Mobile Toxics Human Health Risk Assessment Framework

by

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AUTHOR'S DECLARATION

I hereby declare that I am the sole author of this thesis. This is a true copy of the thesis, including any required final revisions, as accepted by my examiners.

I understand that my thesis may be made electronically available to the public.

Abstract

Emissions from passenger cars, buses, commercial trucks, and motorcycles operated on highways, streets, and roads are major contributors to air pollution. Research led by the United States Environmental Protection Agency (U.S. EPA) identified more than 1000 air toxic compounds in exhaust and evaporative emissions from on-road mobile sources. Under a federal mandate, the U.S. EPA is obligated to regulate the emissions of 187 pollutants, known as Hazardous Air Pollutants (HAPs) or air toxics. HAPs emitted from mobile sources are called Mobile Source Air Toxics (MSATs). Compounds within a subgroup of these MSATs are identified by the U.S. EPA as being carcinogens. Additionally, MSATs cause noncancer serious health effects such as tumor formation, cardiovascular disease, damage to the immune system, neurological disorders, reproductive disorders, and respiratory problems.

The U.S. EPA estimates approximately half of the cancer risk from air toxics is attributed to mobile sources, whereas, 74 % of noncancer health impacts from air toxics are a result of exposure to emissions from mobile sources.

The quantification of these risk risks associated with MSATs remains limited to date. Only 20 of the MSATs have ambient air quality standards to protect human health. This work presents a novel and validated approach to quantify the myriad health risks associated with on-road mobile emissions. This approach is introduced in the form of a pipelined analysis process, which may be employed in existing and new road projects.

The result of this research is a new approach to provide regulators and risk analysts a more detailed awareness of the health impacts of these MSATs in current and future contexts.

A distinguished feature between this framework and conventional analysis is providing the handshake between the different models that generate the on-road mobile source emission inventories, conduct the air dispersion modeling, and run the risk engine to calculate the risk estimates. Furthermore, this framework will overcome existing limitations such as roadway geometry characterization in different models.

Acknowledgements

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Dedication

Dedicated to the memory of my dear uncle, Dr. Sami Al-Yakoob

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List of Acronyms

AADT	Annual Average Daily Traffic
AREL	Acute Reference Exposure Level
ADAFs	Age-dependent Adjustment Factors
ADD	Average Daily Dose
ADM	Air Dispersion Modeling
AEGL	Acute Inhalation Exposure Guidelines
AERMOD	American Meteorological Society / EPA Regulatory Model
AIEC	Acute Inhalation Exposure Criteria
AQH	Acute Hazard Quotient
AT	Averaging Time
BMD	Benchmark Dose
BMDL	Statistical Lower Confidence Limit
BW	Body Weight
CAA	Clean Air Act
CAAA	Clean Air Act Amendment
CAS	Chemical Abstracts Service
CDC	Centers for Disease Control and Prevention
CFR	Code of Federal Regulations
COPC	Chemical of Potential Concern
CSF	Cancer Slope Factor
DPM	Diesel Particulate Matter
E85	Ethanol Fuel Blend of 85 %
EC	Exposure Concentration
ED	Exposure Duration
EF	Exposure Frequency
ELCRs	Excess Lifetime Cancer Risks
ERPG-1	Level 1 Emergency Planning Guidelines
FHWA	Federal Highway Administration
Fv	Fraction of MSAT Air Concentration in Vapor Phase
GE	Google Earth
GUI	Graphical User Interface
HAPs	Hazardous Air Pollutants
HED/HEC	Human Equivalent Dose or Concentration
HEM-3	Human Exposure Model
HHRAP	Human Health Risk Assessment Protocol
HI	Hazard Index

HQ	Hazard Quotient
IRIS	Integrated Risk Information System
LADD	Lifetime Average Daily Dose
LEC_{01}	Exposure Concentration Corresponding to the 95 % Lower Bound on the EC_{01}
LED_{10}	Dose Associated with 10 % Response
LOAEL	Lowest-Observed-Adverse-Effect Level
µg	Microgram
mg	Milligram
MSATs	Mobile Source Air Toxics
MOVES	Motor Vehicle Emissions Simulator Model
MPCA	Minnesota Pollution Control Agency
NAAQS	National Ambient Air Quality Standards
NATA	National Air Toxics Assessment
NCEI	National Centers for Environmental Information
NIH	National Institutes of Health
NOAEL	No-Observed-Adverse-Effect Level
OEHHA	Office of Environmental Health Hazard Assessment
OSW	Office of Solid Waste
PB	Particle-bound
$PM_{2.5}$	Particulate Matter Less than 2.5 Micrometers in Diameter
PM_{10}	Particulate Matter Less than 10 Micrometers in Diameter
POD	Point of Departure
RAIMI	Regional Air Modeling Initiative
RfC	Reference Concentration
RfD	Reference Dose
RVP	Reid Vapor Pressure
TEEL	Temporary Emergency Exposure Limits
TOXNET	Toxicology Data Network
TRAQS	Transportation Air Quality System
U.S. EPA	U.S. Environmental Protection Agency
ULSD	Ultra-low Sulfur Diesel
URF	Unit Risk Factor
USGS	U.S. Geological Survey
UTM	Universal Transverse Mercator
VKT	Vehicle Kilometers Traveled
VOC	Volatile Organic Compound
WHO	World Health Organization
WOE	Weight-of-evidence

Chapter 1: INTRODUCTION

1.1 Scope of Research

In 2012, the World Health Organization (WHO) (World Health Organization, 2016) estimates that global premature death as a result of air pollution exposure is broken down into:

- 72 % due to disease and strokes.
- 14 % due to chronic obstructive pulmonary disease or acute lower respiratory infections.
- 14 % due to lung cancer.

Major sources of air pollution include biogenic sources, products of combustion and incomplete combustion, fugitive emissions, and mobile sources. Mobile sources include cars, buses, trucks, commercial marine vessels, railroads, agricultural equipment, and recreational equipment. Mobile sources consist of two major subcategories: On-road mobile sources, which are the focus of this research and non-road mobile sources. Mobile sources emit more than 1000 chemicals into the atmosphere (United States Environmental Protection Agency, 2006).

The scope of this research is to develop a novel methodology to evaluate human health impacts from traffic emissions and resolve current limitations. These emissions, also referred to as Mobile Source Air Toxics (MSATs), are currently the biggest air pollution health issue in North America and Europe. The research includes a case study with a modeled versus measured analysis to validate the proposed approach. Moreover, peer-reviewed models and proven technologies are employed to advance the understanding of evaluating health impacts, as a result of exposure to MSATs. The outcome is a streamlined MSATs methodology for conducting human health risk assessments. Finally, this methodology can assist policy-makers in making decisions on the basis of science.

1.2 Research Objective

The objective of this research is to develop and validate a new methodology to evaluate human health impacts from exposure to mobile source air toxics (MSATs). This is a challenging research since it must integrate multi-disciplinary fields including the estimation of mobile source emissions of over a thousand air toxics, the atmospheric dispersion of these toxics, the fate and transport of chemicals through various media, the assessment of the dose on humans, and its final combined toxicological risks.

1.3 Thesis Outline

This thesis is divided into six chapters. The chapters are outlined as follows:

- Chapter 1 – Introduction.
- Chapter 2 – Methodology.
- Chapter 3 – Chemicals of Potential Concern.
- Chapter 4 – Multi-pathway Fate-and-transport Risk Assessment.
- Chapter 5 – Analysis of Results and Validation.
- Chapter 6 – Conclusion, Uncertainty Analysis, and Future Work.

1.4 Literature Review

1.4.1 MPCA Statewide Cumulative Risk Study

The Minnesota Pollution Control Agency (MPCA), conducted a statewide screening level human health risk assessment of all inventoried emission sources located in Minnesota. The MPCA conducted the cumulative risk study in accordance with the methodologies and science contained in the 2005 U.S. EPA Human Health Risk Assessment Protocol (HHRAP) (U.S. Environmental Protection Agency, 2005). MPCA contracted with Lakes Environmental Software, to design and develop a state-of-the-science tool that automated the emission inventory, air dispersion modeling, and risk assessment. The tool, MNRISKs, incorporates the U.S. EPA's AERMOD air dispersion model (United States Environmental Protection Agency, 2015) and risk assessment protocols outlined in the HHRAP to predict cancer risk and noncancer hazard indices. In conclusion, the study provided evidence of the viability of this advanced human health risk assessment system, and its approach, for conducting large-scale analyses of air toxics health effects.

As a result of this study, a collection of peer-reviewed articles were published (Pratt, Dymond, Ellickson, & The´, 2012), and confirmed the necessity of evaluating emissions from on-road sources and how they contribute to adverse health effects resulting from acute and / or chronic exposure. MPCA continues to conduct updated risk assessments for the entire state every three years.

Regulatory benefits of investing in a state-of-the-science cumulative human health risk assessment modeling system include the ability to:

1. Identify likely health outcomes in the population including asthma rates, cancers, and other expected disease outcomes. This information can be cross correlated with epidemiology data (e.g., hospital admissions) to further prioritize prevention, intervention and treatment strategies.
2. Develop effective risk communication strategies and informative educational programs based on cumulative health study results.
3. Quantify and compare potential health risks based on evaluation of actual, allowable, and maximum emissions limits.

1.4.2 Regional Air Modeling Initiative (RAIMI) – U.S. EPA Region 6

The Regional Air Impact Modeling Initiative (RAIMI) is a suite of integrated software tools developed by the U.S. EPA Region 6 Compliance Assurance and Enforcement Division. The RAIMI program and modeling tools were developed to conduct community wide cumulative air dispersion and human health risk assessment modeling for hundreds of air toxics being emitted from thousands of air pollution sources. Human health risk assessment modeling includes evaluation of chronic cancer risk and noncancer hazard as well as short term acute exposure. Exposure pathways include direct inhalation and multiple indirect pathways including water, plant, and animal tissue ingestion. The first RAIMI pilot study was conducted in Harris County Texas, which includes the Houston Ship Channel industrial corridor. The RAIMI tool is limited to calculating point source data only. For on-road sources, ambient air concentrations were estimated for six hazardous air pollutants (HAPs) in a study area containing four air monitoring stations. The Human Exposure Model (HEM-3) is the model used to estimate emissions from on-road sources. The RAIMI program was designed to manage the large data sets associated with community wide modeling studies and is ideally suited to calculate cumulative risks while also identifying contributing emission sources, pollutants, and exposure pathways driving risk estimates.

1.5 Framework

This framework will serve as the groundwork for conducting multi-pathway risk assessments due to mobile source air toxics exposure. The framework consists of the following components as shown in Figure 1:

1. Generating the emissions inventory for on-road sources in a readable format by the air dispersion model (e.g., AERMOD).

2. Conducting the air dispersion and deposition modeling for the on-road sources to estimate the downwind ambient air concentrations and deposition fluxes.
3. Calculation of the fate and transport of MSATs in other media (e.g., soil, air, beef, dairy, poultry, etc.).
4. Estimation of exposure scenarios.
5. Estimation of risks via each exposure pathway for each modeled source and MSATs.

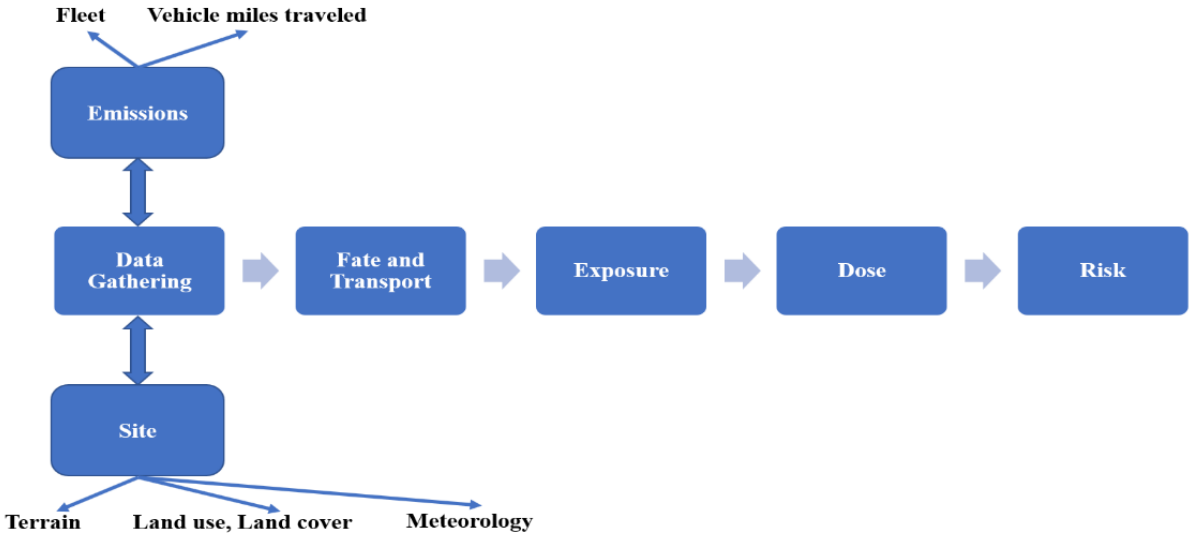


Figure 1: Human Health Risk Assessment Flowchart

The framework builds upon principles outlined in the U.S. EPA Human Health Risk Assessment Protocol (HHRAP) (U.S. Environmental Protection Agency, 2005). It is important to note that availability and completeness of on-road emissions data is of key importance to assess human health risk associated with on-road emissions exposure.

1.6 Mobile Source Air Toxics (MSATs)

The Clean Air Act Amendment of 1990 (United States Environmental Protection Agency, 1990) is a federal law that requires the United States Environmental Protection Agency to regulate the emissions of 187 air toxics (United States Environmental Protection Agency, 2005). Air toxics emitted from on-road sources such as highway vehicles and non-road equipment like farm tractors and road graders are called Mobile Source Air Toxics (MSATs). The research focuses on MSATs emitted from on-road sources. A

percentage of MSATs are found in fuels and emitted by on-road sources into the atmosphere because of fuel evaporation or when unburned fuel passes through the engine. On-road sources also emit MSATs as a consequence of incomplete combustion of fuels or as a by-product of combustion (i.e., secondary combustion products). Another significant source of emission, is the secondary formation of MSATs due to reaction of primary compounds in the atmosphere (Corrêa, et al., 2010). Furthermore, engine and brake wear, abrasion and corrosion of vehicle components, and, impurities in oil or gasoline generate a significant amount of metal MSATs. The U.S. EPA identified 9 compounds emitted from mobile sources, 8 of which are from the 187 air toxics list, in addition to diesel particulate matter as large contributors to overall risk in the 2011 National Air Toxics Assessment (NATA) (United States Environmental Protection Agency, 2011). These compounds are 1,3-Butadiene, Acetaldehyde, Acrolein, Benzene, Ethyl benzene, Formaldehyde, Naphthalene, Polycyclic Organic Matter, and Diesel Exhaust Particles also known as Diesel Particulate Matter (DPM). The Federal Highway Administration (FHWA) (Federal Highway Administration, 2016), classifies the forestated air toxics as priority mobile source air toxics, meaning they are considered high priority, the word "priority" is used in the sense of being the most important, and the designation has legal implications for reporting and inclusion in studies. These MSATs present the greatest threat to public health as per the 2011 NATA. Health problems include cancer and noncancer health risks like immune system and nervous system problems. Policies for mobile source air toxics are limited in scope and incomprehensive when compared to criteria air pollutants regulations (i.e., NAAQS) (United States Environmental Protection Agency, 1970), and many of these MSATs are persistent. Moreover, MSATs concentrations are amplified as they move from one trophic level to the next.

In this research, we use the following terms interchangeably: MSATs, COPCs, and Chemicals. For the air concentrations modeled versus monitored analysis (validation study), we modeled the following MSATs: Benzene, 1,3-Butadiene, Formaldehyde, Toluene, and Xylene since we had monitored and modeled data for on-road vehicle emissions as shown in Figure 2.

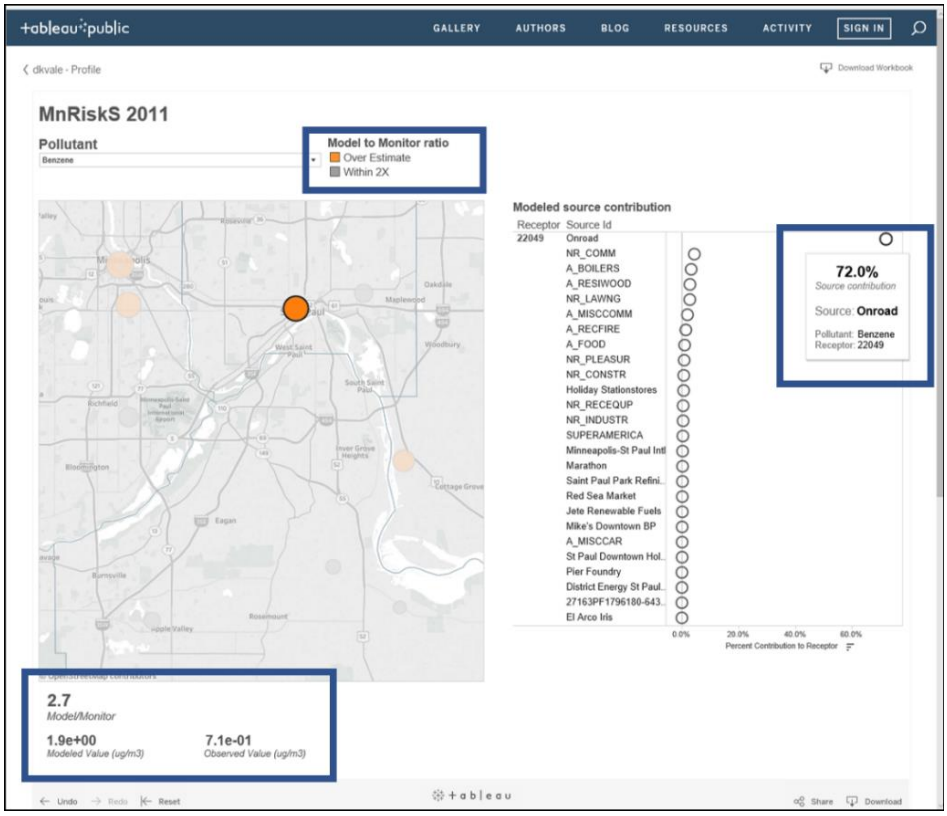


Figure 2: Benzene Emissions Data at Saint Paul – Ramsey Health Center Air Monitoring Station

Figure 2 shows the publicly available Benzene data, the site contains information about the modeled Benzene concentrations, monitored Benzene concentrations, and the on-road sources contribution to the receptor.

In the cumulative risk analysis, the study focused and analyzed MSATs summarized in Table 1. As per the 2011 NATA, Benzene is designated as regional cancer risk driver, Formaldehyde is a national cancer risk driver, and 1,3-Butadiene and hexavalent chromium are national cancer risk contributors.

U.S. EPA classifies Indeno(1,2,3-cd)pyrene as probable human carcinogen and Benzo(a)pyrene as carcinogenic to humans (United States Environmental Protection Agency, 2018).

Table 1: MSATs Included in the Cumulative Risk Analysis

Mobile Source Air Toxic	CAS	Fv[1]	Assumed emitted as vapor, particle, or vapor with portion particle-bound (PB)
Benzene	71-43-2	1	Vapor
Formaldehyde	50-00-0	1	Vapor
1,3-Butadiene	106-99-0	1	Vapor
Hexavalent Chromium	18540-29-9	0	Particle
Indeno(1,2,3-cd)pyrene	193-39-5	0.005	Particle
Benzo(a)pyrene	50-32-8	0.294	29.4 % V, 70.6 % PB

1.7 Models for the Validation Study

The following sections describe the models included in this analysis:

1. U.S. EPA MOVES—Estimates emissions for on-road mobile sources.
2. AERMOD—Regulatory steady-state plume model for air dispersion modeling.
3. Risk Engine (IRAP-h View)—Calculates risk values for multiple MSATs, from multiple on-road sources, at multiple exposure locations.

1.7.1 On-Road Mobile Source Emission Inventory

U.S. EPA's Motor Vehicle Emissions Simulator Model (MOVES) will produce the mobile sources inventory. The MOVES model estimates the emission factors and emissions inventories for the following pollutants: Particulate Matter (PM_{10} and $PM_{2.5}$), Nitrous Oxides, Carbon Monoxide, Greenhouse Gases, Metals, and MSATs, which are the focus for this work. MOVES is capable of modeling continuous releases of mobile source air toxics with over 50 different exhaust and evaporative species. Moreover, the model, accounts for various fuel types (e.g., Diesel, Gasoline, Ethanol fuel blend of 85 % (E85)) used by a range of mobile sources such as cars, buses, motorcycles, and haul trucks. Furthermore, the model includes different emission rates for each combination of sources, age groups, and operating modes and it accurately reflects the various vehicle operating processes, such as running exhaust, crankcase running exhaust, cold start or extended idle, and provides estimates of bulk emissions or emission rates. The modeler enters the vehicle activity data into MOVES as a series of link drive schedules to represent each segments of cruise, deceleration, idle and acceleration of a congested intersection. The 2014 version of the MOVES model (MOVES2014) is the preferred model and U.S. EPA requires all transportation

1 : Fraction of MSAT air concentration in vapor phase

conformity analyses be modeled using MOVES2014 (United States Environmental Protection Agency, 2017).

This is the first step in the risk assessment, as the mobile sources inventory constitutes the feedstock to the air dispersion model.

1.7.2 Air Dispersion and Deposition Modeling

Before conducting any human health risk assessment, it is very important to understand the fate and transport of MSATs, this is done using air dispersion modeling (ADM). Air dispersion modeling is a computer simulation that expresses the relationship between the various variables of a system in mathematical terms. Air dispersion models predict the downwind concentrations of a source, provided, the model is fed with sufficient data such as source characteristics (location, source release parameters, etc.), meteorological conditions, and terrain data. Furthermore, air dispersion modeling, helps us better understand how pollutants amplify existing conditions.

Air dispersion modeling is used to provide fine spatial resolution of emission allocation, determine mobile source emission contributions to ambient air quality, and verify emission inventory.

Air dispersion models estimate the air concentrations and deposition rates of MSATs. These dispersion models also estimate the impact of chemical partitioning into vegetation, soil, and water bodies such as lakes, rivers, and wetlands.

Air dispersion and deposition modeling conducted for this analysis was done using the U.S. EPA preferred model for air dispersion modeling for near-field applications, AERMOD (version16216r), and follows the procedures recommended by the U.S. EPA to conduct human health risk assessment. AERMOD is a steady-state Gaussian plume air dispersion model. Figure 3 shows a schematic of the Gaussian plume.

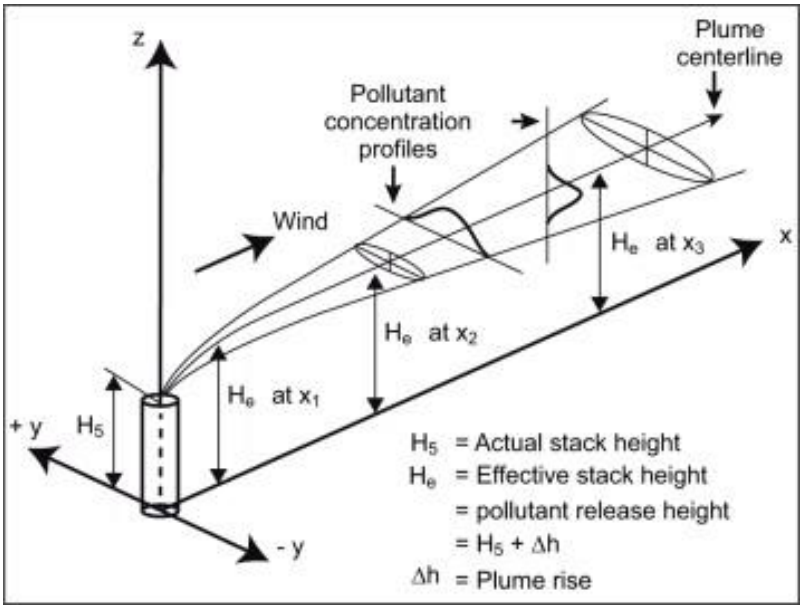


Figure 3: Schematic Representation of Gaussian Plume

The model calculates concentrations based upon the mathematical principles of plume rise, atmospheric dispersion of buoyant (or neutrally-buoyant) effluent, turbulence theory, and Gaussian distribution (Vesilind, Peirce, & Weiner, 2013) as presented in (Equation 1). The output concentrations are the result of the model's calculations which vary depending upon numerous inputs such as source type, input parameters, meteorology, terrain, and more.

$$\boldsymbol{C(x,y,z) = \frac{Q}{2\pi u \sigma_y \sigma_z} exp\left(-\frac{y^2}{2\sigma_y^2}\right)\left(exp\left(-\frac{(z+H)^2}{2\sigma_z^2}\right) + exp\left(-\frac{(z-H)^2}{2\sigma_z^2}\right)\right),} \qquad \textbf{Eq. 1}$$

Where:

$C(x, y, z) =$ concentration at some point in space with coordinates x, y, z

Q= emission rate of the pollution source in grams per second

u= average wind speed in meters per second

σ_y= standard deviation of the plume in the y direction (m)

σ_z= standard deviation of the plume in the z direction (m)

Turbulence parameterization is based on the Monin-Obukhov similarity theory. The model heavily relies on surface weather observations to create vertical profiles of temperature, temperature gradient, wind speed, wind direction, and turbulent velocities in the atmospheric boundary layer. This process is handled

in the U.S. EPA AERMET model, the meteorological preprocessor that generates AERMOD meteorological data files.

Links and roadway segments are terms used interchangeably in this framework and they are sites where similar vehicular activity occurs. Vehicle activity over the length of the links vary due to acceleration, deceleration, cruising and / or idling. Links classify into two types:

1. Running Links such as ramps, intersections, and free-flow highways.
2. Off-Network Links such as locations that include extended idling and / or running emissions like parking facilities, and transit facilities.

For the 47 links we modeled, we conducted the air dispersion modeling using the *Unitized Emission Concept* of a "generic" MSAT as demonstrated in Figure 4, known as the Unit Emission Rate, a term coined by Professor Thé (The' & Weeks, 2007).

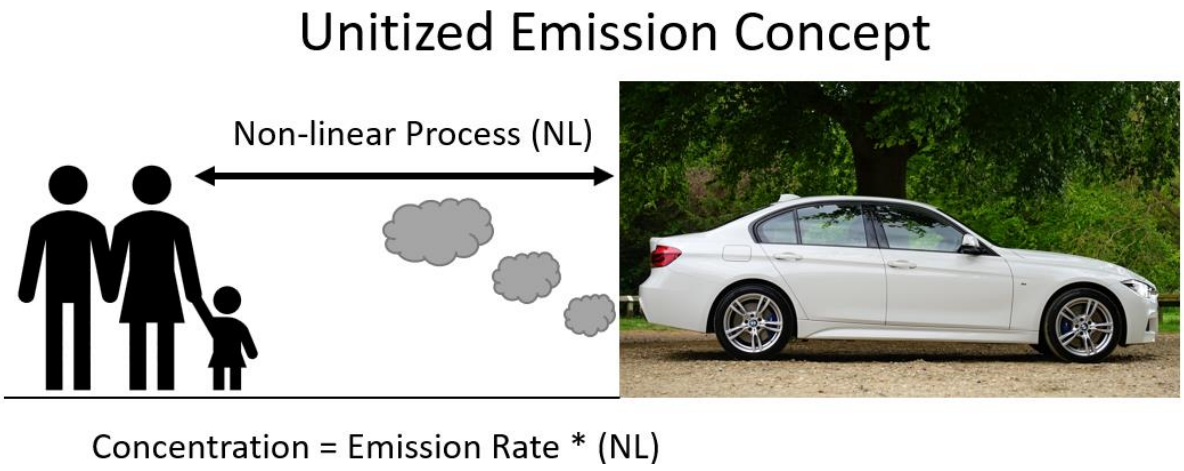


Figure 4: Unitized Emission Concept

The MSATs specific emission rates are multiplied by the appropriate air modeling parameter outside the air dispersion model (i.e., IRAP-h View). IRAP-h View (Lakes Environmental Software, 2018)is the GUI for conducting the multi-pathway human health risk assessment based on the U.S. EPA Human Health Risk Assessment Protocol (HHRAP) (U.S. Environmental Protection Agency, 2005).

Unit emission rate serves two purposes:

1. Minimize calculations imprecision by low emissions values.
2. Eliminate multiple dispersion runs for each modeled MSAT.

To put it more simply, this saves the modeler from having to generate a model run for each MSAT for IRAP-h View, which would be painful for a vehicle that emits more than one MSAT into the environment.

A more detailed description of how we modeled the links will follow in chapter 2.

Many factors contribute to the complexity of this modeling process, such as secondary formation of MSATs in the atmosphere like Acetaldehyde and Formaldehyde (Corrêa, et al., 2010), mechanical and thermal induced turbulence, transformation of roadway segments to spatially aware sources for input in the air dispersion model, and uncertainties in the MSATs emission inventories due to changing operating conditions, higher evaporative losses in warm weather, and changing warm up and idle cycles.

Another challenge identified in this segment of the research is the averaging of mobile source air toxics; criteria pollutants have set ambient air quality concentration standards as per the National Ambient Air Quality Standards (NAAQS) (United States Environmental Protection Agency, 1970). However, MSATs lack these defined health safety levels (Federal Highway Administration, 2016) resulting in a subjective evaluation of how bad they are. As per the HHRAP (U.S. Environmental Protection Agency, 2005) recommendations, we modeled for 1-hour and annual air concentrations for vapor, particle, and particle-bound chemical phases resulting from on-road sources.

1.7.3 Human Health Risk Assessment Protocol

The U.S. EPA OSW Human Health Risk Assessment Protocol (HHRAP) (U.S. Environmental Protection Agency, 2005) was developed as a national guidance for the U.S. EPA and its authorized states on how to perform human health risk assessments at hazardous waste combustion facilities. The purpose of a human health risk assessment is to use existing information to estimate the risk to human health (cancer risks and noncancer hazards) resulting from emissions of hazardous waste combustion units. The document was finalized in September 2015 and was assigned the following U.S. EPA publication number, EPA530-R-05-006.

Even though the HHRAP was developed for combustion facilities, it includes methodologies, equations, and procedures for emissions source parameterization, air dispersion and deposition modeling, and equations and recommended default variable values for estimating media concentrations for assessment of air toxics exposure.

The research adapted, extended, and applied the HHRAP in evaluating health risks due to MSATs exposure.

1.7.4 Human Health Risk Assessment

Mobile source air toxics do not have established National Ambient Air Quality Standards to protect human health, these standards cover mostly criteria pollutants: Nitrogen dioxide, Sulfur dioxide, Carbon monoxide, Ozone, Lead, and Particulate Matter (PM_{10} and $PM_{2.5}$). Many of the MSATs are orders of magnitude more toxic than the regulated criteria pollutants and bioaccumulate in the food chain.

MSATs such as Benzo(a)pyrene and Indeno(1,2,3-cd)pyrene deposit in the environment, where humans are exposed via indirect pathways further contributing to risk already caused by inhalation exposure.

Numerous peer-reviewed studies have clearly demonstrated the largest urban contributor to air quality human health impacts are mobile source emissions (Pratt, Dymond, Ellickson, & The´, 2012).

A risk assessment is necessary when the following factors are present:

1. An event where there is a likelihood of release of contaminants of potential concern.
2. Presence of human and ecological receptors.
3. Existing pathways for human exposure to COPC.

Such assessments, commonly, employ conservative (worst-case) assumptions that produce amplified risk estimates, this doesn't necessarily reflect real-world conditions, but does represent overestimated exposure conditions. If the risk results are within the permissible limit, then there is no need for corrective action. On the other hand, if the assessment outcome is not within the permissible limit, it is imperative to go back to the underlying assumptions to better understand the risk driver and propose abatement measures.

On-road sources release COPCs into the environment where people live, go to school, and work through vehicles tailpipe, evaporative emissions (fuel in tank, fuel lines, and fittings), engine oil spills leaked onto heated parts of the engine, and products of wear and tear such as tire wear and metallic components from the brakes (brake pads and rotors).

A study conducted in 2001 by the American Housing Survey estimates over 35 million people live near (i.e., within 91 meter of a 4-lane road) mobile source emissions (United States Environmental Protection Agency, 2007)

Humans are exposed to these chemicals via multiple pathways (routes) such as inhalation, ingestion of produce, beef, pork, and poultry, or through dermal absorption. Risk resulting from inhalation (direct

pathway) is known as inhalation risk. COPCs such as dioxins and furans (DeRose, 2009) transferred via indirect pathways have a higher risk than the direct pathway risk by orders of magnitude.

Human health risk assessments are clunky and cumbersome. Risk assessments incorporate conservative approaches due to the presence of data gaps. Also, the mechanism of human exposure to MSATs is very convoluted, this is mainly due to the complex MSATs mixtures which contain other pollutants which results in unknown MSATs contributing to the deterioration of human health.

Our aim is to conduct the risk assessment following the methodologies outlined in the Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities (HHRAP) (U.S. Environmental Protection Agency, 2005). HHRAP is a guidance for conducting multi-pathway, site-specific human health risk assessments on stationary combustion sources such as incinerators, furnaces, except for on-road sources.

The equations, methodologies, and science for source parameterization, air dispersion / deposition modeling, and media concentrations in the HHRAP will be custom-tailored to our research in evaluating risk due to MSATs exposure.

The HHRAP implements and incorporates other U.S. EPA guidance resources like:

1. Integrated Risk Information System (IRIS) (United States Environmental Protection Agency, 2018).
2. Exposure factors handbook (United States Environmental Protection Agency, 2011)
3. Methodology for assessing health risks associated with multiple pathways of exposure to combustor emissions.

In conclusion, the HHRAP summarizes the procedures, equations, parameter values, and inputs required to perform risk evaluations, and addresses issues identified while conducting risk assessments.

Air dispersion modeling estimates concentrations and deposition fluxes of MSATs. The resultant files,

1-hour and annual averages are known as AERMOD plot files. The AERMOD plot files serve as input data for the risk engine. IRAP-h View software is the risk engine used in this framework. The science used in the risk engine adheres to the methodologies presented in the HHRAP.

IRAP-h View calculates the risk values (cancer risks and noncancer hazards numerical estimates) for multiple MSATs, from multiple road segments, at multiple exposure locations. This constitutes the final step in our proposed methodology.

1.8 Area of Study

The location of the high traffic roadway segments analyzed is the Interstate-35E and I -94/US 10 in Saint Paul junction Figure 5. Figure 5 was generated by enabling the Google Maps traffic layer. In 2011, annual average daily traffic (AADT) exceeded 183,000 vehicles (Minnesota Department of Transportation) (Minnesota Department of Transportation, 2018). The studied roadway segment is in the Ramsey County, Minnesota.

The reason for selecting these segments is because they rank among the highest AADT in the Ramsey county and due to the presence of near-roadway air monitoring stations (Saint Paul – Ramsey Health Center) within the project area. The Saint Paul – Ramsey Health Center air monitoring station is located at Universal Transverse Mercator (UTM) Easting: 492230.00 m, Northing: 4977478.00 m, and zone 15.

The modeled versus measured section of this framework provides detailed information about the air monitoring station and the mobile source air toxics measured at that location.

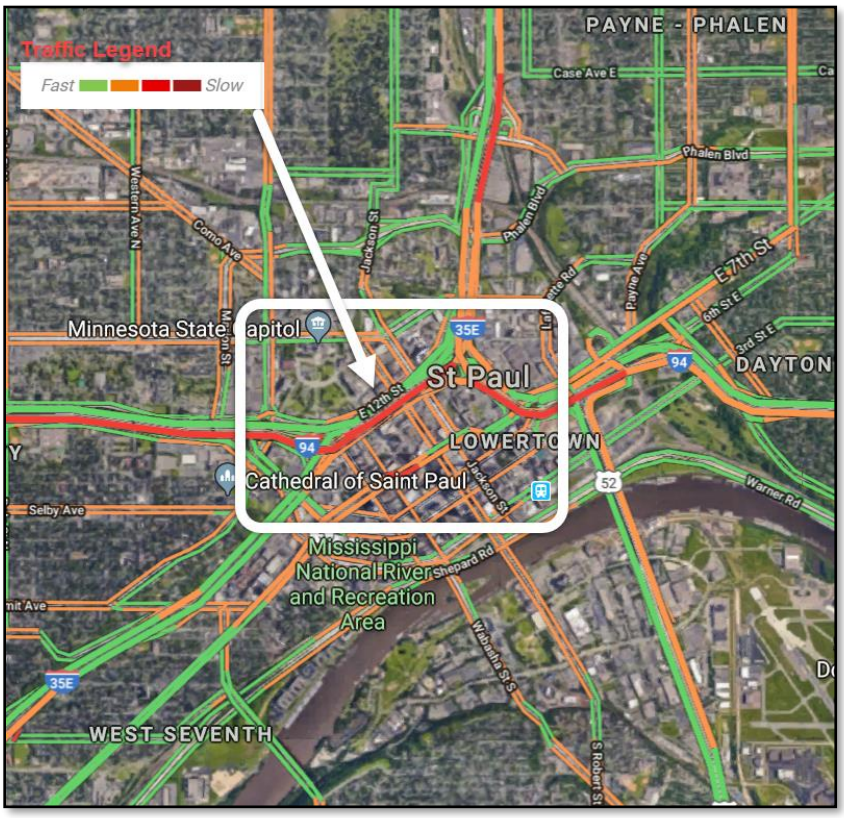


Figure 5: Traffic Conditions Shown in Area of Study

1.9 Transportation Air Quality System (TRAQS)

The Transportation Air Quality System (TRAQS) is an open-source software designed to conduct mobile air quality assessments. TRAQS is designed to automate the modeling process and minimize any limitations of these standalone models by seamlessly integrating both MOVES and AERMOD into a user-friendly and intuitive Graphical User Interface (GUI). Figure 6 visualizes how a modeler can easily enter traffic data such as number of vehicles for each the 16 modeled scenarios. Future work in the pipeline includes the development of a web-based (cloud solution) roadway segments risk assessment module for TRAQS to ensure the modeling community have the tools and expertise necessary to quantify exposure to mobile source air toxics. The author participated in the development and validation of TRAQS.

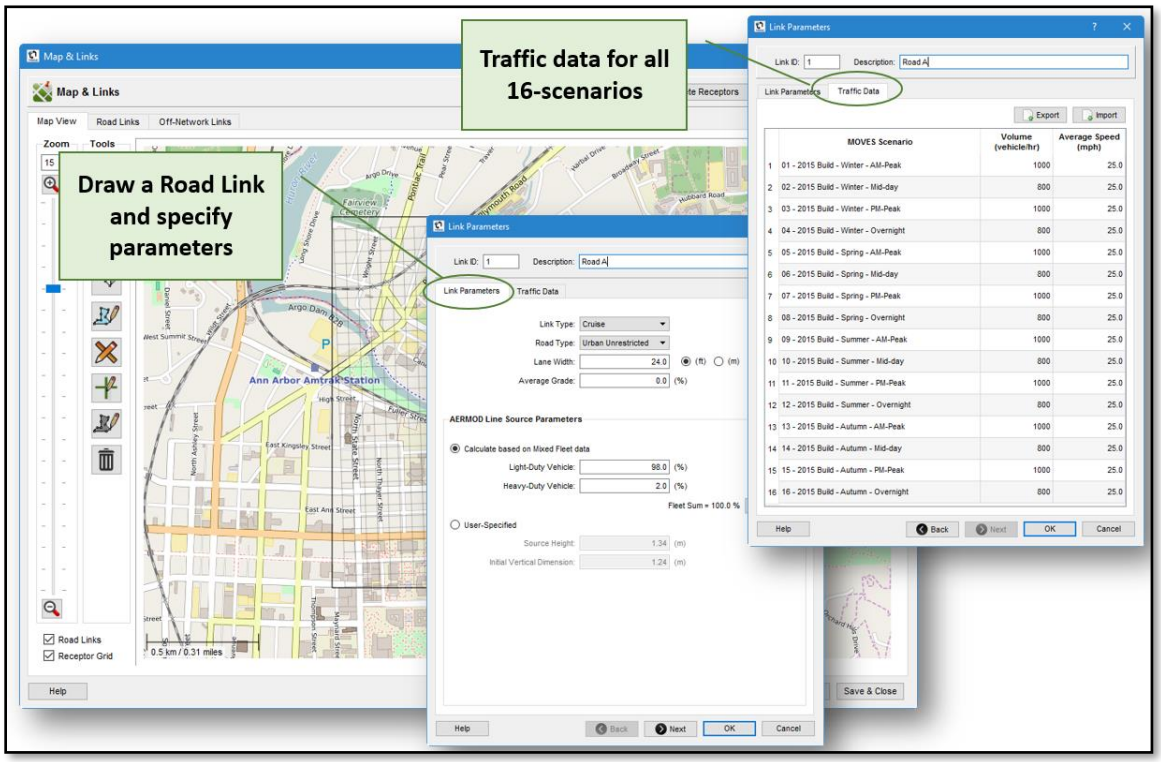


Figure 6: TRAQS – Traffic Data Entry

1.10 National Ambient Air Quality Standards (NAAQS)

The Clean Air Act (CAA) of 1970, a federal law passed by the senate and house of representatives of the United States, requires the U.S. EPA to set standards for air pollutants known to cause health problems and damage to the environment. The federal law included six criteria air pollutants with primary and secondary standards (United States Environmental Protection Agency, 1970). The primary standards are set to provide public health protection and the secondary standards provide public welfare protection. The current NAAQS includes 12 different standards for 7 pollutants, these pollutants are: carbon monoxide, lead, nitrogen dioxide, ozone, particulate matter 10 micrometers or less in aerodynamic diameter, particulate matter 2.5 micrometers or less in aerodynamic diameter, and sulfur dioxide. The amended 1990 Clean Air Act requires the U.S. EPA to regulate the emissions of an additional 187 pollutants, known as Hazardous Air Pollutants (HAPs) or air toxics (United States Environmental Protection Agency, 2005). Only 20 mobile source air toxics, a subgroup of the 187 pollutants, have established ambient air quality standards to protect human health. 9 mobile source air toxics are classified are priority pollutants, these MSATs are 1,3-Butadiene, Acetaldehyde, Acrolein, Benzene, Ethyl benzene, Formaldehyde, Naphthalene, Polycyclic Organic Matter, and Diesel Particulate Matter (Federal Highway Administration, 2016).

Chapter 2: METHODOLOGY

2.1 Methodology Overview

The proposed methodology estimates the impacts from road emissions on human health. The approach goes one step further than current practices. The steps in the methodology are:

1. Estimate emissions from vehicles on road.
2. Compute the transport of these pollutants away from the roads towards human receptors.
3. Assess critical air concentrations of the MSATS – **This is where current practices stop.**
4. Conduct a multi-pathway fate-and-transport analysis of the MSATs.
5. Assess the total dose from direct inhalation and indirect pathways to human.
6. Determine the total cumulative human health impact based on the MSATs doses.

2.2 On-road Emissions

On-road sources are ubiquitous and abundant. The mobile source air toxics emitted from all the on-road sources accumulate and their harmful effects are magnified. Studies have shown that emission exposure has led to increased morbidity and mortality rates for drivers and people living near congested roadways (Zhang & Batterman, 2013).

On-road emissions contribute greatly to emissions inventory. Direct emissions from on-road sources contributed to 10 % of major sources of 2011 air toxics emissions in Minnesota (Minnesota Pollution Control Agency, 2016) as shown in Figure 7 and this is approximately equivalent to 17,700 tons. Light-duty gasoline trucks are the major contributors to the total mobile source air toxics emissions in 2011, representing about 54 % of total emissions for that category (Minnesota Pollution Control Agency, 2016).

On-road emissions must be estimated based on a series of factors. U.S. EPA's MOVES2014 modeling software generates the on-road emissions. MOVES uses a different emissions rate for each combination of source, vehicle age group, and operating mode. The model takes into account a broad array of input such as vehicle kilometers traveled, vehicle types on the link during the specified modeling period, meteorological data, vehicle type / fuel combination, source type population, vehicle age distribution, and so on. Based on the user-specified inputs, the MOVES model performs calculations that simulate the

various vehicle emission processes and generates the results in the form of emission rates or total emissions.

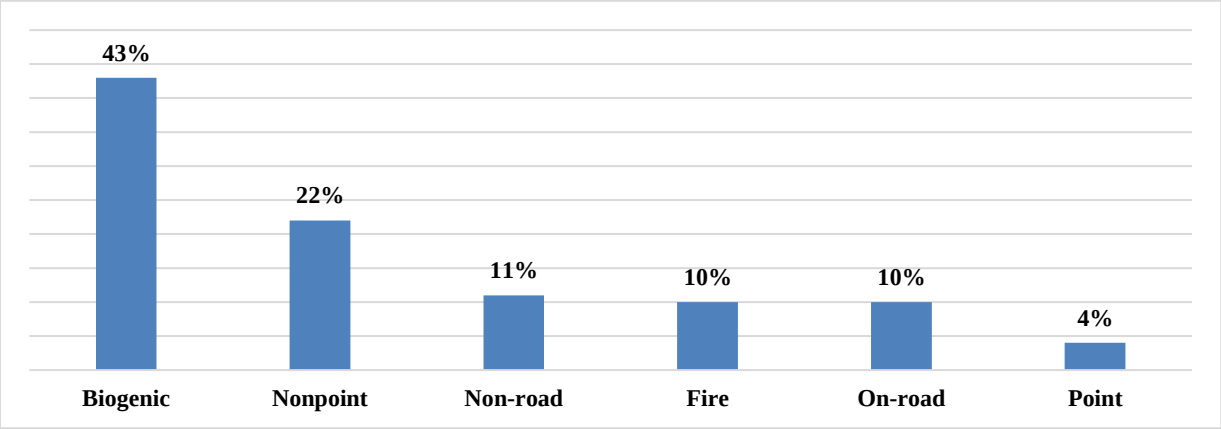


Figure 7: Major Sources of 2011 Air Toxics Emissions in Minnesota

2.3 Modeling Road Segments (Sources)

The U.S. EPA has ruled that AERMOD is the preferred model for on-road sources (United States Environmental Protection Agency, 2016), this preferred status comes as a result of:

1. Availability of modeled versus measured studies.
2. AERMOD is a well-documented model.
3. Availability of test datasets for model evaluation.
4. Sulfur hexafluoride tracer studies demonstrated that AERMOD outperformed CALINE3 and CALINE4.

Extensive data analysis focusing on the highest concentrations and numerous model performance studies concluded that AERMOD is the best performing model for on-road source applications (United States Environmental Protection Agency, 2016).

Good-practice modeling protocols recommend classifying the road itself as the emission source rather than tracking individual vehicles (United States Environmental Protection Agency, 2015).

The Haul Road Workgroup Final Report (United States Environmental Protection Agency, 2012), one of the first published documents of its kind to recommend how to model roads (in this case unpaved ones but roads nonetheless) has become the de facto standard for source characterization.

The procedure to convert the roadway segments into AERMOD emission sources is outlined in the Haul Road Workgroup Final Report (United States Environmental Protection Agency, 2012).

2.4 On-road Emissions Estimation

Methods used to estimate emissions from on-road sources vary. For example, older methods included calculating emissions by multiplying the activity rate (i.e., vehicle kilometers traveled) and the emission factor together. The emission factors are available in Volume II 4th edition of AP-42 (United States Environmental Protection Agency, 1985). Progress in science and technology led to the inception of computer models, which automated and eliminated the tedious hand calculations. The MOBILE model (United States Environmental Protection Agency) was first introduced in 1978 and was superseded by the Motor Vehicle Emission Simulator (MOVES) in 2004. Forty-nine states require the use of the MOVES2014 model to estimate emissions from on-road mobile sources, while California uses the EMFAC2014 model (California Air Resources Board, 2014). The U.S. EPA has yet to approve EMFAC2017. EMFAC2017 was released by the California Air Resources Board in December 2017. This framework can use either model. However, our case study employs the MOVES model.

Mobile sources are divided into two categories:

1. On-road mobile sources.
2. Non-road mobile sources.

Non-road mobile sources include farm tractors, road graders, aircrafts, locomotives, and other categories. The focus of this work is On-road mobile sources. On-road mobile sources are categorized into the groups summarized in Table 2.

Table 2: On-road Mobile Sources Categories

On-Road Mobile Sources
Motorcycle
Passenger Car
Passenger Truck
Light Commercial Truck
Intercity Bus
Transit Bus
School Bus
Refuse Truck
Single Unit Short-haul Truck
Single Unit Long-haul Truck
Motor Home
Combination Short-haul Truck
Combination Long-haul Truck

The MOVES model contains default databases for each county in the United States to perform the emission calculation. Nevertheless, input requirements vary depending on the scope of the project, whether modeling a county or performing a more refined analysis (i.e., Project-Level $PM_{2.5}$ Conformity Analysis).

For transportation conformity analyses, the modeler needs to supply extensive project-specific inputs such as vehicle age distribution, meteorological data, fuel data (fuel usage and fuel formulation), operating mode data, regulatory control programs, vehicle fleet characteristics, temporal distributions, and roadway segments data. Segment data include road length, width, vehicle counts, and road gradient.

Table 3 below, presents vehicle activity occurring on roadway segment and its recommended application.

Table 3: Vehicle Activity in MOVES with Respective Application

MODE Type	Application
Operating Mode	Emission estimates are based on the duration the source spends in various operating modes such as accelerating, cruising, or idling.
Average Speed	Emission estimates are based on default drive cycles.
Link Drive Schedules	Emission estimates are based on user-specified drive cycles.

One significant difference between MOVES and its predecessor is the reliance on operating modes to calculate emissions. MOBILE models were based on aggregate driving cycles and only took into consideration the differences in average speeds. MOVES can model conventional and alternative fuels such as gasoline, diesel, compressed natural gas, liquid petroleum gas, ethanol fuel blend of 85 %, and electricity. Furthermore, MOVES includes a built-in fuel wizard to create custom fuels.

MOVES estimates emissions from the following processes: running exhaust, start exhaust, extended idle, evaporative emissions (permeation, vapor venting, liquid leaks), refueling (vapor loss, spillage), crankcase exhaust, tire wear, and brake wear. MOVES2014 was used to prepare the on-road emissions. The emissions modeling was set to align U.S. EPA's 2011 National Emissions Inventory (United States Environmental Protection Agency, 2011). Emissions for the individual high traffic sources were back-calculated from county total emissions. Then, each source was assigned a fraction of the county's total highway emissions based on its relative annual average daily traffic and heavy-duty percentage.

Calculations are repeated for gasoline and diesel emissions. The algorithm for calculating on-road mobile source emissions is shown in Figure 8.

$$VKT\,on\,a\,segment(average\,daily\,traffic\,per\,km) = Segment\,length\,(km) * AADT$$

$$County\,VKT = \sum_{n=1}^{\#segments\,in\,County} (segment\,VKT)$$

$$Block\,Group\,VKTtotal = \sum_{n=1}^{\#segments\,in\,Block\,Group} (segment\,VKT)$$

But also:

$$Block\,Group\,VKTtotal = Block\,Group\,VKTlow_{\,traffic\,segments} + \sum_{n=1}^{\#high\,traffic\,sources\,in\,Block\,Group} (high\,traffic\,source\,VKT)$$

Thus

$$VKT_{high\,traffic\,source} = Source\,length\,(100\,m\,for\,most) * AADT\,on\,the\,segment\,in\,which\,the\,source\,is\,located$$

Therefore:

$$Block\,Group\,VKTlow_{\,traffic\,segments} = Block\,Group\,VKTtotal - \sum_{n=1}^{\#high\,traffic\,sources\,in\,Block\,Group} (high\,traffic\,source\,VKT)$$

$$Emissions\,from\,each\,source\,on\,a\,high\,traffic\,segment = \frac{County\,Emissions * VKT\,on\,the\,volume\,source}{County\,VKT}$$

Figure 8: Algorithm for Calculating On-Road Mobile Source Emissions

The main assumption of the algorithm is emissions are proportional to vehicle kilometers traveled (VKT).

We analyzed on-road emissions data obtained from the Minnesota Pollution Control Agency (MPCA). The logic behind using ready-made emissions is because MPCA has monitoring and modeled data sets, which are utilized in our modeled versus measured study.

In practice, an ideal method would be to calculate the emissions for each high traffic segment using MOVES emission factors and the individual road segment's annual average daily traffic. This would ensure road segments with similar traffic are assigned similar emissions, which is closer to a real-world scenario.

In conclusion, on-road emissions inventories are very important in trend analysis, regulatory impact assessments, and human health risk exposure modeling.

2.5 Air Dispersion and Deposition Modeling

Air dispersion and deposition modeling in this research was performed using AERMOD (Version 16216r) (United States Environmental Protection Agency, 2015). Version 16216r is the latest U.S. EPA preferred model for air dispersion modeling for near-field impacts which, per U.S. regulations translate to 50 kilometers from the source. The AERMOD model follows the procedures recommended by the U.S. EPA to conduct human health risk assessment as per the Human Health Risk Assessment Protocol (U.S. Environmental Protection Agency, 2005).

AERMOD calculates concentrations at every-source combination for every hour processed using advanced variables to parameterize the atmospheric conditions at each hour.

The AERMOD modeling system includes the following preprocessors:

1. AERMET (which calls AERMINUTE and AERSURFACE) for processing meteorological data.
2. AERMAP (used to import terrain elevations).

AERMOD performs better than its predecessor, Industrial Source Complex Short Term Version 3 (United States Environmental Protection Agency, 2002). This improvement is because of the new and improved algorithms incorporated into AERMOD.

These improvements are a result of new and improved algorithms (United States Environmental Protection Agency, 2004) for:

1. Plume rise and buoyancy.
2. Computation of vertical profiles of wind.
3. Improved approach for characterizing the fundamental boundary layer parameters.

The air dispersion modeling for this framework was performed using AERMOD View 9.5, from Lakes Environmental (Lakes Environmental Software, 2018).

2.6 Sources Parameterization

The roadway segments in this framework are modeled as area sources. The rationale behind characterizing the on-road sources as area sources is:

1. As per the U.S. EPA guidance "When modeling roadway links, experience in the field has shown that area sources may be easier to characterize correctly compared to volume sources. It is acceptable to use either area or volume sources to simulate roadways in AERMOD. Modelers may want to be particularly mindful of making errors when using volume sources." (United States Environmental Protection Agency, 2015).
2. Area sources are not subject to exclusion zones. Exclusion zones are the region ((2.15 * Sigma $Y_{initial}$) + 1 meter)) from the center of the volume source. Where Sigma Y is the initial lateral dimension. In the exclusion zone, the source-receptor calculations are not made, and this may lead to aberrant results. Figure 9 illustrates the exclusion zones in volume sources.
3. Area sources create a uniform emission characterization of a roadway, whereas volume sources don't because they are spaced far apart.

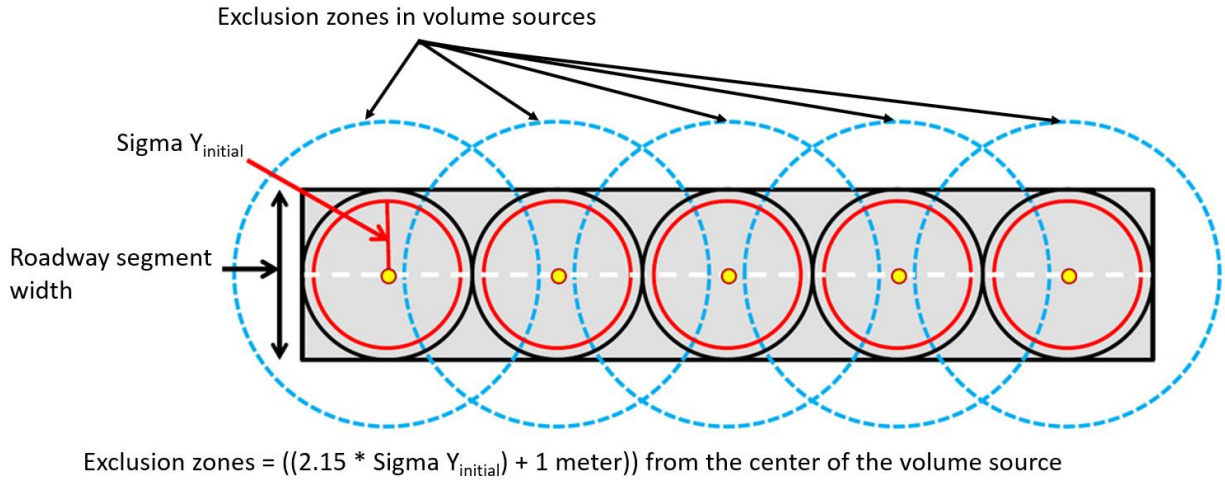


Figure 9: Exclusion Zone in Volume Sources

Table 4 presents the key advantages and disadvantages of area and volume sources.

Table 4: Advantages and Disadvantages of Area and Volume Sources

	Area source	Volume source
Advantages	Can place receptors within the area source	Contains meander algorithm
	Explicitly simulate a uniform emission density across the roadway	A volume source mimics an initial, well-mixed plume
	Avoids the need to determine Sigma Y values	Faster run time than area source
Disadvantages	Does not have the plume meander algorithm	Concentrations are not calculated in a volume source exclusion zone
	Longer run times	-

The meander algorithm is a fundamental part of the AERMOD model point source calculations. It accounts for the perturbations of the plume in the sub-time step periods. It applies to all non-area source types, (e.g., volume sources).

For roadway segments, the general guidance from the U.S. EPA -which is specific to haul roads but applied more liberally by the modeling community- is to use either area or volume sources. More information about the advantages and disadvantages of each source is found in Table 4. This guidance, known-better as the Haul Road Workgroup Final Report (United States Environmental Protection Agency, 2012)is the most widely-used reference for characterizing mobile sources within the confines of AERMOD. The report includes a sensitivity analysis comparing the modeled results when roadway segments were characterized as volume and area sources. Moreover, the report provides recommendations for entering roadway segment emissions into AERMOD. In summary, the modeling basics are:

1. The length of the source is equal to the length of the roadway segment (aspect ratio in AERMOD extended to 100:1 before warning is issued).
2. Plume Height is based on multiplying the vehicle height (not the height at which emissions escape the vehicle) by a factor of 1.7. This accounts for additional turbulence caused by vehicle movement.
3. The release height is equal to the plume height multiplied by 0.5.
4. Plume Width calculations are dependent on the number of lanes, if the road is single lane, then the modeler adds 6 meters to the width of the vehicle. For a two-lane road, the entire width of the road is used plus 6 meters. The Haul Road Workgroup Final Report is available at the following webpage:

   https://www3.epa.gov/ttn/scram/reports/Haul_Road_Workgroup-Final_Report_Package-20120302.pdf

2.7 Unitized Emission Rate

The air dispersion modeling was performed using the Unitized Emission Concept of a "generic" MSAT.

A unit emission is defined as 1 gram of MSAT / second. This unitized emission concept is built on the fundamentals that for an individual on-road source, the air concentration levels and deposition fluxes are linearly correlated with the emission rate of the on-road source. To put it simply, this means increasing emissions from the on-road source by two times, will result in an increase of the concentration at the receptor by two times. The unit emission rate serves two purposes: minimize calculations imprecision by low emissions values and eliminate multiple dispersion runs for the vast number of MSATs.

The concentrations and deposition fluxes for on-road sources modeled using the unitized emission rate concept can be obtained by multiplying the MSAT specific emission rates and the unit emission results together.

For an on-road source that emits multiple MSATs, a single air dispersion run is sufficient.

2.8 Meteorological Data

AERMET, AERMOD's meteorological preprocessor, formats hourly surface observations and twice-daily upper air soundings for use in the AERMOD air quality dispersion model. AERMET has two input pathways for reading surface-level variables: Surface and Onsite. The former reads hourly surface observations via existing archival file formats compiled and published by data repositories such as the National Centers for Environmental Information (NCEI). The latter permits modelers to input data observed at custom measurement equipment from surface-level as well as variables recorded at heights above ground. Onsite data is given priority in AERMET as they are assumed to represent conditions at the point of release. AERMET generates two files:

1. Surface—Contains hourly boundary layer parameter estimates.
2. Profile—Contains multiple-level observations of wind speed and direction, temperature, and fluctuating components of the wind.

The meteorological data used in this framework is collected from the Minneapolis–Saint Paul International Airport station.

The surface parameters (Surface Roughness, Albedo, and Bowen ratio) entered in AERMET were determined for the project location and not the measurement site (Minneapolis–Saint Paul International Airport station). Using surface parameters at the measurement sites is not optimal, experience and research conducted by Professor Thé has proved modeled results improved when surface parameters are used at the source location. Furthermore, the plume is subjected to mixing caused by shear, affected by local surface roughness, and impacted by local convective circulation, caused by local albedo and Bowen ratio.

2.9 Human Health Risk Model

The human health risk model demonstrates the migration of mobile source air toxics (MSATs) through various media such as air, soil, and water. The model then describes the movement of MSATs from water into fish and from soil into vegetables, grain, and forage and then into farm animals tissue where

they are consumed by humans. The air concentration is modeled for evaluation of direct inhalation risk of MSATs. For some air toxics, the risk from indirect exposure pathways is greater than the risk from direct exposure pathways. For example, the indirect risk from dioxin and furans (DeRose, 2009) can be more than 100 times greater than the direct risk (i.e., inhalation risk).

The direct and indirect risks can be quantified into health risks for both cancer effect and noncancer health effects. The main reference that outlines the fate and transport of mobile source air toxics in the environment is the U.S. EPA OSW Human Health Risk Assessment Protocol (U.S. Environmental Protection Agency, 2005).

2.10 Software Used for Modeling

The software used in establishing this framework are summarized below in Table 5.

Table 5: Software Used for Framework

Modeling Task	Software	License Fee
Estimates emissions for on-road mobile sources	U.S. MOVES2014	Freely available from U.S. EPA
Air dispersion modeling	AERMOD View 9.5	$1,599.00
Multi-pathway human health risk assessment	IRAP-h View	$4,895.00
	Total US$	**$6,494.00**

Chapter 3: CHEMICALS OF POTENTIAL CONCERN

3.1 Overview

Mobile Source Air Toxics (MSATs) are generally emitted in smaller quantities than criteria pollutants but are known to cause cancer, developmental effects, reproductive dysfunctions, neurological disorders, inheritable gene mutations, or other chronically or acutely toxic effects in humans with sufficient exposure. The list of 187 HAPs, in Section 112 (b) of the US 1990 Clean Air Act Amendments (CAAA), includes the following MSATs: Benzene, 1,3-Butadiene, Acetaldehyde, Acrolein, Ethyl benzene, Formaldehyde, and Polycyclic Organic Matter.

Diesel Particulate Matter (DPM), an MSAT not included in the list of 187 HAPs is identified as the primary cancer risk of all MSATs in the South Coast Air Basin (South Coast Air Quality Management District, 2000). Numerous peer-reviewed studies and systematic reviews show the strong correlation between being close to a roadway and the increased risk of adverse health effects, a summarized list of these studies is included below:

1. Johns Hopkins Study—Links traffic / curbside concentrations with cancer causing MSATs.
2. The Journal of the American Medical Association Study—Links soot in diesel particulate matter to lung cancer and cardiopulmonary disease.
3. Denver Study—Children living near congested roadways are at great risk of developing leukemia and other forms of cancer.

The 3 studies above are referenced in the "Highway Health Hazards" report produced by the Sierra Club in 2004 (Sierra Club, 2004). The Sierra Club is an environmental group, founded in 1892 in the United States. The main purpose of the group is to protect the environment.

In the risk assessment field, MSATs are referred to as chemicals of potential concern (COPC) and the COPC term is used throughout the thesis to reference MSATs. The following terms are used interchangeable in this framework: MSATs, COPCs, chemicals, and pollutants.

Assessing risk from mobile source air toxics is different from the standard risk assessment performed at superfund sites, energy recovery sites, or renewable energy power plants, because the concentration and deposition fluxes of mobile source air toxics are calculated using models in place of collecting samples from the studied sites. Risk modeling is quick and inexpensive, and a modeler can model for any location with all chemicals of potential concern in question.

The main component of mobile source air toxics assessment is modeling the fate and transport of mobile source air toxics in the environment. Then, the models describe the movement of mobile source air toxics from soil into produce, grain, and forage and then into farm animals where they are consumed by humans.

3.2 Priority MSATs

The Clean Air Act listed 187 chemicals as air toxics, the act requires the United States environmental protection agency to regulate the air toxics in phases. The air toxics program has two phases, technology-based and risk-based. On-road sources are omnipresent and emit many air toxic compounds. A number of these compounds are considered high priority because they increase the burden of chronic and acute diseases (CDC, 2017). The word "priority" is used in the sense of being the most important, and the designation has legal implications for reporting and inclusion in studies. The U.S. EPA identified nine priority mobile source air toxics: non-aromatic carbonyls (Formaldehyde, Acetaldehyde, and Acrolein), 1,3-Butadiene, Benzene, Ethyl benzene, diesel particulate matter, Naphthalene, and Polycyclic Organic Matter. These priority MSATs are among the national and regional-scale cancer risk drivers or contributors and non-hazard contributors from the 2011 NATA. The list of priority MSATs is subject to change and may be amended with future U.S. EPA mandates.

3.3 Origin and Formation

Mobile source air toxics (MSATs) can exist in the vapor phase, particle phase, or as a complex mixture with a portion of the vapor condensed onto the surface of the particulates. After emission, some MSATs undergo reactions in the atmosphere resulting in the formation of other MSATs of complex chemical structure and unknown toxicity endpoints (HEI, 2007). Mobile source air toxics are introduced to the environment via multiple routes. Benzene is formed due to incomplete gasoline and diesel fuel combustion. In addition to incomplete combustion, Benzene escapes to the environment due to evaporative emissions which include permeation, vapor venting or liquid leaks and due to refueling emissions such as vapor loss or spillage. Evaporative and refueling emissions contribute significantly to atmospheric Benzene concentrations. Carbonyls such as Formaldehyde, Acetaldehyde, and unsaturated carbonyls like Acrolein are formed due to incomplete combustion of fuels too. Secondary processes occurring in the atmosphere such as photochemical reactions of volatile organic compounds also contribute to increasing Acetaldehyde ambient concentrations (Kimbrough, Palma, & Baldauf, 2014). Motor vehicles contribute to 4 % of the nationwide Formaldehyde emissions (United States Environmental Protection Agency, 2011) and 14 % of Acrolein nationwide emissions is from vehicle exhaust (Kimbrough, Palma, & Baldauf, 2014). In addition to the said MSATs, other sources of air toxics

included emitted oils, burned or unburned via the exhaust. Finally, on-road sources emit metal mobile source air toxics as a result of vehicle wear and tear, mechanical deterioration of catalysts, and impurities found in oil or fuels.

3.4 Integrated Risk Information System (IRIS)

The U.S. EPA maintains the Integrated Risk Information System (IRIS) (United States Environmental Protection Agency, 2018), an electronic database that contains information on human health effects from exposure to various chemicals of potential concern in the environment. IRIS contains information on individual chemicals, with detailed and thorough quantitative information on inhalation reference concentrations for chronic non-carcinogenic effects and oral reference doses. The oral reference dose is an estimate with associated uncertainties of a daily exposure to sensitive receptors like the human population, that is likely to cause harm during a lifetime. The database contains information on oral cancer slope factors and inhalation unit risks for potential health effects of exposure to carcinogenic chemicals. In this framework, IRIS is the main reference for cancer and noncancer assessments for the mobile source air toxics. Table 22 summarizes the main toxicity values used in the risk assessment.

3.5 National Air Toxics Assessment (NATA)

The national air toxics assessment is a nationwide risk assessment. The NATA provides a snapshot of the outdoor air quality and risks associated with exposure to air toxics. The assessment is conducted by the U.S. EPA. The goal of NATA is to identify air toxics which are of greatest potential concern in terms of contribution to population risk. In this framework, we reference the 2011 NATA (United States Environmental Protection Agency, 2011). The NATA focuses on risk resulting from inhalation of emitted air toxics and does not consider indirect pathways as a result of exposure other than inhalation (i.e., ingestion exposure pathway). Results from the NATA help in prioritizing pollutants and emission sources and identify hotspots of interest for detailed analyses.

The U.S. EPA identified 9 compounds emitted from mobile sources, 8 of which are from the 187 air toxics list, in addition to diesel particulate matter as large contributors to overall risk in the 2011 National Air Toxics Assessment (NATA).

The 2011 NATA risk results are summarized below:

- *National cancer risk driver*—Formaldehyde: Risk exceeds 10 in a million for 25 million people.

- *Regional cancer risk drivers*—Benzene, Chloroprene, Coke Oven Emissions: Risk exceeds 1 in a million for 1 million people or risk exceeds 100 in a million for 10,000 people.
- *National cancer risk contributor*—1,3-Butadiene, Acetaldehyde, Carbon tetrachloride, Hexavalent Chromium, Ethyl benzene, Naphthalene: Risk exceeds 1 in a million for 25 million people.
- *Regional cancer risk contributor*—1,3-Dichloropropene, 1,4-DichloroBenzene, Arsenic compounds, Ethylene oxide, Nickel compounds, Polycyclic aromatic hydrocarbon / Polycyclic Organic Matter: Risk exceeds 1 in a million for 1 million people.
- *National noncancer hazard drivers*—Acrolein, Chlorine, Diesel PM.
- *Regional noncancer hazard drivers*—Hexamethylene diisocyanate.

3.6 Epidemiological Studies

A growing number of epidemiological studies show that air toxics emitted from on-road sources are statistically associated with adverse health effect. Human population working, living or attending school close to major roadways have shown to exhibit adverse health effects like respiratory effects, premature mortality, childhood cancer or neurological effects (United States Environmental Protection Agency, 2007).

In the past 20 years, more than 30 studies on MSATs exposure and health impacts were published (United States Environmental Protection Agency, 2007). The studies include human epidemiology and animal toxicology experiments. The federal highway administration (FHWA) supported and initiated several epidemiological studies.

These studies are listed below:

1. The National Near Roadway MSAT Study—MSAT and mobile source $PM_{2.5}$ in Las Vegas, Nevada and Detroit, Michigan study.
2. Diesel Emissions—Diesel Emissions and Lung Cancer study.
3. Mobile Source Air Toxic Hot Spot—Camden, New Jersey and Buffalo Peace Bridge Study.
4. Air Toxics Assessment in North Denver.
5. Particulate matter study in Kansas City.

The FHWA site has detailed information on the listed studies: https://www.fhwa.dot.gov/environMent/air_quality/air_toxics/policy_and_guidance/msat/page04.cfm

3.7 Weight-of-evidence Cancer Classification

The U.S. EPA published the first set of guidelines for carcinogen risk assessment in 1986 and this is the outcome of two decades of research and scientific experience. The science behind risk assessment and toxicity testing is constantly evolving and as a result, the U.S. EPA updated the guidance in 2005.

In the process of hazard identification of carcinogens; human data, animal data, and supporting evidence are used together synergistically to characterize the weight-of-evidence (WOE) regarding the agent's potential as a human carcinogen.

The current guidelines, finalized in 2005, recommend expressing weight-of-evidence by *narrative statements* in preference of only hierarchical categories, and expressing them separately for the oral and inhalation routes (United States Environmental Protection Agency, 2005).

Figure 10 summarizes the general categories recognized by the U.S. EPA's 2005 and 1986 Guidelines for Carcinogen Risk Assessment.

Chemicals of potential concern classified as Group A and B carcinogens or (Carcinogenic to Humans or Likely to be Carcinogenic to Humans) are generally evaluated as carcinogens.

U.S. EPA 2005	U.S. EPA 1986
Carcinogenic to Humans	Group A: (Carcinogenic to Humans)
Likely to be Carcinogenic to Humans	Group B: (Probably Carcinogenic to Humans)
Suggestive Evidence of Carcinogenic Potential	Group C: (Possibly Carcinogenic to Humans)
Inadequate Information to Assess Carcinogenic Potential	Group D: (Not Classifiable as to Human Carcinogenicity)
Not Likely to be Carcinogenic to Humans	Group E: (Evidence of Non-carcinogenicity for Humans)

Figure 10: Guidelines for Carcinogen Risk Assessment

3.8 MSATs included in Cumulative Risk Analysis

The cumulative risk analysis section includes the following MSATs: Benzene, Formaldehyde, Hexavalent Chromium, 1,3-Butadiene, Indeno(1,2,3-cd)pyrene, and Benzo(a)pyrene. Each chemical has a CAS registry number. The CAS registry number is a unique numerical identifier for chemical substances. This section summarizes the toxicity values for each MSAT. The information presented in this section is obtained from the Integrated Risk Information System (IRIS).

3.8.1 Benzene

- CAS: 71-43-2
- Molecular weight: 78.114 g / mol
- 2D Structure:

Figure 11: 2D Structure of Benzene

- Cancer Assessment:

Table 6: Cancer Assessment for Benzene

WOE Characterization	Framework for WOE Characterization
A (Human carcinogen)	Guidelines for Carcinogen Risk Assessment
Known / likely human carcinogen	Proposed Guidelines for Carcinogen Risk Assessment

- Tumor Sites:

Table 7: Tumor Sites

Type	Tumor Site	Tumor Type
Quantitative Estimate of Carcinogenic Risk from Oral Exposure	Hematologic	Leukemia
Quantitative Estimate of Carcinogenic Risk from Inhalation Exposure	Hematologic	Leukemia

- Noncancer Assessment:

Table 8: Noncancer Assessment for Benzene

	Critical Effect Systems	Basis	Confidence
Reference Dose for Oral Exposure	Immune	Decreased lymphocyte count	Medium
Reference Concentration for Inhalation Exposure	Immune	Decreased lymphocyte count	Medium

3.8.2 Formaldehyde

- CAS: 50-00-0
- Molecular weight: 30.026 g / mol
- 2D Structure:

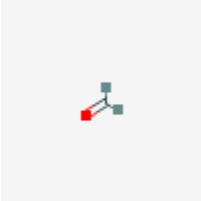


Figure 12: 2D Structure of Formaldehyde

- Cancer Assessment:

Table 9: Cancer Assessment for Formaldehyde

WOE Characterization	Framework for WOE Characterization
B1 (Probable human carcinogen - based on limited evidence of carcinogenicity in humans)	Guidelines for Carcinogen Risk Assessment

- Tumor Sites:

Table 10: Tumor Sites

Type	Tumor Site	Tumor Type
Quantitative Estimate of Carcinogenic Risk from Inhalation Exposure	Respiratory	Squamous cell carcinoma

- Noncancer Assessment:

Table 11: Noncancer Assessment for Formaldehyde

	Critical Effect Systems	Basis	Confidence
Reference Dose for Oral Exposure	Urinary, Gastrointestinal, Other	Reduced weight gain, histopathology in rats	Medium

3.8.3 1,3-Butadiene

- CAS: 106-99-0
- Molecular weight: 54.092 g / mol
- 2D Structure:

Figure 13: 2D Structure of 1,3-Butadiene

- Cancer Assessment:

Table 12: Cancer Assessment for 1,3-Butadiene

WOE Characterization	Framework for WOE Characterization
Carcinogenic to humans	Revised Draft Guidelines for Carcinogen Risk Assessment

- Tumor Sites:

Table 13: Tumor Sites

Type	Tumor Site	Tumor Type
Quantitative Estimate of Carcinogenic Risk from Inhalation Exposure	Hematologic	Leukemia

- Noncancer Assessment:

Table 14: Noncancer Assessment for 1,3-Butadiene

	Critical Effect Systems	Basis	Confidence
Reference Concentration for Inhalation Exposure	Reproductive	Ovarian atrophy	Medium

3.8.4 Hexavalent Chromium

- CAS: 18540-29-9
- Molecular weight: 51.996 g / mol
- Cancer Assessment:

Table 15: Cancer Assessment for Hexavalent Chromium

WOE Characterization	Framework for WOE Characterization
A (Human carcinogen) (Inhalation route)	Guidelines for Carcinogen Risk Assessment
D (Not classifiable as to human carcinogenicity) (Oral route)	Guidelines for Carcinogen Risk Assessment
Carcinogenic potential cannot be determined (Oral route)	Proposed Guidelines for Carcinogen Risk Assessment
Known / likely human carcinogen (Inhalation route)	Proposed Guidelines for Carcinogen Risk Assessment

- Tumor Sites:

Table 16: Tumor Sites

Type	Tumor Site	Tumor Type
Quantitative Estimate of Carcinogenic Risk from Inhalation Exposure	Respiratory	Lung cancer

- Noncancer Assessment:

Table 17: Noncancer Assessment for Hexavalent Chromium

	Critical Effect Systems	Basis	Confidence
Reference Concentration for Inhalation Exposure	Respiratory	Nasal septum atrophy	Low
Reference Concentration for Inhalation Exposure	Respiratory	Lactate dehydrogenase in bronchioalveolar lavage fluid	Medium

3.8.5 Indeno(1,2,3-cd)pyrene

- CAS: 193-39-5
- Molecular weight: 276.338 g / mol
- 2D Structure:

Figure 14: 2D Structure of Indeno(1,2,3-cd)pyrene

- Cancer Assessment:

Table 18: Cancer Assessment for Indeno(1,2,3-cd)pyrene

WOE Characterization	Framework for WOE Characterization
B2 (Probable human carcinogen - based on sufficient evidence of carcinogenicity in animals)	Guidelines for Carcinogen Risk Assessment

- Noncancer Assessment:

The Reference Dose for Oral Exposure (RfD) and Reference Concentration for Inhalation Exposure (RfC) were not assessed under the IRIS program. Since neither the RfD nor the RfC were not assessed under the IRIS program, no hazard quotients were calculated.

3.8.6 Benzo(a)pyrene

- CAS: 50-32-8
- Molecular weight: 252.316 g / mol
- 2D Structure:

Figure 15: 2D Structure of Benzo(a)pyrene

- Cancer Assessment:

Table 19: Cancer Assessment for Benzo(a)pyrene

WOE Characterization	Framework for WOE Characterization
Carcinogenic to humans	Guidelines for Carcinogen Risk Assessment

- Tumor Sites:

Table 20: Tumor Sites

Type	Tumor Site	Tumor Type
Quantitative Estimate of Carcinogenic Risk from Oral Exposure	Gastrointestinal	Forestomach, esophagus, tongue, and larynx tumors
Quantitative Estimate of Carcinogenic Risk from Inhalation Exposure	Gastrointestinal, Respiratory	Squamous cell neoplasia in the larynx, pharynx, trachea, nasal cavity, esophagus, and forestomach

- Noncancer Assessment:

Table 21: Noncancer Assessment for Benzo(a)pyrene

	Critical Effect Systems	Basis	Confidence
Reference Dose for Oral Exposure	Developmental	Neurobehavioral changes	Medium
Reference Concentration for Inhalation Exposure	Developmental	Decreased embryo / fetal survival	Medium

3.9 Toxicity Assessment

The correlation between exposure to a chemical of potential concern and the probability and magnitude of developing an adverse health effect is estimated using a toxicity assessment. Toxicity values such as *oral reference dose* (RfD), *cancer slope factor* (CSF), *reference concentration* (RfC), *unit risk factor* (URF), and *acute inhalation exposure criteria* (AIEC) are extracted from studies and experiments conducted on laboratory animals and human epidemiological studies. Toxicity values are very important in estimating the quantitative risk related to MSATs exposure and the potential for adverse health impacts. The toxicity values used in the risk calculations for this framework are defined in Table 22.

The toxicity variables used to evaluate MSATs are summarized in Table 23.

The toxicity values used in the calculations for this study are selected from references following the U.S. EPA-approved hierarch (United States Environmental Protection Agency, 2003). The tiers are listed below:

1. U.S. EPA Integrated Information System.
2. U.S. EPA Provisional Peer-reviewed Toxicity Values.
3. U.S. EPA and non-U.S. EPA sources, with priority to recent and publicly available sources with proper citation.

As for the acute values, the hierarchy is listed below (U.S. Environmental Protection Agency, 2005):

1. Cal / EPA Acute RELs.
2. Acute Inhalation Exposure Guidelines (AEGL-1).
3. Level 1 Emergency Planning guidelines (ERPG-1).
4. Temporary Emergency Exposure Limits (TEEL-1).
5. Acute Inhalation Exposure Guidelines (AEGL-2).

Hierarchical approaches are needed because acute criteria values are COPC-specific, and no single organization or method has developed acute criteria values or benchmarks for all the COPCs.

Table 22: Toxicity Values Definition

Toxicity Values	Definition
Oral Reference Dose (RfD)	Estimate (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime.
Oral Cancer Slope Factor (CSF)	Value used to evaluate potential carcinogenic health effects resulting from long-term oral exposure to COPC.
Reference Concentration (RfC)	Value used to evaluate potential noncarcinogenic health effects resulting from long-term inhalation exposure to COPC.
Unit Risk Factor (URF)	The upper-bound excess lifetime cancer risk estimated to result from continuous exposure to an agent at a concentration of 1 µg / m^3 in air.
Acute Inhalation Exposure Criteria (AIEC)	Value used to evaluate short-term or acute effects from direct inhalation of vapor phase and particle phase COPCs.

Table 23: Summary of Chemical-Specific Toxicity Values

COPC	CAS #	Oral RfD mg/kg-day	Oral CSF $(mg/kg\text{-}day)^{-1}$	RfC mg/m^3	URF $(\mu g/m^3)^{-1}$	AIEC mg/m^3
Benzene	71-43-2	4.00E-03	5.50E-02	3.00E-02	7.80E-06	1.30E+00
Formaldehyde	50-00-0	2.00E-01	1.30E-05	9.80E-03	1.30E-05	9.40E-02
1,3-Butadiene	106-99-0	**NA**	3.40E+00	2.00E-03	5.88E-05	6.60E-01
Hexavalent Chromium	18540-29-9	3.00E-03	0.00E+00	8.00E-06	1.20E-02	3.00E-04
Indeno(1,2,3-cd)pyrene	193-39-5	**NA**	7.30E-01	**NA**	1.10E-04	5.00E-01
Benzo(a)pyrene	50-32-8	3.00E-04	7.30E+00	2.00E-06	1.10E-03	6.00E-01

Table 24: Cancer Assessment - Extrapolation Method

	Extrapolation Method[2]	
COPC	**Oral Slope Factor**	**Inhalation Unit Risk**
Benzene	Linear extrapolation of human occupational data	Low-dose linearity utilizing maximum likelihood estimates
Formaldehyde	Not Assessed under the IRIS Program	Linearized multistage procedure, additional risk
1,3-Butadiene	Information reviewed but value not estimated	Linear extrapolation from LEC_{01} (0.254 ppm); LEC_{01} derived from linear relative rate model (RR = 1 + (B)(x)) using lifetable analysis with leukemia incidence data; an adjustment factor of 2 was applied
Hexavalent Chromium	Information reviewed but value not estimated	Multistage, extra risk
Indeno(1,2,3-cd) pyrene	Not assessed under the IRIS Program	Not assessed under the IRIS Program
Benzo(a)pyrene	Time-to-tumor dose-response model with linear extrapolation from the POD ($BMDL_{10HED}$) associated with 10 % extra cancer risk	Time-to-tumor dose-response model with linear extrapolation from the POD ($BMCL_{10HED}$) associated with 10 % extra cancer risk

[2] The Toxicological Review of each MSAT is available at: https://www.epa.gov/iris

The extrapolation methods summarized in Table 24 are important as these methods affect the uncertainty which plays a key role in risk interpretation and risk management options. Also, because of the lack of adequate human data to estimate risk, numeric and biologic extrapolations are conducted. Furthermore, extrapolation methods prevent underestimation of risk.

Table 25 contains a summary of the Dose-Response terms used in the aforementioned extrapolation methods.

Table 25: Dose-Response Dictionary

LOAEL	Lowest-Observed-Adverse-Effect Level. Lowest dose at which significant adverse effects are observed.
NOAEL	No-Observed-Adverse-Effect Level. Highest dose at which no significant adverse effects are observed.
BMD	Benchmark Dose. An exposure to a low dose of a substance that is linked with a low (1-10 %) risk of adverse health effects, or the dose associated with a specific biological effect.
BMC	Same as above (for inhaled substances).
BMDL	Statistical lower confidence limit (usually 95 %) on the BMD.
Critical effect	The first adverse effect or its known precursor that occurs to the most sensitive species as the dose rate of an agent increases.
POD	Point of departure (The dose-response point that marks the beginning of a low dose extrapolation.
LEC_{01}	Exposure concentration corresponding to the 95 % lower bound on the EC01.
HED/ HEC	Human equivalent dose or concentration.
LED_{10}	Dose associated with 10 % response.

The following doses: NOAEL, LOAEL, BMDL, and LED_{10} are used as points of departure for decision-making purposes in risk assessments.

- Importance information about the toxicity values used in this framework:

    1. The AIEC values for 1,3-Butadiene and Hexavalent Chromium were extracted from Cal / EPA Acute RELs as per the recommended hierarchical approach (U.S. Environmental Protection Agency, 2005). The Hexavalent Chromium value was not available in HHRAP database because this MSAT is not associated with combustion which is the basis of the HHRAP.

    2. For Benzo(a)pyrene, we extracted the RfC and RfD values from the U.S. EPA IRIS (United States Environmental Protection Agency, 2018). The lowest values of each set were selected, because it is recommended to take the more conservative approach when faced with multiple values.

    3. Benzo(a)pyrene is identified as mutagenic by the U.S. EPA in the regional screening levels tables (United States Environmental Protection Agency, 2017). Early-life exposure to carcinogenic MSATs such as Benzo(a)pyrene with a mutagenic mode of action may result in a greater contribution to cancers appearing later in life (United States Environmental Protection Agency, 2007). To account for MSATs identified as mutagenic, age-dependent adjustment factors are applied to the oral and inhalation slope factors evaluated over a lifetime exposure.

    4. Benzo(a)pyrene is highly lipophilic (Gerde, et al., 1997) and therefore would propagate through the animal tissue pathways.

Chapter 4: MULTI-PATHWAY FATE-AND-TRANSPORT RISK ASSESSMENT

4.1 Overview

Mobile source air toxics risk assessment is a qualitative and quantitative evaluation of the risk posed to human health by the actual or potential presence of air toxics emitted from on-road mobile sources. The process of the mobile source air toxics multi-pathway fate-and-transport risk assessment is summarized in Figure 16. Our research adapted, extended, and applied the U.S. EPA Human Health Risk Assessment Protocol in the determination of potential adverse health risks from exposure to MSATs. Current practices don't go beyond assessing critical air concentrations of MSATs. Our proposed methodology takes a giant leap forward and estimates the total cumulative human health risk posed by the aggregated exposures of on-road mobile sources. The risk engine used in this framework is IRAP-h View (Lakes Environmental Software, 2018).

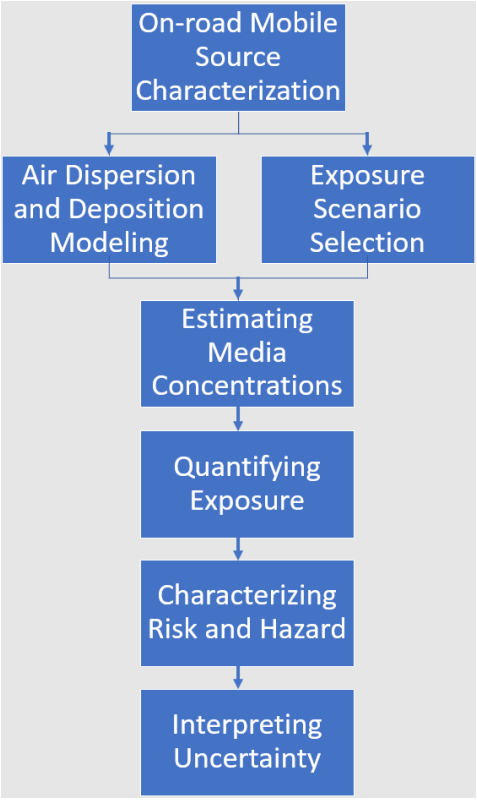


Figure 16: MSATs Human Health Risk Assessment Process

4.2 Risk Modeling

IRAP-h View is the graphical user interface for conducting a comprehensive multi-pathway human health risk assessment based on the U.S. EPA Human Health Risk Assessment Protocol (HHRAP) (U.S. Environmental Protection Agency, 2005). The software estimates the risk to human health (cancer risks and noncancer hazards) resulting from emissions of on-road mobile sources.

The air dispersion modeling was performed using the U.S. EPA preferred air dispersion model, AERMOD. AERMOD View, a graphical user interface for the AERMOD model, was utilized for visualization of modeled data. We utilized the "Risk Mode" option within AERMOD View which allows modelers to setup the project in adherence to the HHRAP guidelines. AERMOD generates 1-hour and annual average ground-level pollutant concentrations and annual average deposition rates. The input and output files prepared during the model run were processed in a format compatible with the risk engine, IRAP-h View.

In our risk modeling, the focus was on the mobile source air toxics (MSATs) summarized in Table 26. The *Fv parameter*, is the fraction of MSAT air concentration in vapor phase. The MSATs originating from on-road sources disseminate and deposit into the environment as either vapor, particle, or particle-bound phases. Air dispersion modeling conducted with AERMOD View's "Risk Mode" estimates air concentrations and deposition fluxes for vapor phase MSATs, particle phase MSATs, and particle-bound MSATs.

Table 26: Modeled Mobile Source Air Toxics

Mobile Source Air Toxic	Fv	Assumed emitted as vapor, particle, or vapor with portion particle-bound (PB)
Benzene	1	Vapor
Formaldehyde	1	Vapor
1,3-Butadiene	1	Vapor
Hexavalent Chromium	0	Particle
Indeno(1,2,3-cd)pyrene	0.005	Particle
Benzo(a)pyrene	0.294	29.4 % V, 70.6 % PB

The chemical phases are described in the HHRAP (U.S. Environmental Protection Agency, 2005) and are categorized as follows:

1. Highly volatile organic MSATs occur only in the vapor phase (Fv = 1.0).
2. Most metals and organic MSATs with very low volatility (fraction of MSAT in vapor phase, Fv less than 0.05) occur as *particles.*
3. The remaining organic MSATs occur with a portion of the vapor condensed onto the surface of particulates (i.e. particle-bound).

The Fv values for the MSATs presented in Table 26 are available in HHRAP Appendix A-2. The emissions phase assumption was determined from the Fv value in agreement with the HHRAP guidelines.

Air dispersion and deposition modeling in AERMOD was performed in 3 modes:

1. Vapor phase.
2. Particle phase.
3. Particle-bound phase.

Running AERMOD in 3 modes generates the following plot files:

1. Vapor phase output: air concentration, dry deposition rate, and wet deposition rate.
2. Particle phase output: air concentration, dry deposition rate, and wet deposition rate.
3. Particle-bound output: air concentration, dry deposition rate, and wet deposition rate.

MSAT-specific parameters such as diffusivity in air, diffusivity in water, leaf cuticular resistance, and Henry's Law Constant are required to model vapor phase dispersion and deposition. The parameter values required to model the vapor phase dispersion and deposition are available in the U.S. EPA Deposition Parameterizations document (United States Environmental Protection Agency, 2002).

As for modeling the particle and particle-bound phases dispersion and deposition, the modeler needs to provide particle size distributions for particles originating from the on-road sources. The fate of particles is determined by the particle size. Larger particles deposit closer to the on-road source, whereas, smaller particles remain suspended in the air for prolonged periods.

The rate at which dry and wet removal processes deposit particles onto the Earth's surface depends on particle size and particle density (United States Environmental Protection Agency, 2004). The AERMOD model uses the mass-based particle size distribution to apportion the mass of particle phase MSATs according to particle size (United States Environmental Protection Agency, 2004).

Finally, when modeling MSATs particle-bound dispersion and deposition, the AERMOD model calculates the area available for MSATs to condense onto the surface of particles. The AERMOD model uses the surface area-based particle size distribution to apportion the mass of particle bound MSATs according to particle size.

The AERMOD air parameters output is based on unitized modeling and is summarized in Figure 17.

	Emission Phase	AERMOD Output Type	Description of Highest Unitized
1	Vapor Phase	Concentration	Hourly air concentration from vapor phase
2	Vapor Phase	Concentration	Annual average air concentration from vapor phase
3	Vapor Phase	Dry Deposition	Annual dry deposition rate from vapor phase
4	Vapor Phase	Wet Deposition	Annual wet deposition rate from vapor phase
5	Particle Phase	Concentration	Hourly air concentration from particle phase
6	Particle Phase	Concentration	Annual air concentration from particle phase
7	Particle Phase	Dry Deposition	Annual dry deposition rate from particle phase
8	Particle Phase	Wet Deposition	Annual wet deposition rate from particle phase
9	Particle-Bound	Concentration	Hourly air concentration from particle-bound
10	Particle-Bound	Concentration	Annual air concentration from particle-bound
11	Particle-Bound	Dry Deposition	Annual dry deposition rate from particle-bound
12	Particle-Bound	Wet Deposition	Annual wet deposition rate from particle-bound

Figure 17: AERMOD Risk Mode Air Parameter Output

Air dispersion modeling is not sufficient to determine the adverse health effects associated with MSATs exposure, many of the mobile source air toxics reach humans by indirect pathways (non-inhalation). A case in point, is through the deposition of MSATs onto soil, impacting produce. Above ground produce is impacted by the following possible mechanisms: direct deposition of particles, vapor transfer, and root uptake. Human health is ultimately affected due to the consumption of the contaminated produce.

The main novelty of this framework is performing a multi-pathway fate-and-transport analysis of the MSATs and the assessment of the total cumulative risk.

The risk computation process is described below:

1. AERMOD produces 2 plot files; 1-hour and annual, and a risk input file for each source. The annual average values are used to estimate the potential for adverse health effects from chronic exposure and the maximum 1-hour values are generated to estimate the health effects from acute exposure. The AERMOD output is based on unitized modeling as described in section 2.7.
2. The batcher program is used to perform multiple model runs automatically.
3. The Air-2-Risk program compiles all the sources with their source-specific and pathway-specific contour plot files (based on unitized air modeling data for particle, particle-bound, and vapor phase).
4. The output from the Air-2-Risk program is imported into IRAP-h View.
5. Once imported into IRAP-h View, the appropriate air modeling phases are applied to the MSATs based on their physicochemical properties (i.e., fraction of MSAT air concentration in vapor phase).
6. MSAT-specific emissions are added in IRAP-h View.
7. The MSATs concentrations and risk impacts are calculated.

The risk results computation process is illustrated in Figure 18.

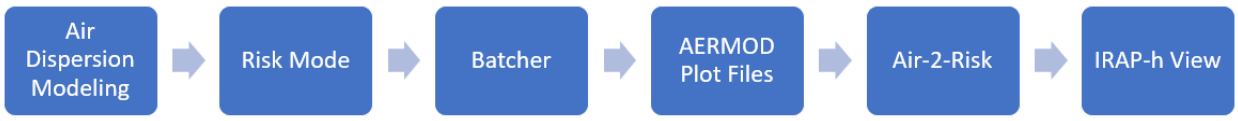


Figure 18: Risk Results Computation Process

IRAP-h View does not account for background concentrations. The HHRAP (U.S. Environmental Protection Agency, 2005) methods account for exposure to background concentrations in how the regulatory target levels are applied which are believed to be conservative enough to account for the influence of background exposures.

IRAP-h View is the only system to encapsulate all the complexities of the HHRAP.

4.3 Exposure Scenarios

Exposure scenarios serve to define the combination of exposure pathways and exposure parameters applied to complete the risk and hazard calculations. For the purposes of this study exposure scenarios include the resident adult, resident child, farmer adult, farmer child, fisher adult, and fisher child scenarios. Table 27 summarizes the human receptor populations and exposure pathways evaluated in this framework. In addition to the mentioned scenarios, we also evaluated the acute receptor scenario.

The acute receptor scenario evaluates short-term 1-hour maximum MSATs air concentrations. This scenario only applies to the inhalation of vapors and particulates.

Table 27: Evaluated Human Exposure Scenarios

	Resident		**Farmer**		**Fisher**	
Exposure Pathways	Adult	Child	Adult	Child	Adult	Child
Inhalation of Vapors and Particulates	I	I	I	I	I	I
Incidental Ingestion of Soil	I	I	I	I	I	I
Ingestion of Homegrown Produce	I	I	I	I	I	I
Ingestion of Homegrown Beef	N	N	I	I	N	N
Ingestion of Milk from Homegrown Cows	N	N	I	I	N	N
Ingestion of Homegrown Chicken	S	S	I	I	S	S
Ingestion of Eggs from Homegrown Chickens	S	S	I	I	S	S
Ingestion of Homegrown Pork	N	N	I	I	N	N

The characters in Table 27 denote the following:

- I: Pathway included in exposure scenario.
- N: Pathway not included in exposure scenario.
- S: Site-specific exposure setting characteristics.

The drinking water pathway was not evaluated in this framework because it relies on the impracticable assumption that the source of all drinking water is untreated surface water.

4.3.1 Sensitive Receptors

We identified the sensitive receptors, synonymous with risk receptors, using the risk receptor identification tool in IRAP-h View software. The tool shown in Figure 19, identifies the grid notes with the maximum value for each air parameter summarized in Figure 17. This approach ensures that the maximum risk locations area selected for analysis, reducing the addition of inessential risk receptors, and minimizing computational time. Figure 20 displays the identified risk receptor and their location in the project domain.

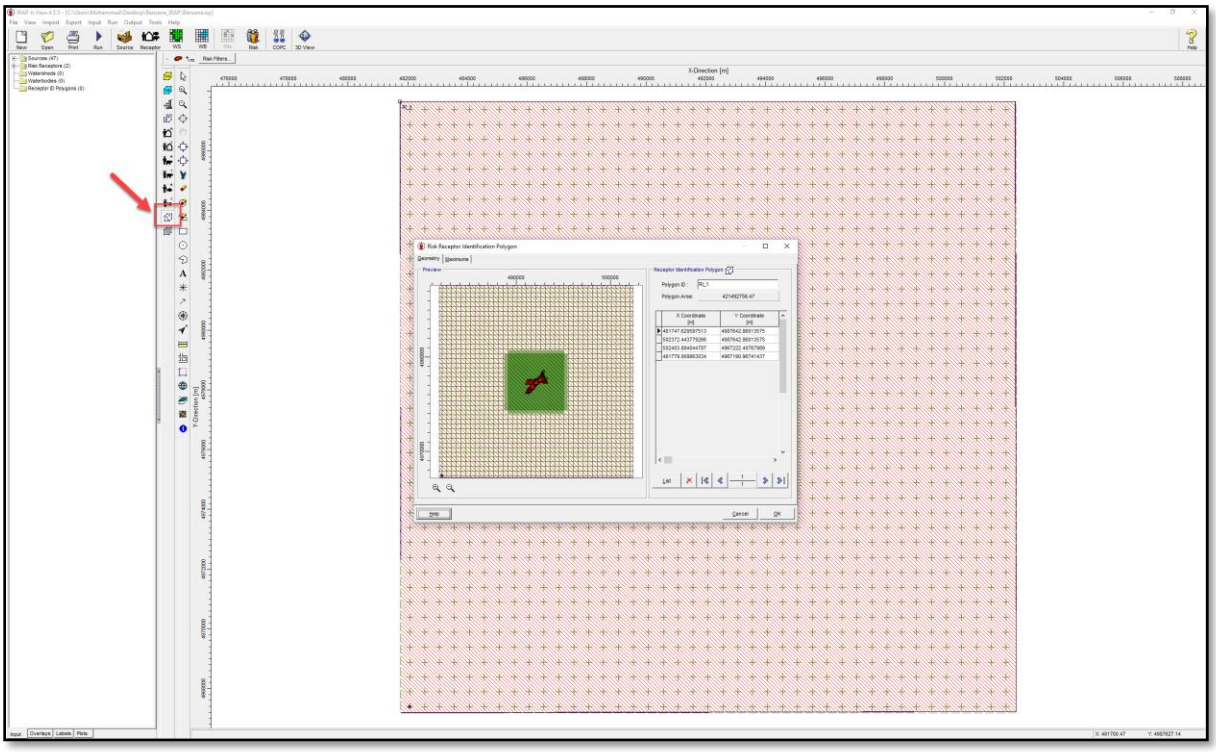


Figure 19: Risk Receptor Identification Tool

It comes as no surprise the sensitive receptors locations are near the sources with the highest emission rates. When conducting multi-pathway human health risk assessments, it is imperative to include additional sensitive receptors such as schools, playgrounds, nursing homes, and health care facilities.

For this study, we selected Saint Paul – Ramsey Health Center as a sensitive receptor.

Sensitive Receptor	Easting (m)	Northing (m)
Saint Paul - Ramsey Health Center	492230.04	4977478.54
Sensitive Receptor 1	492167.91	4977485.5
Sensitive Receptor 2	492567.91	4977785.5
Sensitive Receptor 3	491967.91	4977385.5

Figure 20: Sensitive Receptors Locations

4.4 Estimating Media Concentration

Estimating media concentrations includes the calculation of:

1. Air Concentrations in Air for Direct Inhalation.
2. Concentrations in Soil.
3. Concentrations in Produce.
4. Concentrations in Meats and Eggs.

Details regarding the equations and recommended default variable values for estimating media concentrations (e.g., air, soil, produce) are not duplicated in this thesis but can be found in Chapter 5 and Appendix B of the U.S. EPA Human Health Risk Assessment Protocol. The COPC concentrations are estimated based on the COPC-specific emission rates and the unitized ambient air concentrations and deposition fluxes computed using AERMOD.

4.5 Quantifying Exposure

This part of the assessment describes the factors to be evaluated in quantifying exposure in each of the evaluated exposure scenarios described in section 4.3 of this framework. The calculation of specific exposure rates for each chemical of potential concern for each exposure pathway includes:

1. Estimated COPC media concentrations.
2. Exposure Frequency (EF).
3. Exposure Duration (ED).
4. Averaging Time (AT).
5. Receptor Body Weight.

4.5.1 Exposure Frequency (EF)

The main assumption for the exposure frequency is that the receptors in each evaluated exposure scenario are assumed to be exposed to all the exposure scenario-specific exposure pathways **8400 hours per year (350 days)** (U.S. Environmental Protection Agency, 2005). This is based on a conservative estimate that all receptors spend a minimum of two weeks at a location other than the exposure scenario location.

4.5.2 Exposure Duration (ED)

The time span that a receptor is exposed via a specific exposure pathway is defined as the exposure duration. After an emission source stops emitting, the receptor is no longer exposed to COPCs via the direct inhalation exposure pathway, however, the receptor is still exposed via the indirect exposure pathways for as long as the receptor continues to be in the assessment area. The exposure duration values recommended by the U.S. EPA are summarized in Table 28.

Table 28: Exposure Duration Values

Exposure Duration Values		
Recommended Exposure Scenario Receptor	**Value (Years)**	**Source**
Resident Child	6	Human Health Risk Assessment Protocol
Resident Adult	30	
Fisher Adult	30	
Fisher Child	6	
Farmer Adult	40	
Farmer Child	6	

4.5.3 Averaging Time (AT)

The U.S. EPA Human Health Risk Assessment Protocol recommends that averaging time for noncarcinogenic pollutants is the **exposure duration in years *multiplied* by 365 days**, however, for carcinogenic pollutants, the effect of which may have long latency periods; the recommended averaging time is **70 years.** The averaging times for noncarcinogenic pollutants and carcinogenic pollutants are consistent with the mechanism of action for each of the health effects endpoints which vary from one COPC to another. The carcinogenic pollutants averaging time is built on the basic assumption that a high dose received by a receptor over a shorter period is comparable to a corresponding low dose spread over a lifetime of the receptor.

4.5.4 Receptor Body Weight

The average body weight values used in this framework are 70 kilograms (154 pounds) for an adult and 15 kilograms (33 pounds) for a child. These are the U.S. EPA Human Health Risk Assessment Protocol recommended values.

4.6 Risk Characterization Methodology

The risk assessment modeling was performed based on the U.S. EPA Human Health Risk Assessment Protocol (HHRAP) (U.S. Environmental Protection Agency, 2005). The HHRAP provides guidance on how to conduct air dispersion modeling and perform multi-pathway fate-and-transport risk assessments. Moreover, the HHRAP includes guidance on calculation of cancer risks and noncancer health effects.

The HHRAP includes appendices, containing MSAT-specific fate, transport, and health benchmarks; as well as equations for estimating media concentrations and cancer and noncancer health effects.

4.6.1 Quantitatively Estimating Cancer Risk

This section describes the equations and risk methodologies used to calculate cancer risk.

4.6.1.1 Inhalation Cancer Risk

The equation for calculating inhalation risk is presented in (Equation 2).

$$\boldsymbol{Cancer\ Risk_{Inhalation} = EC \times URF_i} \qquad \textbf{Eq. 2}$$

$$\boldsymbol{EC = \frac{C_a \times EF \times ED}{AT \times 365\ days/year}} \qquad \textbf{Eq. 3}$$

Where:

$\boldsymbol{Cancer\ Risk_{Inhalation(i)}}$

$=$ Individual lifetime cancer risk through direct inhalation of COPC carcinogen (unitless)

$\boldsymbol{EC}$

$=$ Exposure concentration ($\mu g/m^3$)

$\boldsymbol{URF_i}$

$=$ Inhalation Unit Risk Factor $(\mu g/m^3)^{-1}$

$\boldsymbol{C_a}$

$=$ Total COPC air concentration ($\mu g/m^3$)

$\boldsymbol{EF}$

$=$ Exposure frequency $(\frac{\text{days}}{\text{year}})$

$\boldsymbol{ED}$

$=$ Exposure duration (year)

$\boldsymbol{AT}$

$=$ Averaging time (year)

4.6.1.2 Cancer Risk from Indirect Exposure Pathways

The equation for calculating cancer risk from indirect exposure pathways is presented in (Equation 4).

$$\boldsymbol{Cancer\ Risk = LADD \times CSF} \qquad \textbf{Eq. 4}$$

Where:

$\boldsymbol{Cancer\ Risk}$

$=$ The potential for cancer risk from indirect (i. e. , ingestion) exposure pathways (unitless)

$\boldsymbol{LADD}$

$=$ Lifetime average daily dose $\left(\frac{\text{mg}}{\text{kg} - \text{day}}\right)$

$\boldsymbol{CSF}$

$=$ Cancer slope factor $\left(\frac{\text{mg}}{\text{kg} - \text{day}}\right)^{-1}$

4.6.1.3 Total Cancer Risk

The equation used to calculate the total cancer risk at receptors that are exposed to multiple chemicals of potential concern within an individual exposure pathway is presented in (Equation 5).

$$\boldsymbol{Cancer\ Risk_T = \sum_i Cancer\ Risk_i} \qquad \textbf{Eq. 5}$$

Where:

$\boldsymbol{Cancer\ Risk_T}$

$=$ Total cancer risk for a specific exposure pathway

$\boldsymbol{Cancer\ Risk_i}$

$=$ Cancer risk for each COPC i for a specific exposure pathway

4.6.1.4 Cumulative Cancer Risk

The equation used to calculate the cumulative cancer risk from multiple exposure pathways is presented in (Equation 6). The cumulative cancer risk posed to a sensitive receptor is the sum of total risks from each individual exposure pathway.

$$\boldsymbol{Cumulative\ Cancer\ Risk_T = \sum_i Cancer\ Risk_T} \qquad \textbf{Eq. 6}$$

Where:

$\boldsymbol{Cumulative\ Cancer\ Risk_T}$

$=$ Cumulative cancer risk from multiple exposure pathways

$\boldsymbol{Cancer\ Risk_T}$

$=$ Cumulative cancer risk for exposure pathway T

4.6.2 Quantitatively Estimating Noncancer Hazard

This section describes the equations and risk methodologies used to calculate noncancer effects.

4.6.2.1 Inhalation Hazard Quotient

The equation for calculating inhalation hazard quotient is presented in (Equation 7).

$$\boldsymbol{HQ_{inhalation}} = \frac{\boldsymbol{EC} \times \mathbf{0.001}}{\boldsymbol{RfC}} \qquad \textbf{Eq. 7}$$

Where,

$$\boldsymbol{EC} = \frac{\boldsymbol{C_a} \times \boldsymbol{EF} \times \boldsymbol{ED}}{\boldsymbol{AT} \times \mathbf{365}\ \boldsymbol{days/year}} \qquad \textbf{Eq. 8}$$

Where:

$\boldsymbol{HQ_{Inhalation}}$

= Hazard quotient for direct inhalation of COPC noncarcinogen (unitless)

$\boldsymbol{EC}$

= Exposure concentration ($\mu g/m^3$)

$\mathbf{0.001}$

= Units conversion factor mg/g

$\boldsymbol{RfC}$

= Reference concentration (mg/m^3)

$\boldsymbol{C_a}$

= Total COPC air concentration ($\mu g/m^3$)

$\boldsymbol{EF}$

= Exposure frequency ($\frac{\text{days}}{\text{year}}$)

$\boldsymbol{ED}$

= Exposure duration (year)

$\boldsymbol{AT}$

= Averaging time (year)

4.6.2.2 Noncancer health effects associated with indirect exposure

The calculation for noncancer health effects associated with indirect exposure to chemicals of potential concern is presented in (Equation 9).

$$\boldsymbol{HQ} = \frac{\boldsymbol{ADD}}{\boldsymbol{RfD}} \qquad \textbf{Eq. 9}$$

Where:

$\boldsymbol{HQ}$

$=$ Hazard quotient (unitless)

$\boldsymbol{ADD}$

$=$ Average daily dose $\left(\frac{\text{mg}}{kg - day}\right)$

$\boldsymbol{RfD}$

$=$ Reference concentration (mg/m^3)

4.6.2.3 Hazard Index

The equation used to calculate the total chronic hazard linked to exposure to all chemicals of potential concern through a single exposure pathway is presented in (Equation 10) and is known as the Hazard index. The main assumption of this method is that health effects of the different chemicals are additive.

$$\boldsymbol{HI} = \sum_i \boldsymbol{HQ_i} \qquad \textbf{Eq. 10}$$

Where:

$\boldsymbol{HI}$

$=$ Hazard index for a specific exposure pathway

$\boldsymbol{HQ_i}$

$=$ Hazard quotient for COPC i

4.6.2.4 Cumulative Hazard Index

The equation used to calculate the hazard a receptor might be exposed to chemicals of potential concern associated with noncancer health effects through more than one exposure pathway, and from multiple on-road sources is presented in (Equation 11) and is known as the cumulative hazard index. A sensitive receptor's cumulative hazard is the sum of hazards from each individual exposure pathway.

$$\textbf{Cumulative } \boldsymbol{HI} = \sum \boldsymbol{HI} \qquad \textbf{Eq. 11}$$

Where:

$\boldsymbol{Cumulative\ HI}$

$=$ Cumulative hazard index from all scenario $-$ specific exposure pathways

$\boldsymbol{HI}$

$=$ Hazard index for a specifc exposure pathway

4.6.3 Noncancer Hazard (Acute Inhalation)

The equation for calculating acute (short term) inhalation hazard quotient is presented in (Equation 12).

$$\boldsymbol{AQH_{inhalation}} = \frac{\boldsymbol{C_{acute}} \times \mathbf{0.001}}{\boldsymbol{AIEC}} \qquad \textbf{Eq. 12}$$

Where:

$\boldsymbol{AQH_{Inhalation(i)}}$

$=$ Acute hazard quoteint for inhalation of COPC (unitless)

$\boldsymbol{C_{acute}}$

$=$ Acute air concentration ($\mu g/m^3$)

$\boldsymbol{AIEC}$

$=$ COPC acute inhalation exposure criteria (mg/m^3)

4.7 Cancer and Noncancer Numeric Target Levels

To quantify potential health risks, target levels are commonly established by the regulatory agency to gauge the magnitude of risk which in turn influences decisions regarding the management of risk. The definitions of Cancer risk, Noncancer hazard, and the numeric target levels for each are summarized in Tables 29 and 30.

Table 29: Cancer and Noncancer Hazard Definitions

	Definition
Cancer Risk	The probability of an individual developing cancer as result of exposure to an MSAT concentration. For example, a risk of 1E-05 is interpreted to mean that an individual has up to a one in 100,000 chances of developing cancer during their lifetime from the exposure being evaluated.
Hazard Quotient	The potential for developing noncancer health effects. A hazard is not a probability but, rather a comparison (calculated as a ratio) of a receptor's potential exposure relative to standard exposure level. The standard exposure level which includes the reference concentration (RfC) which is an estimated daily concentration of an MSAT in air, the exposure to which over a specific exposure duration poses no appreciable risk of adverse health effects, even to sensitive populations.

For this study and based on the U.S. EPA Region 6 Addendum (United States Environmental Protection Agency, 1998), the risk thresholds numeric target values set in IRAP-h View are summarized in Table 30. After the risk modeling is complete, the final risk and hazard values will be displayed in red if they exceed the defined threshold. If the final risk and hazard values are lower than the defined threshold, the value will be displayed in blue.

The values used in IRAP-h View are the recommended values published in the U.S. EPA Region 6 Addendum (authors of the U.S. EPA Human Health Risk Assessment Protocol). In this framework, these values will be referred to as U.S. EPA HHRAP target levels.

Table 30: Numeric Target Levels

Description	Value	Source
Cancer Risk Threshold	1.00E-05	U.S. EPA Region 6 (HHRAP)
Hazard Quotient Threshold	0.25	

The numeric risk values are not a discrete indicator of observed harmful effects. If the modeled risk values fall within the ranges in Table 30, it might be concluded, without further analysis, that a proposed action does not present an unacceptable risk. However, a calculated risk value that exceeds the targets in Table 30, would not indicate that the proposed action is not safe or that it presents an unacceptable risk. Rather, a risk calculation that exceeds the target values triggers further consideration of the underlying scientific basis and uncertainties associated with the risk calculation.

Chapter 5: ANALYSIS OF RESULTS AND VALIDATION

This chapter presents and discusses the risk results generated using the described methodologies in Chapter 2. Risk results are a statistically derived probability that an adverse health effect will happen at a defined exposure level. A case study is used to validate the modeled air dispersion results with actual measured results at the Saint Paul – Ramsey Health Center air monitoring station in Minnesota. This serves as the modeled versus measured study to see the effectiveness of the generated results in addressing the scope of research in Chapter 1.

5.1 Air Monitoring

Air monitoring stations are the golden standard for acquiring detailed information about air quality at a specific location. Measured ambient air concentrations from these stations are pivotal in validating how air dispersion models perform in studies, known as modeled versus measured analyses. Moreover, the measured air concentrations are used in estimating residential adult and residential child inhalation cancer risk and noncancer hazards.

5.2 Saint Paul – Ramsey Health Center Monitoring Site

The near-road air monitoring data used in this framework for the validation study is from the Saint Paul – Ramsey Health Center Monitoring Site in Saint Paul, Minnesota. The station is located at (UTM) Easting: 492230.00 m, Northing: 4977478.00 m, and zone 15. The main monitoring objective of the station is to record air toxics measurements. The station base elevation is 251 meters, above sea level, and the monitors are positioned on the north side of the building approximately 60 meters south of the I-94 corridor and interchange with I-35E (Figures 21). The station measures the following pollutants: $PM_{2.5}$, PM_{10}, metals, Carbon monoxide, Carbonyls, Ozone, Sulfur dioxide, Nitrogen oxides, Asbestos, and Volatile organic compounds (Minnesota Pollution Control Agency, 2015).

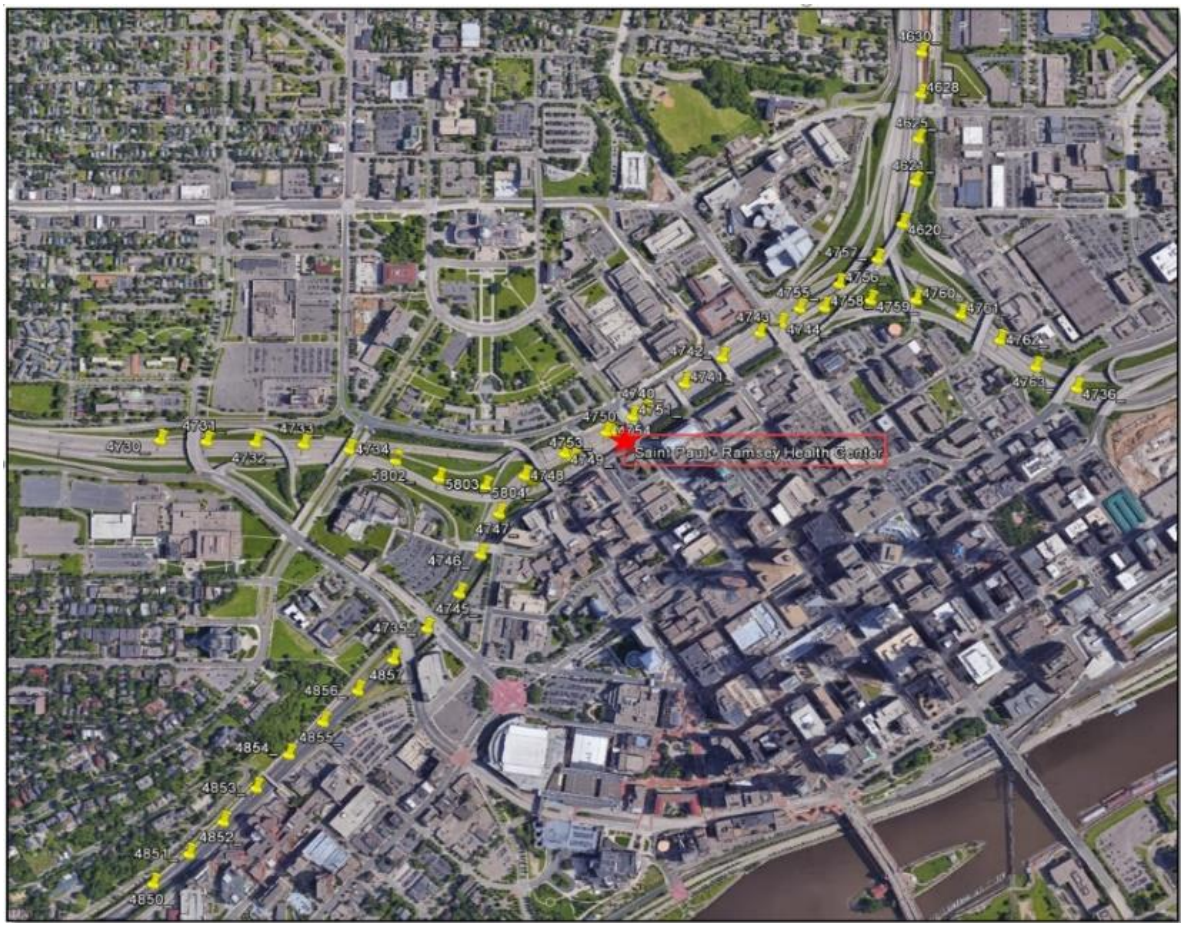


Figure 21: Saint Paul – Ramsey Health Center Monitoring Site

The air monitoring station is denoted by a red star in Figure 21. The yellow pins in Figure 21 represent the evaluated on-road mobile sources in the case study.

Figure 22-a illustrates the proximity of the air monitoring station from the roadway evaluated in this study. The monitoring equipment is displayed in Figure 22-b.

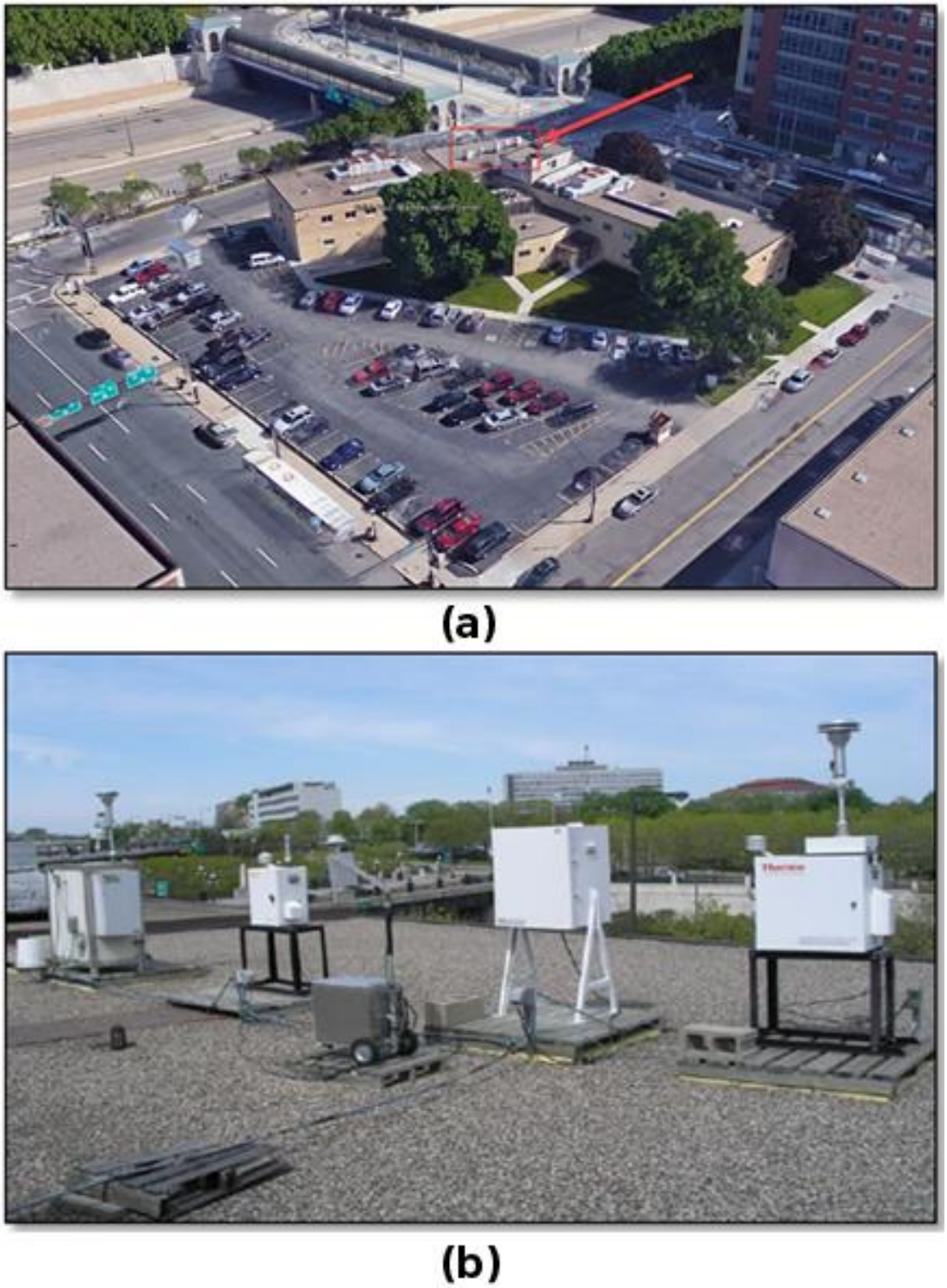


Figure 22: Saint Paul – Ramsey Health Center Monitoring Equipment

5.3 Roadway Segments

The 47 road segments modeled in this validation exercise are categorized as high traffic roadway segments. As mentioned earlier in section 1.7 (Area of Study), these roadway segments rank among the highest annual average daily traffic (AADT) in the Ramsey County, Minnesota. Table 31 summarizes the AADT and the corresponding number of roadway segments modeled. The roadway segments represent the modeled sources, and the total number of sources modeled are 47.

Table 31: Number of Roadway Segments Modeled and Corresponding AADT

AADT	Number of Roadway Segments Modeled
183,520	3
150,784	8
143,840	9
134,912	11
74,400	5
56,544	11
Total	47

One major impediment in quantifying health impacts from on-road emissions is roadway geometry characterization. The MOVES model, used for estimating emissions from on-road sources is geospatially unaware. To put it more simply, the MOVES model sees two roads with same vehicle volumes in two different geolocations with different weather conditions as one and the same.

Air dispersion models like AERMOD, require precise coordinates (e.g., Universal Transverse Mercator) to accurately model the dispersion of air toxics from mobile sources. Data in the coordinate system is used to characterize terrain, receptors, and mobile sources (modeled as area sources).

Figure 23 illustrates the road geometry characterization difference between the mobile emission models and the air dispersion models. Furthermore, the methodology to characterize the road segments in the emission models is different from the one in the air dispersion models.

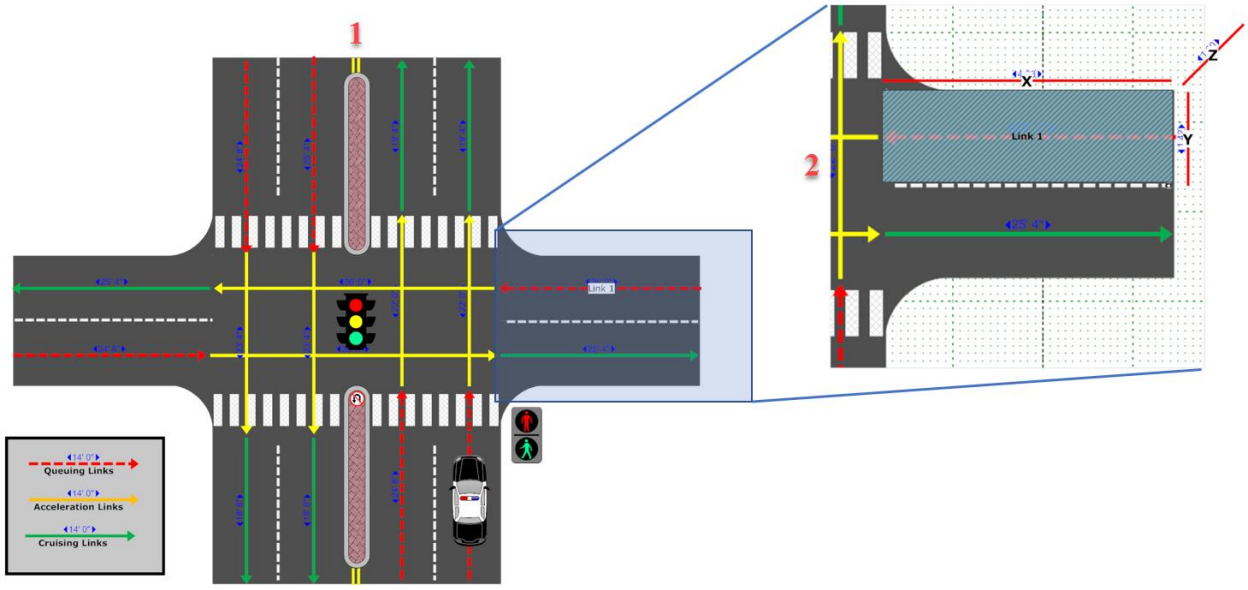


Figure 23: Difference Between Models

In Figure 23, the left-hand side illustration “1” represents a road in the mobile emissions realm, and how it is only characterized by vehicular activity (queuing links, acceleration links, and cruising links), with no consideration of precise coordinates.

Alternatively, the right-hand side illustration “2” renders the road as a set of precise coordinates within a known datum (e.g., World Geodetic System 1984 or North American Datum 1983).

Figures 24 and 25 illustrate how the road segments are spatially defined in the air dispersion model using area sources.

Figure 24 shows the project area with the base map overlay enabled and how the road segments are properly aligned with the air dispersion emission sources (red dotted lines representing area sources).

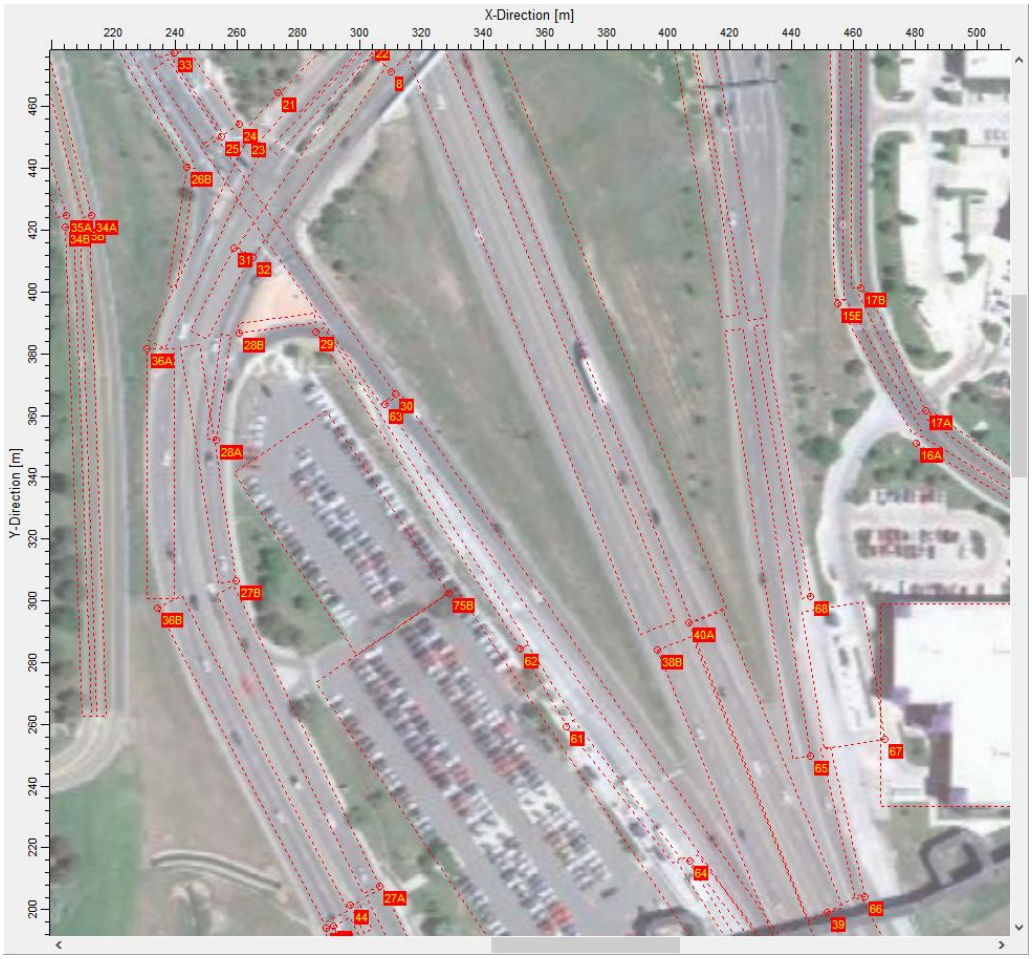


Figure 24: Roadway Segments Divided Based on Geometry and Vehicle Volumes

Figure 25 displays the area sources with the base map overlay inactive.

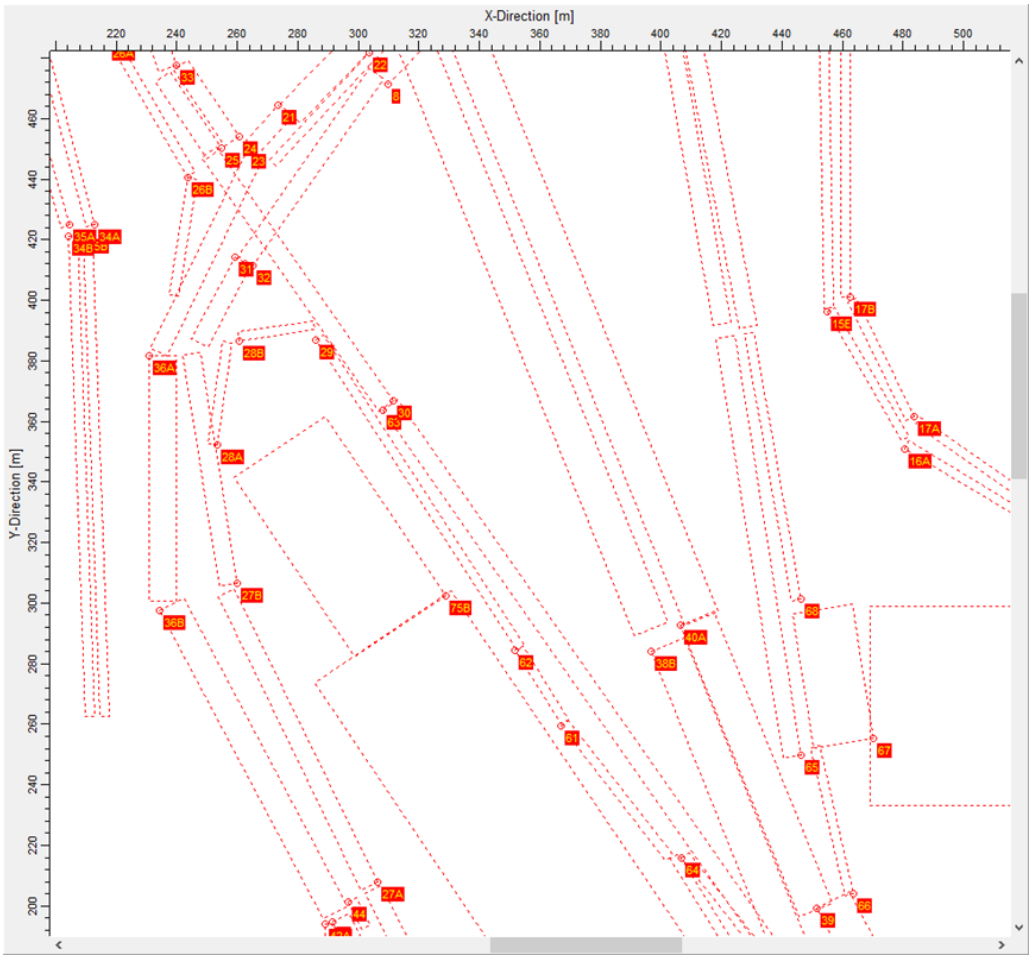


Figure 25: Roadway Segments Divided Based on Geometry Without a Base Map

5.4 Sources Parameterization and Project Setup

This section details the inputs and model setup and includes information on source characterization, AERMOD model options and settings, modeled mobile source air toxics, meteorological data, terrain, and risk grid setup.

5.4.1 Source Characterization

The roadway segments in this framework are modeled as area sources (See more details in section 2.6), the 47 roadway segments correspond to 47 area sources along the Interstate-35E and I-94/US 10 in the Saint Paul junction (See Figure 26). The area sources are represented by the red shading in Figure 26.

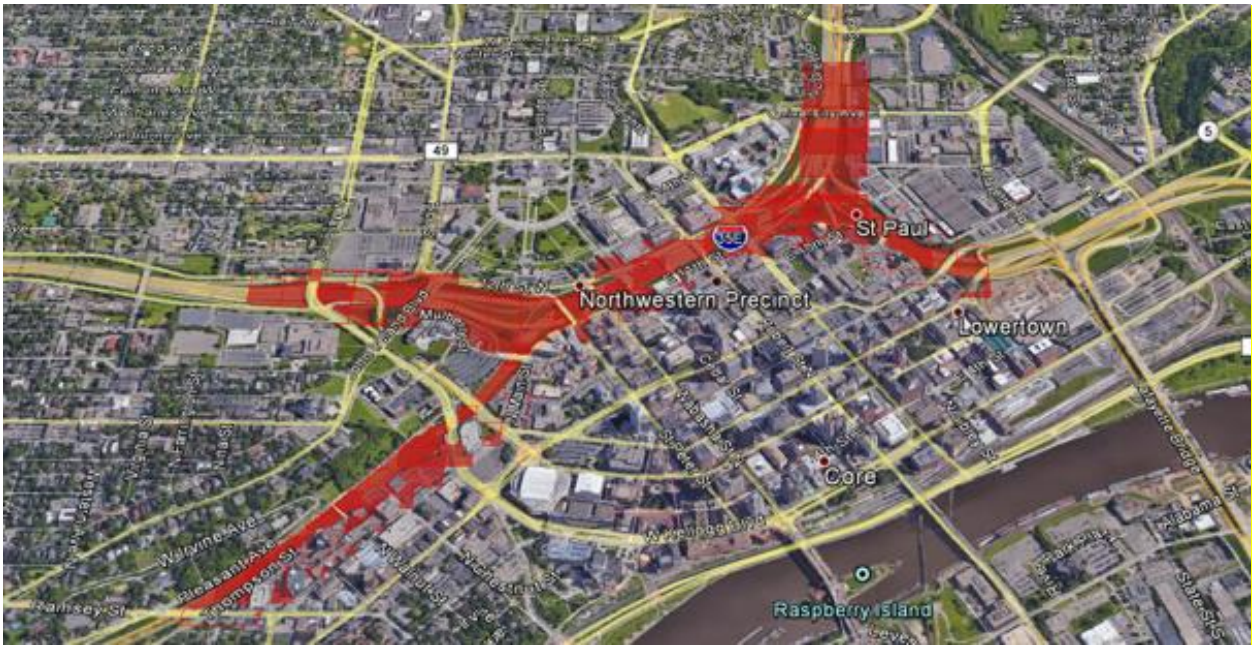


Figure 26: Area of Study - Interstate-35E and I-94/US 10 in Saint Paul Junction

5.4.2 AERMOD Model Options and Settings

The model input parameters for the 47 roadway sources in AERMOD are summarized in Table 32.

Table 32: AERMOD Model Options and Settings

Model Option	Setting
Model Options	Non-Regulatory Default Options Selected FASTAREA, AREADPLT Adjusted Friction Velocity
Calculation Type	Concentration, Total Deposition, Dry Deposition, and Wet Deposition
Emission Rate	1 gram of MSAT / second (Unitized)
Dispersion Coefficient	Rural
MSATs	See Section 5.4.3 for details
Averaging Periods	1-hour, Annual
Sources	See Table 33
Building Downwash	Not evaluated. Area sources are not part of the building downwash considerations
Receptors	See Section 5.4.6 for details
Terrain	Flat
Met Data Files	2011 hourly met data is employed for the final assessment of on-road mobile emissions impact

The geolocations of the on-road sources are expressed in the Universal Transverse Mercator coordinates system and are summarized in Table 33. This project is located at UTM Zone 15 T, the letter T is the latitude band. The base elevation values are calculated by the AERMAP preprocessor within the AERMOD modeling system (United States Environmental Protection Agency, 2015).

Table 33: Source Location

Area Source	Base elevation	X	Y
	meter	meter	meter
1	238.89	491144.23	4976404.75
2	237.65	491218.46	4976471.68
3	237.46	491289.17	4976542.77
4	238.76	491356.42	4976616.48
5	241.8	491424.11	4976690.37
6	240.85	491493.15	4976762.5
7	241	491565.35	4976831.51
8	240.54	491638.07	4976900.15
9	240.06	491708.64	4976970.94
10	239.2	491775.19	4977048.11
11	244.13	491818.73	4977135.26
12	268.85	491145.27	4977392.96
13	267.56	491242.65	4977390.26
14	266.43	491345.16	4977387.3
15	263.15	491446.08	4977383.2
16	259.46	491544.27	4977371.62
17	240.75	492756.45	4978284.52
18	236.42	492753.62	4978186.87
19	238.19	492748.41	4978084.64
20	237.23	492741.15	4977985.03
21	228.86	492713.69	4977889.51
22	232.21	493082.64	4977512.46
23	232.38	492996.05	4977560.03
24	232.84	492920.48	4977622.55
25	241.78	492248.95	4977521.64
26	262.56	491640.66	4977347.37
27	257.96	491732.09	4977306.99

Area Source	Base elevation	X	Y
	meter	meter	meter
28	252.3	491829.48	4977288.41
29	239.4	492329.02	4977581.51
30	250.21	491858.22	4977227.65
31	234.03	492836.99	4977680.8
32	241.03	492164.43	4977463.34
33	238.26	492408.77	4977637.88
34	239.94	492453.49	4977659.3
35	234.26	492660.12	4977808.84
36	235.74	492741.35	4977713.56
37	240.78	492546.53	4977696.16
38	237.41	492644.39	4977713.48
39	234.29	492577.28	4977749.9
40	236.32	492493.68	4977694.56
41	241.59	491910.09	4977312.89
42	237.56	491927.83	4977299.2
43	240.42	492138.78	4977446.34
44	239.04	491994.09	4977363.94
45	240.41	492012.34	4977351.8
46	241.53	492081.98	4977411.18
47	241.28	492093.16	4977413.92

5.4.3 Modeled Mobile Source Air Toxics

The mobile source air toxics modeled for the air concentrations modeled versus monitored analysis (validation study) include: Benzene, 1,3-Butadiene, Formaldehyde, Toluene, and Xylene. For the cumulative risk modeling, we included the following MSATs: Benzene, Formaldehyde, 1,3-Butadiene, hexavalent chromium, Indeno(1,2,3-cd)pyrene, and Benzo(a)pyrene. The motivation behind this selection is explained in Section 1.5.

5.4.4 Meteorological Data

The meteorological data used in this framework is collected from the Minneapolis–Saint Paul International Airport station. The distance between the station and the validation site is 12.7 kilometers. The surface station details are summarized below in Table 34.

Figure 27 displays a wind rose plot generated using WRPLOT View from Lakes Environmental (Lakes Environmental Software, 2018). The wind rose illustrates the frequency of occurrence of winds in each of the specified wind direction sectors and wind speed classes at the Minneapolis–Saint Paul International Airport station. Wind roses are useful because they provide the probability of downwind impacts. The station receives prevailing winds from the southeast. Wind speed and direction variability heavily influence transport of traffic-related air toxics and thus exposure. Studies found the average concentrations of mobile source air toxics in a neighborhood decreased as wind speeds increased. This is mainly due to horizontal dilution and vertical dilution, which is a function of mixing layer height (Kim, Lee, Woo, & Bae, 2015).

Table 34: Surface Station Details

	Station Details
Name	MINNEAPOLIS ST PAUL INTERNATIONAL AIRPORT, MN US
Network:ID	GHCND: USW00014922
UTM Easting	481922.53
UTM Northing	4969989.91
Elevation	265.8 m
Data Coverage	100 %

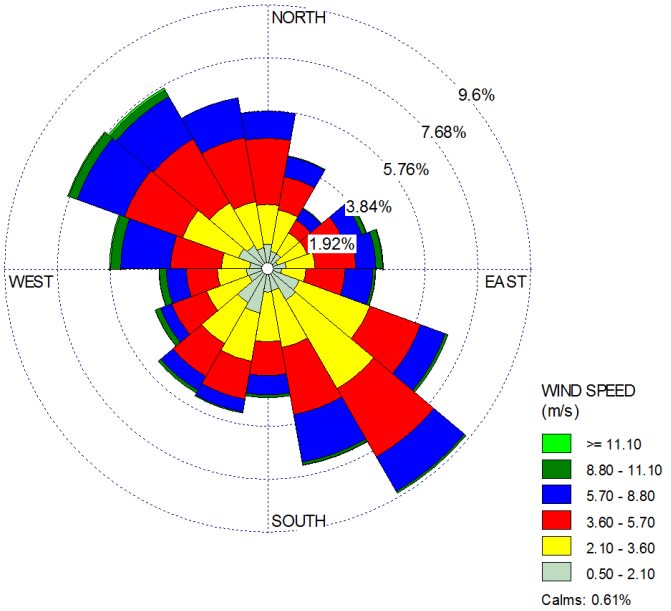


Figure 27: Wind Rose Plot for the Minneapolis–Saint Paul Int'l Airport Meteorological Station, 2011

Total No. of Hours:	8760
Average Wind Speed:	4.09 m/s
Calm Records:	53
Calm Winds Frequency:	0.61%
Data Availability:	99.84%
Incomplete/Missing Records:	14
Total Records Used:	8746

Figure 28: Station Data Quality

Figure 28 shows the data quality of the Minneapolis–Saint Paul International Airport meteorological station. Calm is a record reflected with a wind speed of 0.0 meter / second and a wind direction of 0 degrees.

5.4.5 Terrain

The project area is relatively flat. The average elevation of the state of Minnesota is 236 meters above sea level, and the overall state elevation ranges from 183 to over 700 meters (NETSTATE, 2016). The elevation profile in Figure 29 was extracted from Google Earth. The flat project area and the distance from coastal areas make the AERMOD model the best candidate to model such conditions. This is because AERMOD uses steady-state conditions throughout the modeling domain and will not resolve sub-domain features like sea breeze. The project center (yellow arrow in Figure 29) is located at Universal Transverse Mercator (UTM) Easting: 492230.00 m, Northing: 4977478.00 m, and zone 15. The numbers on the elevation profile in Figure 29 correspond to the following elevations in meters:

1. 227
2. 236
3. 243
4. 251
5. 260

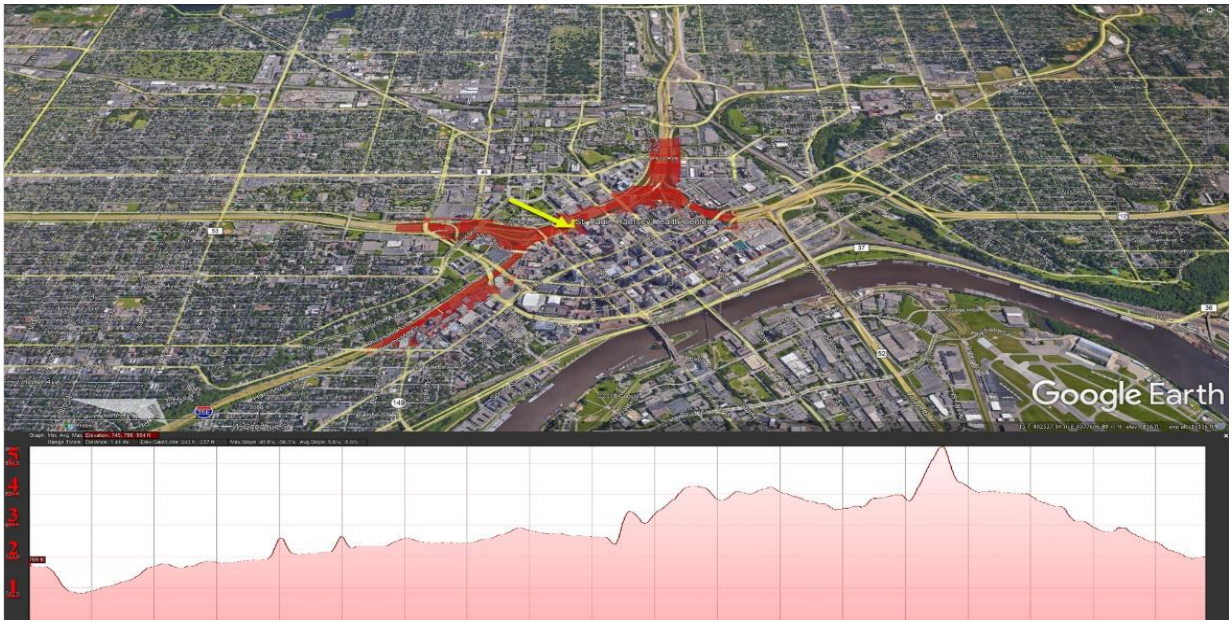


Figure 29: Project Area Elevation Profile

The terrain data used in this study is produced by the United States Geological Survey (USGS) (United States Geological Survey, 2015). The USGS NED GeoTIFF files, originate from the National Elevation Dataset. This format is recommended by the U.S. EPA for regulatory purposes. The AERMAP model executable supports the USGS NED GeoTIFF files and generates terrain information for the project area. In addition, AERMAP generates the height scale and base elevation for both the receptors and the on-road sources.

5.4.6 Risk Grid Setup

The risk grid setup for the air dispersion and deposition modeling performed in this framework is defined according to the U.S. EPA Human Health Risk Assessment Protocol (U.S. Environmental Protection Agency, 2005). The risk grid is a group of discrete Cartesian receptors, where human health impacts are computed. The risk grid is square in shape and the origin is at the center of the project domain. This grid is composed of two tiers, in the first tier the spacing between the receptors is 100 meters and the second tier spacing is 500 meters. In the air dispersion modeling realm, a receptor is defined as a specific location where the concentration is calculated at the ground level as opposed to some height above the ground. AERMAP, the model preprocessor for handling terrain elevations, processes receptors and sources to interpolate the elevations at each receptor point and format these in a way that can be absorbed by AERMOD. Figure 30 shows the layout of the risk grid in the modeling software graphical user interface.

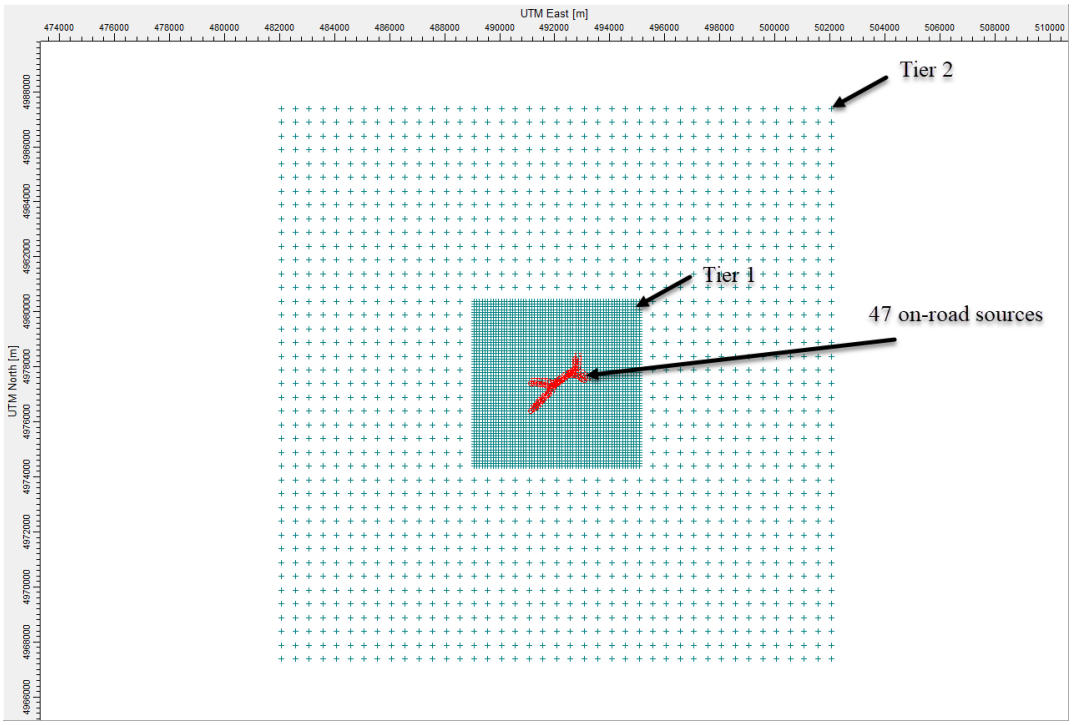


Figure 30: Risk Grid

5.5 Validation Study

This section includes the modeled versus measured study air concentrating results. After, we completed the air dispersion modeling, we compared the results (modeled results) with the actual monitored results at the near-road air monitoring station, Saint Paul – Ramsey Health Center Monitoring Site. Comparing modeled and measured concentrations is an elemental step in evaluating how the model is performing. The use of data from an air monitoring station to evaluate the model is known as validation of the model or "calibration". This is an iterative process, where the model is run and calibrated using measured data. Calibration is employed to reduce uncertainty and improve model performance.

We modeled the following MSATs: Benzene, 1,3-Butadiene, Formaldehyde, Toluene, and Xylene. Figure 31 shows the results of the study.

Parameter	Benzene	1,3 - Butadiene	Formaldehyde	Toluene	Xylenes
MPCA modeled (µg/m^3)	1.37	0.20	0.94	5.03	3.18
Monitored (µg/m^3)	0.71	0.14	2.200	1.600	0.77
Research Group modeled Conc. (µg/m^3)	0.64	0.08	0.69	2.58	1.69
MPCA: Modeled / Monitored	1.93	1.41	0.42	3.14	4.13
Research Group: Modeled / Monitored	0.91	0.60	0.32	1.61	2.19

Figure 31: Validation Study Results

In Figure 31, the parameters represent the following:

1. MPCA modeled: *Modeled concentrations performed by Minnesota Pollution Control Agency (MPCA).*
2. Monitored: *Observed concentrations at Saint Paul – Ramsey Health Center Monitoring Site.*
3. Research Group Modeled Conc.: *Modeled concentrations performed by the author of the thesis.*
4. MPCA: Modeled / Monitored: *The ratio of the MPCA modeled to observed concentrations.*
5. Research Group: Modeled / Monitored: *The ratio of the concentrations modeled by the author to the observed concentrations at Saint Paul – Ramsey Health Center Monitoring Site.*

The ratios presented in Figure 31 conform with the often-quoted *factor of two accuracy*, a ratio recognized in the air dispersion modeling field. The studies of model accuracy are discussed in greater detail in Section 9 of the Appendix W "Revision to the Guideline on Air Quality Models: Adoption of a Preferred General Purpose (Flat and Complex Terrain) Dispersion Model and Other Revisions; Final Rule" (United States Environmental Protection Agency, 2005).

The modeled versus measured study was reproduced independently and compared well with the MPCA study. A validated framework like this will be a great contribution to the assessment of the adverse impacts of toxic vehicle emissions.

5.6 Risk and Hazard Characterization

This section presents the risk and hazard characterization and the risk results. This section is broken down into the following subsections:

- Risk and Hazard Estimates.
- MSATs Evaluated.
- Scenarios.
- Pathways.
- Sensitive Receptor Locations.
- Chronic Cancer, Chronic Noncancer Hazard, and Acute Hazard.

5.6.1 Risk and Hazard Estimates

Risk, in terms of exposure to air emissions, is the probability that a human receptor will develop cancer, based on the unique set of exposure pathways, resulting dose and toxicity assumptions applied. As a probability, a risk of 1×10^{-5} is interpreted to mean that an individual has up to a one in 100,000 chances of developing cancer during their lifetime. From equation 4, the cancer risk is equivalent to lifetime average daily dose (mg / kg-day) multiplied by the MSAT-specific cancer slope factor $(\text{mg / kg-day})^{-1}$. The lifetime average daily dose (LADD) equals the product of the dose, exposure duration, and exposure frequency divided by the averaging time. Greater detail is found Section 4.6.1 (Quantitatively Estimating Cancer Risk). In contrast, Hazard is the potential for developing noncancer health effects as a result of the exposure being evaluated. Hazard is calculated as a comparison of calculated human exposure levels with the applicable pertinent toxicity benchmark for noncarcinogenic health endpoints. The hazard quotient is the ratio of the average daily dose (mg / kg-day) to the reference dose $(\text{mg / kg-day})^{-1}$.

5.6.2 MSATs Evaluated

The MSATs evaluated in this framework are Benzene, Formaldehyde, 1,3-Butadiene, Hexavalent chromium, Indeno(1,2,3-cd)pyrene, and Benzo(a)pyrene.

5.6.3 Scenarios

The human receptor population evaluated in this framework include the following scenarios:

- Resident Adult.
- Resident Child.
- Farmer Adult.
- Farmer Child.
- Fisher Adult.
- Fisher Child.

The exposure scenarios as recommended by the Human Health Risk Assessment Protocol (HHRAP) (U.S. Environmental Protection Agency, 2005) for the human health risk assessment framework are summarized in Table 27.

5.6.4 Pathways

The routes of exposure evaluated in this framework are shown in Figure 32.

The exposure scenario accounts for the combination of exposure pathways that a receptor may be exposed to Mobile Source Air Toxics (MSATs) in the environment. Dermal exposure to MSATs in the soil was not included in the risk calculations because of the insignificant contribution of the dermal exposure route to overall risk (U.S. Environmental Protection Agency, 2005).

Exposure Pathways	Symbol
Inhalation of Vapors and Particulates	
Incidental Ingestion of Soil	
Ingestion of Homegrown Produce	
Ingestion of Homegrown Beef	
Ingestion of Milk from Homegrown Cows	
Ingestion of Homegrown Chicken	
Ingestion of Eggs from Homegrown Chickens	
Ingestion of Homegrown Pork	

Figure 32: Routes of Exposure

5.6.5 Sensitive Receptor Locations

The receptor locations for the risk analysis were determined using the "Risk Receptor Identification Tool" in IRAP-h View. These locations are the grid nodes where the unitized air toxic concentrations and deposition fluxes were maximized. Table 35 summarizes the sensitive receptors identified in the study.

Table 35: Sensitive Receptor Locations (UTM Coordinate System)

Sensitive Receptor	Easting (m)	Northing (m)
Saint Paul - Ramsey Health Center	492230.04	4977478.54
Sensitive Receptor 1	492167.91	4977485.5
Sensitive Receptor 2	492567.91	4977785.5
Sensitive Receptor 3	491967.91	4977385.5

5.6.6 Chronic Cancer, Chronic Noncancer Hazard

The following sections present the results of the quantitative risk assessment and the air concentration contours. The risk results include the evaluation of potential excess lifetime cancer risks (ELCRs) and noncancer hazards estimated for each receptor population, for combined MSATs and overall exposure pathways.

The U.S. EPA finds ELCRs less than 1.00E-05 and noncancer hazard indices of less than 0.25 acceptable (value was modified to account for life style and background concentrations). For each mobile source air toxic evaluated in this framework, the following information is displayed:

- Unitized contour.
- MSAT-specific risk summary at Saint Paul - Ramsey Health Center.
- Total cancer risk broken down by source contribution for the resident scenario.

5.6.7 IRAP-h View Importing Source Coordinates

In the AERMOD modeling system, the X and Y coordinates (UTM Coordinate System) for the vertex of the area source occur in the southwest quadrant of the source, however, IRAP-h View imports the southwest vertices of the area source and NOT the centroid. This results in shifting the visual reference location of the sources by 50 meters in Google Earth (GE). Results of the modeling analysis ***<u>are not affected</u>*** by this shift. Figures 33 and 34 show this shift. The shifted sources are represented by the three orange boxed regions in Figure 34.

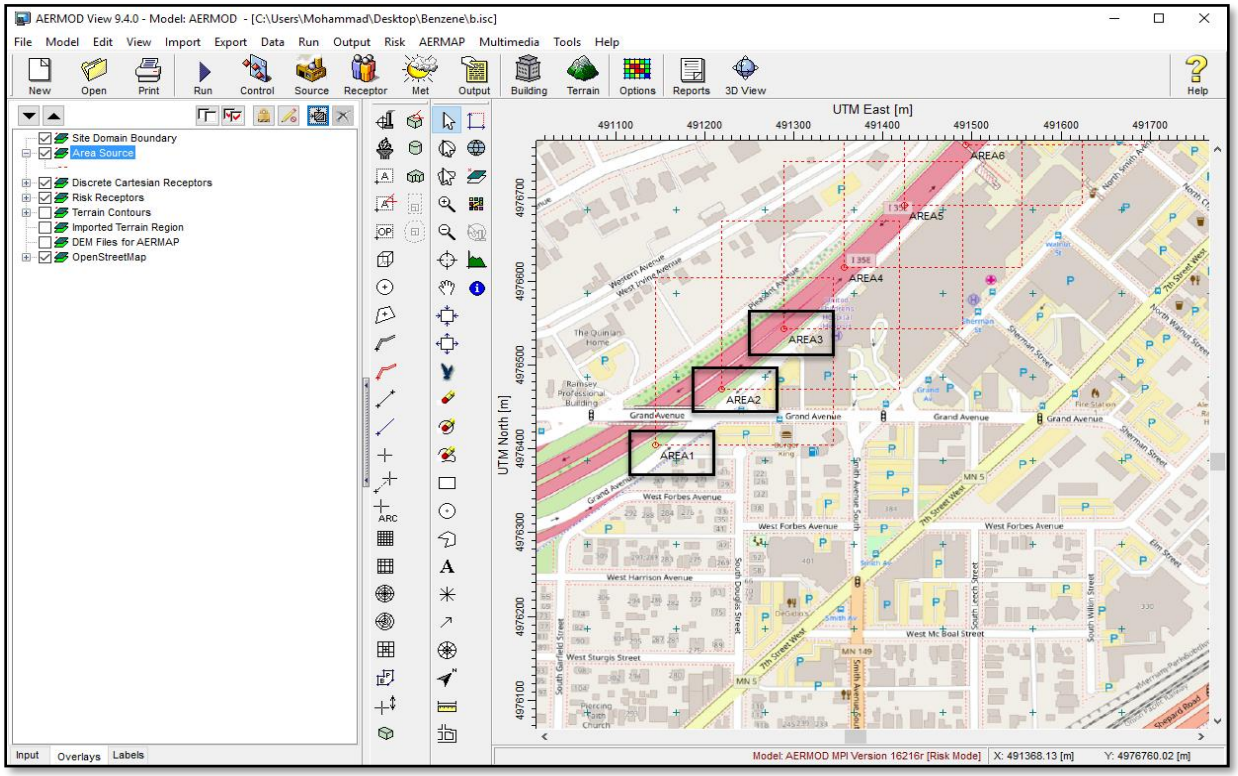


Figure 33: AERMOD Graphical User Interface

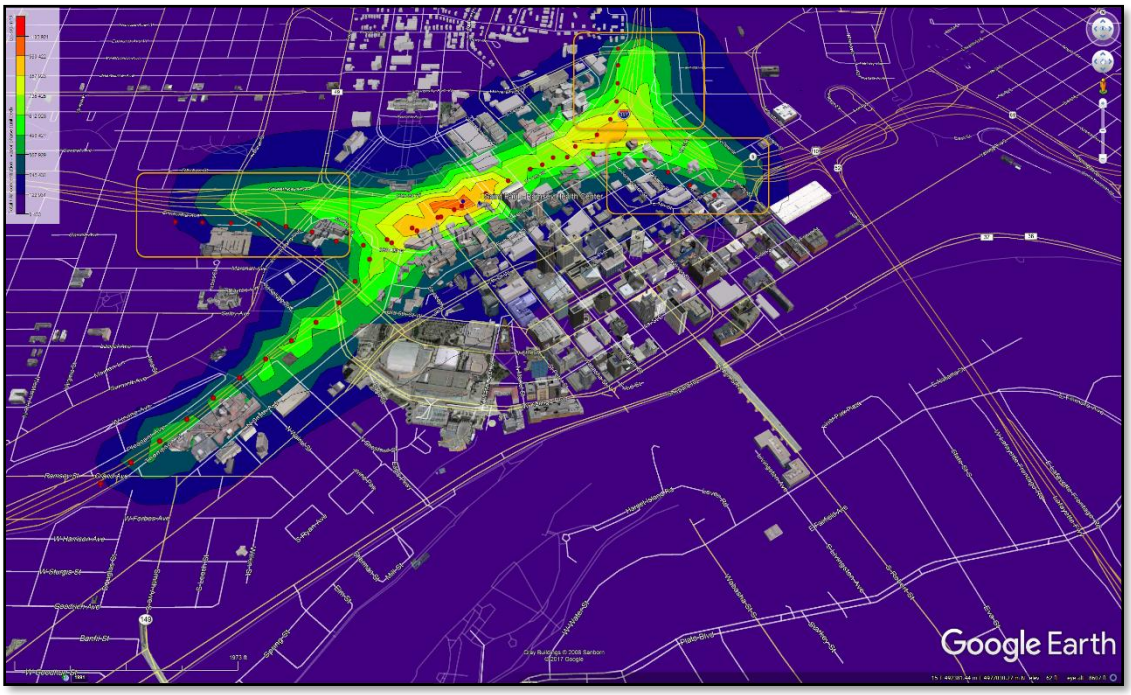


Figure 34: IRAP-h View GE Export

5.6.7.1 Benzene

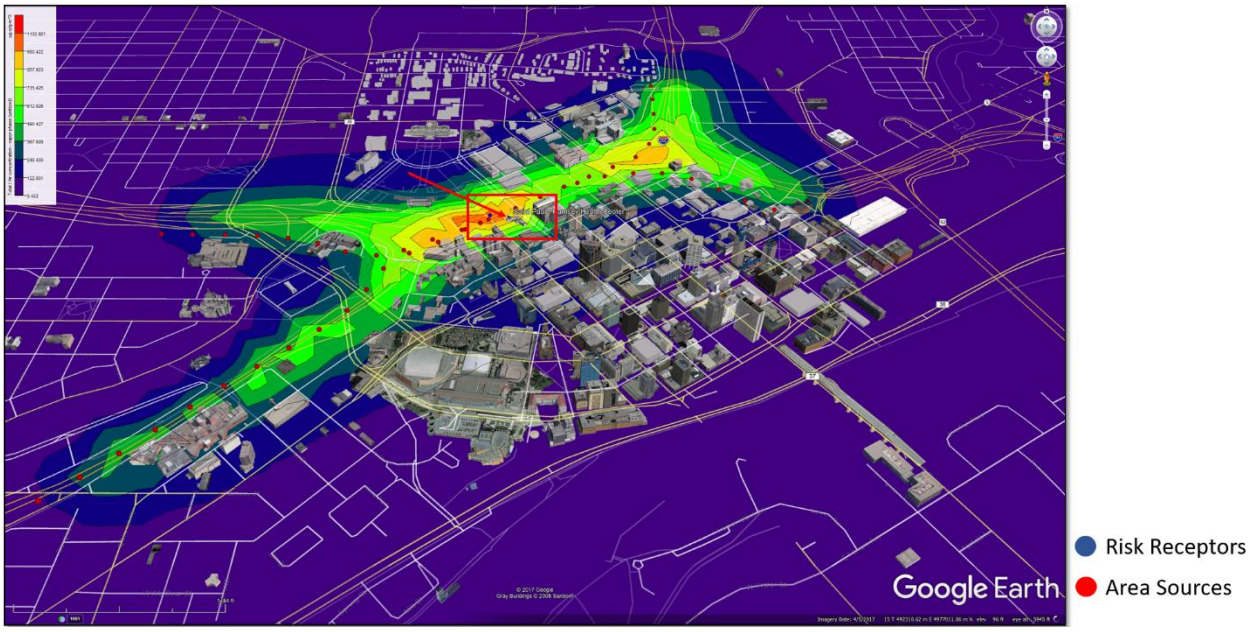


Figure 35: Benzene – Total / Air Conc. – Vapor Phase (Unitized)

In Figure 35, the red dots represent the area sources (on-road mobile sources), and the blue dots are the risk receptors (sensitive receptors) summarized in Table 35. The red arrow shows the Saint Paul - Ramsey Health Center risk receptor. Figure 35 shows the results for the unitized Benzene – Total / Air Conc. – Vapor Phase. The maximum air concentration is 1102.9 µg-s / g-m^3.

Risk	Resident Adult	Resident Child	Farmer Adult	Farmer Child	Fisher Adult	Fisher Child
Risk Receptor: Saint Paul - Ramsey Health Center						
Cancer	2.079E-06	4.158E-07	2.772E-06	4.158E-07	2.079E-06	4.158E-07
Hazard	2.073E-02	2.073E-02	2.073E-02	2.073E-02	2.073E-02	2.073E-02

Figure 36: Benzene – Risk Summary

The risk results in Figure 36 are color-coded, the blue color indicates the risk is below the U.S. EPA HHRAP target levels, whereas, the red indicates, the risk number exceeds the U.S. EPA HHRAP target levels allowed threshold. The cancer risk results and noncancer hazards for Benzene are below the target levels.

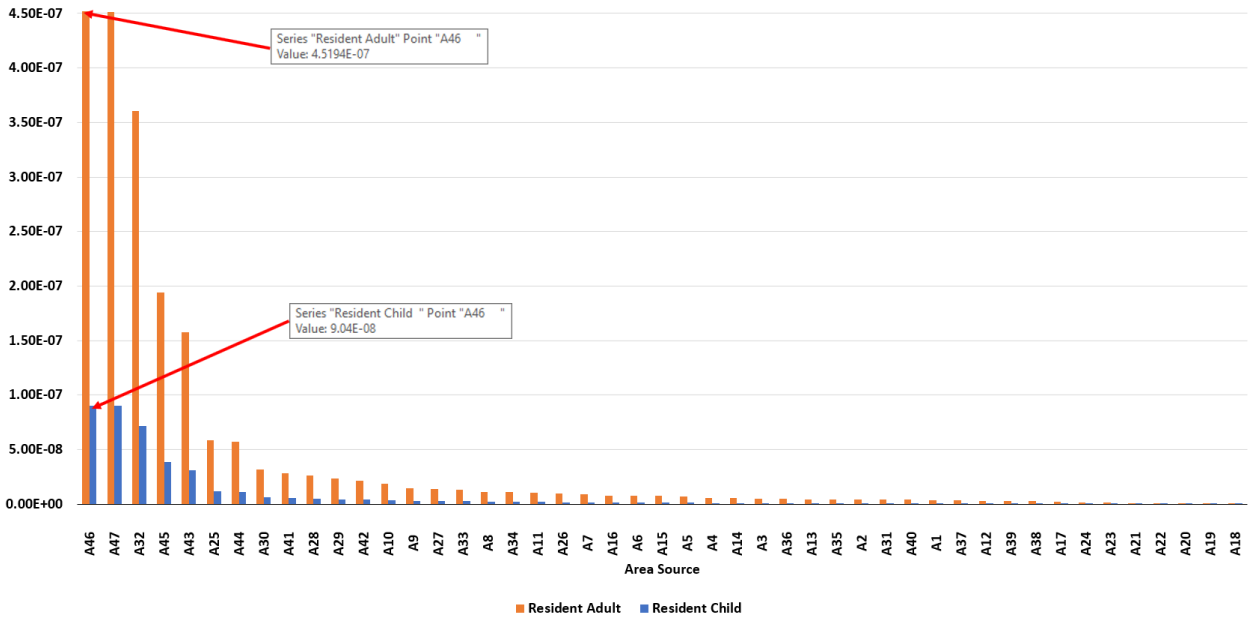


Figure 37: Total Cancer Risk by Source at Saint Paul - Ramsey Health Center

Figure 37 shows the contribution of each source to the total overall cancer risk for the adult and child scenarios. This is consistent with the fact that sources that emit more, contribute more to the overall risk at a specific location. The risk for the adult and child scenarios is not additive.

5.6.7.2 Formaldehyde

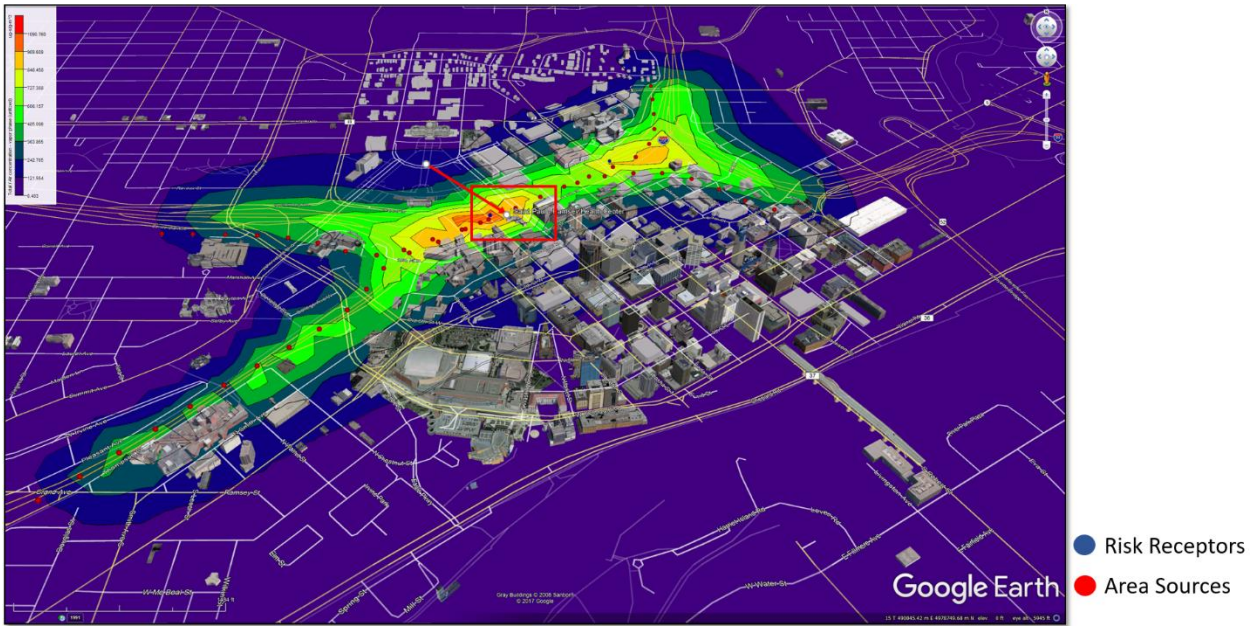


Figure 38: Formaldehyde – Total / Air Conc. – Vapor Phase (Unitized)

Figure 38 shows the results for the unitized Formaldehyde – Total / Air Conc. – Vapor Phase. The maximum air concentration is 1090.7 µg-s / g-m^3.

Risk	Resident Adult	Resident Child	Farmer Adult	Farmer Child	Fisher Adult	Fisher Child
Risk Receptor: Saint Paul - Ramsey Health Center						
Cancer	3.687E-06	7.374E-07	4.916E-06	7.375E-07	3.687E-06	7.374E-07
Hazard	6.800E-02	6.837E-02	6.810E-02	6.855E-02	6.800E-02	6.837E-02

Figure 39: Formaldehyde – Risk Summary

The cancer risk and noncancer hazard results for Formaldehyde at the Saint Paul - Ramsey Health Center are all below the U.S. EPA HHRAP target levels.

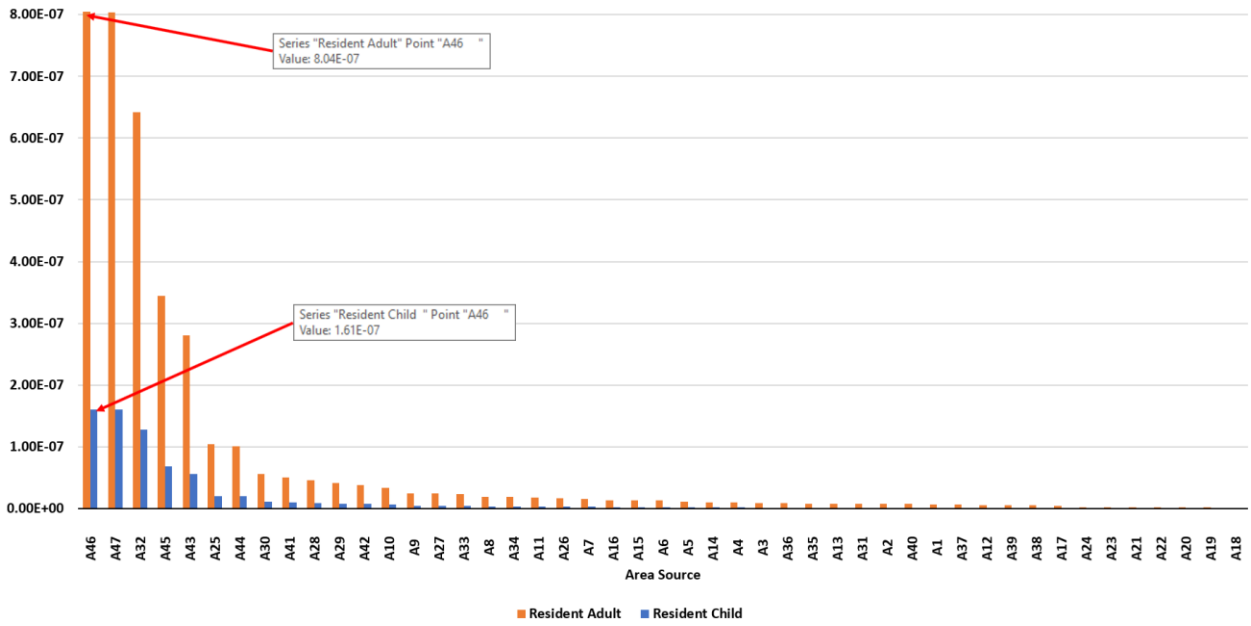


Figure 40: Total Cancer Risk by Source at Saint Paul - Ramsey Health Center

Figure 40 shows the contribution of each source to the Formaldehyde total overall cancer risk for the adult and child scenarios.

5.6.7.3 1,3-Butadiene

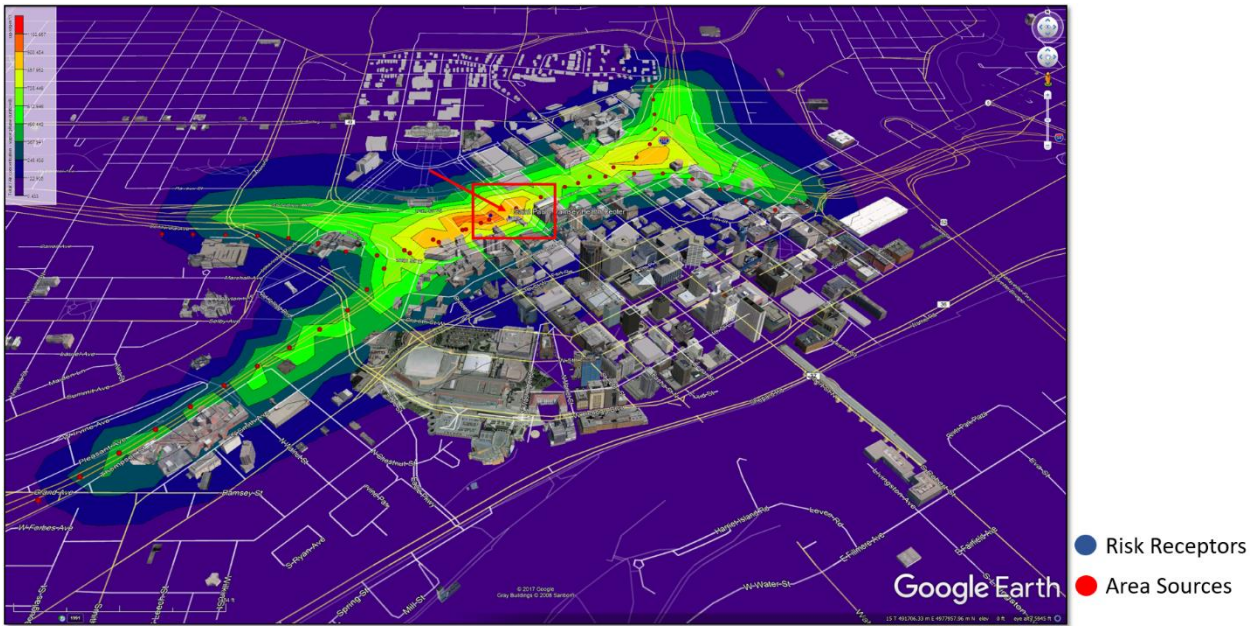


Figure 41: 1,3-Butadiene – Total / Air Conc. – Vapor Phase (Unitized)

Figure 41 shows the results for the unitized 1,3-Butadiene – Total / Air Conc. – Vapor Phase. The maximum air concentration is 1102.9 µg-s / g-m^3.

Risk	Resident Adult	Resident Child	Farmer Adult	Farmer Child	Fisher Adult	Fisher Child
Risk Receptor: Saint Paul - Ramsey Health Center						
Cancer	2.043E-06	4.087E-07	2.725E-06	4.087E-07	2.043E-06	4.087E-07
Hazard	4.054E-02	4.054E-02	4.054E-02	4.054E-02	4.054E-02	4.054E-02

Figure 42: 1,3-Butadiene – Risk Summary

As shown in Figure 42, the cancer risk and noncancer hazard results for 1,3-Butadiene at the Saint Paul - Ramsey Health Center are all below the U.S. EPA HHRAP target levels.

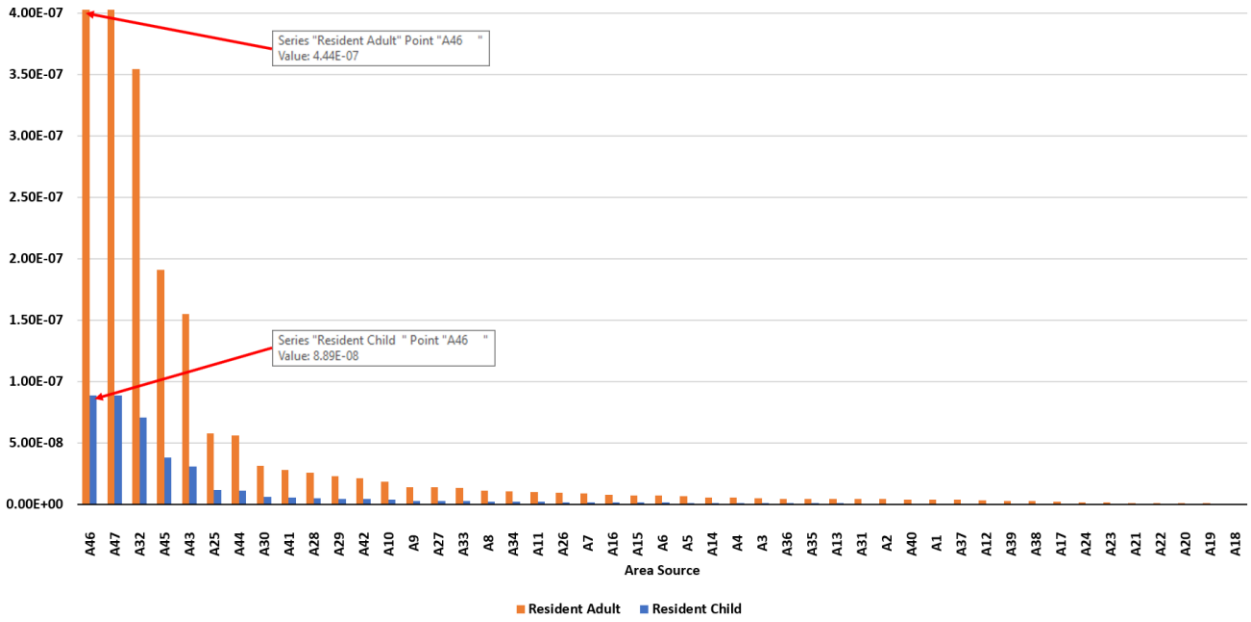


Figure 43: Total Cancer Risk by Source at Saint Paul - Ramsey Health Center

Figure 43 shows the contribution of each mobile source to the 1,3-Butadiene total overall cancer risk for the adult and child scenarios.

5.6.7.4 Hexavalent Chromium

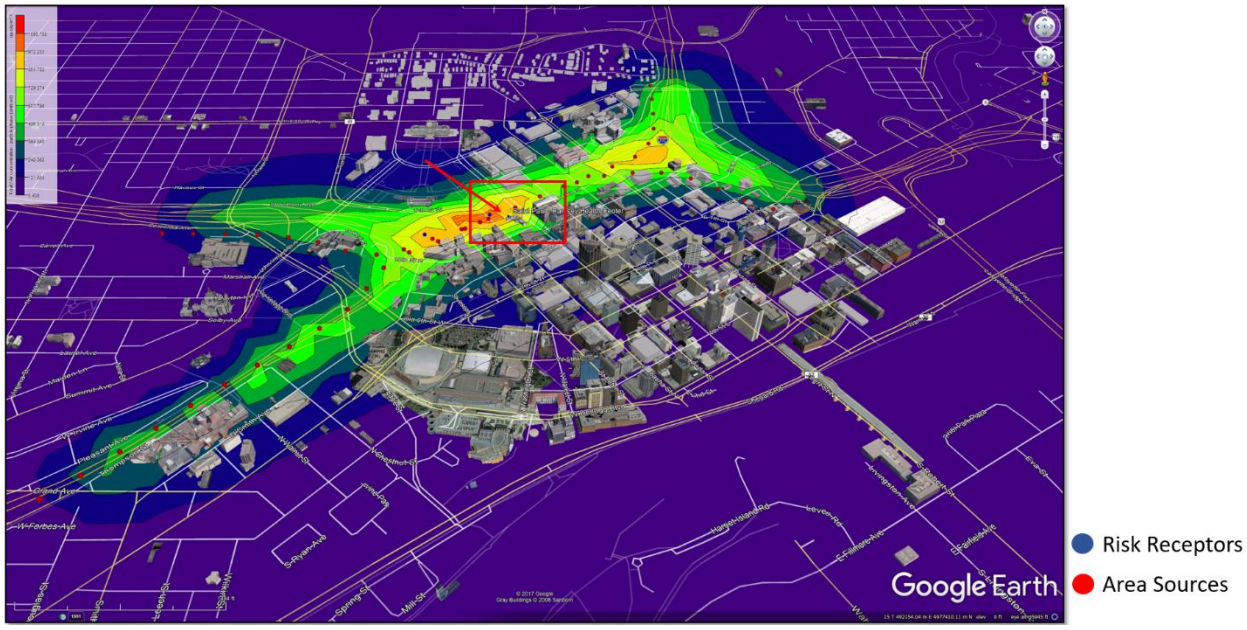


Figure 44: Hexavalent Chromium – Total / Air Conc. – Particle Phase (Unitized)

Figure 44 shows the results for the unitized Hexavalent Chromium – Total / Air Conc. – Particle Phase. The maximum air concentration is 1093.7 µg-s / g-m^3.

Risk	Resident Adult	Resident Child	Farmer Adult	Farmer Child	Fisher Adult	Fisher Child
Risk Receptor: Saint Paul - Ramsey Health Center						
Cancer	4.574E-09	9.147E-10	6.098E-09	9.147E-10	4.574E-09	9.147E-10
Hazard	1.112E-04	1.113E-04	1.116E-04	1.119E-04	1.112E-04	1.113E-04

Figure 45: Hexavalent Chromium – Risk Summary

Figure 45 summarizes the risk results for Hexavalent Chromium. All the cancer risk and noncancer hazard results at the Saint Paul - Ramsey Health Center are below the U.S. EPA HHRAP target levels.

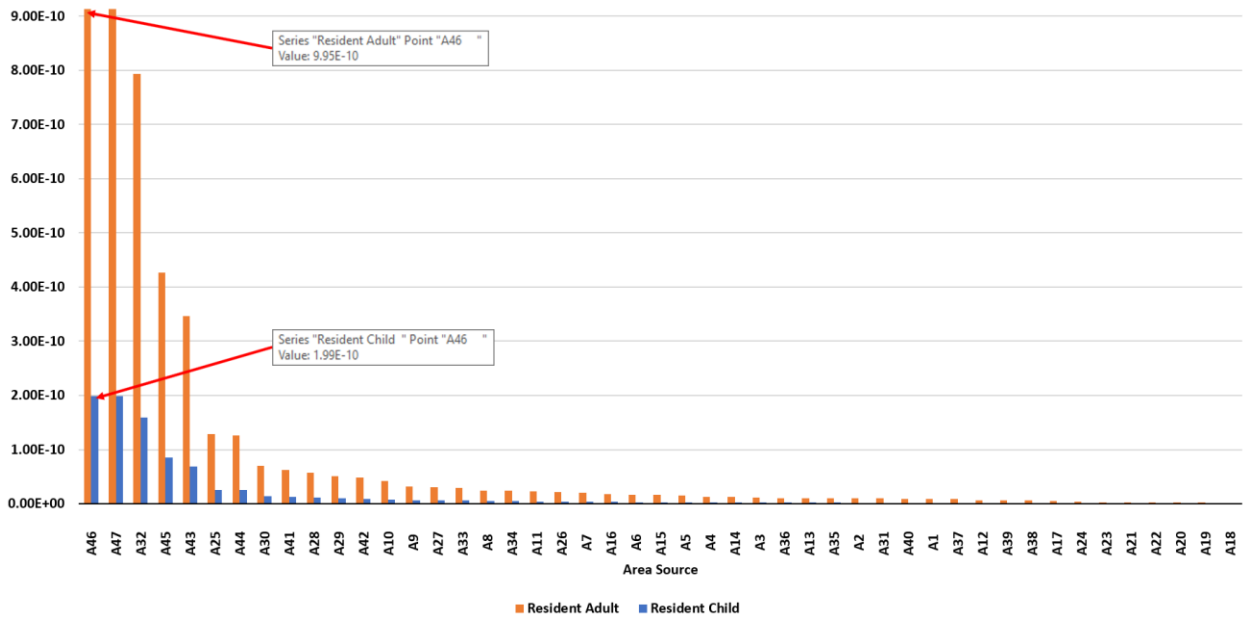


Figure 46: Total Cancer Risk by Source at Saint Paul - Ramsey Health Center

Figure 46 shows the contribution of each mobile source to the Hexavalent Chromium total overall cancer risk for the adult and child scenarios.

5.6.7.5 Indeno(1,2,3-cd)pyrene

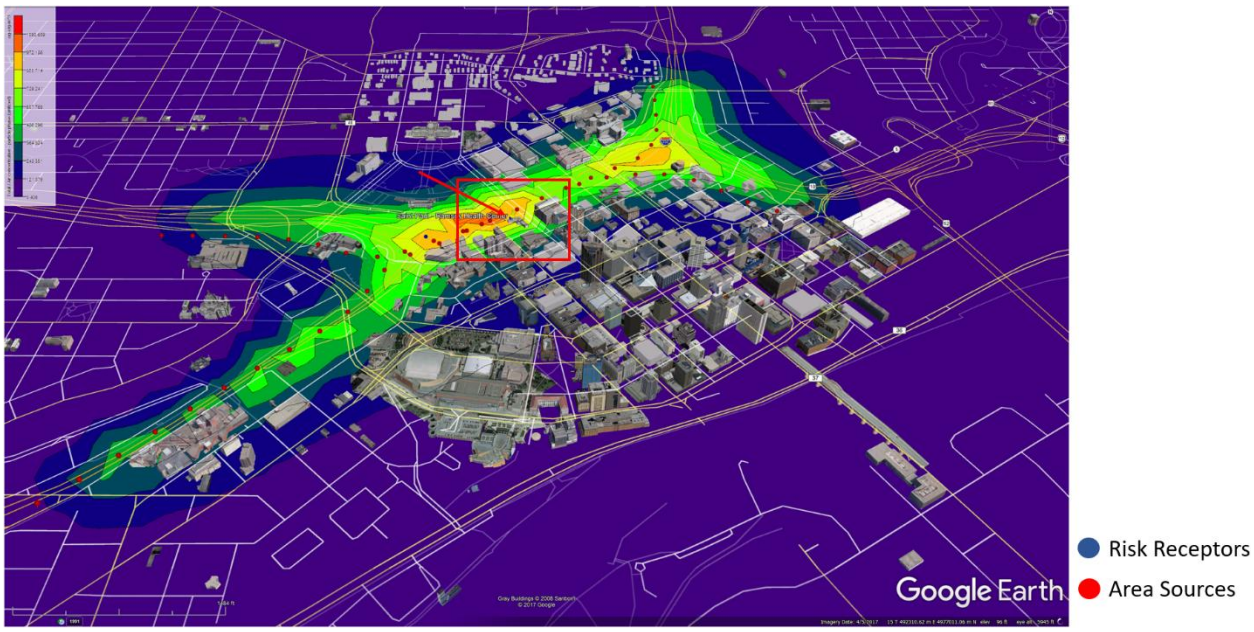


Figure 47: Indeno(1,2,3-cd)pyrene – Total / Air Conc. – Particle Phase (Unitized)

Figure 47 shows the results for the unitized Indeno(1,2,3-cd)pyrene – Total / Air Conc. – Particle Phase. The maximum air concentration is 1093.6 µg-s / g-m^3.

Risk	Resident Adult	Resident Child	Farmer Adult	Farmer Child	Fisher Adult	Fisher Child
Risk Receptor: Saint Paul - Ramsey Health Center						
Cancer	2.621E-08	9.122E-09	7.777E-07	1.695E-07	2.621E-08	9.122E-09
Hazard	-	-	-	-	-	-

Figure 48: Indeno(1,2,3-cd)pyrene – Risk Summary

As shown in Figure 48, the cancer risk results at the Saint Paul - Ramsey Health Center are below the U.S. EPA HHRAP target levels. As neither the RfD nor the RfC were assessed under the IRIS program, hazard quotients were not calculated for Indeno(1,2,3-cd)pyrene. Indeno(1,2,3-cd)pyrene is a common risk driver, however, that's from large combustion sources, so for mobile sources, with much lower emissions, the expected risk ranges from 1.00E-07 to 1.00E-09.

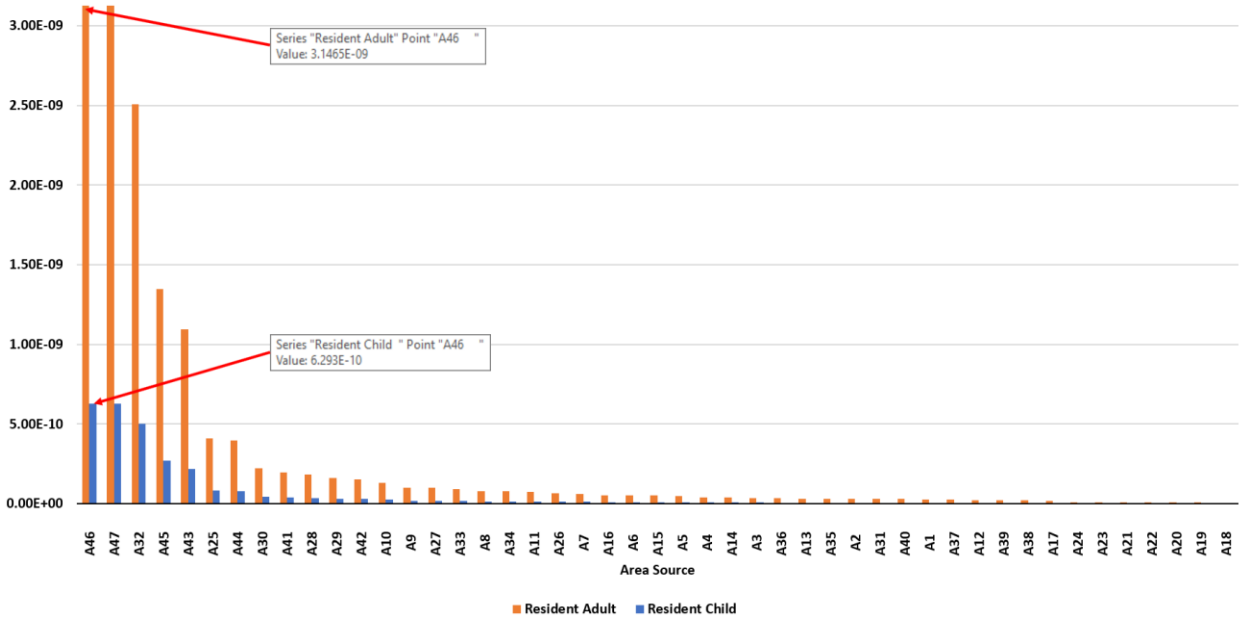


Figure 49: Inhalation Cancer Risk by Source at Saint Paul - Ramsey Health Center

Figure 49 shows the contribution of each on-road mobile source to the Indeno(1,2,3-cd)pyrene inhalation cancer risk.

5.6.7.6 Benzo(a)pyrene

Figure 50: Benzo(a)pyrene – Total / Air Conc. – Particle Bound (Unitized)

Figure 50 shows the results for the unitized Benzo(a)pyrene – Total / Air Conc. – Particle Bound Phase. The maximum air concentration is 1092 µg-s / g-m^3.

Risk	Resident Adult	Resident Child	Farmer Adult	Farmer Child	Fisher Adult	Fisher Child
Risk Receptor: Saint Paul - Ramsey Health Center						
Cancer	6.468E-07	2.478E-07	1.656E-04	3.554E-05	6.468E-07	2.478E-07
Hazard	2.816E-01	2.823E-01	4.135E-01	4.703E-01	2.816E-01	2.823E-01

Figure 51: Benzo(a)pyrene – Risk Summary

As shown in Figure 51, the cancer risk results at the Saint Paul - Ramsey Health Center exceed the U.S. EPA HHRAP target levels for the farmer scenario (adult and child). The noncancer hazard levels for all scenarios exceeds the U.S. EPA HHRAP target levels. Early-life exposure to carcinogenic mobile source air toxics with a mutagenic mode of action like Benzo(a)pyrene may result in a greater contribution to cancers appearing later in life.

To account for this, age-dependent adjustment factors (ADAFs) developed by the U.S. EPA are applied to the oral and inhalation slope factors for Benzo(a)pyrene.

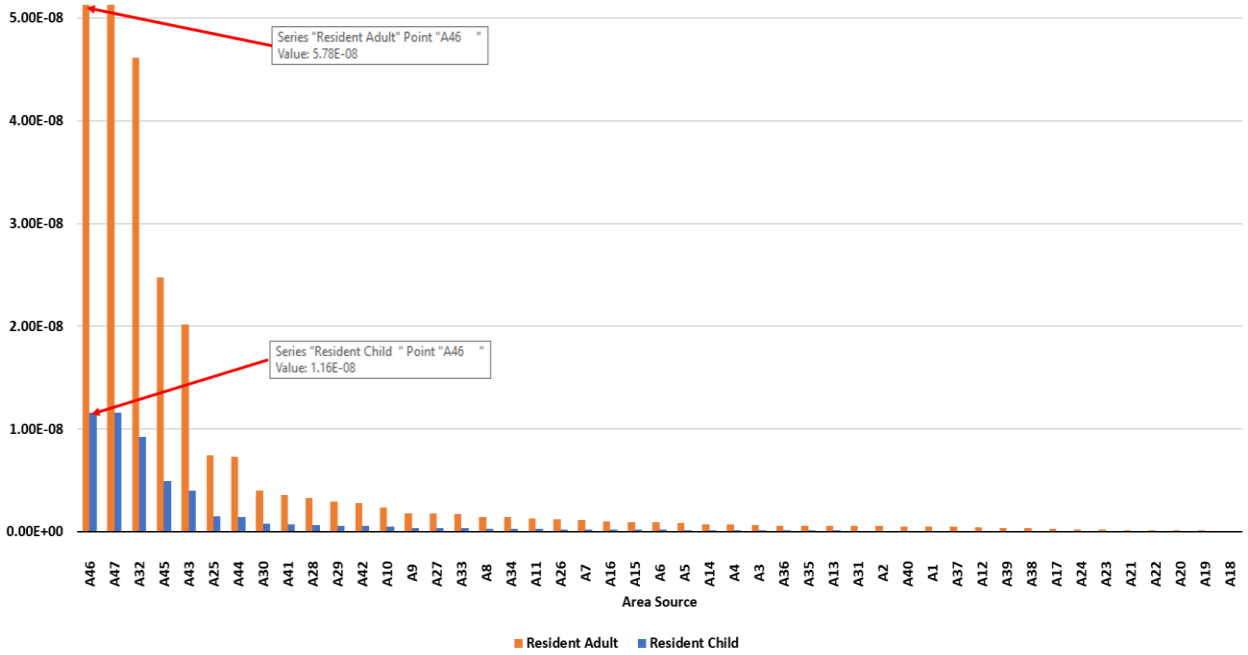


Figure 52: Inhalation Cancer Risk by Source at Saint Paul - Ramsey Health Center

Figure 52 shows the contribution of each of the 47 on-road mobile source to the Benzo(a)pyrene inhalation cancer risk.

5.6.8 Acute Hazard

Maximum 1-hour air concentrations generated using the AERMOD model are used to estimate the potential for noncancer human health effects resulting from short-term exposure to pollutants via the inhalation pathway. The 1-hour air concentrations are compared to acute inhalation exposure criteria (AIEC) to estimate the acute exposure potential. The hierarchical approach used to select the appropriate AIEC values are summarized in Section 3.9 (Toxicity Assessment). Table 36 provides a summary of the acute hazard quotients calculated for the MSATs included in the risk assessment.

Table 36: Acute Health Hazards (Unitless)

MSAT	Scenario
	Acute Inhalation
Benzene	1.59E-02
Formaldehyde	2.25E-01
1,3-Butadiene	4.08E-03
Hexavalent Chromium	9.81E-05
Indeno(1,2,3-cd)pyrene	2.03E-05
Benzo(a)pyrene	3.05E-05

Chapter 6: CONCLUSION, UNCERTAINTY ANALYSIS, AND FUTURE WORK

This chapter provides the results of the cumulative risk study, uncertainty analysis, conclusion, and finally, recommendations for future research.

6.1 Cumulative Risk

Cumulative risk is the set of risk present from the combined exposure to a variety of agents. Aggregate exposure describes that exposure from all sources of all risk agents. Cumulative risk is analogous with a human receptor breathing various mobile source air toxics (MSATs) over a lifetime exposure, in our case, being exposed to the modeled MSATs: Benzene, Formaldehyde, 1,3-Butadiene, Hexavalent chromium, Indeno(1,2,3-cd)pyrene, and Benzo(a)pyrene.

The cumulative risk results, cancer and hazard, for each of the considered MSATs were generated by finding the summation of risk (cancer risk and noncancer hazard) at each of the four sensitive receptors.

The cumulative risk results, excess lifetime cancer risks, and noncancer hazard indices are presented below.

6.1.1 Cumulative Risk (Cancer)

Excess Lifetime Cancer Risks (across all pathways)						
Sensitive Receptor / Exposure Scenarios	Resident		Farmer		Fisher	
	Adult	Child	Adult	Child	Adult	Child
Saint Paul - Ramsey Health Center	8.49E-06	1.82E-06	1.77E-04	3.73E-05	8.49E-06	1.82E-06
Sensitive Receptor 1	9.41E-06	2.03E-06	1.97E-04	4.14E-05	9.41E-06	2.03E-06
Sensitive Receptor 2	7.50E-06	1.61E-06	1.56E-04	3.29E-05	7.50E-06	1.61E-06
Sensitive Receptor 3	7.89E-06	1.69E-06	1.64E-04	3.45E-05	7.89E-06	1.69E-06

Region 6 Target Levels

Above Threshold

Below Threshold

Figure 53: Excess Lifetime Cancer Risks (across all pathways)

Figure 53 shows the cumulative cancer risk across all pathways. The cancer probabilities for the farmer scenario (adult and child) exceed the U.S. EPA HHRAP target levels at all sensitive receptors. The farmer is assumed to be exposed to mobile source air toxics through the following exposure pathways:

1. Direct inhalation of vapors and particulates.
2. Incidental ingestion of soil.
3. Ingestion of homegrown produce.
4. Ingestion of homegrown beef.
5. Ingestion of milk from homegrown cows.
6. Ingestion of homegrown chicken.
7. Ingestion of eggs from homegrown chickens.
8. Ingestion of homegrown pork.

Below are the individual air toxics excess lifetime cancer risks.

6.1.1.1 Benzene Excess Lifetime Cancer Risks

Sensitive Receptor / Exposure Scenarios	Resident		Farmer		Fisher	
	Adult	Child	Adult	Child	Adult	Child
Saint Paul - Ramsey Health Center	2.08E-06	4.16E-07	2.77E-06	4.16E-07	2.08E-06	4.16E-07
Sensitive Receptor 1	2.30E-06	4.60E-07	3.07E-06	4.60E-07	2.30E-06	4.60E-07
Sensitive Receptor 2	1.84E-06	3.67E-07	2.45E-06	3.67E-07	1.84E-06	3.67E-07
Sensitive Receptor 3	1.94E-06	3.87E-07	2.58E-06	3.87E-07	1.94E-06	3.87E-07

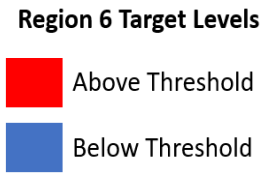


Figure 54: Benzene Excess Lifetime Cancer Risks (across all pathways)

6.1.1.2 Formaldehyde Excess Lifetime Cancer Risks

Sensitive Receptor / Exposure Scenarios	Resident		Farmer		Fisher	
	Adult	Child	Adult	Child	Adult	Child
Saint Paul - Ramsey Health Center	3.69E-06	7.37E-07	4.92E-06	7.37E-07	3.69E-06	7.37E-07
Sensitive Receptor 1	4.07E-06	8.14E-07	5.42E-06	8.14E-07	4.07E-06	8.14E-07
Sensitive Receptor 2	3.25E-06	6.49E-07	4.33E-06	6.50E-07	3.25E-06	6.49E-07
Sensitive Receptor 3	3.42E-06	6.84E-07	4.56E-06	6.84E-07	3.42E-06	6.84E-07

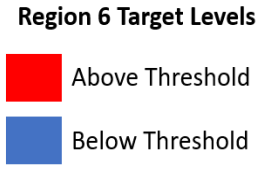


Figure 55: Formaldehyde Excess Lifetime Cancer Risks (across all pathways)

6.1.1.3 1,3-Butadiene Excess Lifetime Cancer Risks

Sensitive Receptor / Exposure Scenarios	Resident		Farmer		Fisher	
	Adult	Child	Adult	Child	Adult	Child
Saint Paul - Ramsey Health Center	2.04E-06	4.09E-07	2.72E-06	4.09E-07	2.04E-06	4.09E-07
Sensitive Receptor 1	2.26E-06	4.52E-07	3.01E-06	4.52E-07	2.26E-06	4.52E-07
Sensitive Receptor 2	1.81E-06	3.61E-07	2.41E-06	3.61E-07	1.81E-06	3.61E-07
Sensitive Receptor 3	1.90E-06	3.81E-07	2.54E-06	3.81E-07	1.90E-06	3.81E-07

Region 6 Target Levels

Above Threshold

Below Threshold

Figure 56: 1,3-Butadiene Excess Lifetime Cancer Risks (across all pathways)

6.1.1.4 Hexavalent Chromium Excess Lifetime Cancer Risks

Sensitive Receptor / Exposure Scenarios	Resident		Farmer		Fisher	
	Adult	Child	Adult	Child	Adult	Child
Saint Paul - Ramsey Health Center	4.57E-09	9.15E-10	6.10E-09	9.15E-10	4.57E-09	9.15E-10
Sensitive Receptor 1	5.05E-09	1.01E-09	6.73E-09	1.01E-09	5.05E-09	1.01E-09
Sensitive Receptor 2	4.03E-09	8.06E-10	5.38E-09	8.06E-10	4.03E-09	8.06E-10
Sensitive Receptor 3	4.25E-09	8.50E-10	5.67E-09	8.50E-10	4.25E-09	8.50E-10

Region 6 Target Levels

Above Threshold

Below Threshold

Figure 57: Hexavalent Chromium Excess Lifetime Cancer Risks (across all pathways)

6.1.1.5 Indeno(1,2,3-cd)pyrene Excess Lifetime Cancer Risks

Sensitive Receptor / Exposure Scenarios	Resident		Farmer		Fisher	
	Adult	Child	Adult	Child	Adult	Child
Saint Paul - Ramsey Health Center	2.62E-08	9.12E-09	7.78E-07	1.70E-07	2.62E-08	9.12E-09
Sensitive Receptor 1	3.07E-08	1.10E-08	9.75E-07	2.13E-07	3.07E-08	1.10E-08
Sensitive Receptor 2	2.40E-08	8.50E-09	7.42E-07	1.62E-07	2.40E-08	8.50E-09
Sensitive Receptor 3	2.47E-08	8.64E-09	7.42E-07	1.62E-07	2.47E-08	8.64E-09

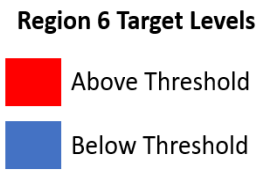


Figure 58: Indeno(1,2,3-cd)pyrene Excess Lifetime Cancer Risks (across all pathways)

6.1.1.6 Benzo(a)pyrene Excess Lifetime Cancer Risks

Sensitive Receptor / Exposure Scenarios	Resident		Farmer		Fisher	
	Adult	Child	Adult	Child	Adult	Child
Saint Paul - Ramsey Health Center	6.47E-07	2.48E-07	1.66E-04	3.55E-05	6.47E-07	2.48E-07
Sensitive Receptor 1	7.44E-07	2.90E-07	1.84E-04	3.95E-05	7.44E-07	2.90E-07
Sensitive Receptor 2	5.85E-07	2.26E-07	1.46E-04	3.14E-05	5.85E-07	2.26E-07
Sensitive Receptor 3	6.06E-07	2.33E-07	1.53E-04	3.29E-05	6.06E-07	2.33E-07

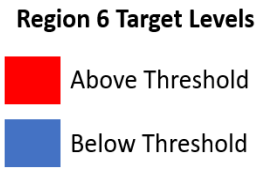


Figure 59: Benzo(a)pyrene Excess Lifetime Cancer Risks (across all pathways)

Amongst the modeled air toxics in this framework, Benzo(a)pyrene was the only air toxic with excess lifetime cancer risk across all pathways exceeding the U.S. EPA HHRAP target levels. The exceedances occurred in the farmer scenario, adult and child.

6.1.2 Cumulative Risk (Hazard Indices)

Noncancer Hazard Indices (across all pathways)						
Sensitive Receptor / Exposure Scenarios	Resident		Farmer		Fisher	
	Adult	Child	Adult	Child	Adult	Child
Saint Paul - Ramsey Health Center	0.41	0.41	0.54	0.60	0.41	0.41
Sensitive Receptor 1	0.45	0.45	0.60	0.66	0.45	0.45
Sensitive Receptor 2	0.36	0.36	0.48	0.53	0.36	0.36
Sensitive Receptor 3	0.38	0.38	0.50	0.56	0.38	0.38

Region 6 Target Levels

Above Threshold

Below Threshold

Figure 60: Noncancer Hazard Indices (across all pathways)

Figure 60 shows the cumulative noncancer hazard indices across all pathways. The hazard indices for all exposure scenarios exceed the U.S. EPA HHRAP target levels at all sensitive receptors. The resident and fisher are assumed to be exposed to mobile source air toxics through the exposure pathways discussed below. The farmer scenario was discussed in Section 6.1.1.

The resident exposure scenario accounts for the combination of exposure pathways that a receptor may be exposed to in an urban or rural (non-farm) setting. The resident is assumed to be exposed to mobile source air toxics through the following exposure pathways:

1. Direct inhalation of vapors and particulates.
2. Incidental ingestion of soil.
3. Ingestion of homegrown produce.

The fisher exposure scenario accounts for the combination of exposure pathways that a receptor may be exposed to in an urban or rural setting where fish is the main component of the receptor diet. The fisher is assumed to be exposed to mobile source air toxics through the following exposure pathways:

1. Direct inhalation of vapors and particulates.
2. Incidental ingestion of soil.
3. Ingestion of homegrown produce.
4. Ingestion of fish.

In the subsequent sections, we present the individual air toxics noncancer hazard indices.

6.1.2.1 Benzene Noncancer Hazard Indices

Sensitive Receptor / Exposure Scenarios	Resident		Farmer		Fisher	
	Adult	Child	Adult	Child	Adult	Child
Saint Paul - Ramsey Health Center	2.07E-02	2.07E-02	2.07E-02	2.07E-02	2.07E-02	2.07E-02
Sensitive Receptor 1	2.29E-02	2.29E-02	2.29E-02	2.29E-02	2.29E-02	2.29E-02
Sensitive Receptor 2	1.83E-02	1.83E-02	1.83E-02	1.83E-02	1.83E-02	1.83E-02
Sensitive Receptor 3	1.93E-02	1.93E-02	1.93E-02	1.93E-02	1.93E-02	1.93E-02

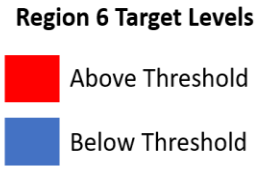


Figure 61: Benzene Noncancer Hazard Indices (across all pathways)

6.1.2.2 Formaldehyde Noncancer Hazard Indices

Sensitive Receptor / Exposure Scenarios	Resident		Farmer		Fisher	
	Adult	Child	Adult	Child	Adult	Child
Saint Paul - Ramsey Health Center	6.80E-02	6.84E-02	6.81E-02	6.86E-02	6.80E-02	6.84E-02
Sensitive Receptor 1	7.51E-02	7.56E-02	7.52E-02	7.58E-02	7.51E-02	7.56E-02
Sensitive Receptor 2	5.99E-02	6.03E-02	6.00E-02	6.05E-02	5.99E-02	6.03E-02
Sensitive Receptor 3	6.31E-02	6.35E-02	6.32E-02	6.37E-02	6.31E-02	6.35E-02

Region 6 Target Levels

Above Threshold

Below Threshold

Figure 62: Formaldehyde Noncancer Hazard Indices (across all pathways)

6.1.2.3 1,3-Butadiene Noncancer Hazard Indices

Sensitive Receptor / Exposure Scenarios	Resident		Farmer		Fisher	
	Adult	Child	Adult	Child	Adult	Child
Saint Paul - Ramsey Health Center	4.05E-02	4.05E-02	4.05E-02	4.05E-02	4.05E-02	4.05E-02
Sensitive Receptor 1	4.49E-02	4.49E-02	4.49E-02	4.49E-02	4.49E-02	4.49E-02
Sensitive Receptor 2	3.58E-02	3.58E-02	3.58E-02	3.58E-02	3.58E-02	3.58E-02
Sensitive Receptor 3	3.78E-02	3.78E-02	3.78E-02	3.78E-02	3.78E-02	3.78E-02

Region 6 Target Levels

Above Threshold

Below Threshold

Figure 63: 1,3-Butadiene Noncancer Hazard Indices (across all pathways)

6.1.2.4 Hexavalent Chromium Noncancer Hazard Indices

Sensitive Receptor / Exposure Scenarios	Resident		Farmer		Fisher	
	Adult	Child	Adult	Child	Adult	Child
Saint Paul - Ramsey Health Center	1.11E-04	1.11E-04	1.12E-04	1.12E-04	1.11E-04	1.11E-04
Sensitive Receptor 1	1.23E-04	1.23E-04	1.23E-04	1.24E-04	1.23E-04	1.23E-04
Sensitive Receptor 2	9.80E-05	9.81E-05	9.84E-05	9.87E-05	9.80E-05	9.81E-05
Sensitive Receptor 3	1.03E-04	1.03E-04	1.04E-04	1.04E-04	1.03E-04	1.03E-04

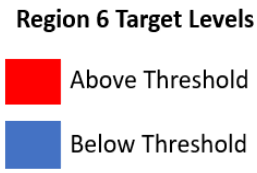


Figure 64: Hexavalent Chromium Noncancer Hazard Indices (across all pathways)

6.1.2.5 Indeno(1,2,3-cd)pyrene Noncancer Hazard Indices

Since neither the RfD nor the RfC were assessed under the IRIS program, hazard quotients were not calculated for Indeno(1,2,3-cd)pyrene.

6.1.2.6 Benzo(a)pyrene Noncancer Hazard Indices

Sensitive Receptor / Exposure Scenarios	Resident		Farmer		Fisher	
	Adult	Child	Adult	Child	Adult	Child
Saint Paul - Ramsey Health Center	2.82E-01	2.82E-01	4.14E-01	4.70E-01	2.82E-01	2.82E-01
Sensitive Receptor 1	3.10E-01	3.11E-01	4.57E-01	5.20E-01	3.10E-01	3.11E-01
Sensitive Receptor 2	2.48E-01	2.48E-01	3.64E-01	4.14E-01	2.48E-01	2.48E-01
Sensitive Receptor 3	2.61E-01	2.62E-01	3.83E-01	4.36E-01	2.61E-01	2.62E-01

Region 6 Target Levels

Above Threshold

Below Threshold

Figure 65: Benzo(a)pyrene Noncancer Hazard Indices (across all pathways)

As shown from Figure 65, all the hazard indices exceed the U.S. EPA HHRAP target levels except for the resident scenario and fisher scenario at sensitive receptor 2. This comes as no surprise, because Benzo(a)pyrene is a potent carcinogenic substance and is very toxic to organs and tissues after biotransformation (Livingston, 2012).

6.2 Risk Driver Analysis

The risk driver analysis is systematic process for answering the following question "Where does the risk come from?". The risk driver analysis process is illustrated in Figure 66.

This section will include the steps conducted to find the cancer risk and noncancer hazard drivers.

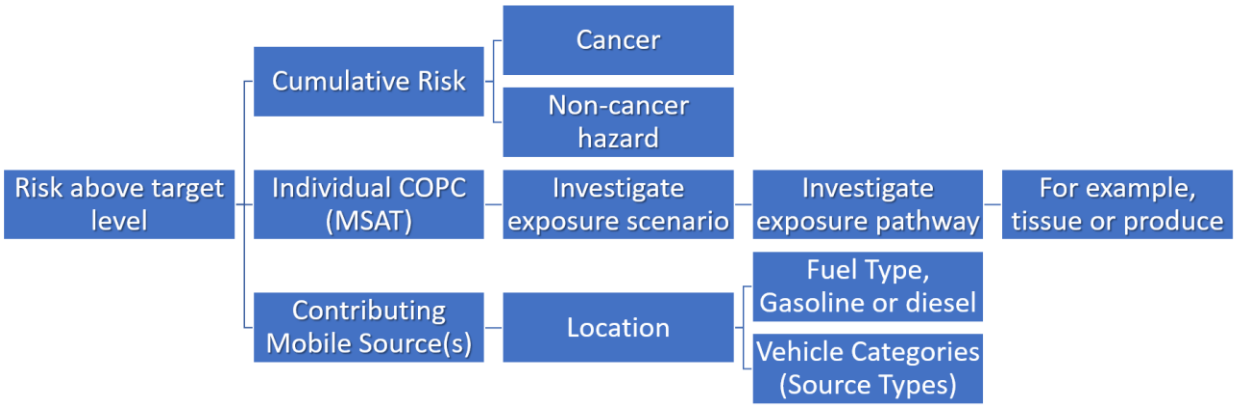


Figure 66: Risk Driver Analysis Process

6.2.1 Contributing Mobile Sources

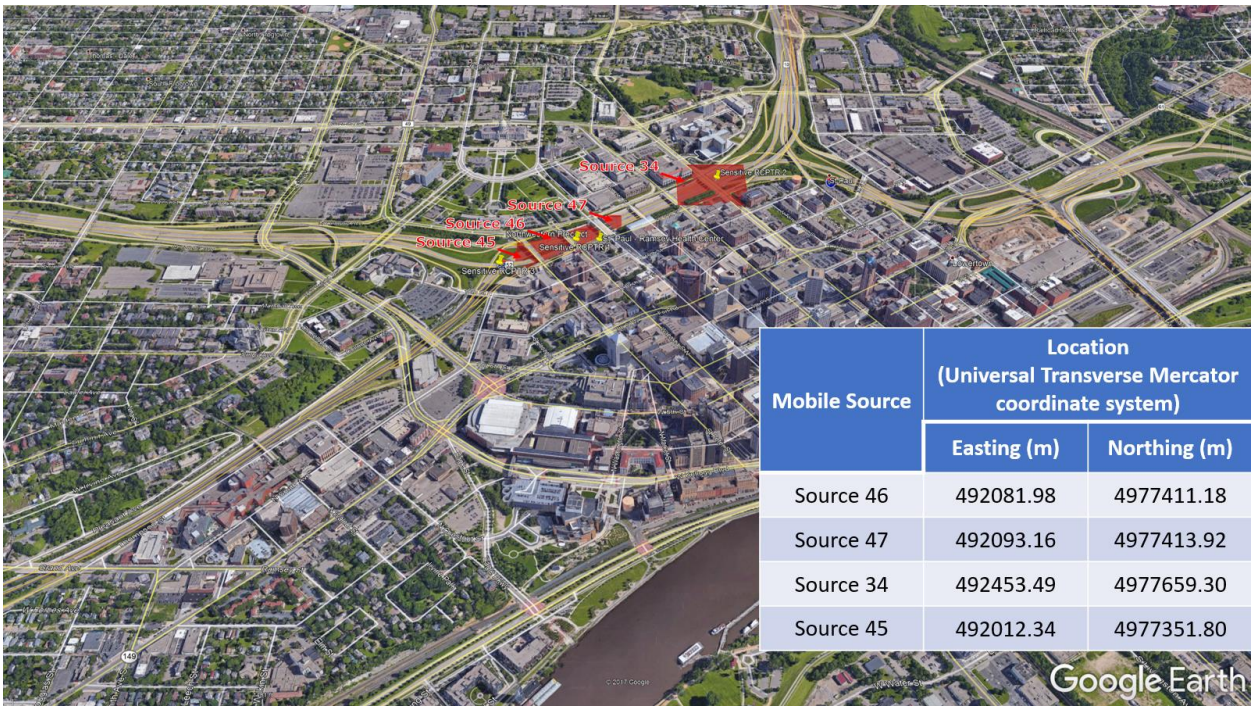
The exposure scenarios with Cumulative Risk (Cancer) exceeding the U.S. EPA HHRAP (U.S. Environmental Protection Agency, 2005) target levels at the sensitive receptors are listed below:

- Farmer adult at all 4 sensitive receptors (Saint Paul – Ramsey Health Center, and sensitive receptors 1, 2, and 3).
- Farmer child at all 4 sensitive receptors.

The on-road mobile source contributions to the maximum risk estimates (Cancer Risk) are summarized as follows in descending order:

1. Source 46 @ Saint Paul - Ramsey Health Center – Risk Value: 3.664E-05
2. Source 47 @ Saint Paul - Ramsey Health Center – Risk Value: 3.661E-05
3. Source 34 @ Sensitive Receptor 2 – Risk Value: 3.62E-05
4. Source 45 @ Sensitive Receptor 1 – Risk Value: 3.55E-05
5. Source 46 @ Sensitive Receptor 1 – Risk Value: 3.46E-05

Figure 67 shows the location of the on-road mobile sources contributing to the maximum risk estimates.



Mobile Source	Location (Universal Transverse Mercator coordinate system)	
	Easting (m)	Northing (m)
Source 46	492081.98	4977411.18
Source 47	492093.16	4977413.92
Source 34	492453.49	4977659.30
Source 45	492012.34	4977351.80

Figure 67: On-road Mobile Sources Locations

The yellow pins in Figure 67 are the sensitive receptors and the table embedded in the figure shows the UTM coordinates for the 4 on-road mobile sources.

6.2.2 Emission Rates

The gasoline and diesel emission rates for the four identified roadway segments in (Section 6.2.1) are summarized in Figure 68.

Mobile Source	Emissions (gram per second) - GASOLINE	Emissions (gram per second) - DIESEL
Source 46	1.59E-07	1.51E-07
Source 47	3.78E-07	3.61E-07
Source 34	4.23E-07	4.04E-07
Source 45	3.78E-07	3.61E-07

Figure 68: Emission Rates - Risk Driver Analysis

The correlation between the emission rate and the air concentrations is directly proportional.

6.2.3 Vehicle Fleet Composition

The fleet composition for the area of study is shown in Figure 69.

Highways Performance Monitoring System (HPMS) Vehicle Class	Fleet composition
Motorcycles	0.66%
Passenger Cars	92.49%
Buses	0.30%
Single Unit Trucks	1.70%
Combination Trucks	4.85%

Figure 69: Vehicle Fleet Composition

The majority of cars in the area of study are passenger cars, comprising almost 93 % of the overall fleet.

6.2.4 Cancer Risk Driver Analysis

1. The cancer risk driver is ***Benzo(a)pyrene***.
2. Risk exceeds the U.S. EPA HHRAP target levels across all pathways in all four sensitive receptors in the farmer exposure scenario as shown in Figure 59.
3. Pathway(s) risk:
    a. **Farmer Adult:** Cancer risk milk (1.416E-04) and cancer risk beef (3.966E-05).
    b. **Farmer Child:** Cancer risk milk (3.529E-05).
4. Pathway(s) intake:
    a. **Farmer Adult:** Intake milk (3.541E-05 mg/kg-day).
    b. **Farmer Child:** Intake milk (5.881E-05 mg/kg-day).

6.2.5 Noncancer Hazard Risk Driver Analysis

1. The noncancer hazard risk driver is ***Benzo(a)pyrene.***
2. All the hazard indices across all pathways exceed the U.S. EPA HHRAP target levels except for the resident scenario and fisher scenario at sensitive receptor 2 as shown in Figure 65.
3. Pathway(s) risk:
    a. **All exposure scenarios:** Inhalation.

It is important in the risk assessment process to take a common-sense approach by asking questions like "Is it realistic that a farmer living across from a highway is really eating all these animals?". The location of the highest cancer risk occurred West of the Interstate-35E and I-94/US 10 in the Saint Paul junction which is currently zoned commercial and contains a medical complex composed of St. Paul - Ramsey Health Center and HealthEast St. Joseph's Hospital, and government buildings such as Minnesota National Guard, Minnesota World War II Memorial, and Cedar Street Armory - Minnesota National Guard, and finally residential buildings like Fitzgerald Condominiums. In summary, the *maximum* risk impacts are predicted to occur at sensitive receptor Saint Paul - Ramsey Health Center (See Table 37).

Table 37: Maximum Risk Summary

	Location (UTM)			
Sensitive Receptor	Easting	Northing	Exposure Scenario	Total Cancer
Saint Paul - Ramsey Health Center	492081.98	4977411.18	Farmer Adult	3.664E-05

On-road mobile sources contributing to maximum risk estimates can be summarized as follows:

- Source 46
- Source 47
- Source 34
- Source 45

For these reasons, it is highly unlikely that the farmer indirect exposure pathways are complete. Therefore, while excess risk was predicted, in reality the study area we evaluated, Saint Paul – Ramsey Health Center in the State of Minnesota, this is highly unlikely. While actual risk is not expected in the study area for the reasons stated, findings of this research effort are important because it establishes that exposure to Benzo(a)pyrene from MSATS is plausible as there are other places throughout the world where mobile emissions are higher, and the farmer exposure scenario and intact indirect pathways actually exist.

The outcome of this research is expected, as mechanistic studies of Benzo(a)pyrene provide substantial evidence that the metabolism of Benzo(a)pyrene is highly correlated with mutagenic agents (DNA reactive) which affect genes whose corruption can induce tumor growth. DNA reactive agents are electrophiles that interact with DNA resulting in DNA adducts and DNA strand breakage (McQueen, 2010). The mutational events include formation of DNA adducts and mutations in oncogenes and antioncogene due to human multi-pathway exposure to Benzo(a)pyrene.

Extensive epidemiologic evidence of correlation and causal mechanistic evidence of cancer development or its precursors in multiple species provides the basis for characterizing Benzo(a)pyrene as “carcinogenic to humans”.

6.3 Uncertainty Analysis

The framework's overall assumptions were conservative and are expected to overestimate the excess lifetime cancer risks and noncancer hazards associated with exposure to air toxics emitted from on-road sources. In this section, a detailed description of the uncertainties associated with the model input and model predictions are presented. Risk assessment is a very complex process, incorporating the combination of many models, although, advanced and accepted models are used to complete the assessments, uncertainties do occur. Many factors contribute to these uncertainties, including the lack of scientific knowledge of release and fate of mobile toxics into the surroundings, variability in sensitive receptors due to many factors like genes, age, and activity level. Additionally, current methodology only considers outdoor concentrations. These factors will cause limitations and propagate all the uncertainties across all elements of the model. In this analysis, there are several areas of uncertainty. They are summarized below:

1. Traffic data: Traffic volumes and driver behaviors (drive cycles).
2. Vehicle emissions: Age, condition, and make of vehicles.
3. Vehicle emissions apportionment: Some emissions were apportioned to individual roadway segments and other emissions were apportioned to census block groups.
4. Release of mobile source air toxics into the environment and background concentrations.
5. The fate and transport of the mobile source air toxics. This includes complex processes and reactions happening in the atmosphere and varying with different meteorological conditions (presence of sunlight and volatile organic compounds, secondary formation of Formaldehyde). These processes cannot be predicted with 100 percent accuracy.
6. Limited information on personal air toxics exposure.
7. Lack of data on mechanistic studies of air toxics.
8. Extrapolating from less-than-lifetime exposures to lifetime exposure.

Uncertainties associated with the risk assessment portion of this framework are classified into four types (Finkel, 1990):

1. Variable.
2. Model.

3. Decision-rule.
4. Variability.

6.3.1 Variable Uncertainty

Variable uncertainty is present when variables in the equations cannot be measured precisely, due to the following:

1. Limitation of equipment used.
2. Spatial and / or temporal variances between the quantities being measured.
3. Sample errors. These types of errors are critical for small sample sizes.

6.3.2 Model Uncertainty

Model uncertainty transpires with the integration of various models used in the mobile source air toxics human health risk assessment. Model uncertainty includes:

1. Human carcinogenicity is tested using animal models as surrogates.
2. Extrapolation of animal studies to humans for testing human carcinogenicity.
3. Air dispersion models used to estimate the fate and transport of mobile source air toxics in the environment.

Extrapolation of animal studies to humans is not an exact science resulting in many uncertainties into the risk factor because of the variability in sensitivity between different species. In addition, computer models used for air dispersion models are a simplified scenario and do not replicate real-life scenarios.

6.3.3 Decision-rule Uncertainty

This type of uncertainty is a major concern when it comes to risk management decisions. The outcome of risk management decisions influences policy and pivotal decisions. An example, the need to balance different social concerns when setting an acceptable risk level. Furthermore, this includes important decisions like which mobile source air toxics need to be included in the risk assessment and the selection of values for inhalation rates, body weight, lifespan, and health benchmarks like RfD and RfC.

6.3.4 Variability

This type of uncertainty is attributed to chemical, biological, and physical variations. An example of variability is, members of a study population are different in body weight. Variability cannot be minimized but it's characterization can be improved.

In practice, uncertainty is reducible by:

1. Improving emissions inventory science. This includes complete speciation of mobile source air toxics, particle distributions, and temporal variability.
2. Running the air dispersion models on small domains. Then post-processing the results to combine all concentration / deposition results for all the individual runs.
3. Refining on-road mobile source contributions to overall emissions (Source apportionment studies).
4. Application of age-dependent adjustment factors to address human variability.
5. Improvements in toxicity benchmarks.
6. Incorporation of more dose-response experimentations in the chemical databases (TOXNET (National Institutes of Health, 2018) and IRIS).
7. Improved characterization and parameterization of mobile source air toxics dispersion and movement in the environmental medial (soil, water, and air) leading to exposure to sensitive receptors.
8. Additional measurements of semi-volatile organic compounds for model validation and calibration.
9. Employing probabilistic techniques.

6.3.5 Probabilistic Techniques

Statistical methods are employed to treat uncertainties, two approaches are discussed in our research effort:

1. Employing appropriate statistical parameters to express variables.
2. Employing probability distributions of variables to propagate variable value uncertainties through the equations used in the risk assessment.

The statistical parameters technique involves using measures like the mean and standard deviation. Selection of the appropriate statistic measures is dictated by the availability of data.

The probability distribution technique is conducted by aggregating the individual distributions with the equations used in the analysis to calculate the probability of cancer or hazard. This technique involves employing numerical methods such as the Monte Carlo analysis. The Monte Carlo analysis estimates the risk by solving the model equations and under different variable selections. With each iteration, the risk values are estimated, and the values are randomly sampled from the specified distributions for each set of variables. The result is a distribution of the risk (cancer risk or noncancer hazard). In summary, the Monte Carlo analysis is conducted as follows:

1. Obtain input parameter distributions.
2. Sample randomly the distributions.
3. Execute the deterministic simulation, using the samples obtained in step two.
4. Run step two again for the required number of times depending on the required detail for the risk assessment.

The Monte Carlo analysis has limitations such as not distinguishing between variability and uncertainty. Furthermore, the tails of the Monte Carlo risk results are very sensitive to the input distributions.

6.4 Monte Carlo Simulation

The research employs recommended point values for all the parameters, as per the U.S. EPA HHRAP (U.S. Environmental Protection Agency, 2005). This is the case, for example, for adult weight of 75 kg, breathing and consumption rates. However, the population is composed of different individuals with a distribution of parameter values. Future work should employ accepted distributions of the parameter values in a Monte Carlo approach. This way, the final risk will also be composed of distributions. For regulatory purposes, regulatory agencies could establish a 95 percentile for decision points. The probabilistic approach, Monte Carlo simulation is advantageous because the results of the estimated risk is obtained as range of values with respect to the variability and uncertainty of these factors. The distributions for the input factors (breathing rate, consumption rates, exposure duration etc.) are input to the model, and after applying the Monte Carlo simulation the estimated risk is expressed as a relative frequency histogram in contrast to a single point estimate.

For example, carcinogenic and non-carcinogenic risk estimates from mobile sources emitting Benzo(a)pyrene in an urban area for one exposure scenario: adult resident. The pathways through which the sensitive receptor, adult resident, is exposed to Benzo(a)pyrene include direct exposure through inhalation of vapors and indirect exposure through ingestion of drink water from surface water sources, ingestion of homegrown produce, and incidental ingestion of soil. Five input distributions are used in the model: exposed vegetable, leaf vegetable, root vegetable, and protected vegetable ingestion rates, daily breathing rate, and drinking water ingestion rate. The Monte Carlo simulation is performed with a sampling size equal to 1000 for all random variables.

The output from the simulation, histograms of cancer risk and hazard quotient. The total cancer risk and total hazard quotient are divided into two parts: risk from direct inhalation exposure and risk from indirect exposure. Then, cumulative density functions of cancer risk and hazard quotient are derived from the histograms. The 50th and 95th percentiles are calculated from the cumulative density functions, where the 50th percentile is the total probability of 50 % of the samples and the 95th percentile is the total probability of 95 % of the samples. The results from the Monte Carlo simulation are compared with the results from deterministically estimated cancer risk and hazard quotient with point values recommended by environmental agencies like California's Office of Environmental Health Hazard Assessment (OEHHA) and the United States Environmental Protection Agency (U.S. EPA). For example, the point values estimates recommended by OEHHA:

- **High-end point values** corresponding to the 95^{th} percentile of the exposure factor distribution and thus representing the greatest exposed individual.
- **Average point values** corresponding to the 50^{th} percentile of the exposure factor distribution and thus representing an average exposed individual.

Comparison of Monte Carlo simulation output and point value estimates defines how reliable the results for the estimated risk using probabilistic approaches.

As a result, two-point values need to be established by regulating agencies. High-end and average point values estimates representing the average and maximum exposed individual.

6.5 Conclusion

The main goal of this research effort is to develop a validated systematic approach to assess the human health impacts associated with exposure to mobile source air toxics (MSATs). Existing methodologies typically focus on criteria pollutants and the inhalation exposure pathway and ignore mobile source air toxics. Additionally, the majority of air quality studies do not evaluate the fate and transport of MSATs, and the important indirect exposure pathways. Research has shown that lipophilic MSATs such as Benzo(a)pyrene, which are transferred via indirect exposure pathways pose a higher risk to human health by orders of magnitude than MSATs transferred via direct inhalation.

Cancer risk and noncancer health effects (i.e., hazard), cannot be directly measured but rather must be modeled to predict resulting human health effects. This research effort set out to integrate the various disconnect between the models and provide the integration necessary to automate the overall modeling process. A validation study was conducted to quantify model performance and uncertainty that is inherent at each step in the risk assessment framework. The results of the study showed reasonable agreement between the measured and modeled values, lending credibility to the framework and methods employed.

Results from the risk driver analysis study indicate that cumulative cancer risk exceeds the U.S. EPA HHRAP target levels for the farmer scenario (adult and child) at all sensitive receptors (Saint Paul – Ramsey Health Center, Sensitive Receptor 1, Sensitive Receptor 2, and Sensitive Receptor 3). As for the cumulative noncancer risk, all the hazard indices across all pathways exceed the U.S. EPA HHRAP target levels at all four sensitive receptors.

From the case study's risk driver analysis presented in this research, it emerges that Benzo(a)pyrene is the cancer and hazard risk driver. Benzo(a)pyrene is highly lipophilic (Gerde, et al., 1997) and tend to bioaccumulate in the food chain as confirmed in the cancer risk driver analysis (Section 6.2.4), thereby presenting a potentially high risk through the consumption of food exposed to Benzo(a)pyrene. The milk and beef pathways are the cancer risk drivers. As for the noncancer risk, the inhalation pathway was the risk driver.

The presented methodology, successfully developed and validated, can be used to improve on current practices providing the handshake between the different models and assisting in conducting multi-pathway assessment of human health risk posed by mobile source air toxics.

Furthermore, this research can be used to improve public health. The proposed framework allows regulatory agencies to perform risk-based prioritization and develop targeted mitigation strategies (i.e., emissions controls, vehicle inspections, etc.) toward the sources that are causing or contributing to the risk. Since we can predict the mobile source air toxics specific health affects including cancer and noncancer including target organ and disease outcomes, that information can be used to ensure the doctors network, diagnosis, and treatment regime are optimized to identify and respond to the types of adverse health effects that will result from geographically correlated population exposures. For example, there are very strong correlations between certain mobile source air toxics exposure and increases in asthma or asthma-related health outcomes. Asthma trips to the emergency rooms are one of the costliest impacts on the health care system.

Furthermore, air quality forecasting can be used to lower mobile source air toxics exposure by informing the public and providing them with the information necessary to protect themselves. For example, where to buy properties to reduce total exposure to air toxics. Our framework is the tool for researchers to use the scientific methods presented in this research to conduct human health risk assessments in a streamlined and validated manner.

6.6 Machine Learning Techniques Applied in Risk Assessment

Future work includes incorporation of a machine learning system which will determine the health risks associated with MSATs exposure. A wide variety of input variables control the health risks associated with MSATs exposure. Table 38 outlines a variety of input variables which could be considered. Given the availability of data regarding both MSATs prevalence and the health risks associated with them, we believe it is possible to train such a model. That is, for example, using publicly-available U.S. EPA and CDC / NIH data it should be possible to establish a robust risk assessment model using a supervised neural network. Using the U.S. EPA data as inputs to the neural network and the CDC / NIH data as outputs, an optimal model relating MSATs to their associated health risks can be algorithmically obtained. Providing future MSATs measurement data and epidemiological data (dose-response) this model can be continuously updated to refine and improve the risk assessment.

Table 38: Inputs for Processes of Machine Learning

Inputs	Examples
Fuel and Vehicle Types	Gasoline – Passenger Car, Diesel Fuel – Light Commercial Truck
Vehicle Volumes	Temporal and Spatial Variation of Traffic
Pollutants and Processes	Benzene / Running Exhaust, Indeno(1,2,3-cd)pyrene / Crankcase Running Exhaust
Vehicle Age Distribution	Source Age Fraction (2011 Gasoline – Passenger Cars make up 20 % of fleet)
Meteorology	Wind speed, Temperature, Relative Humidity, Monin – Obukhov Length (Turbulence)
Source Parameters	Source Release Parameters (Location, Release Height, Emission Rate)
Air Parameters	Hourly Air Concentration – Particle Phase, Air Concentration – Particle Bound
Site Parameters	Average Annual Evapotranspiration, Average Annual Irrigation, Soil Mixing Depth
Scenario Parameters	Averaging Time for Carcinogens, Body Weight, Exposure Duration, Inhalation Exposure Duration, Exposure Frequency
COPC Site Parameters	Metabolism Factor, Soil Bioavailability Factor
Risk Receptors	Locations, Scenarios (Resident Adult, Resident Child)

6.7 Recommendations and Directions for Future Research

The objective of this research is not to develop policies on how to mitigate emission from on-road mobile sources such as limiting gasoline volatility (RVP limits) or imposing the use of ultra-low sulfur diesel (ULSD) as this will be an exercise in futility, but to provide a systematic approach which can be used to estimate cumulative risk from such sources. The inconsistencies in existing methodologies led to haphazard reporting due to the many uncertainties included and workflow barriers.

Recommendations for future research include provision of better and frequent fleet measurements such as vehicle counts and classification, vehicle age distribution, and fuel formulation data. This is a very crucial step in the risk assessment process and complete emission inventories are correlated with highly reliable risk estimates.

Having complete and detailed emissions inventories helps reduce error in the air dispersion modeling process, and subsequently, increases confidence in the risk estimation process. Better insight of how on-road mobile sources impact air quality also known as source apportionment studies improves the air dispersion model.

Furthermore, incorporating more real-world values contributes to robust emission inventories and reduced intrinsic deficiencies within the integration of the multiple models (MOVES2014, AERMOD, and IRAP-H View). Also, calibrating the model to real-world values, then modeling and trying to determine the underlying calculations that influence the results contributes to modeled values closer to real-life examples.

Finally, refinement and addition to the chemical databases such as TOXNET and IRIS (used in this research) increases precision. An example of lack of data encountered in this research, the **RfD** reference dose for oral exposure and **RfC** Reference Concentration for Inhalation Exposure for Indeno(1,2,3-cd) pyrene were not assessed under the IRIS program.

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Appendix A:
Published and Conference Papers

- **Published paper(s)**

    a. TRAQS Combined Interface for Project-Level Mobile Air Quality Analysis. Matthews, B., Johnson, M., Munshed, M., Chamberlin, R., Thé, J. (2015). EM, April 2015.

- **Conference paper(s)**

    a. Mobile Toxics Human Health Risk Assessment Framework. Munshed, M., Thé, J, Fraser, R. (2017). A&WMA's 110th Annual Conference & Exhibition, 2017.

    b. Mobile Toxics Human Health Risk Assessment Framework. Munshed, M., Thé, J, Fraser, R. (2016). Combustion Institute - Canadian Section, 2016.