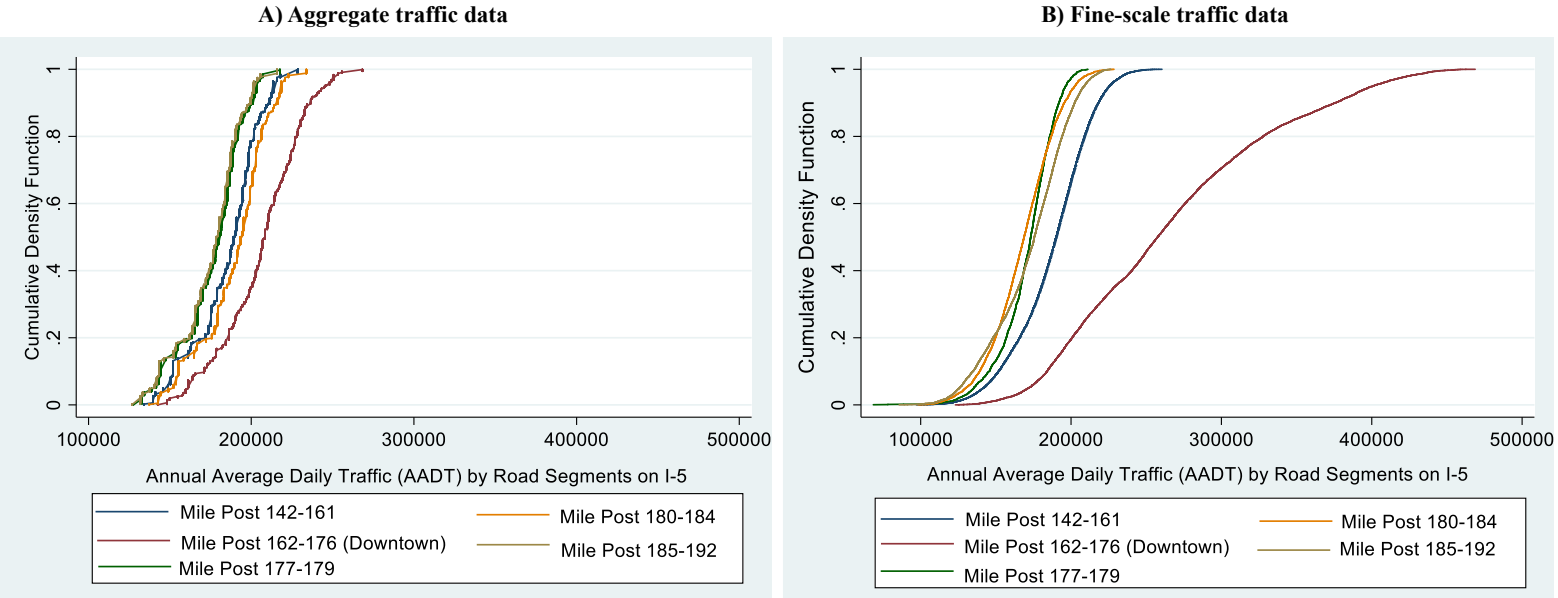


Figure 2.5 Distribution of the annual average daily speed on weekdays by location on I-5 in the fine-scale traffic data

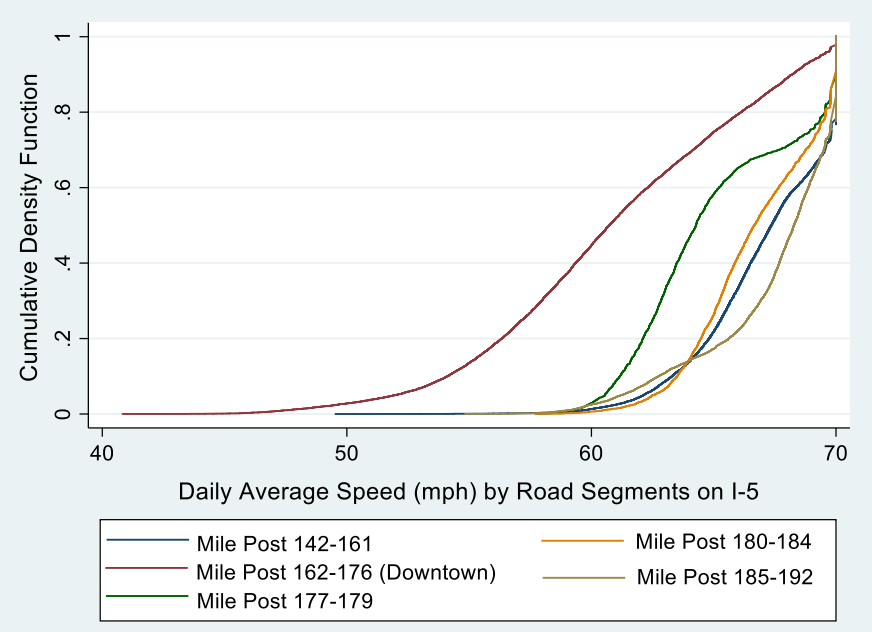


Table 2.2 Observed and modeled of daily mean (±SD) concentrations of NO_x and $PM_{2.5}$ at receptor locations and AQS monitoring stations by traffic input data

Pollutant	Mean (SD)			Spearman Correlation		
	Observed	Modeled aggregate	Modeled fine-scale	Observed vs. aggregate data	Observed vs. fine-scale data	Modeled aggregate vs. fine-scale data
NO_x (No_y), ppb						
Receptors within 1,000 m	---	28.9 (±16.5)	30.8 (±19.9)	---	---	0.96
Receptors within 150 m	---	52.3 (±29.5)	55.7 (±34.9)	---	---	0.96
EPA-AQS 530330080	20.5(±17.6)	24.4 (±25.8)	26.5 (±26.1)	0.56	0.52	---
$PM_{2.5}$, $\mu g/m^3$						
Receptors within 1,000 m	---	1.5 (±0.9)	1.7 (±1.1)	---	---	0.96
Receptors within 150 m	---	2.7 (±1.5)	3.1 (±1.9)	---	---	0.96
EPA-AQS 530330048	7.6 (±4.4)	2.1 (±2.0)	2.9 (±2.5)	0.29	0.33	---
EPA-AQS 530330057	9.5 (±5.7)	1.2 (±1.3)	1.5 (±1,7)	0.45	0.47	---
EPA-AQS 530330037	4.9 (±2.9)	1.4 (±1.5)	1.8 (±1.8)	0.51	0.51	---
EPA-AQS 530332004	8.2 (±5.1)	0.7 (±1.0)	0.8 (±1.2)	0.50	0.51	---
EPA-AQS 530610005	7.1 (±5.2)	1.1 (±1.4)	1.1 (±1.3)	0.45	0.45	---

Notes: In the Central Puget Sound, there are six AQS stations. Of those, one station (Beacon Hill AQS ID: 530330080) at 850 m from I-5 measures hourly Reactive Oxides of Nitrogen (NO_y) and five measure $PM_{2.5}$ (Olive Boren AQS ID: 530330048, Duwamish AQS ID: 5300330057, Bellevue AQS ID 530330037, Kent & Central AQS ID 530332004 and Lynwood AQS ID: 530610005. Since, between 2007 and 2013 the monitoring of NO_2 in the Puget Sound was replaced by monitoring of NO_y (ie., NO_y: Nitric Oxide (NO) + NO_2 + other oxidized nitrogen species) as an indicator of NO_x, we used the measured concentrations of NO_y to compare our modeled NO_x concentrations.

Figure 2.6 Distribution of the annual average concentration of NO_x and $PM_{2.5}$ by input traffic data and distance from the highways

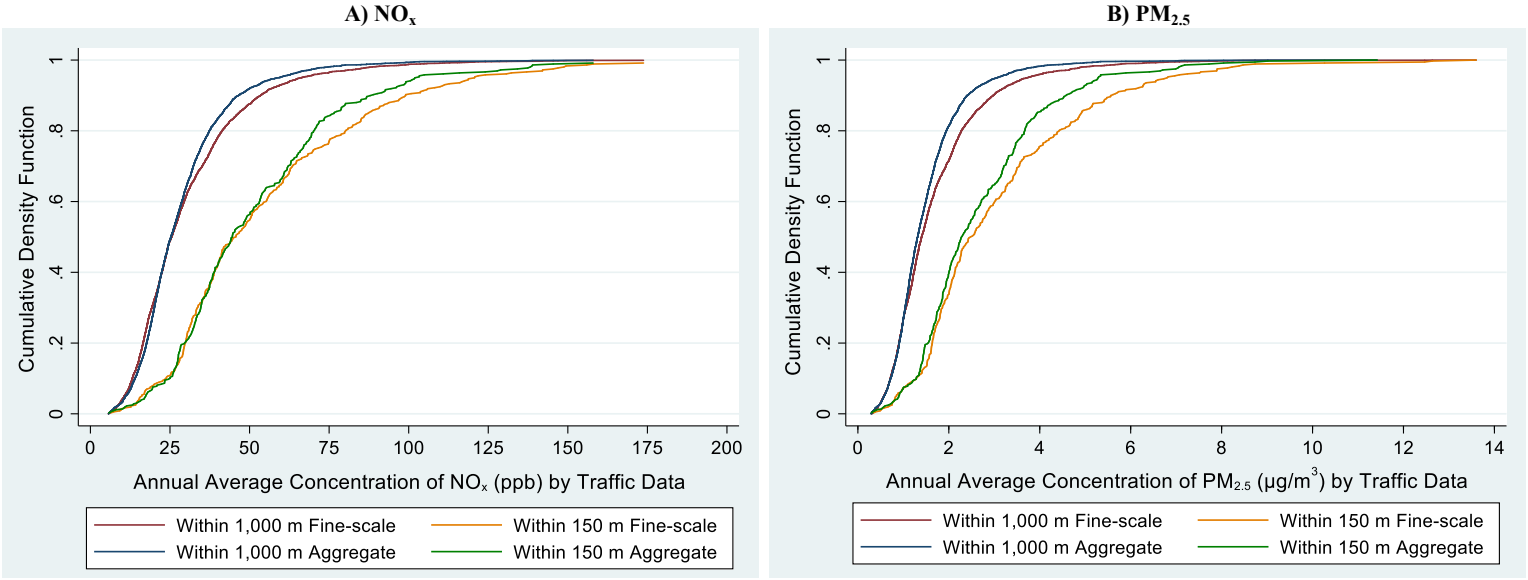


Figure 2.7 Spatial distribution of the annual average concentration of NO_x and $PM_{2.5}$ by input traffic data and difference in concentration

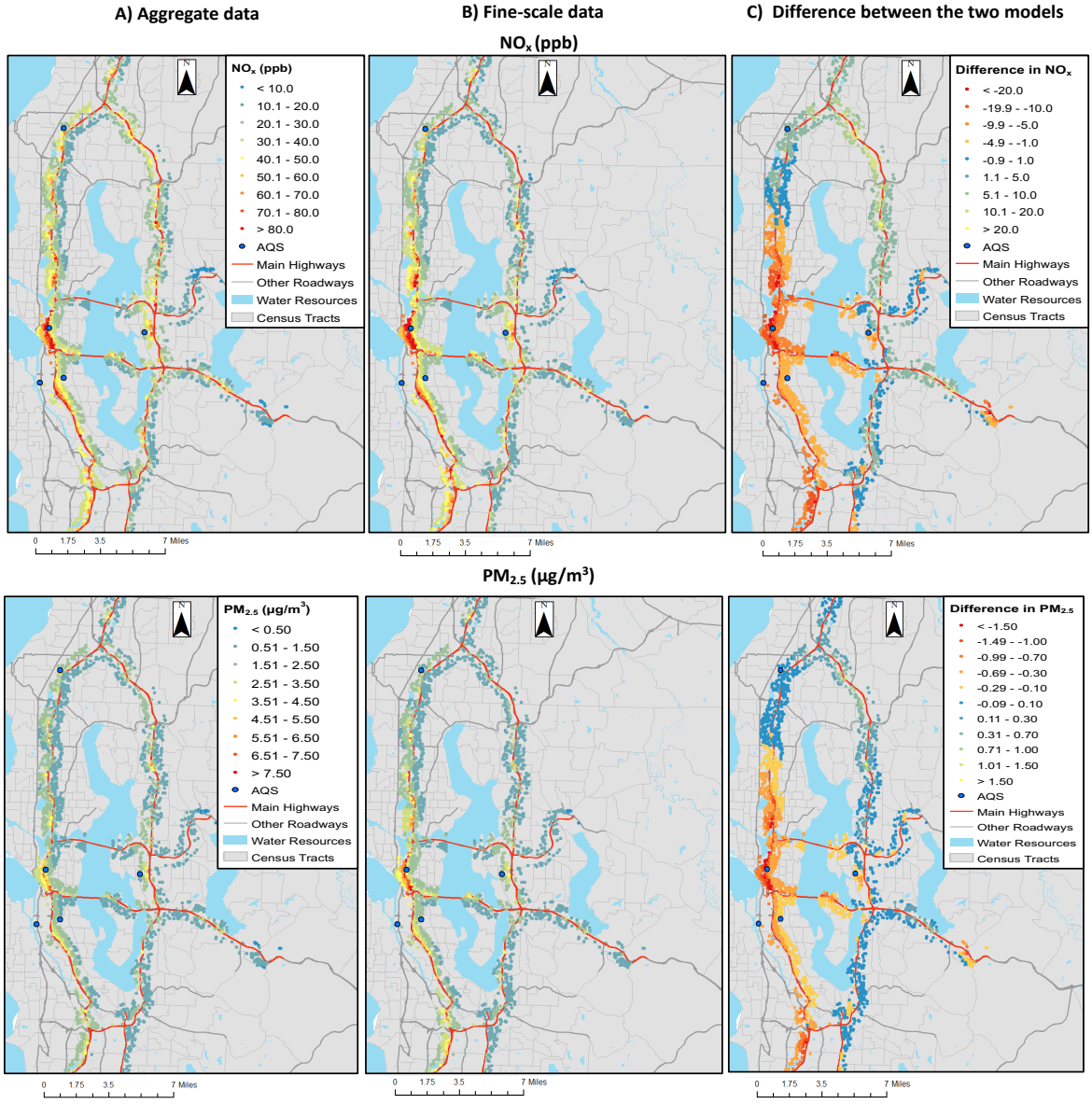


Figure 2.8 Distribution of the difference in modeled concentrations of NO_x and $PM_{2.5}$ between the two traffic input data by distance from the highways

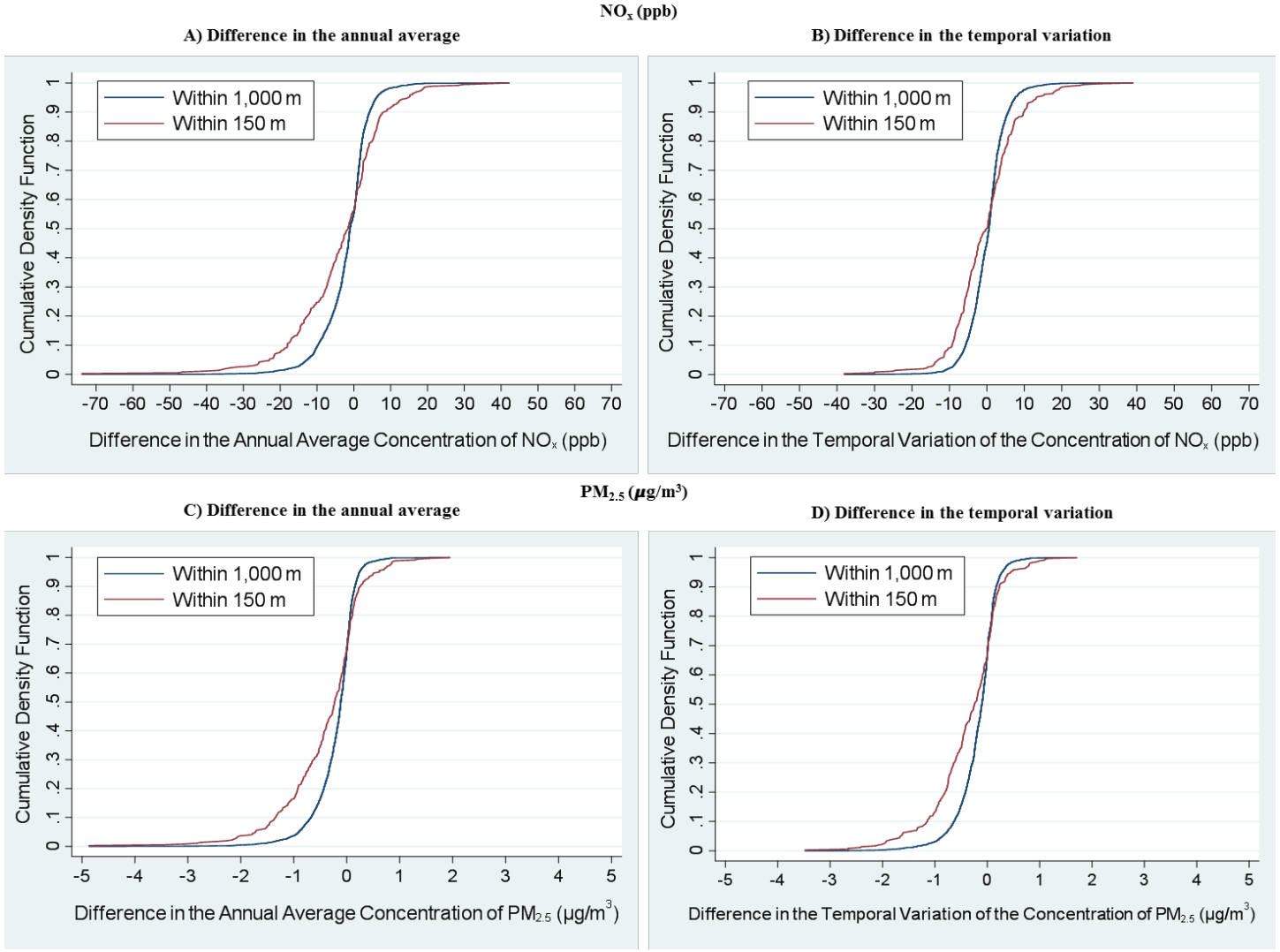
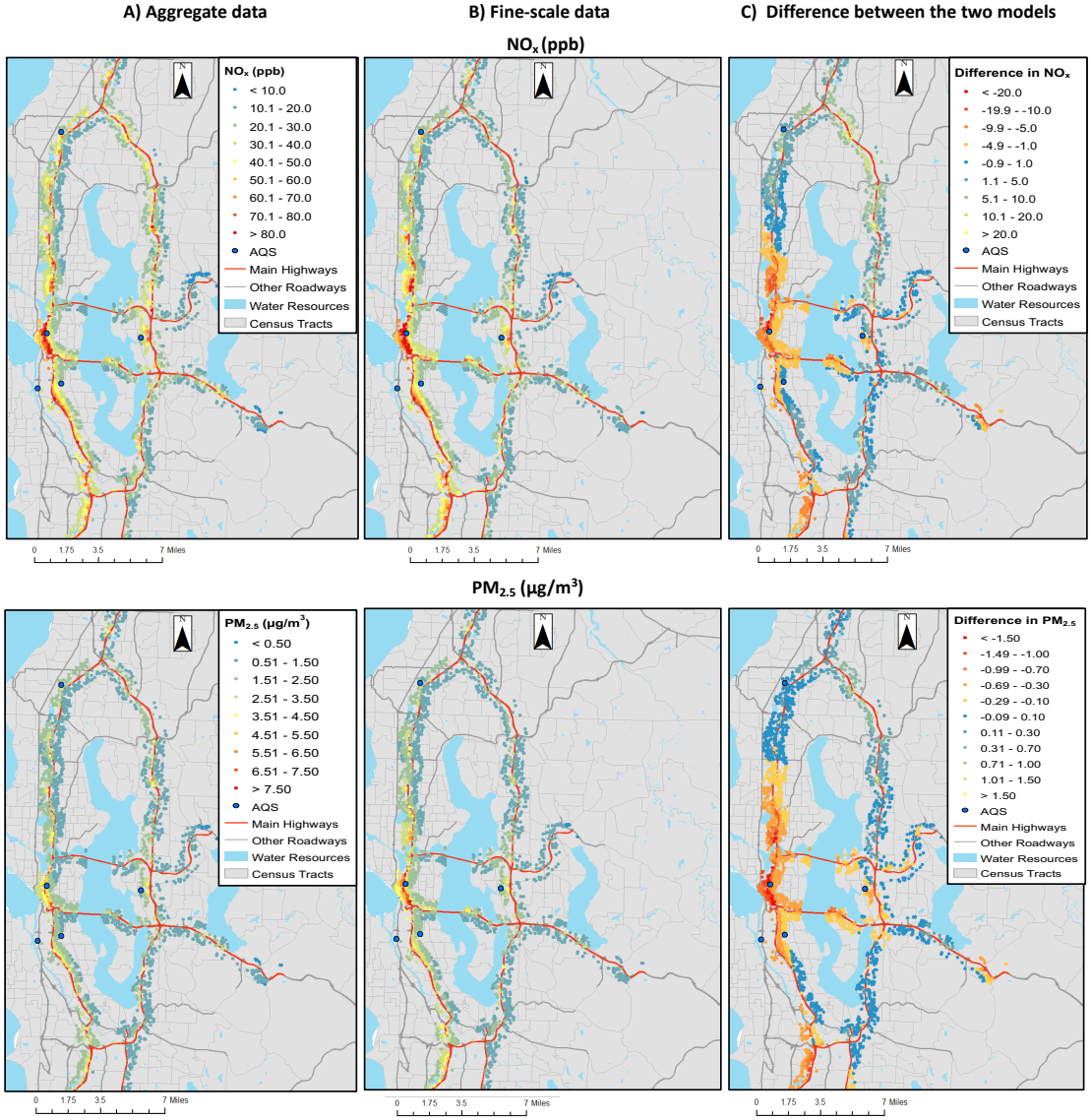


Figure 2.9 Spatial distribution of temporal variation (i.e., between-day standard deviations) of the daily concentration of NO_x and $PM_{2.5}$ over the year by traffic input data



Appendix

Table S. 2.1 Hourly average (SD) traffic volumes for weekdays by traffic input data, highway and rush hours

Highway	Aggregate traffic data			Fine-scale traffic data		
	Morning rush hours (6:00-8:59 AM)	Afternoon rush hours (4:00-6:59 PM)	Off-peak (Mid-day and Night)	Morning rush hours (6:00-8:59 AM)	Afternoon rush hours (4:00-6:59 PM)	Off-peak (Mid-day and Night)
I-5	10,000 (± 3,000)	15,000 (± 1,000)	7,000 (± 4,000)	12,000 (± 4,000)	13,000 (± 3,000)	8,000 (± 5,000)
I-405	9,000 (± 3,000)	13,000 (± 2,000)	6,000 (± 4,000)	8,000 (± 2,000)	9,000 (± 2,000)	5,000 (± 4,000)
I-90	7,000 (± 2,000)	10,000 (± 1,000)	5,000 (± 3,000)	10,000 (± 3,000)	10,000 (± 3,000)	5,000 (± 4,000)
SR-167	5,000 (± 2,000)	7,000 (± 700)	3,000 (± 2,000)	7,000 (± 2,000)	7,000 (± 1,000)	5,000 (± 3,000)
SR-520	3,000 (± 1,000)	5,000 (± 1,000)	2,000 (± 2,000)	8,000 (± 2,000)	8,000 (± 2,000)	4,000 (± 3,000)

Figure S. 2.1 Average vehicle speed by hour and location on I-5

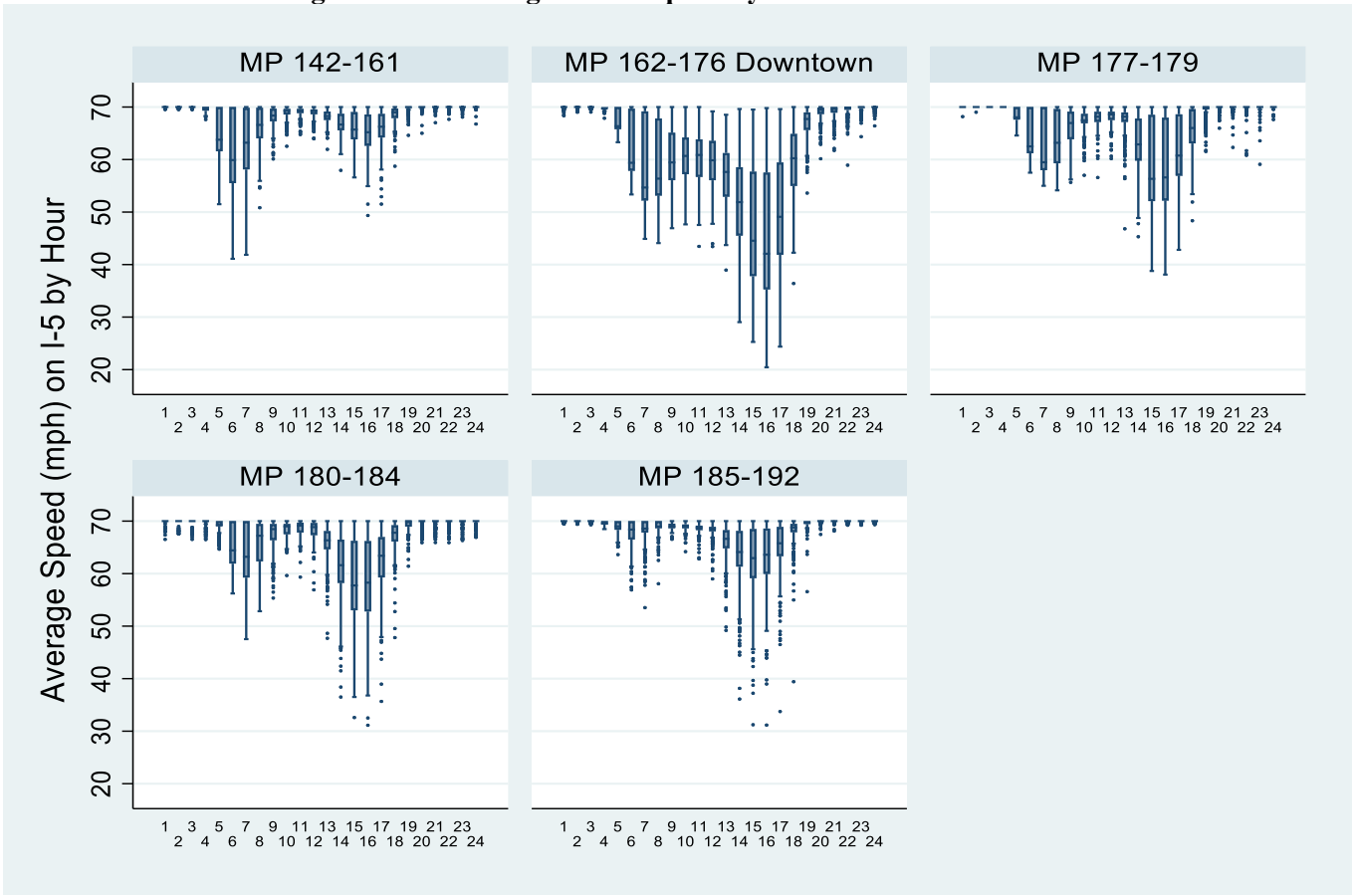
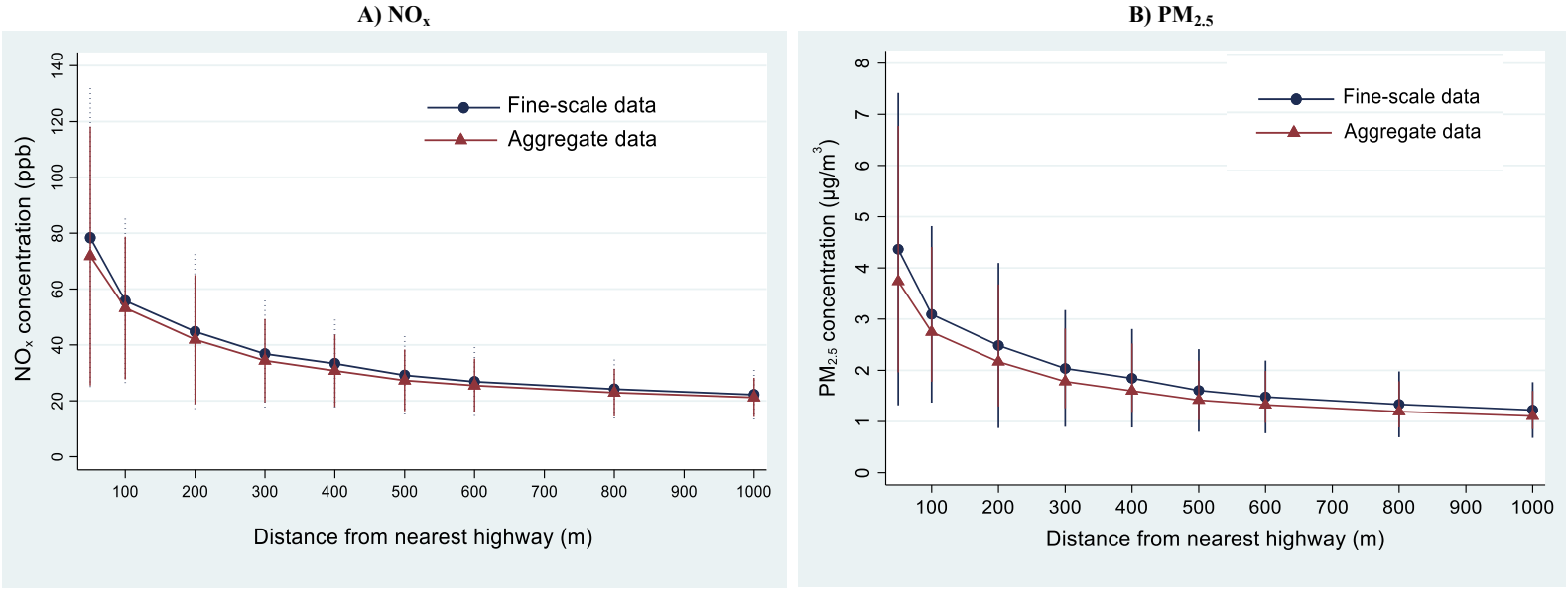


Figure S. 2.2 Spatial gradient of the annual average concentration of NO_x and $PM_{2.5}$ by traffic input data



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Chapter 3

Exposures to Primary $PM_{2.5}$, NO_x And Black Carbon Generated by Highway Traffic and the Risk of Daily Mortality in Near Road Communities: A Case-Crossover Study

Introduction

In urban areas, vehicle emissions are one of the main contributors to poor air quality. (1) Despite policies designed to control vehicle emissions, exposures to traffic-related air pollution continue to be associated with a broad range of acute and chronic health outcomes (1–9) In fact, exposure to fine particle matter ($PM_{2.5}$) from road transportation has been estimated to lead to more than 180,000 premature deaths globally due to ischemic heart disease, stroke, lower respiratory infection, chronic obstructive pulmonary disease, and lung cancer. (10) This exposure has also been estimated to be responsible for more than 5 million of Disability Adjusted Life Years (DALY's), which represent almost 0.2% of the total global burden of disease.

Concentrations of traffic-related air pollutants are often the highest within hundreds of meters of highly trafficked roadways. (1,11) In the United States, more than 11 million people live within 150 meters from major highways (12) and up to 45% of people live within 300 meters from major highways in large urban areas. (1) Within these buffers there is an overrepresentation of minority racial and ethnic groups and low-income populations. (13–18) Research has demonstrated that greater long-term exposures to air pollutants in the near-road environment put these populations at greater risk of chronic health outcomes. (2–4,19,20) Additionally, they likely experience more triggering of clinical and subclinical events due to short-term elevations in traffic-related air pollution. (21–23)

Disruptions to typical traffic patterns such as accidents, construction, or policing can impact traffic congestion and thus vehicle emissions in near road communities. In spite of these sources of variability, most epidemiological studies are ill equipped 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 monitoring stations. (24–28) However, central monitoring stations are too scarce and spatially disperse to capture any small-scale variation of traffic-related air pollution in the near-road environment. Furthermore, traffic-related air pollutants captured by urban-scale monitoring stations are often correlated with regional and other local sources over time due to the effect of meteorology. This makes it more challenging to disentangle the specific contribution of vehicle emissions to near-road exposures to traffic-related air pollution.

Only one study to our knowledge has investigated the health impacts to near-road communities of short-term variations in traffic patterns assessed with high spatial and temporal resolution. (29) That work used a unique source of spatiotemporal traffic congestion measured each half-mile and every five-minutes on the five major highways in the Washington Puget Sound to examine associations with mortality. It found that greater traffic congestion levels were associated with increased risk of cerebrovascular and respiratory mortality for decedents adjacent to a highway. That study, however, only used an indicator for nearby traffic congestion and did not account for the influence of meteorology on the dispersion of traffic-related air pollutants in the nearby communities. We extend that study by leveraging a physically-based air quality model and high spatiotemporally resolved traffic volume and vehicle speed data. Specifically, we employ the Mobile Vehicle Emissions Simulator (MOVES), which allows for the estimation of primary air pollutants generated by traffic, along with the Research Line (RLINE) dispersion

model to estimate concentrations of traffic-generated air pollution with high temporal and spatial resolution. Ideally, this should allow us to disentangle the specific role of vehicle emission from other sources of air pollution.

Our objective was to improve our understanding of the health impacts of air pollution generated by highway traffic in populations living in close proximity to highways. Specifically, we investigated whether short-term variations in primary $PM_{2.5}$, NO_x and black carbon (BC) generated by highway traffic (herein referred to as primary traffic-generated $PM_{2.5}$, NO_x and BC) were associated with an increased risk of mortality among the general population living near these major highways as well as among different racial, ethnic and socioeconomic groups.

Methods

Study design

We conducted a time-stratified case-crossover study to quantify the association between short-term variations in primary traffic-generated $PM_{2.5}$, NO_x and BC, and non-accidental, cardiovascular, respiratory and cerebrovascular mortality among individuals living within 1 km of all highways in the Central Puget Sound region of Washington State between 2009 and 2013 (Figure 3.1). 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. (31) Following the methodological literature, we compared exposures to primary traffic-generated $PM_{2.5}$, NO_x and BC in deceased subjects immediately prior to the time of death (case period) to their own exposures during control periods selected from all days within the same month as the case period, matched by day of week. For instance, if a death occurred on a Monday in February 2010 then the control days for that death were all other Mondays in

February 2010 before and after the case period. This approach prevents bias due to long-term temporal trends, time-independent confounding, and overlap bias. (32–34)

This study was reviewed and approved by the Institutional Review Board of the University of Michigan.

Outcome ascertainment

We included all non-accidental deaths in the Puget Sound among persons living within 1 km of our highways of interest between 2009 and 2013. We identified cases using death certificates from the Washington Department of Health (WADOH) with underlying causes of death derived from *The International Classification of Disease 10th* revisions as all non-accidental mortality (ICD10: A00-R99), respiratory diseases (ICD-10: J00-J98), cardiovascular disease (CVD) (ICD10: I01-I52, I60-I69), and cerebrovascular disease (ICD-10: I60-I69). We included death certificates from individuals with a valid date of death and residential addresses geocoded by the WSDOH. In-patients, hospice deaths, and cases with undefined place of death were excluded from this analysis because residential address may not accurately reflect exposures for these patients during both their case and control periods.

Exposure assessment

The exposure assessment approach used in this study was described in detail in Chapter 2. Briefly, we incorporated highly resolve spatiotemporal traffic volume and speed data available each half-mile and every 5-mintues for all highways (I-5, I-405, I-90, SR-167 and SR-520) in the Puget Sound from the Transportation Center of the University of Washington (TRAC-UW) into the Research Line Source Dispersion Model (RLINE, v1.2) (30) to predict hourly concentrations

of primary traffic-generated $PM_{2.5}$, NO_x and BC at each decedent's residential address. RLINE is a line-source dispersion model developed for the U.S. Environmental Protection Agency to model near-road exposure with high temporal and spatial resolution. (30) The concentrations of primary, chemically inert air pollutants originating from roadways are found by approximating the line as a series of point sources, where the contribution of every point source along the line is computed with the Gaussian steady-state plume formulation.

In our modelling, we followed the approach proposed by Cook et al (35) and Snyder et al. (36) Specifically, we used measured hourly traffic volume and vehicle speed for each half-mile road-segment along with information on local distributions of the vehicle fleet, age distribution, miles traveled and fuel types from the Washington State Department of Transportation (WSDOT). Using these input data and the Motor Vehicle Emissions Simulator (MOVES, version 2014) (37), we generated hourly $PM_{2.5}$, NO_x and BC emission factors (grams/vehicle-mile) for each road segment. Because each direction of these highways showed different traffic patterns across space and time, we modeled emissions factors for each direction uniquely. Then, using the numerical integration of RLINE and the beta 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 each residential address of the deceased subjects. The hourly meteorological parameters used by RLINE (i.e., friction velocity-u*, convective velocity-w*, surface roughness height-z_0, Monin-Obukhov length-L, moisture, albedo, cloud cover, and temperature) were obtained from the Puget Sound Clean Air Agency (PSCAA) from the meteorological station at the Renton Airport and processed with AERMET. Additionally, the roadway network data were obtained from US Census TIGER/Line Shapefiles and manipulated in ArcGIS, version 10.1 (ESRI).

Using the hourly-modeled concentration of primary traffic-generated $PM_{2.5}$, NO_x and BC from all of the highways at each residential address, we calculated 1, 6, 12, 24, and 48-hour moving averages of the pollutants concentration based on time of death. We defined the average concentrations of traffic-generated pollution during the 24-hours before each case and control periods as our primary exposure.

Covariates

Time-dependent covariates are of primary interest for a case-crossover design, while time-invariant factors are matched on by design. Thus, we obtained temperature, relative humidity, dew point and rain from the weather station at the Renton airport to control for confounding for daily meteorology. (NOAA: ftp://ftp.ncdc.noaa.gov/pub/data/noaa) We also obtained weekly cases of influenza validated by laboratory tests from outpatient surveillance data from the State of Washington. (38) Additionally, to control for potential confounding by holidays, we included in our models a dummy variable for New Year's Day, Memorial Day, Independence Day, Labor Day, Thanksgiving and Christmas as well as 2 days before and after each holiday. In secondary analyses, we explored other notable days such as Super Bowl Sunday. We also used $PM_{2.5}$ concentrations measured at five EPA Air Quality Monitoring Stations (AQS) in our study area (Figure 3.1) to account for potential confounding by other regional sources of pollution. In an attempt to isolate pollution from regional sources from emissions from the studied highways, we calculated the residuals from a regression of predicted concentrations from the highways of interest at each AQS station and measured levels. We then averaged these hourly residuals across all AQS monitoring stations and estimated 24-hour moving averages of $PM_{2.5}$ background concentrations for each case and control period. In sensitivity analyses we also explored using

unadjusted averages from the AQS monitors as an indicator of background levels of pollution.

Although in a case-crossover study confounding by time-independent covariates is controlled for by design, we were interested in evaluating effect modification by covariates at the individual and neighborhood level. Therefore, we extracted information on age, race/ethnicity, and education from the death certificates. Following the approach described by Diez-Roux et al (39), we also created a composite score of neighborhood disadvantage for each decedent address (NDS) by combining census variables from the American Community Survey (2009-2013) representing domains of wealth/income 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 managerial or professional education; percentage of non-Hispanic whites; percentage of non-Hispanic blacks, etc.) Finally, we created an indicator of comorbidity for individuals with diabetes mellitus (ICD10: E10 and E11), overweight/obesity (ICD10: E66), hypertension (ICD10: I10-I16) or respiratory disease in the contributory, but not underlying, causes of death.

Data analysis

We calculated summary statistics to describe the distribution of causes of death, individual and neighborhood-level socio-demographic characteristics, and within-person variations in meteorology, $PM_{2.5}$ background concentrations, and concentrations of primary traffic-generated $PM_{2.5}$, NO_x and BC during the case and control periods. We performed conditional logistic regression stratifying on each cause of death to assess whether short-term variations in primary traffic-generated $PM_{2.5}$, NO_x and BC at the residential address are associated with higher odds of non-accidental, cardiovascular, cerebrovascular and respiratory mortality. We estimated odds

ratios (ORs) and their 95% confidence intervals (95% CI) for the association of each exposure and cause of death associated with one interquartile range (IQR) increase in the 24-hour moving average (lag 01) of the modeled concentrations of primary traffic-generated $PM_{2.5}$, NO_x and BC. In all analyses, we controlled for the 24-hour moving average of ambient temperature and relative humidity prior to each case and control period using natural cubic splines. We selected 3 degree of freedom and knots at 90th percentile of temperature and relative humidity for most causes of death to minimize the Akaike Information Criterion (AIC). For cardiovascular mortality, however, based on AIC an additional knot at the 10th percentile of temperature was also included. We controlled for the 24-hour moving average of $PM_{2.5}$ background concentrations based on time of death in all models as a linear variable. Weekly influenza counts were also included in all models using natural cubic splines with 2 degree of freedom. Public holidays were entered in the models as a dichotomous variable.

In secondary analyses, we assessed whether persons who were older, non-white, of low socioeconomic status (SES) (based on individual and neighborhood-level SES) or with comorbidities have a greater risk of non-accidental, respiratory, cardiovascular and cerebrovascular mortality associated with short-term increments of primary traffic-generated $PM_{2.5}$, NO_x and BC. To test these hypotheses, we included interaction terms with each pollutant in our regression models. We categorized age as <75 and ≥75 years and, due to a small sample size for some race and ethnic groups, we dichotomized race/ethnicity as non-Hispanic white and non-white (i.e., African-American, Hispanic, Asian, Native Americans, Hawaiian, other Pacific Islander).). Individual-level SES was based on educational attainment and categorized as college, graduate degree or some college or technical school, high school or GED, and less than

high school. Neighborhood-level SES was categorized according to quartiles as high (-20.5 to 0.10), middle (0.10 to 5.04), and low disadvantage (5.04 to 19.2).

In sensitivity analysis, we examined associations with exposures to primary traffic-generated $PM_{2.5}$, NO_x, and BC at 1, 6, 12 and 48-hour moving averages before death. We examined the robustness of our estimated associations to the use of different approaches to obtain $PM_{2.5}$ background concentrations. We also assessed effect modification by time, rain, and season. Since RLINE performs better for flat roadways without different elevations in terrain or surrounding buildings, we also assessed whether or not differences in elevation between segments of the highways and the receptors location influenced our estimations. Finally, we evaluated the association of our main outcomes and $PM_{2.5}$ concentrations measured at the AQS monitors in the region. Data management was performed in Stata statistical software version 14.1 (Stata Corp) and ArcGIS version 10.1 (ESRI). All statistical analyses were performed in R statistical software, version 3.1.1.

Results

Between 2009 and 2013 there were 15,659 deaths due to non-accidental causes in the Central Puget Sound among persons living within 1 km from our five major highways. Among these deaths 5,780 (36%) were excluded for occurring as in-patients at a hospital, hospice, or undefined place and 50 (0.3%) were excluded due to incomplete data to obtain the 24-hours moving average of modeled concentrations of traffic-generated air pollutants. Thus, we had 9,829 (63%) deaths that met our inclusion criteria (Table 3.1). Of those, 8, 33, and 5% had respiratory, cardiovascular, and cerebrovascular listed as the underlying cause of death, respectively. Decedents were predominantly non-Hispanic whites (82%) and female (54%) with

a mean age of 79 (±15) years and college or some college as their maximum level of education attainment (50%). On average decedents resided in neighborhoods with more than 27% of adults who completed college, 46% employed in management, business, or science occupations, $66,000 (±26,000) median household incomes, and less than 6% unemployed. Neighborhoods were also predominantly white (59%). (Table 3.2) Among decedents within 1km of a highway, 2,867 (29%) lived within 300 meters of a highway. These individuals showed similar distributions of individual and neighborhood-level sociodemographic characteristics.

Table 3.3 shows the distribution of exposure to traffic-generated air pollutants and time varying covariates between case and control periods by cause of death. Among all decedents residing within 1 km from major highway there were no differences between case and control periods in mean exposures to primary traffic-generated $PM_{2.5}$, NO_x and BC from neighboring highways in the 24-hours before death ($PM_{2.5}$: 1.7 $\mu g/m^3$, NO_x: 26.4 ppb and BC: 0.7 $\mu g/m^3$). Similarly, no differences were found between case and control periods among decedents within 300 meters from highways though concentrations were consistently higher among these individuals than those living further away (Case vs. Control: $PM_{2.5:}$ 2.4 vs. 2.5 $\mu g/m^3$; NO_x: 37.5 vs. 38.1 ppb; BC: 1.0 $\mu g/m^3$ vs. 1.0 $\mu g/m^3$). Among decedents of cardiovascular and cerebrovascular mortality, concentrations of air pollutants for the control periods were slightly higher than case periods and these differences were more pronounced among decedents within 300 meters. There were no differences between case and control periods for any decedents on mean $PM_{2.5}$ background concentrations, temperature, relative humidity and laboratory confirmed cases of influenza.

After adjusting for time varying covariates and background $PM_{2.5}$ concentrations, we found no evidence of associations between all non-accidental mortality and exposure to primary

traffic-generated $PM_{2.5}$, NO_x and BC from nearby highways among residents within 1 km from a highway (Table 3.4). However, after stratifying by cause of death, we found between a 2 and 3% higher odds of respiratory mortality for each IQR increase in 24-hour average traffic-generated pollution levels from nearby highways before time of death although this finding could not be distinguished from no association. Conversely, an inverse association was found for cardiovascular mortality, with lower odds of cardiovascular mortality for each IQR increase in the 24-hours average of primary traffic-generated air pollutants before time of death ($PM_{2.5}$ and NO_x ORs: 0.96, 95% CI: 0.91-0.99 and BC: 0.95, 95%CI: 0.91-1.00). Stronger, but less precise, associations were also found for cerebrovascular mortality with lower odds of death found associated with higher exposures to traffic-generated pollution levels ($PM_{2.5}$, NO_x and BC ORs: 0.92, 95% CI: 0.81-1.03).

As shown in Table 3.4, stronger associations were found among residents within 300 m of a highway, with higher odds of increased respiratory mortality ($PM_{2.5}$: 1.12, 95% CI: 0.98-1.27; NO_x: 1.13, 95% CI: 0.98-1.29; BC: 1.09, 95% CI: 0.97-1.26) and lower odds of cardiovascular ($PM_{2.5}$ and BC: 0.93, 95% CI: 0.87-0.99; NO_x: 0.92 95% CI: 0.86-0.98), and cerebrovascular mortality ($PM_{2.5}$: 0.79, 95% CI: 0.64-0.97, NO_x and BC: 0.78, 95% CI: 0.64-0.96) with greater exposures to primary traffic-generated pollutants from nearby highways.

Sensitivity analyses (Supplemental Table S. 3.1) showed similar results with different time windows of exposure (i.e., 1, 6, 12 and 48-hour moving averages) as those found for the 24-hour moving average. Similarly, all associations were robust to alternate approaches to adjust for $PM_{2.5}$ background concentrations and excluding decedents residing near to elevated road-segments 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 (ORs

ranging from: 0.98 to 0.99, 95% CIs 0.89 to 0.97 – 1.03 to 1.12). Similarly, we found no evidence of effect modification by time, rain and season.

As shown in Table 3.5, we found no evidence of effect modification of our main associations of interest by comorbidity or sociodemographic characteristics.

Discussion

In this study, we incorporated highly resolved spatiotemporal traffic data into a dispersion model to estimate exposure to investigate associations between mortality and short-term exposures to primary traffic-generated air pollutants in communities near highly trafficked highways in the Puget Sound, Washington State. We found no conclusive evidence of an increased risk of mortality associated with short-term exposures to primary $PM_{2.5}$, NO_x and BC generated by highway traffic. However, we observed some suggestive evidence that greater short-term exposures to primary $PM_{2.5}$, NO_x and BC generated by highway traffic was associated with higher odds of respiratory mortality, with a stronger association for residents nearest to the highways. In contrast, and contrary to our hypothesis, we found reduced odds of cardiovascular and cerebrovascular mortality associated with higher levels of these primary traffic-generated air pollutants with stronger associations among those living closest to the highways. To our knowledge this is the first epidemiological study that estimated highly spatially and temporally resolved concentrations of primary traffic-generated air pollutants to better understand the short-term health impacts of exposures on the near-road communities.

Our observed associations for respiratory mortality for decedents within 1 km of highways (OR: 1.02, 95% CI: 0.94 to 1.11) are consistent with previous research showing positive associations with short-term exposure to traffic-related air pollutants such as NO_2 and

BC, with some studies showing a stronger association for respiratory mortality than for all non-accidental and cardiovascular mortality. (40–43). Additionally, the stronger associations we found for decedents within 300 m from a highway (OR: 1.12, 95% CI: 0.98 to 1.27) are of similar magnitude as those found in studies linking an increased risk of respiratory hospitalization and emergency department visits with 10 $\mu g/m^3$ increase in $PM_{2.5}$ that are in the range of 1-8%. (43)

This is not the first study to find limited associations between short-term exposures to ambient air pollutants and mortality in the Seattle area. Sullivan and colleagues (44) found no association between ambient $PM_{2.5}$ at 3 AQS monitors in the region with primary cardiac arrest. Zhou and colleagues found no association with all-cause, cardiovascular and respiratory mortality in the warm season (April-September) using $PM_{2.5}$ concentrations at one AQS station in Seattle (i.e., Beacon Hill). However, in that study significant associations for the three outcomes were found for the cold season (October-March). (45) Similar to our findings for primary traffic-generated air pollution, we did not find evidence of an association between exposure to ambient concentrations of $PM_{2.5}$ measured at the AQS monitors and our main outcomes of interest among all decedents.

Associations counter to our hypotheses were found for cardiovascular and cerebrovascular mortality, with higher levels of primary traffic-generated $PM_{2.5}$, NO_x and BC associated with lower odds of mortality for these specific causes. These reduced odds were even stronger for residents in the near proximity to the highways. These findings differ from most previous research that have consistently linked short-term exposure to traffic-related air pollution and cardiovascular and cerebrovascular mortality. (26,27,46) as well as controlled human exposure and toxicological studies, which have linked these exposures to subclinical changes in

health such as heart rate variability (HRV), (47–50) blood coagulation markers, and vasomotor function. (48,49) Only a few investigations have found similar results to ours, showing a slightly reduced risk of mortality associated with exposure to higher levels of specific markers of air pollution from traffic. (51–53)

Possible explanations for our counter-to-hypothesized findings for cardiovascular and cerebrovascular mortality are competing risks or unmeasured confounders. For example, research has suggested that traffic-generated noise levels decrease but air pollution levels increase at low vehicle speeds such as during traffic congestion. (54) Hence, it might be that our findings for decreased cardiovascular and cerebrovascular mortality with increased traffic-generated air pollution may be confounded by noise. This hypothesis is consistent with our findings given that we would expect negative confounding for cardiovascular, cerebrovascular, and total mortality but not with respiratory mortality, which has not been linked to traffic noise Unfortunately, data on time-varying ambient noise was not available to be included in our analysis. Similarly, ozone concentrations decrease with increased NO_x concentrations but explanation is less consistent with negative confounding for cardiovascular but not respiratory mortality, which is more strongly associated with ozone. Unfortunately, RLINE does not model secondary pollutants such as ozone to test this hypothesis.

Interestingly, we did not observe that socioeconomic factors modify the association between exposure to primary traffic-generated air pollutants and mortality. Additionally, we did not find that individual-level factors such as age, comorbidities (i.e., hypertension, diabetes mellitus, overweight/obesity) modify the association between exposure to primary traffic-generated $PM_{2.5}$, NO_x and BC and mortality. This differs from findings of some (28) but not all studies. (27,55,56) Unlike our study, however, most studies of traffic-related air pollution that

have identified significant effect modification by socioeconomic characteristics have found that more deprived populations often live closer to roads whereas we found slight greater proportion of wealthier individuals and neighborhoods closer to highways. (57–60)

There are some limitations of our study. Although our modeling approach allowed us to estimate exposure to primary traffic-generated air pollution with high temporal and spatial resolution, all models inherently have errors. Specifically, the performance of a dispersion model like RLINE is sensitive to the accuracy of the model input data such as meteorology, traffic activity, and emissions inventories. (61) In general, we expect our data to be strong, however, a known limitation of RLINE is that it does not model the influence of different elevations in terrain or surrounding buildings on the dispersion of air pollutants. This might be expected to be important in Seattle. However, this factor did not appear to influence our estimations, since after excluding decedents who resided near to elevated road segments relative to the land surface, we did not find changes in the observed associations. Moreover, in the region there is vegetation along some segments of the five highways included in this study. Several studies have shown a significant reduction in traffic-generated air pollutants behind a roadside vegetation barrier. (62–65) Although the current RLINE formulation allows us to model solid noise barriers, there is no feature to model the effect of near-road vegetation on dispersion of traffic-generated air pollutants. It may be that the presence of barriers reduces the impacts of traffic-related exposures on health. Another possible limitation is in regards to meteorology. Previous research has shown that RLINE predictions might be sensitive to the selection of the meteorological inputs. (61) While we used the information from one weather station to represent meteorological conditions in the region and its influence on dispersion of traffic-generated air pollutants, our analysis of other meteorological stations in the region showed similar wind profiles and meteorological

parameters as those from Renton airport station that would support the consistency of our modeled concentrations. In all cases, we would expect that any exposure measurement error from these causes or others should be non-differential based on the case-crossover design, such that the result will likely underestimate any true association. (66,67)

Another limitation of our study is that we did not have data on ambient noise for the study area to account for this confounding in our regression analysis. While several studies have demonstrated that confounding between noise and air pollution is often not of great significance (68), we cannot eliminate the possibility that confounding is important in this near road environment. We also did not have information on the 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 concentration of air pollutants at the residential address may reflect relevant exposure levels before death. First, most adults in the US spend a great majority of their time (69%) in a residence (69) and those who were near death likely spend more time at home than others. In addition, we excluded in-patient and hospice deaths for whom residential address may not accurately reflect exposure for case or control periods. Finally, despite the fact that we had enough power (i.e., 80%) to detect a minimum OR of 1.025 for total non-accidental mortality, we were not fully powered to detect associations with our cause-specific outcomes. Similarly, due to few cases we could not evaluate associations with more specific causes of mortality such as asthma, chronic obstructive pulmonary disease, myocardial infarction, and ischemic stroke, which have all been strongly associated with exposure to ambient air pollution. (1,70–72)

Despite these limitations, this study has important strengths. The primary contribution of this work is our exposure assessment approach. Instead of using regional monitoring stations or

even aggregated traffic data (e.g. Annual Average Daily Traffic-AADT) across time and space as most studies have done, we used a dispersion-based air quality model to capture the influence of localized differences in traffic patterns on the ambient concentrations of traffic generated air pollutants in near-road populations. Even more importantly, 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 resolution. Since such contributions have been shown to improve the accuracy of modeled concentrations of $PM_{2.5,}$ BC, (36) and NO_x, we expect reduced measurement error. (61,73) Additionally, our detailed vehicle speed data allowed us to capture the influence of complex traffic patterns such as stop-and-go congestion and short-term traffic jams. This allowed us to disentangle the contribution of exposure to traffic in a way that cannot be achieved with the use of more conventional, aggregated metrics of exposure.

The use of our improved exposure assessment approach seemed to be an important addition. This was especially true for the cerebrovascular deaths, which had previously been shown to be positively associated with the number of congested minute-kilometers near a decedent's home in our previous work but not in this study (29). On the other hand, our findings for all non-accidental, cardiovascular and respiratory mortality are consistent with previous work conducted by Pedde M., et al, 2017 in that we found no association for all non-accidental mortality and higher odds of respiratory mortality associated with greater levels of traffic congestion, with a dose-response relationship with distance from the highways. They also found reduced odds of cardiovascular mortality associated with higher congested minute-kilometers, especially among residents adjacent to a highway (within 150 or 300). Inconsistent results for cerebrovascular mortality between the two studies likely is due to the influence of meteorology and other physical factors determining dispersion of vehicle emissions as we observed low

correlations between our two exposures estimates, especially among cerebrovascular deaths (R_{sp}: 0.24).

In conclusion, this study found no conclusive evidence of an increased risk of mortality associated with exposure to primary $PM_{2.5}$, NO_x and BC generated by highway traffic in near-road populations. Although there were some suggestive stronger associations of greater odds of respiratory mortality with higher concentrations of traffic-generated air pollutants among population in close proximity to the highways, we observed unexpectedly reduced odds of mortality with greater exposure for cardiovascular and cerebrovascular mortality. This study provides insight of the use a novel approach to estimate the near-road exposure to traffic-generated air pollution.

Figure 3.1 Study area and decedents living near major highways in the Puget Sound area, Washington state over the study period (2009-2013).

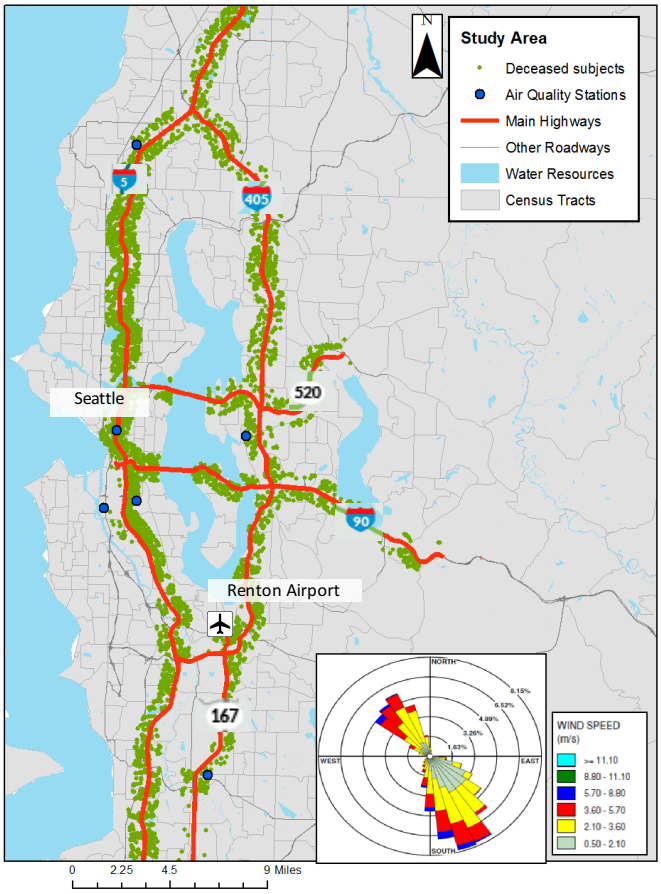

Figure 3.1 shows the study area (Puget Sound). Our five major highways are shown as red lines and other roads as dark gray lines. Decedent locations within 1 km of our highways are shown as green dots and the air quality monitoring stations where we obtained background $PM_{2.5}$ concentrations as blue dots. The prevailing wind direction came from Southeast and South-southeast.

Table 3.1 Causes of death and individual-level sociodemographic characteristics for decedents near major highways in the Puget Sound Region, Washington State, 2009-2013

Causes of mortality and individual-level sociodemographic characteristics	All subjects within 1,000 meters		Subjects within 300 meters	
	n = 9,829		n = 2,867	
	N	**%**	**N**	**%**
Causes of mortality				
All-cause non-accidental	9,829	100	2,867	100
Respiratory	737	7.5	193	6.7
Cardiovascular	3,235	32.9	972	33.9
Cerebrovascular	479	4.9	134	4.7
Individual-level sociodemographic characteristics	**Mean (±SD) or N (%)**			
Age (years)	78.9	(±14.9)	80	(±14.8)
Sex				
Female	5,286	53.8	1,622	56.6
Male	4,543	46.2	1,245	43.4
Race/ethnicity				
Non-Hispanic white	8,043	81.8	2,367	82.5
Non-white	1,768	18	496	17.3
Unknown	18	0.2	4	0.1
Education				
≥Some college, technical school	4,877	49.6	1,493	52
High school diploma	3,436	34.9	971	33.9
Less than high school	1,255	12.8	337	11.8
Unknown	261	2.7	66	2.3

Table 3.2 Neighborhood-level sociodemographic characteristics for decedents near major highways in the Puget Sound Region, Washington State, 2009-2013

Neighborhood-level sociodemographic characteristics	All subjects within 1,000 meters		Subjects within 300 meters	
	n = 9,829		n = 2,867	
	Mean (SD) or Median (IQR)			
Percentage adult residents who completed high school	17.8	8.3	17	8
Percentage adult residents who completed college	27.4	10.3	28.4	10.6
Percentage employed residents with executive, managerial occupations	45.7	16.6	47.3	16
Median value of housing units (in thousand dollars)	335	(266 - 436)	338	(274 - 436)
Median Household incomes (in thousand dollars)	61	(48 - 83)	60	(48 - 83)
Percentage household with interest, dividend, or rental income	29.5	11.7	30.3	11.2
Percentage non-Hispanic white	59.2	16.9	60.9	6
Percentage non-Hispanic black	7.4	7.8	6.9	7.3
Percentage Hispanic	9.3	6.7	8.6	6
Percentage residents over 16 years of age unemployed	5.7	2.4	5.4	2.6
Percentage below poverty level	13.7	9.3	13.9	9.8

Table 3.3 Descriptive statistics for concentrations of traffic-generated air pollutants by cause of death and time-varying covariates for decedents near major highways in the Puget Sound Region, Washington State, 2009-2013

Pollutant and cause of death	All decedents within 1,000 meters n = 9,829			Decedents within 300 meters n = 2,867		
	Case	Control	Within-person SD[a]	Case	Control	Within-person SD[a]
$PM_{2.5}$, μg/m^3						
All non-accidental	1.7	1.7	(±1.1)	2.4	2.5	(±1.5)
Respiratory	1.6	1.6	(±1.1)	2.4	2.2	(±1.4)
Cardiovascular	1.6	1.7	(±1.1)	2.3	2.5	(±1.5)
Cerebrovascular	1.6	1.6	(±1.1)	2.0	2.4	(±1.5)
NO_x, ppb						
All non-accidental	26.3	26.4	(±17.1)	37.5	38.1	(±23.1)
Respiratory	25.4	24.8	(±16.2)	38.0	33.4	(±21.4)
Cardiovascular	25.6	26.5	(±17.1)	35.6	38.5	(±23.1)
Cerebrovascular	24.4	25.7	(±16.9)	31.2	38.0	(±22.7)
Black Carbon, μg/m^3						
All non-accidental	0.7	0.7	(±0.5)	1.0	1.0	(±0.6)
Respiratory	0.7	0.7	(±0.4)	1.0	0.9	(±0.6)
Cardiovascular	0.7	0.7	(±0.5)	1.0	1.0	(±0.6)
Cerebrovascular	0.6	0.7	(±0.5)	0.8	1.0	(±0.6)
Time-Varying Covariates						
$PM_{2.5}$ background, μg/m^3	6.4	6.4	(±2.8)			
Temperature, Fahrenheit	52.3	52.3	(±4.4)			
Relative humidity, %	68.7	68.9	(±9.3)			
Weekly cases of influenza	80.0	79.3	(±28.6)			

[a] Standard deviation calculated as the within-person variability and averaged across all subjects

Table 3.4 Association between an IQR increase in traffic-generated $PM_{2.5}$, NO_x and BC and specific-causes of mortality (OR, 95% CI) among all decedents, by distance from highways

	Pollutant*					
	$PM_{2.5}$		NO_x		BC	
	OR[a]	95% CI	OR[a]	95% CI	OR[a]	95% CI
Association with modeled concentrations						
All decedents within 1,000 m						
All non-accidental	0.99	(0.97- 1.01)	0.99	(0.97 - 1.02)	0.99	(0.97 - 1.01)
Respiratory	1.02	(0.94 - 1.11)	1.03	(0.94 - 1.13)	1.02	(0.94 - 1.12)
Cardiovascular	0.96	(0.91 - 0.99)	0.96	(0.91 - 0.99)	0.95	(0.91 - 1.00)
Cerebrovascular	0.92	(0.81 -1.03)	0.92	(0.81 - 1.03)	0.92	(0.82 - 1.03)
Decedents within 300 m						
All non-accidental	0.99	(0.95 - 1.02)	0.99	(0.95 - 1.02)	0.98	(0.95 - 1.02)
Respiratory	1.12	(0.98 - 1.27)	1.13	(0.98 - 1.29)	1.09	(0.97 - 1.26)
Cardiovascular	0.93	(0.87 - 0.99)	0.92	(0.86 - 0.98)	0.93	(0.87 - 0.99)
Cerebrovascular	0.79	(0.64 - 0.97)	0.78	(0.63 - 0.96)	0.78	(0.64 - 0.95)

*IQR: PM2.5: 1.7, NO_x: 26.4 ppb and BC: 0.7 $\mu g/m^3$

[a] Adjusted by temperature, relative humidity, influenza, holidays, rain and $PM_{2.5}$ background concentrations

Table 3.5 Association between an IQR increase in traffic-generated $PM_{2.5}$, and specific-causes of mortality by different racial, ethnic, and socioeconomic groups.

Subgroup	All non-accidental			Respiratory			Cardiovascular			Cerebrovascular		
	OR	95% CI	P-value	OR	95% CI	P-value	OR	95% CI	P-value	OR	95% CI	P-value
Age												
<75 years	0.97	(0.93-1.01)	0.284	1.09	(0.93-1.26)	0.32	0.90	(0.83-0.98)	0.138	0.74	(0.49-1.13)	0.289
≧75 years	1.00	(0.97-1.03)		0.99	(0.88-1.11)		0.97	(0.93-1.02)		0.93	(0.82-1.05)	
Comorbidity												
Yes	1.00	(0.96-1.05)	0.576	1.08	(0.94-1.24)	0.324	0.97	(0.89-1.06)	0.773	0.91	(0.74-1.12)	0.986
No	0.99	(0.96-1.01)		0.99	(0.88-1.11)		0.95	(0.91-1.00)		0.91	(0.79-1.06)	
Education												
College or some college	0.99	(0.96-1.03)		1.03	(0.90-1.18)		0.95	(0.89-1.02)		0.88	(0.74-1.04)	
High school diploma	0.99	(0.95-1.03)	0.926	1.02	(0.88-1.17)	0.562	0.99	(0.92-1.06)	0.257	1.01	(0.82-1.25)	0.562
Less than high school	0.99	(0.93-1.06)		0.88	(0.65-1.17)		0.87	(0.76-1.00)		0.88	(0.66-1.19)	
Race												
Non-Hispanic white	0.98	(0.95-1.01)	0.099	1.05	(0.95-1.17)	0.399	0.94	(0.94-0.99)	0.086	0.92	(0.81-1.05)	0.779
Non-white	1.03	(0.98-1.08)		0.87	(0.87-1.10)		1.02	(1.02-1.09)		0.89	(0.68-1.15)	
Neighborhood deprivation score												
Low	0.99	(0.94-1.06)		1.14	(0.91-1.43)		1.01	(0.91-1.12)		0.86	(0.67-1.10)	
Middle	0.99	(0.95-1.03)	0.985	1.06	(0.91-1.23)	0.375	0.95	(0.89-1.02)	0.409	0.96	(0.78-1.18)	0.781
High	0.99	(0.95-1.03)		0.97	(0.85-1.09)		0.94	(0.88-1.01)		0.91	(0.76-1.08)	

[c] Adjusted by temperature, relative humidity, influenza, holidays and $PM_{2.5}$ background concentrations

Appendix

Table S. 3.1 Association between an IQR increase in traffic-generated $PM_{2.5}$, and specific-causes of mortality (OR, 95% CI) among all decedents by time window of exposure and distance from highways

	Moving averages for $PM_{2.5}$ *							
	1-hour		6-hour		12-hours		48-hours	
	OR[a]	95% CI	OR[a]	95% CI	OR[a]	95% CI	OR[a]	95% CI
All decedents within 1,000 m								
All non-accidental	0.99	(0.99-1.00)	0.99	(0.99-1.01)	0.99	(0.98-1.01)	0.98	(0.96-1.00)
Respiratory	1.01	(0.99-1.02)	1.02	(0.98-1.05)	0.99	(0.94-1.04)	0.95	(0.87-1.02)
Cardiovascular	0.98	(0.98-1.00)	0.99	(0.96-1.01)	0.97	(0.93-1.00)	0.96	(0.91-1.01)
Cerebrovascular	0.97	(0.95-1.00)	0.99	(0.97-1.01)	0.94	(0.88-1.01)	0.99	(0.92-1.06)
Decedents within 300 m								
All non-accidental	1.00	(0.99-1.00)	1.00	(0.99-1.01)	0.99	(0.98-1.01)	0.99	(0.96-1.02)
Respiratory	1.01	(0.98-1.03)	1.04	(0.98-1.10)	1.03	(0.95-1.11)	0.99	(0.89-1.13)
Cardiovascular	0.99	(0.98-1.01)	0.98	(0.95-1.02)	0.95	(0.89-0.99)	0.94	(0.87-1.01)
Cerebrovascular	0.94	(0.88-1.00)	0.99	(0.97-1.01)	0.89	(0.77-1.05)	0.95	(0.83-1.09)

*IQR: PM2.5: 1.7;

[a] Adjusted by temperature, relative humidity, influenza, holidays, rain and $PM_{2.5}$ background concentrations

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Chapter 4

Air Pollution and Health Benefits from Cleaner Vehicles and Increased Active Transportation: A Health Impact Assessment Approach for Seattle, WA

Introduction

A reliance on private vehicles over public transportation and physically active modes of transportation poses several important challenges to public health. For example, motorized road transportation impacts environmental quality through air pollution and greenhouse gas emissions. (1) In fact, air pollution from motorized road transportation has been estimated to cause 184,000 deaths globally, which represents 0.3% of deaths from all causes. (2) Motorized road transportation has been also identified as the eighth-leading risk factor of global health lost. (2) In the United States, where more than three-quarters of the population drives alone during their commutes and less than 3% walk or bicycle as a means of transportation (3), air pollution from roadway motor vehicles has been estimated to cause 53,000 premature deaths. (4) Hence, in recent years there has been a growing interest in designing transportation policies that promote cleaner vehicles and alternatives to private motor vehicle use. (1)

In the United States motorized road transportation is an important contributor to emissions of primary air pollutants. (5) In Chapter 2, we have shown that these emissions result in exposures to traffic air pollutants in communities within hundreds of meters from major roads. This has important implications for public health since nearly 45% of the United States population lives within 300 meters of major roads in urban areas where the concentration of

traffic-related air pollutants is the highest. (5,6) Hence, there has been an emphasis on improving vehicle technology to reduce on-road vehicle emissions.

Active modes of transportation such as walking and bicycling have also been identified as a strategy to reduce on-road vehicle emissions from private motor vehicle use. (1) Given that in the United States almost 41% of all car trips are less than 2 miles (3.2 km), a distance that is typically walked or biked in European cities, (7) replacing these short car trips with active transportation could translate to substantial environmental health benefits in the United States population. (8–10) In addition to lowering vehicle emissions, this may have important health benefits due to physical activity since studies have found that 32% of the United States population is fully physically inactive. (11) Such inactivity puts the population at higher risk of chronic conditions such as ischemic heart disease, ischemic stroke, diabetes, colon cancer, and breast cancer. (1,12,13)

Health impact assessment (HIA) has become an approach that uses and array of data sources, analytic methods, and inputs from stakeholders to ensure that public health is taken into consideration in policy decisions such as those related to transportation. (14–17) Past studies from Europe have used HIA to show that shifting private car use to walking and bicycling should translate into positive net health benefits by reducing all-cause mortality and chronic diseases. (18–33) Although several HIA studies of the transportation sector have been conducted in the United States, few have focused on evaluating the health benefits of promoting active transportation, reducing car trips, and increasing electric vehicle (EV) use. (8–10,34–37) Although these studies have also found potential health benefits from transportation policies, more research is needed to fully understand the impact of transportation policies across a range of different locations, populations, and policy scenarios. Furthermore, studies are needed that

examine the impact of transportation scenarios on pollutants other than just $PM_{2.5}$ since motor vehicle emissions contribute a larger fraction of pollutants such as NO_x, which leads to ozone, a wide spread problem. Finally, questions remain regarding the air pollution and health benefits resulting from an increased use of cleaner vehicles such as EVs as opposed to an exclusive shift in active modes of transportation. Collectively, this scientific evidence can be used to guide the design of urban transportation policies that have the greatest public health benefits.

In this study, we utilize some key components of the HIA framework to quantify the air pollution ($PM_{2.5}$ and NO_x) and health benefits of urban transportation policies that promote electric vehicle use and replacement of short car trips with walking and bicycling. We compare these interventions to a business as usual scenario (BAU). Changes in CO_2 emissions due to the transportation scenarios were also examined. We evaluated these policies in the urban area of Seattle, Washington since regional and local agencies have committed resources for transportation plans to increase active transportation and improve infrastructure to support EVs use. In this HIA, we used a dispersion-based air quality modeling to predict exposures in 2035 under our three different policy scenarios. We also estimated changes in physical activity due to walking and bicycling.

Methods

Study design

A complete HIA begins with the evaluation of the need for an evaluation in the *screening phase.* This is followed by the identification of the target population and stakeholders, health effects, pathways of interest, and methods during a *scoping phase*. Then, investigators model changes in exposure and health effects and propose alternatives to minimize adverse health effects in the

assessment phase and *recommendations phase*, respectively. Finally, communicating results and recommendations to decision-makers and monitoring the implementations of these recommendations occurs during the *reporting* and *monitoring* phases. (17) In this study, we constrained the scope to the assessment phase of the HIA framework. However, by basing our transportation policy scenarios on the well-defined Washington Transportation Plan 2035 (WTP-2035) (38) and the Drive Clean Seattle Strategy (39) we relied upon previously conducted screening and scoping work conducted by the Seattle Office of Sustainability and Environment. (39)

The conceptual basis of the HIA model that we used in this study is the comparative risk assessment (CRA) model proposed by the World Health Organization, in which a change in the disease burden is estimated from a shift in the exposure distribution from a baseline scenario to an alternative scenario. (40) This study followed the approaches proposed by Woodcock J., et al; de-Nazelle, A., et al; and Rojas-Rueda D., et al. (1,18,26). Specifically, we predicted concentrations of primary $PM_{2.5}$ and NO_x from on-road vehicle emissions and estimated the amount of physical activity experienced by the population of Seattle in 2035 under three different urban transportation policy scenarios. We then compared estimates of mortality from all-causes and specific causes (i.e., cardiovascular, stroke and respiratory) for the Seattle population under scenarios using more EVs and active transportation as compared to a BAU case.

Scenarios of urban transportation

To inform the potential impacts of a range of realistic policies, we examined the air pollution, physical activity, and health impacts of three transportation policy scenarios on the population of

Seattle in 2035. These scenarios were based at least in part on the Washington Transportation Plan 2035 (WTP-2035) (38) and the Drive Clean Seattle Strategy from the Seattle Office of Sustainability and Environment. (39) For all scenarios we used traffic conditions in 2010 in Seattle reported by the WSDOT and projected them to 2035 (38,41).

Under the first scenario, the BAU, we used the forecast of overall motor vehicle usage from the Washington State Department of Transportation, which projects that the vehicles miles traveled (VMT) in Washington for 2035 will increase 19%, concurrent with the projected trends in population growth and economic improvement for the area . (38,41) As the base case, we assumed no additional efforts to increase the percentage of electric vehicles in the fleet since 2010 nor any changes in the percentage of trips using active modes of transport. In Scenario 2, we assumed the same vehicles miles traveled (VMT) and active transportation as the BAU but assumed that by 2035 35% of the gasoline passenger cars and light duty tracks will be replaced by EVs. In Scenario 3, we assumed the same improvements in the vehicle fleet technology as described in Scenario2 but also assumed that 50% of car trips less than 1.5 miles (2.4 km) will be replaced by walking and 50% of car trips between 1.5 and 5 miles (2.4 - 5.6 km) will be replaced by bicycling. For all scenarios, including the BAU, we assumed improved fuel economy for the fleet based on the Joint National Standards (2017-2025) of the USEPA and the National Highway Traffic Safety Administration. (42)(43) These standards, which were developed to reduce green-house-gases (GHG) emissions and improve fuel economy of passenger cars and light duty trucks, are the most recent available standards for motor vehicles.

Estimation of changes in concentrations of air pollution

To quantify differences in concentrations of primary $PM_{2.5}$ and NO_x from traffic as well as CO_2

emissions for each scenario, we conducted dispersion-based air quality modeling using an approach that we have used previously for modeling current day conditions in the Central Puget Sound Region and has been described in detail elsewhere. (Chapter 2). Broadly, we used the Motor Vehicle Emissions Simulator (MOVES, version 2014)(44) and the Research Line-Source Dispersion Model (RLINE v1.2) (45) with inputs representing conditions for each transportation scenario. We then predicted concentrations of NO_x and $PM_{2.5}$ at receptor locations throughout Seattle urban area. These locations were randomly selected from residential parcels in each census tract to reflect a population-weighted exposure.

As inputs to our air quality models and to represent conditions in 2035, we used EPA projections on the vehicle fleet mix distribution and fuel formulations for King County in 2035. (MOVES, user-guide) Next, we scaled up the traffic volumes (i.e., Annual Average Daily Traffic-AADT) in 2010 for all minor and principal arterials and major highways (I-5, I-405, I-90, SR-520 and SR-167) from the Seattle Department of Transportation (SDOT) by 19% based on population growth and the WSDOT VMT forecast in Washington for 2035. We assumed that vehicle speeds did not change from 2010. For both Scenarios 2 and 3, we replaced 35% of gasoline passenger cars and light duty tracks by electric vehicles. In Scenario 3, we further reduced the VMT on principal and minor arterials by 50% for all car trips less than 5 miles using information on origins and destinations of car trips in Seattle from the Puget Sound Travel Survey. (46) Data of the geography of the road network for minor and principal arterials in Seattle was obtained from the SDOT (http://data-seattlecitygis.opendata.arcgis.com/datasets/seattle-streets) For major highways, we used the geography from the Tiger products of the US Census Bureau.

Using these projected input data by scenario and MOVES, we generated hourly emission factors (grams/vehicle-mile) for NO_x, $PM_{2.5}$ and CO_2 for the roadways of interest. Then, we combined these emission factors with local meteorology using the numerical integration of RLINE to model hourly concentrations of primary NO_x and $PM_{2.5}$ under each scenario at our receptor locations. Since there is no meteorological data for the future, we used the conditions measured in 2010 at the Renton Airport station operated by the National Oceanic and Atmospheric Administration (NOAA) (ftp://ftp.ncdc.noaa.gov/pub/data/noaa) as a year with typical conditions. Since we are estimating changes on air pollution on the annual average scale we anticipate using data for a typical year will not affect our estimated air pollutant concentrations. These data were processed through the AERMET program by the Puget Sound Clean Air Agency.

To compare pollution levels across scenarios, we calculated annual concentrations for each scenario at all receptor locations for each scenario and estimated a percentage change as compared to the BAU scenario. For inclusion in our health analyses, we averaged pollutant concentrations of all receptor locations within census tracts and weighted these estimates by the age and sex distribution of the population by census tract to develop a population-weighted annual average concentration for each scenario. We selected census tracts as our unit of aggregation since this is an area from the U.S Census Bureau where estimations of health outcomes and traffic air pollution can be made accurately. (47) This assumes that people living within the census-block experience the same air pollution level. We further assumed that non-traffic related sources of $PM_{2.5}$ and NO_x were constant across scenarios.

Estimation of changes in physical activity

We used data from the Puget Sound Travel Survey (46) to estimate age- and sex-specific travel distances by mode of active transportation for the BAU scenario. To convert distances walked or bicycled into travel times we used the mean and standard deviations of age- and sex-specific walking and bicycling speeds from published studies. (18,48) Then shifting 50% of short car tips to active transportation we obtained mean travel time distributions by distance, age, and sex for walking and bicycling in Scenario 3. These travel time distributions were converted into distributions of metabolic equivalents (METs) using tabulated data for specific activities and speeds (i.e., 6.8 METs are assigned to bicycling to/from work at a self-selected pace and 3.5 METs for walking at a moderate pace). (49,50) We used the geometric mean of MET hours per week as the summary statistic by age and sex groups and as the measure of exposure to physical activity due to active transportation given the log-normal distribution of these data.

Because the relationship between physical activity and all-cause and cardiovascular mortality is curvilinear with the greatest benefits for moving from low to moderate levels of activity, (51,52) we added our estimates of physical activity from active transportation to data for non-transportation related physical activity. Specifically, we calculated the geometric mean and standard deviation of the sum of weekly minutes of leisure-time moderate and vigorous physical activity plus occupational physical activity by age and sex for the West Region from the National Health Interview Survey for the Center of Disease Control and Prevention and converted these into METs (h)/week. (49,53) We assumed that the non-transportation related physical activity was constant across all scenarios.

Estimation of projected health impacts for each transportation scenario

We quantified the projected health impacts for all non-accidental, cardiovascular or

cardiopulmonary mortality in adults of the general population attributable to changes in air pollution between the BAU and each alternate transportation scenario. To quantify the health effects attributable to changes in physical activity from active transportation, we examined all-cause and cardiovascular mortality. These outcomes were selected based on the best evidence available in the scientific literature. (5,54–57) For physical activity we focused on adults less than 65 years of the general population because we expected that this group would be the most likely to adopt active transport over driving for short trips.

We estimated the Population Attributable Fraction (PAF) to reflect the fraction of the deaths in a population that are due to the exposure. This calculation includes the concentration-response function expressed as risk ratios (RRs), and the population distribution of exposure under the BAU scenario and the policy scenarios. (58) To translate the obtained PAFs into mortality burdens, we multiplied the PAFs by projected Seattle mortality rates by age, sex, and cause and summed across all strata by specific cause. To project Seattle mortality rates for 2035, we adjusted the specific mortality rates of 2010 to the projected age and sex structure of the population in the region for 2035 based on projection from the Center for Disease Control and Prevention (https://wonder.cdc.gov/population.html). Since in this study we focused only on the health and not economic impacts of our transportation scenarios we did not include any discounting. (9,18,40).

Although previous studies conducted in the Seattle area have quantified associations between exposure to traffic-related air pollution and mortality, we used exposure-response functions derived from a wider body of scientific literature, which is considered a better practice for this type of evaluation. In particular, for the health impacts of $PM_{2.5}$, we used the exposure-response functions from the reanalysis of the Harvard six cities and the American Cancer Society

Study (ACS). (59–62) Because less scientific literature is available for NO_x, we used exposure-response functions from a recent publication of the ACS study recommended in the EPA's Integrated Science Assessment for Oxides of Nitrogen. (55,63) To avoid double counting, we used reported associations of $PM_{2.5}$ adjusted by NO_2 and NO_2 adjusted by $PM_{2.5}$. To translate our NO_x modeled concentrations to NO_2 we applied a ratio of 0.75 (NO_2/NO_x) based on previous findings in the study area (Chapter 2). We assumed a linear concentration-response function for the association of exposure to NO_2 and $PM_{2.5}$ with our health endpoints since concentrations in this area are within a range where most epidemiological studies have not identified a threshold concentration below which ambient air pollutants do not have an effect on health. (64)

For associations between physical activity (METs) and all-cause mortality, we used the exposure-response function derived from the meta-analysis conducted by Woodcock et al. (57). For associations of physical activity (METs) with cardiovascular mortality we used the meta-analysis by Hammer et al. (65) Since the exposure-response function between physical activity and all-cause and cardiovascular mortality is non-linear, we used a 0.25 power transformation of total physical activity with all-cause mortality and a 0.5 power transformation with cardiovascular mortality following recommendations by Woodcock et al. (23,57)

Modeling and sensitivity analysis

We quantified the heath impacts of air pollution and physical activity for our three scenarios as described above using Stata statistical software version 15.1 (Stata Corp). We also included uncertainty analyses around the parameter estimates using Monte Carlo simulations, allowing for the estimation of confidence intervals based on 95% of the model runs (CI 95%). Using the a lognormal distribution of the annual average concentrations of $PM_{2.5}$ and NO_2 as well as physical

activity, we generated exposure levels and exposure-response functions in 10,000 simulations for a random sample of 10,000 subjects. Then, from these simulated runs we obtained the 95% confidence intervals for our estimations.

Finally, we ran sensitivity analyses to evaluate the robustness of our results, including the consideration of different concentration-response functions for $PM_{2.5}$ such as those reported in the EPA's Integrated Science Assessment for PM. (66) For active transportation, we performed sensitivity analysis using a linear exposure-response function between transportation-related physical activity and mortality. We also evaluated shorter travel times for our scenario with active transportation.

Results

As shown in Table 4.1, both proposed transportation intervention scenarios (Scenarios 2 and 3) resulted in an overall reduction in the emissions of NO_x and $PM_{2.5}$. Under Scenario 2, there was an 8% in NO_x and 11% reduction in $PM_{2.5}$ while under Scenario 3 there was 9% reduction in NO_x and a 19% reduction in $PM_{2.5}$ as compared with the BAU scenario. Moreover, we estimated that Scenario 2 and Scenario 3 would result in reductions in atmospheric CO_2 emissions from on-road traffic by 26% and 30%, respectively as compared with the BAU. We also estimated that switching to EV would reduce the mean annual average concentrations of NO_x and $PM_{2.5}$ from on-road traffic at receptors across the Seattle urban area by an average of 0.27 ppb (10.9%) and 0.04 $\mu g/m^3$ (8.6%), respectively, as compared to the BAU scenario. Adding a shift of 50% of car trips less than 1.5 miles to walking and 1.5 to 5 miles to bicycling increased reductions in NO_x and $PM_{2.5}$ to 0.32 ppb (12.8%) and 0.08 $\mu g/m^3$ (18.2%) as compared to the BAU.

As shown in Table 4.2, shifting 50% of short car trips to active transportation would

increase the daily average transport-related walking and bicycling times from 7.7 and 2.0 minutes in the BAU scenario to 13.9 and 21.4 minutes under Scenario 3, respectively. In addition, the mean distance traveled per trip for walking and bicycling would increase from an average of 0.7 and 0.3 miles to 0.6 and 2.8 miles, respectively. (Table 4.2).

As for the health benefits, our results indicate that the transportation intervention scenarios would reduce adverse health outcomes associated with exposure to NO_x and $PM_{2.5}$ in the adult population of the Seattle urban area. (Table 4.3) Specifically, we estimated that for an adult population of 691,000 inhabitants, reductions in NO_x from on-road traffic would result in 10 (95% CI: -1, 21) and 11 (95% CI: -1, 25) fewer premature deaths per year for all non-accidental mortality in Scenario 2 and Scenario 3, respectively, as compared with the BAU. These NO_x reductions would also prevent 4 (95% CI: -2, 10) and 5 (95% CI: -2, 11) premature deaths per year for cardiovascular mortality in Scenario 2 and Scenario 3, respectively, as compared with the BAU. Fewer health benefits were estimated for reductions in $PM_{2.5}$ from on-road traffic, with 1 (95% CI: -1,3) and 2 (95% CI: -2, 5) premature deaths avoided for all non-accidental mortality for Scenario 2 and Scenario 3 as compared to the BAU, respectively. These predicted reductions in all non-accidental premature mortality were primarily driven by reductions in cardiopulmonary deaths for both scenarios. (Table 4.3)

We also found that increments in physical activity due to switching to active transportation in Scenario 3 had large contributions to health. As shown in Table 4.3, increasing active transportation would result in 49 (95% CI: 19-71) premature deaths avoided per year for all-cause mortality, of which 21 (95% CI: 9-25) were attributable to reductions in cardiovascular mortality.

Overall, the total health gains estimated for Scenario 3, which includes active

transportation were 62 (95% CI: 28-84) avoided deaths as compared with 11 (95% CI: 0-22) fewer premature deaths from the Scenario 2.

In sensitivity analyses, we found that our results were largely robust to different concentration-response functions for air pollution. In particular, using different concentration-response functions for $PM_{2.5}$ resulted in premature death estimates for all-cause non-accidental mortality that were within the range of our initial findings (e.g.; 1-2 prevented deaths for Scenario 2 and 2-4 prevented deaths for Scenario 3). For physical activity, we found that our results were more sensitive to the exposure-response function. In particular, using a linear exposure-response function for walking and bicycling related-physical activity resulted in overall benefits that were up to 3 times greater than those we estimated using a non-linear exposure-response function (e.g.; avoided deaths for all-cause mortality in Scenario 3: 153, 95% CI: 83-176). In addition, assuming shorter travel times, especially for bicycling (e.g.; 11 minutes and a distance of 1.6 miles) resulted in smaller health benefits (avoided premature deaths: 32, 95% CI: 19-45) though these values were within the range of our results assuming longer travel times.

Discussion

Our results showed that transportation interventions scenarios, based largely on policies proposed by local authorities, would translate to sizeable health benefits in the general population of Seattle, Washington. We estimated that switching 35% of all gasoline cars and passenger trucks to electric engines and shifting 50% of car trips less than 5 miles to walking and bicycling would result in 62 fewer premature deaths each year. While these estimates included 13 fewer deaths due to reduced exposure to NO_x and $PM_{2.5}$ from traffic, the vast majority of the health benefits of these transportation policies are due to the increased physical activity of

walking and bicycling for active transportation.

Overall, the 62 avoided premature deaths for all-cause mortality associated with our transportation scenarios have a public health impact that is roughly equivalent to a 6% increase in the prevalence of those quitting smoking within five years. According to the study by Messer K et al, this is consistent with cessation rates for smoking with an average of 6.7% of adults in the United States having quit smoking for 6 months or longer. (67) In addition to local health benefits, these transportation scenarios demonstrated their contribution to achieving local goals to reduce carbon emissions, with approximately 30% less CO_2 emissions under both scenarios, which are obtained mainly by switching to EV.

Our results are consistent with previous research that documented health benefits of replacing car use by active transportation and reducing vehicle emissions. Similar to our results, most of these studies found larger health benefits of increased physical activity as compared to improved air quality. (8–10,18,19,26–28,34–36,68) Comparing our results to those of Grabow et al, who conducted a HIA in 11 metropolitan areas of the Midwestern United States, we found very similar reductions in $PM_{2.5}$ for shifting 50% of short car trips to active transportation. We found reductions of 0.04 $\mu g/m^3$ in $PM_{2.5}$ attributable to removing these short trips in comparison to their findings of reductions between 0.01-0.05 $\mu g/m^3$. They also reported reductions in the daily maximum 8 hours of O_3 of 0.05-0.23 ppm. In terms of their finding of health benefits, for a population of 31.3 million people in the Midwestern region, Grabow M et al found declines in all-cause mortality due to less air pollution exposure and increased physical activity due to bicycling of about 1,295 fewer deaths per year. (8) Even larger estimates were reported by the study by Maizlish N et al for the San Francisco Bay area where they found overall net health benefits of 2,413 avoided premature deaths per year due to reduced $PM_{2.5}$ exposure and increase

physical activity due to active transportation among a population of 7.1 million. (9) Although we found somewhat smaller benefits for $PM_{2.5}$ than the Maizlish study after adjusting for population size, this might be explained by our use of concentration-response functions of $PM_{2.5}$ and NO_2 adjusted by co-pollutants to avoid double counting whereas older studies used estimates from single pollutant studies. Other possible explanations for differences in the estimated health benefits across studies might be the assumptions used such as the percentage of car trips replaced by active transportation, inclusion or not of cleaner vehicles, as well as the amount of physical activity assumed under the different scenarios.

Another contribution of this work is that we newly quantified the impacts of transportation policies on reducing population exposure to NO_x. This is important since we found greater health benefits of reduced exposure to NO_x than those found for $PM_{2.5}$. One potential explanation for this finding is that transportation policies have greater impacts on air pollutants more tightly related with traffic emissions such as NO_x, for which 33% of total emissions are contributed by on-road motor vehicles as compared with $PM_{2.5}$ for which traffic emissions only contribute to 12% of the total emissions. (5)

One important note on our findings is that our health benefit estimates for air pollution used concentration-response functions for the United States derived from the American Cancer Study. However, previous epidemiological studies conducted in the Seattle region have found largely inconsistent results for the association between exposures to air pollution and mortality. (69–71) If these previous associations reflect the true conditions in Seattle, we would expect that the health benefits of the interventions evaluated in this study would be smaller than reported here. However, given that we found that the health impacts were mainly due to the active transportation and physical activity component of the transportation policies, our results still

suggest important benefits from the transportation policies evaluated.

Another note of importance is that we assumed that EVs do not generate tailpipe emissions and thus have a reduced contribution to community exposures to air pollution. Yet, while EVs do not directly burn fossil fuels to power their engines, the electricity used to power those engines is often derived from coal or other polluting sources. In fact, one analysis from 2014 documented that the overall population burden of air pollutants from EVs was only lower than gasoline vehicles when clean sources of energy were used. (72) Since greater efforts have been placed on renewable sources of energy, however, a more recent analysis has estimated that the air pollution impacts of EVs in the future will be 60% lower than the EVs driven today. (73) This report similarly noted that in spite of the fact that manufacturing of EVs has been associated with higher GHG emissions than conventional gasoline-powered vehicles, (74) the excess manufacturing emissions for EVs are usually compensated by cleaner emissions from driving after 6 to 16 months. (73) Thus provided that clean energy sources continue to replace their dirtier counterparts for electricity generation, EVs will continue to provide population health benefits although individuals near the power plants may have some additional burden.

Another issue of note is that the transportation policies proposed in this study would require infrastructure investments to facilitate access to charging technology and support pedestrians and bicycle traffic. Nonetheless, the resulting health benefits may outweigh the costs of such as policies. For instance, the Seattle master plan for bicycles projected that the addition of nearly 100 miles of protected bicycle lanes, 250 miles of neighborhood greenways, and bicycle parking facilities would cost to the city of Seattle nearly $72.6 million for the first five years (2017-2021). (75) In contrast, health cost savings of around $212 millions has been estimated from a similar active transportation scenario for cities in the Midwestern Unite States

with similar population density as Seattle. (8) Thus, the potential health care saving from avoiding premature mortality could cover the cost of adding bicycle and pedestrian infrastructure. In fact, the intervention scenarios assessed in this study are highly feasible since Seattle has experienced an increasing trend in EV ownership as well as active transportation, with an increase of almost 350%, 48%, and 38% for EV sales, bicyclists, and pedestrians in the last 6 years, respectively. (39,76)

As with all studies, our work is not without limitations. For one, we did not assess all transportation-related exposures and outcomes. Specifically, we did not assess any adverse outcomes due to increased exposure to air pollutants for those walking or bicycling next to busy roadways, or any changes in the risk of traffic injuries. As such, our net health benefits could be overestimated. Most previous studies, however, have found that the health benefits of physical activity have far exceeded the adverse health impacts of walking and bicycling next to air polluting traffic so this impact may be minimal. (31,32,77) Moreover, previous studies have found that the total net health benefits of promoting active transportation outweigh the adverse effects of traffic injuries. (9,10,19,26) In contrast, we may have underestimated our health benefits by not including secondary pollutants such as O_3 or secondary aerosol particles formed in the atmosphere in our modeling. Another possible limitation of this study is that the EPA projections for fuel formulations and vehicle fleet distribution for 2035 may be overly optimistic. These projections followed the EPA Tier 3 for Motor Vehicle Emissions and Fuel Standards (78) as well as the Final Rule to Further Reduce GHGE and Improve Fuel Economy for Model Years 2017 to 2025 for Light-Duty Vehicles. (43) In sensitivity analyses where we modeled vehicle emissions using 2020 data, we found that vehicle emissions rates in 2035 represent approximately 32% of 2020 emissions rates. Thus, if by 2035 there is not compliance with the

current EPA and NHTSA light-duty vehicle GHG emissions and fuel economy regulations our modeled concentrations of $PM_{2.5}$ and NO_x for 2035 will be underestimated. However, we do not expect this to influence our health benefits estimates since we assumed the same standards across our transportation scenarios and focused on differences in concentration between each intervention scenario and the BAU scenario. Finally, we note that the health benefits associated with transportation interventions are expected to occur gradually over time (79) and we assessed the benefits at 2035 assuming that active transportation and physical activity took place in previous years of implementation.

Despite these limitations, this study has important strengths. The primary contribution of this work is our air quality modeling approach. Instead of assuming uniform reductions of VMT due to shifting car trips to active transportation, as most studies have done, we used information on the census tracts of origin and destination of all car trips in Seattle from the Puget Sound Travel Survey. (46) In addition, by using receptors locations throughout the city in our dispersion model, we were able to estimate changes in exposures that would happen throughout the city. Finally, as was mentioned above a strength of this study was the quantification of the impacts of transportation policies on reducing population exposure to NO_x since most previous studies have focused on $PM_{2.5}$.

In conclusion, this study demonstrated that moving towards cleaner vehicles and active transportation can help to improve not only air quality in urban areas but also reduce burden of disease and improve population health. Most health benefits were the result of increased physical activity due to increased active transportation though the use of cleaner vehicles was important for greenhouse gases emissions.

Table 4.1 Modeled daily vehicle miles of travelled, annual average concentration of NO_x and $PM_{2.5}$ and CO_2 emissions by transportation scenario

Variable	Scenario 1: BAU	Scenario 2: Electric vehicles	Scenario 3: Electric vehicles and active transportation
Daily Vehicle Miles of Traveled	11,602,000	11,602,000	10,536,000
Percent reduction from BAU		0.0	9.2
Emissions			
NO_x			
Emissions, tons/year[a]	372.2	342.6	337.5
Percent reduction from BAU		7.9	9.3
$PM_{2.5}$			
Emissions, tons/year[a]	29.8	26.6	24.1
Percent reduction from BAU		10.7	19.3
Atmospheric CO_2			
Emissions, thousands of tons/year[a]	1,068.7	793.4	744.1
Percent reduction from BAU		25.7	30.2
Concentrations			
NO_x, ppb			
Mean concentration (SD)	2.5 (1.2)	2.2 (1.1)	2.2 (1.1)
Mean reduction from BAU (SD)		0.3 (0.1)	0.3 (0.2)
Percent reduction from BAU		10.9	12.9
$PM_{2.5}$, $\mu g/m^3$			
Mean concentration (SD)	0.4 (0.2)	0.4 (0.2)	0.4 (0.2)
Mean reduction from BAU (SD)		0.04 (0.02)	0.08 (0.04)
Percent reduction from BAU		9.3	18.6

[a]Metric tons

Table 4.2 Baseline data and modeled travel times and distances for active transportation

Variable	Scenario 1: BAU	Scenario 3: Electric vehicles and active transportation
Mean daily travel times* **(SD),** minutes		
Walk	7.7 (5.5)	13.9 (4.2)
Bicycle	2.0 (5.2)	21.4 (8.2)
Mean distance traveled, miles		
Walk	0.4	0.6
Bicycle	0.3	2.8
Average speed, MPH		
Walk	2.8	2.8
Bicycle	7.9	7.9

*Geometric mean of weekly physical activity

Table 4.3 Estimated reductions in mortality per year among adults of the general population from reduced exposure to NO_x, $PM_{2.5}$ and increased physical activity due to transportation intervention scenarios as compared with the BAU scenario

Health benefits	Scenario 2: Electric vehicles	Scenario 3: Electric vehicles and active transportation
Air pollution		
NO_2, ppb[a]		
Change in concentration[b] (SD)	0.21 (0.10)	0.24 (0.12)
Premature deaths avoided		
All non-accidental	10 (-1, 21)	11 (-1, 25)
Cardiovascular	4 (-2, 10)	5 (-2, 11)
$PM_{2.5}$, µg/m^3		
Change in concentration[b] (SD)	0.04 (0.02)	0.08 (0.04)
Premature deaths avoided		
All non-accidental	1 (-1, 3)	2 (-2, 7)
Cardiopulmonary	1 (0, 3)	3 (0, 6)
Total air pollution	11 (0, 22)	13 (1, 28)
Physical Activity		
Minutes increased in physical activity[c]		25
Premature deaths avoided		
All-cause		49 (19, 71)
Cardiovascular		21 (9, 25)
Total deaths avoided		
Air pollution and physical activity	11 (0, 22)	62 (28, 84)

[a]NO_2 concentrations were obtained applying a ratio of 0.75 to NO_x concentrations, [b]Mean reduction in the annual average concentration of NO_x (ppb) or $PM_{2.5}$ (µg/m3) in each intervention transportation scenario as compared with the BAU across census tracts. [c] Mean increased minutes in physical activity per person. Population of adults and the expected mortality rate for all non-accidental mortality in Seattle adults for 2035 are 691,000 inhabitants and 644.3/100,000, respectively. Similarly, the population of adults 18-64 years and mortality rate for all-cause mortality (i.e., including injuries) are 540,000 inhabitants and 233.4/100,000, respectively.

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Chapter 5

Discussion

Summary and implications of main findings

This dissertation aimed to better understand the exposure and health impacts of the complex spatiotemporal variations in motor-vehicle traffic patterns on near-road communities and consider how policies might be used to minimize exposures and improve health. From an exposure assessment and environmental epidemiology standpoint, it provides evidence that improvements are needed over standard approaches when assessing near-road exposures to air pollution. Furthermore, from a decision-making perspective this dissertation provides evidence that transportation policies may mitigate the population health burdens of motorized transportation.

In Chapter 2, we estimated air quality modeling results using a source of fine-scale spatiotemporally resolved traffic activity data as compared with more aggregated traffic data on concentrations of pollutants in near road communities. We found that highly variable traffic volumes and speeds across space and time result in similarly complex spatiotemporal variations of air pollutant concentrations in near-road communities. These variations in traffic volumes, vehicle speed, and air pollutant concentrations differ between different highways as well as within individual highways. Even though some of these variations were well captured by traditional aggregated traffic input data, the use of fine-scale scale traffic data adds to the ability of the air quality modeling to capture greater temporal and spatial variability of air pollutant concentrations, especially in communities closest to highly congested highways.

The results in Chapter 2 represent important contributions of this study to the scientific literature. In contrast to prior work, we modeled concentrations of air pollution from highway traffic using fine-scale spatiotemporally detailed traffic data, which has been uncommon in the environmental health science literature due to the lack of available detailed traffic information. (12–15) Although previous studies have argued that aggregated traffic data over space and time may adequately capture the variability of vehicle emissions and air pollutant concentrations, (12,16) we provided strong evidence of large differences in concentrations between the two sources of traffic data over space and time, especially for communities closest to highways and among those with more complex traffic patterns. This highlights the benefits of using more detailed traffic information, especially since using fine-scale spatio-temporal input data only increased the computational demands of our model by 17% as compared to the aggregate data. As such, our study brings the attention of the scientific community to the importance of considering the fine-scale spatiotemporal complexities in traffic patterns and its influence on predicted population exposures to air pollution from highway traffic. Although future research is needed to test this hypothesis, our exposure assessment approach may potentially reduce measurement error and thus minimize bias in epidemiology analyses and increase the power to identify associations. From a regulatory standpoint, our study also brings attention to the importance of improving traffic monitoring to inform traffic regulation.

In Chapter 3, we built upon the work in Chapter 2 by using our models with highly resolved spatiotemporal traffic data in an epidemiological study of associations between air pollutants from highway traffic ($PM_{2.5}$ and NO_x) and daily mortality in communities near highly trafficked highways. Results of this work indicated no conclusive evidence of an increased risk of mortality associated with short-term exposures to air pollutants generated by highway traffic.

However, there was some suggestive evidence that greater short-term exposures to these air pollutants were associated with greater odds of respiratory mortality, with stronger associations for residents nearest to the highways. Contrary to our hypothesis, however, we found reduced odds of cardiovascular and cerebrovascular mortality with higher levels of these air pollutants, again with stronger associations among those living closest to the highways. We hypothesized that competing risks or unmeasured confounding by traffic noise may explain these counter-to-hypothesis findings.

On the other hand, we also did not find any association of our main outcomes of interest with ambient concentrations of $PM_{2.5}$ at AQS monitors. In addition, the fact is that this is not the first study to find limited associations between exposures to ambient air pollutants and mortality in the region. This raises the question as to if there are individual or context-level conditions in this region that may play a role in reducing the risk of this population against the harmful effects of exposure to air pollution. While we did not find evidence that individual and neighborhood-level SES or comorbidities modified our studied associations, additional research could explore other factors such as diet, health behaviors, and the built environment, among others, that might balance the risks of air pollution. If such modifiers did exist, they could contribute to further understanding of the pathways between exposure to air pollutants and health outcomes as well as to provide lights on possible interventions to reduce the risk of mortality associated with air pollution in other populations.

Despite our lack of conclusive results, this study has implications for the scientific literature. The primary contribution of this work is our modeling approach to assess population exposure to air pollutants from highway traffic. Unlike most studies that have used data from regional monitoring stations or aggregated traffic data across space and time, we used highly

resolved traffic data along with a dispersion model to estimate spatiotemporally resolved exposures to air pollutants generated by highway traffic in communities near highways. This exposure assessment approach allowed us to disentangle the contribution of exposure from traffic from other sources in a way that cannot be achieved with the use of more conventional, aggregated metrics of exposure such as those obtained from air quality monitoring stations (AQS). This is important since our modeled concentrations of air pollutants from highway traffic were only moderately correlated with measurements at AQS monitors in the region.

Given the larger literature suggesting important health implications of population exposure to air pollutants from traffic as well as the multiple pathways whereby transportation may affect population health, in Chapter 4, we assessed the health benefits of urban transportation policies scenarios designed to reduce on-road vehicle emissions and increase active transportation. Specifically, we looked at the adoption of electric vehicles (EVs) and replacement of short car trips with walking and bicycling as compared with a business as usual scenario (BAU) for 2035. Results indicated that transportations scenarios promoting EVs and walking and bicycling would result in declines of vehicle miles of travelled from private car use, lower concentrations of primary traffic-generated air pollutants ($PM_{2.5}$ and NO_x) in the community, as well as reduced CO_2 vehicle emissions as compared with a BAU scenario. Replacing short car trips with walking and bicycling would also increase physical activity levels in adults of the general population. Collectively, these reductions would result in fewer premature deaths per year in adults of the general population. Since most health benefits found in this study were the result of increased physical activity due to increased active transportation, transportation policies focused on promoting active transportation and discouraging private vehicle use are likely most efficient at achieving public health goals.

While our findings are not new, an important contribution of this study is our air quality modeling approach. Most previous studies have assumed a uniform reduction of vehicle miles of traveled (VMT) due to shifting car trips to active transportation across a study area. (17–19) In contrast, in our evaluation we used local information on travel patterns and traffic flow across the city allowing us to estimate with spatial resolution localized changes in air pollutant concentrations across scenarios. This approach could help to further estimate whether or not the impacts of transportation policies are homogenously distributed across the city and by characteristics of the neighborhoods and the population in future work.

Another important contribution of this study is that this is one of the few papers to look at the health impacts transportation policies promoting active transportation in the United States. Most previous studies have been done in the context of European countries (17,18,20–23) with baseline rates of walking and bicycling that are much higher than in the United States, where 76.6% of the population drives alone to commute and only 2.8% and 0.6% walk or bicycle, respectively. (24) Hence, interventions promoting changes in transportation behaviors, if successful, could result in greater environmental and health gains in the United States context. As such, this study provides some insights to inform the decision-making of the transportation sector about integrated and realistic transportation policies that could be implemented to help in air pollution control, reductions in green-house-gases emissions, and improvements to public health. Moreover, this study provides information on how transportation policies that encourage active transportation could also help to achieve weekly physical activity goals such as those proposed by the World Health Organizations. (25)

Collectively the results of this dissertation highlight the potential environmental and health implications of motor vehicles in communities. In particular, this dissertation shows

evidence that traffic volumes and flows, and thus vehicle emissions, vary dramatically across locations and times resulting in similar highly spatiotemporal variable air pollutant concentrations in communities near highways. However, most traffic activity monitoring of urban settings in the United States do not capture most of this temporal and spatial variability. Given that we demonstrate that there is a range in the spatiotemporal variability of traffic by location, state implementation plans could identify areas where incorporating additional traffic monitors would be beneficial to capture high variations in traffic patterns or inform traffic regulations. Our results suggest that such monitoring could have the highest value in sections of highways with high traffic density in highly populated areas. Furthermore, this dissertation suggests that some improvements may be needed in environmental health and epidemiologic studies when assessing population exposure to traffic-generated air pollutants to account for highly variable traffic patterns. Finally, this dissertation illustrates that transportation policies may help to control air pollution exposures, mitigate climate change and improve population health. Our results support, that given the high predominance of on-road motorized transportation in the United States, even small changes towards active transportation could translate to large benefits on the population burden of disease.

Strengths and limitations

This dissertation has several strengths. First and foremost, we used novel traffic volume and speed data highly spatiotemporally resolved. These data were available for each half-mile and every 5-minutes for each direction of the major highways in our study region, which is unique in the exposure science and environmental epidemiology literature due to lack of available information with such characteristics. Having leveraged these traffic data with the mobile source

vehicle emissions simulator (MOVES) and a dispersion-based air quality model (RLINE), we were able to model ambient concentrations of air pollutants generated by highway traffic with high spatial and temporal resolution. The contribution of these data in modeling population exposures to air pollution generated by highway traffic was evident especially for communities living very close to highly congested highways.

Another strength of this dissertation and linked to our novel traffic data is our dispersion-based air quality modeling approach. (12,26) In particular, by combining our detailed traffic activity data with MOVES, we developed hourly emission rates for key traffic-generated pollutants for individual half-mile road segments across highways in our study area. Then, by combining these segment-by-segment emissions factors with meteorology in the Research-line source dispersion model (RLINE) we estimated near-road exposures to air pollutants generated by highway traffic across our study area while accounting for localized differences in traffic and, small-scale variations in vehicle emissions. By intersecting traffic activity with emissions and resultant pollutant levels, we could disentangle the contributions from traffic to other sources of air pollution for our epidemiologic and health impact assessment (HIA) analyses. This approach also allowed us to predict concentrations under hypothetical future scenarios. In summary, this air quality modeling approach was the overarching resource for this dissertation that allowed us to contribute to different disciplines in public health such as exposure science, epidemiology and risk assessment and to further understand the environmental and health impacts of urban transportation.

In spite of the strengths of this dissertation, there are some limitations. First, we focused this work (Chapters 2 and 3) exclusively on highway traffic and its contribution to concentrations of air pollutants in nearby communities. Future work might be needed to explore if our findings

hold for the impacts of non-highway roads such as principal and minor arterials that also may have important contributions in ambient concentrations of air pollutant from traffic. Second, despite the strengths of our modeling approach, RLINE is a model that inherently has errors and its performance is sensitive to many factors such as the model input data and assumptions regarding the impact of the local terrain. Despite there is not a perfect validation dataset to confirm that our estimates of traffic-generated pollution were accurate, we anticipate our estimates will be strong since we focus on concentrations within 1 km of the main highways where RLINE has been shown to perform best. In addition, our modeled concentrations showed generally strong correlations with observations at AQS monitors. Similarly, differences in terrain did not appear to influence our observed associations, since we did not find changes in health estimations after accounting for different elevations in our sensitivity analyses. Third, we were able to capture only primary air pollutants concentrations generated by highway traffic, since RLINE does not model chemical reactions in the atmosphere. Thus, we could not estimate concentrations of secondary pollutants such as NO_2, ozone or aerosols. Nor were we able to assess the impacts of traffic noise and ozone, which may confound our analysis in Chapter 3 or act as additional potential pathways between urban transportation and health benefits in Chapter 4. Fourth, given the nature of the data that we had for our association analysis in Chapter 3 (i.e., cases only via death certificates), we were only able to analyze short-term associations between exposures to air pollutants from highway traffic and mortality rather than associations with long-term exposures. Finally, in our HIA we did not quantify additional transport-related risks such as traffic injuries.

Future directions

There are several directions for future research that I could pursue to expand our understanding of the relationship between on-road urban transportation and public health. First, we had hypothesized that having rich spatiotemporal traffic activity data to model concentrations of traffic-generated air pollutants with high temporal and spatial resolution would likely decrease exposure measurement error and thus bias in health model estimates. We also anticipated that these highly variable data would also increase variability of exposure estimates and thus the power to detect associations as compared with the use of typical aggregate measures. However, we did not test these hypotheses explicitly as part of this dissertation. Therefore, we plan future analyses to conduct a simulation study to assess the added benefit in terms of power and bias of both short and long-term health risk estimates that may result from using fine-scale traffic data as compared to other more traditional measures of exposures. For instance, in the analysis of the short-term risk estimates, we could compare our finely resolved air pollutant concentrations with the modeled concentrations obtained from the aggregate traffic data as well as with fluctuations of observations at the nearest AQS monitors. For the long-term risk estimates, we could add to the previous comparisons exposures derived from an inverse distance weighting of levels from the AQS monitors. A simulation approach will be a key component of this future research since this method involve repeated random sampling from probability distributions (i.e., Binomial, Poisson, etc.) to evaluate the performance and accuracy of a variety of methods with regard to a known truth, while allowing us to estimate measures of accuracy (e.g., power, bias, mean squared error and coverage of the estimates of interest). (30) We could also potentially evaluate the importance of these impacts within different subsets of the population to identify for whom these data are most important. Similarly, our future work could explore if there are demographic

and socioeconomic disparities in exposure to traffic-generated air pollution in near-road communities to expand our knowledge of environmental justice issues in the region.

Our future work might also be focused on replicating the methods that we used in Chapter 3 in other regions of the country where there is traffic data of similar fine-scale temporal and spatial resolution as the data used in this study. For instance, in California there are data with high spatiotemporal resolution that to our knowledge have not been used yet in epidemiologic analysis. This replication could help us to explore if the lack of conclusive evidence of an association with traffic-generated pollution from highways and mortality in near road communities in our study was due to our specific region or because there is no association with exposure to primary air pollutants from traffic. Moreover, we could obtain other datasets that have information on both cases and controls in order to explore the long-term health effects of our traffic-generated air pollution concentrations in near road communities.

As for evaluation studies of the health benefits of transportations policies, we could imagine exploring the socioeconomic or race/ethnicity disparities in exposures changes and the population health impacts from the transportation policies. These are likely important factors that need to be further understood to propose more solid and equitable transportation policies. Moreover, additional research may be needed to identify how transportation policies may produce unintended negative effects on certain areas. For instance if the addition of sidewalks or bicycling lanes result in displaced traffic to other areas. Future work could also expand on additional pathways between transportation policies and health impacts by including analyses of traffic injuries as well as personal exposures to traffic-generated air pollution among people changing behavior towards active modes of transportation. In summary, there is a broad range of potential fields of research in this area of transportation policies and public health.

Conclusions

This dissertation showed that spatiotemporal variations between and within highways in traffic patterns are complex and result in similarly complex spatiotemporal variations of air pollutant concentrations in near road communities. The use of fine-scale spatiotemporally resolved traffic input data allow for enhanced characterization of the temporal and spatial variability of air pollutants concentrations in certain communities, especially those closest to highly congested highways. These data further allowed us to investigate potential associations between short-term exposures to traffic-generated pollutants from highways and mortality in near road communities using existing data. We were also able to project future benefits to the broader community of transportation policies that reduce long-term exposures to traffic-generated pollutants from switches to cleaner vehicles and more active transportation. While we did not find conclusive evidence of an increased risk of mortality associated with short-term exposures to air pollutants generated by highways traffic in near-road communities, we project that transportation interventions oriented towards cleaner vehicles and more active transportation would improve air quality and reduce mortality over the long-term, while also reducing green-house gases emissions

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