

A New Cumulative Air Toxics Risk Assessment for Mobile Sources Introducing Stochastic Human Health and Deterministic Ecological Methods

by

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Examining Committee Membership

The following served on the Examining Committee for this thesis. The decision of the Examining Committee is by majority vote.

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Author's Declaration

This thesis consists of material all of which I authored or co-authored: see Statement of Contributions included in the 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.

Statement of Contributions

The body of this thesis is based on a collection of peer-reviewed, published work.

Mohammad Munshed is the sole author of Chapters 1 and 6. Chapters 2, 3, 4, and 5 are adapted from published journal articles. All chapters were written under the supervision of Professors Roydon Fraser and Jesse Van Griensven Thé.

Citations for each chapter are provided in the accompanying table, with corresponding author contributions outlined below. For all published articles, **I am the lead author.**

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- **Chapter 2:** I designed and implemented the deterministic methodology, conducted the analysis and investigation, and wrote the final manuscript. Supervision and review by Professors Roydon Fraser and Jesse Van Grivensen Thé.
- **Chapter 3:** I designed and implemented the methodology, conducted the analysis and investigation, and wrote the final manuscript. The emissions methodology section was partially

written by Ashraf Ramadan. Supervision and review by Professors Roydon Fraser and Jesse Van Grivensen Thé.

- **Chapter 4:** I designed and implemented the Ecological Health Assessment Methodology, conducted the analysis and investigation, and wrote the final manuscript. Ali Elkamel contributed to conceptualization and participated in manuscript review and editing. Supervision and review by Professors Roydon Fraser and Jesse Van Grivensen Thé.
- **Chapter 5:** I designed and implemented the Mobile Source Air Toxics stochastic methodology, conducted the analysis and investigation, and wrote the final manuscript. Supervision and review by Professors Roydon Fraser and Jesse Van Grivensen Thé.

Abstract

Air toxics emitted from mobile and stationary sources continue to pose substantial risks to both human and ecological health, particularly in urban areas and environmentally sensitive regions. However, many existing risk assessment frameworks rely on deterministic assumptions, narrowly focus on select exposure pathways, and often overlook ecological impacts. This dissertation addresses these limitations by developing and applying both deterministic and stochastic methodologies to assess air toxics risk across diverse sources, spatial scales, and exposure scenarios.

One of the key objectives of this research is to introduce a new stochastic risk assessment method for mobile source air toxics (MSATs) that provides a more detailed and comprehensive characterization of health risks. Structured as a manuscript-based thesis, it comprises four peer-reviewed journal articles, each presenting a novel methodology demonstrated through real-world case studies. This structure ensures a logical progression from foundational risk assessment methodologies to more comprehensive, multi-source, multi-pathway, and probabilistic approaches, providing a holistic and integrated evaluation of air toxics risk.

The first article presents a new deterministic human health risk assessment methodology for MSATs. It integrates emissions modeling using MOVES, receptor-specific air dispersion modeling with AERMOD, and multi-pathway exposure estimation across inhalation and ingestion routes. In the Saint Paul, Minnesota case study, benzo(a)pyrene cancer risks exceeded the target threshold at two urban receptors: 4.59×10^{-5} (45.9 in 1 million) at the Saint Paul–Ramsey Health Center and 2.02×10^{-5} (20.2 in 1 million) at the Anderson Office Building. Ingestion contributed significantly to total exposure. These findings highlight the importance of cumulative risk analysis and the need for probabilistic methods to capture inter-individual variability.

The second article extends the methodology to assess cumulative, multi-pathway human health risks from stationary sources across Kuwait. It evaluates emissions from glycol dehydration units, wastewater treatment plants, and oil and gas operations using a national emissions inventory and dispersion and deposition modeling across three air quality zones. While most risk levels were within regulatory thresholds, cancer risk at the Ahmadi Hospital site exceeded the 1×10^{-5} target threshold for adults, with benzo(a)pyrene—the cancer risk driver—contributing a risk of 1.09×10^{-5} (10.9 in 1 million). This case study demonstrates the value of geographically resolved modeling for informing national-scale mitigation strategies.

The third article introduces the Ecological Health Assessment Methodology (EHAM), addressing a previously underexplored dimension of air toxics risk. EHAM combines fate and transport modeling, habitat-specific food web development, and bioaccumulation analysis to characterize ecological risks from air toxics. Applied across Kuwait's air quality zones, EHAM revealed elevated risks in the coastal region, particularly for carnivorous shorebirds, with an ecological screening quotient of 3.12×10^3 driven by the biomagnification of benzo(a)pyrene. By contrast, risks were negligible in the inland and production zones. The EHAM supports both screening-level and comparative ecological risk assessments and enables ecological evaluation at the national scale, extending beyond localized industrial sources to encompass broader emission landscapes.

The fourth article builds on the earlier methodologies and develops the first stochastic human health risk assessment methodology specifically tailored to MSATs. Using Monte Carlo simulation, it quantifies variability in key exposure parameters such as body weight, inhalation rate, exposure duration, and dietary intake. In the Saint Paul case study, the model produced full cancer risk distributions for adults and children. Computed cancer risks ranged from 5.47×10^{-4} to 4.98×10^{-2} (95th percentile) for adults, and from 2.95×10^{-4} to 2.28×10^{-2} (95th percentile) for children. To contextualize, the upper bound of 4.98×10^{-2} for adults is comparable to the lifetime odds of dying from all preventable causes of death (1 in 19). The model identified high-exposure scenarios and vulnerable subpopulations often obscured in deterministic assessments.

Together, these contributions form an integrated risk architecture that advances emissions estimation, dispersion modeling, exposure characterization, and probabilistic risk quantification for human and ecological receptors. The methodologies developed in this research provide a comprehensive foundation for air quality engineers, public health scientists, and environmental regulators.

By improving the representation of variability, cumulative exposure, and pathway integration, this work strengthens the credibility, transparency, and practical utility of air toxics risk assessment to support informed environmental decision-making.

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Dedication

To my parents, whose love and sacrifices have shaped every step of this journey.

To my wife, who stood beside me with patience, strength, and love through it all.

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List of Abbreviations

AHQinh	Acute hazard quotient – inhalation
AIEC	Acute inhalation exposure criteria
AirToxScreen	Air Toxics Screening Assessment
API	American Petroleum Institute
AQZ	Air quality zone
BTEX	Benzene, toluene, ethylbenzene, xylene
E85	Fuel blend: 85% ethanol, 15% gasoline/hydrocarbon
ECOTOX	Ecotoxicology Knowledgebase
EFSA	European Food Safety Authority
EHAM	Ecological Health Assessment Methodology
ELCRs	Excess lifetime cancer risks
ESQ	Ecological Screening Quotient
FCM	Food-chain Multiplier
FIFRA	Federal Insecticide, Fungicide, and Rodenticide Act
HHRAP	Human Health Risk Assessment Protocol
ISCST3	Industrial Source Complex Short Term 3
LULC	Land Use Land Cover
MNRISKs	Minnesota Statewide Risk Screening
MSATs	Mobile Source Air Toxics
NCRSG	National cancer risk by source groups
NOAELs	No-Observed-Adverse-Effect Levels
POPs	Persistent Organic Pollutants
RAIMI	Regional Air Impact Modeling Initiative
SCIPUFF	Second-order Closure Integrated PUFF Model
SRTM	Shuttle Radar Topography Mission

List of Symbols

ADD	Average daily dose (via inhalation/ingestion)	mg/kg-day
AT	Averaging time used in dose calculation	days
BAF	Bioaccumulation factor	unitless
BCF	Bioconcentration factor	unitless
C _o	Observed pollutant concentration	μg/m ³
C _{air}	Air concentration of pollutant	μg/m ³
C _{medium}	Pollutant concentration in exposure medium	mg/kg
C _p	Modeled concentration from dispersion model	μg/m ³
CSF	Cancer slope factor	(mg/kg-day) ⁻¹
ED	Exposure duration	years
EF	Exposure frequency	days/year
ET	Exposure time	hours/day
EEL	Estimated exposure level	mg/kg-day
ESQ	Ecological screening quotient	unitless
FCM	Food-chain multiplier	unitless
F _v	Fraction of MSATs in vapor phase	unitless
IngR	Ingestion rate	kg/day
InhR	Inhalation rate	m ³ /day
μg/m ³	Micrograms per cubic meter	N/A
PDFs	Probability density functions	unitless
p5, p50, p95	5th, 50th, and 95th percentiles	context-specific
Q-Q plot	Quantile–Quantile plot (distribution comparison)	N/A
TRV	Toxicity reference value	mg/kg-day
URF	Unit risk factor for inhalation exposure	(μg/m ³) ⁻¹

Chapter 1. Introduction

1.1 Background

Air pollution remains a significant environmental and public health concern, particularly in urban areas, where emissions from mobile and industrial sources contribute substantially to air toxics exposure [1, 2, 3]. The World Health Organization (WHO) estimates that approximately 7 million premature deaths annually are caused by air pollution exposure [4]. Among air toxics, mobile source air toxics (MSATs) are a major contributor to adverse human health effects, including leukemia, lung cancer, cardiovascular, and hematological diseases [5, 6, 7, 8, 9].

Unlike industrial emissions, MSATs pose an elevated health risk due to their proximity to densely populated areas and high-exposure environments near roadways, schools, and hospitals [1, 10, 11, 12]. The U.S. Environmental Protection Agency (EPA) has identified over 1,160 compounds in the exhaust and evaporative emissions from on-road and non-road mobile sources [13, 14]. These toxic compounds extend beyond direct inhalation, depositing onto soil, vegetation, and water bodies, contributing to indirect exposure via ingestion and dermal pathways [3]. However, regulatory agencies do not effectively monitor most air toxics, with fewer than 70 of the 1,160 identified MSATs actively monitored, and ambient air quality standards for MSATs remain largely absent [15, 16].

Current multi-pathway human health risk assessments for mobile source air toxics rely exclusively on deterministic methods, using worst-case or average exposure values [3]. These approaches fail to capture inter-individual variability, heightened vulnerabilities of sensitive subpopulations, and episodic high-exposure events, potentially leading to inaccurate risk estimates. As a result, these deterministic methodologies may either underestimate or overestimate both cancer and noncancer risks for the exposed population.

While the human health risks associated with air toxics are well documented, ecological risk assessment remains largely underexplored. Industrial emissions, atmospheric deposition, and air toxics transport contribute to toxic accumulation in terrestrial and aquatic ecosystems, leading to bioaccumulation, trophic transfer, and ecosystem-wide disruptions. This is particularly relevant for vulnerable ecological zones such as coastal and desert ecosystems, where air toxics can persist in the environment and enter the food web [17].

1.2 Motivation

Despite growing recognition of the risks associated with air toxics, several critical gaps remain unaddressed in current risk assessment frameworks, limiting public health protection and effective regulatory enforcement.

1.2.1 Gaps in Air Toxics Human Health Risk Assessment

1. Stochastic Modeling Gap in MSATs Risk Assessment
 - a. No prior peer-reviewed studies have applied stochastic modeling to assess health risks from MSATs, highlighting a major gap in the characterization of exposure and associated health risks.
 - b. Current deterministic methods rely on fixed exposure estimates, failing to capture variability in real-world exposure scenarios.
2. Inadequate Consideration of Multi-Pathway Exposure and Cumulative Health Risks
 - a. Current assessments primarily focus on inhalation exposure, neglecting ingestion pathways, which contribute significantly to cumulative health risks.
 - b. Multi-source exposures from both mobile and non-mobile sources are infrequently integrated into risk assessments, limiting the understanding of cumulative exposure impacts.
 - c. Air toxics exposure hot spots and spatiotemporal variations are not well characterized in existing studies.
3. Regulatory and Monitoring Limitations
 - a. The U.S. EPA has identified over 1,160 compounds in mobile source emissions, yet fewer than 70 are actively monitored.
 - b. There are no ambient air quality standards specific to most MSATs, leading to challenges in regulatory enforcement and public health protection.

1.2.2 Gaps in Ecological Risk Assessment

1. Limited Ecological Risk Assessment of Air Toxics
 - a. Existing ecological risk frameworks (e.g., U.S. EPA's SLERAP) were designed for hazardous waste combustion facilities and do not account for diverse emission sources, such as glycol dehydration units in oil and gas operations, wastewater treatment plants, and industrial flares.
 - b. Most ecological risk studies are confined to localized industrial emissions, lacking large-scale assessments that integrate both terrestrial and aquatic ecosystems at a country-wide level.
2. Inadequate Understanding of Food Web Contamination and Bioaccumulation
 - a. Air toxics can enter ecosystems through atmospheric deposition, yet their long-term effects on biodiversity and trophic transfer remain underexplored.
 - b. Contaminant fate and transport within interconnected ecosystems (e.g., coastal and desert environments) are poorly understood.

Addressing these research gaps is critical for advancing air toxics risk assessment methodologies, supporting informed regulatory decision-making, and enhancing public and ecological health protection.

1.3 Research Objectives

This thesis aims to advance air toxics risk assessment through the following objectives:

1. Develop the first deterministic cumulative and multi-pathway human health risk assessment methodology designed for MSATs. This contribution integrates emissions estimation, air dispersion modeling, and multi-pathway exposure characterization (inhalation and ingestion), providing a foundational framework for subsequent probabilistic modeling (First Article).
2. Extend human health risk assessment by evaluating cumulative multi-source exposure to air toxics via inhalation, ingestion, and other pathways. This includes assessing the combined effects of multiple pollutants, sources, and exposure routes, which are often overlooked in current assessments (Second Article).
3. Develop a novel ecological risk assessment methodology for air toxics by integrating multi-species exposure across different environmental compartments. This includes creating food web models tailored to local ecosystems, facilitating regional and country-wide risk evaluations, and extending existing methodologies to assess cumulative ecological risks from multiple emission sources (Third Article).
4. Compare stochastic and deterministic approaches for MSATs risk assessment to evaluate the added value of probabilistic modeling in characterizing exposure variability and uncertainty (Fourth Article).
5. Develop and apply the first stochastic risk assessment methodology tailored for on-road mobile source emissions, addressing a critical gap in environmental health risk assessment by quantifying variability in MSAT exposures across diverse population groups (Fourth Article).

1.4 Thesis Contributions

This dissertation introduces five main contributions to air toxics risk assessment:

1. A novel deterministic methodology for cumulative, multi-pathway human health risk assessment of MSATs, integrating emissions, dispersion, and exposure modeling to support MSAT-specific risk estimation (First Article).
2. An extended multi-source and multi-pathway human health risk assessment methodology for air toxics, incorporating cumulative risk assessments across multiple exposure routes and geographical scales (Second Article).
3. A novel ecological health assessment methodology for air toxics (EHAM), adapted from the U.S. EPA's framework to expand its applicability beyond hazardous waste combustion facilities to additional emission sources, such as glycol dehydration units and wastewater treatment plants. EHAM facilitates detailed ecological risk analyses across multiple geographical scales, enabling comparative assessments (Third Article).
4. A comparative evaluation of deterministic and stochastic risk assessment methodologies for MSATs, demonstrating the added value of probabilistic modeling in characterizing exposure variability and uncertainty (Fourth Article).
5. The first stochastic risk assessment methodology developed and applied for on-road mobile source emissions, addressing a critical gap in environmental health risk assessment by quantifying variability in MSAT exposures across diverse population groups (Fourth Article).

These methodologies collectively advance air toxics risk assessment, addressing key gaps in both human and ecological health domains. This research provides a more comprehensive understanding of exposure variability, cumulative and multi-pathway risks, and ecosystem impacts. It enables practical interventions to reduce air toxics exposure, particularly for populations living near major roadways. The findings equip air quality engineers, risk analysts, and regulators with data-driven tools to strengthen risk mitigation strategies, inform urban planning decisions, and develop targeted policies that improve environmental conditions. Ultimately, this work contributes to safeguarding public health, reducing disparities in air pollution exposure, and promoting long-term well-being in affected communities.

1.5 Thesis Outline

This thesis follows a manuscript-based format, composed of four peer-reviewed published journal articles, which I coauthored as the lead author (Please refer to the Statement of Contributions included in the thesis).

This thesis follows a structured progression designed to enhance the assessment of air toxics risks by integrating multiple methodologies across different exposure sources. It begins with a deterministic risk assessment for mobile source air toxics (MSATs) (Chapter 2), establishing a methodological foundation. The research then expands its scope by assessing cumulative human health risks from industrial and oil and gas emissions (Chapter 3), recognizing that mobile sources are only one part of the broader air toxics landscape.

Moving beyond human exposure, Chapter 4 introduces an ecological risk assessment, addressing gaps in understanding the environmental impacts of air toxics on terrestrial and aquatic ecosystems. Finally, Chapter 5 develops and applies the first stochastic risk assessment methodology for MSAT exposure, using Monte Carlo simulations to quantify variability and uncertainty in human health risk. This probabilistic approach improves exposure characterization, identifies high-risk populations, and supports targeted mitigation strategies, addressing limitations of deterministic models in capturing real-world exposure variability.

This structure ensures a logical progression from foundational risk assessment methodologies to more comprehensive, multi-source, multi-pathway, and probabilistic approaches, providing a holistic evaluation of air toxics risk.

These journal articles contribute to the main research objectives and are structured as follows:

1. Chapter 1: Introduction
 - a. The Introduction establishes the background, motivation, research objectives, and contributions of this thesis. It highlights the existing gaps in air toxics risk assessment, justifying the need for methodological advancements in both human health and ecological risk assessments.
 - b. Additionally, it explains the structured progression of the research and the rationale behind the sequencing of the four journal articles.

2. Chapter 2: Methodology for Mobile Toxics Deterministic Human Health Risk Assessment and Case Study (*Journal Article 1*, published by Munshed et al. [3])
 - a. This chapter presents a new deterministic methodology for assessing cumulative and multi-pathway human health risks from MSATs. The approach integrates emissions estimation, air dispersion modeling, and multi-pathway exposure characterization via inhalation and ingestion. It provides a baseline framework for exposure assessment and supports the transition to stochastic modeling.
 - b. A case study in Saint Paul, Minnesota, demonstrates the method's applicability by validating modeled concentrations against observed data and identifying elevated benzo(a)pyrene cancer risks. It also highlights the limitations of deterministic methods and motivates the shift to probabilistic modeling introduced in Chapter 5.
3. Chapter 3: Extending Multi-Pathway Human Health Risk Assessment from Regional to Country-Wide—A Case Study on Kuwait (*Journal Article 2*, published by Munshed et al. [18])
 - a. This chapter shifts the focus from mobile source air toxics to stationary source emissions, specifically evaluating human health risks from industrial emissions, wastewater treatment plants, glycol dehydration units, and oil and gas operations.
 - b. The research implements a country-wide cumulative risk assessment, incorporating multi-source and multi-pathway exposures. Unlike Chapter 2, which focuses on MSATs, this research assesses toxic emissions from non-mobile sources, comparing risks across different regions and identifying dominant chemical risk drivers for sensitive receptors.
4. Chapter 4: Country-Wide Ecological Health Assessment Methodology for Air Toxics: Bridging Gaps in Ecosystem Impact Understanding and Policy Foundations (*Journal Article 3*, published by Munshed et al. [17])
 - a. This chapter introduces a new ecological health assessment methodology (EHAM) and integrates fate and transport modeling to assess ecosystem-wide impacts. It evaluates the bioaccumulation and trophic transfer of air toxics within terrestrial and aquatic environments, addressing critical gaps in ecological risk assessment.

- b. While this research applies EHAM to industrial emissions, the methodology is adaptable to include mobile source air toxics, provided the necessary data are available.
- 5. Chapter 5: Advancing Human Health Risk Assessment Through a Stochastic Methodology for Mobile Source Air Toxics (*Journal Article 4*, published by Munshed et al. [19])
 - a. The final journal article develops and applies a new stochastic risk assessment methodology for MSAT exposure, utilizing Monte Carlo simulations to quantify variability and uncertainty in human health risk characterization. By incorporating probabilistic modeling, this research enhances exposure characterization, identifies high-risk populations, and informs cost-effective resource allocation.
 - b. This research also identifies dominant MSAT cancer risk drivers, providing insights for targeted mitigation strategies to reduce public health risks.
- 6. Chapter 6: Conclusion
 - The Conclusion synthesizes key findings and identifies future research directions in human and ecological risk assessment.

Chapter 2. Methodology for Mobile Toxics

Deterministic Human Health Risk Assessment and Case Study

Chapter 1 outlined the background, motivation, and research gaps in human health risk assessment related to MSATs, emphasizing the need for a comprehensive methodology to evaluate exposure and associated risks. Chapter 2 builds on this by introducing a deterministic risk assessment framework that integrates emissions estimation, air dispersion modeling, and cumulative and multi-pathway exposure assessment with risk quantification to address these gaps. This methodology is validated using real-world data and applied in a case study to quantify cancer and noncancer risks from on-road emissions, establishing a foundation for future probabilistic modeling.

2.1 Introduction

In 2012, the World Health Organization (WHO) estimated that 7 million premature deaths per year were caused by exposure to air pollution [4]. Exposure to air pollution causes adverse effects such as strokes, chronic obstructive pulmonary disease (COPD), acute lower respiratory infections, and lung cancer [20]. In addition to the alarming numbers published in the WHO report, more than 43% of Americans live in areas with poor air quality, putting their lives at risk [21]. Air pollution costs the global economy trillions of dollars annually due to healthcare expenditures and lost work and is one of the leading causes of morbidity and mortality [22, 23].

Sources of air pollution are of biogenic and anthropogenic origins, such as emissions resulting from energy production by combustion, industrial processes, metallurgical industry, residential heating, waste treatment, and mobile sources. Mobile sources are divided into two categories: on-road, including trucks, passenger cars, buses, and motorcycles, and non-road, including commercial marine vessels, airplanes, railroads, agricultural tools, snow equipment, and recreational vehicles. On-road mobile sources emit more than 1162 compounds into the atmosphere via tailpipe and evaporative emissions [13, 14].

On-road mobile sources, which are the focus of this chapter, also emit non-exhaust emissions from the release of gasoline vapor in the fuel storage system [24], fluid leaks (e.g., fuel, lubricants, and refrigerants), particulate matter from brake dust and tire wear, and re-entrained road dust [25].

Numerous peer-reviewed studies have clearly demonstrated that mobile source emissions are the most significant contributor to air pollution in urban areas, and they represent a risk to human and ecological health [26, 27, 28, 29, 30, 31]. In addition to the previously mentioned studies, the 2014 National Air Toxics Assessment (NATA), the EPA's most recent large-scale and recurring risk assessment, showed that the United States national cancer risk from exposure to air toxics emitted from on-road sources was approximately "4-in-1 million" [32]. This means that if one million people were continuously exposed to the same concentration of MSATs for 70 years, four individuals would likely develop cancer from this exposure. On-road sources contributed to 12.43% of the 2014 NATA national cancer risk by source groups (NCRSG) as the second leading contributor.

In the United States, more than 13% of the population live, attend school, or work within 100 m of a major road [11]. Kingsley et al. [10] showed that in 2005–2006, more than 6 million students attended schools within 250 m of a major road. MSATs and criteria pollutant concentrations are higher near the roadway, with the maximum concentrations typically occurring within the first 100 to 150 m of a roadway [12, 33]. For example, according to the Mobile Source Air Toxics report published by the Health Effects Institute [13], near-road monitoring and air monitoring network data showed that the highest levels of ambient benzene and formaldehyde concentrations were measured at urban roadside monitors.

Depending on many factors, such as the air toxics chemical structure, daily meteorological conditions, vehicle fleet mix, presence of barriers, and the terrain surrounding the roadway, ambient concentrations of MSATs can be found within 600 m of a roadway [12]. Additionally, the EPA's transportation conformity and hot-spot guidance recommend placing air dispersion modeling calculation points (receptors) to estimate concentration impacts at distances as close as five meters from a roadway edge [34].

Unlike criteria pollutants, air toxics lack effective ambient air quality standards or guidelines in Canada or the United States that are directly applicable to protecting public health or the surrounding environment [35, 36]. Moreover, regulatory agencies around the world do not monitor the vast majority

of air toxics but monitor only a small fraction, less than 70, of the list of thousands of air toxics being emitted from on-road sources [15, 16].

This work employs the chronic and acute toxicity approach [37] to estimate the multi-pathway cancer risk and noncancer hazard from exposure to MSATs. This approach, coupled with air dispersion modeling and estimation of population exposures, addresses the absence of ambient air quality standards for most of the MSATs and the lack of monitoring by regulatory agencies by determining through modeling the health impacts resulting from extended and acute exposure to MSATs.

Humans, known in risk assessment studies as human receptors, may come into contact with air toxics via two main exposure routes or pathways: (1) direct via inhalation and (2) indirect via ingestion of contaminated water, produce, and animals. Examples of indirect pathways include deposition of air toxics onto the soil and produce such as lettuce and tomatoes. Additionally, biotic uptake and accumulation from contaminated soil or water can occur, such as irrigation water transporting soil deposits containing air toxics and resulting in uptake by fish and livestock, including cattle, swine, chickens, and sheep. In addition, the incidental ingestion of soil itself is considered an important indirect exposure pathway. Soil contamination is attributed to direct discharge to the soil, atmospheric deposition of air toxics, or transport from other media (e.g., through runoff). Consideration of indirect exposure pathways is critical for certain air toxics. For example, the indirect risk from dioxins and furans can be orders of magnitude higher than the direct risk [38]. Heavy-duty diesel vehicles emit furans and dioxins [39, 40, 41, 42]. Furans and dioxins are highly toxic compounds that contain the dibenzofuran nucleus and dibenzo-p-dioxin nucleus, respectively. 2,3,7,8-Tetrachlorodibenzodioxin (TCDD) is the most toxic and most comprehensively studied dioxin [43]. Moreover, TCDD is highly lipophilic and tends to bioaccumulate in the food chain [44], thereby presenting a potentially elevated risk through the consumption of food exposed to TCDD, further contributing to the risk already caused by inhalation exposure to MSATs.

This work presents a novel and practical (streamlined and integrated) methodology to assess the significant human health impacts associated with chronic and acute exposure to MSATs by calculating the total inhalation cancer risk and the total inhalation hazard quotient. Furthermore, this methodology computes the cumulative cancer risk and the air toxics hazard index due to exposure via indirect pathways such as ingestion. Finally, the methodology is verified using monitored data.

2.1.1 Literature Review

Previous work performed on assessing human health impacts from exposure to air toxics includes the National Air Toxics Assessment [32], Regional Air Impact Modeling Initiative [45, 46], and MPCA Statewide Cumulative Risk Study [47], which are briefly described here.

The most recent large-scale and recurring risk assessment is the National Air Toxics Assessment (NATA). A NATA is conducted every three years, and the most current one is from 2017. NATAs provide a snapshot of the outdoor air quality and risks associated with exposure to air toxics. The main objective of the NATA is to identify air toxics that are of the highest potential concern in terms of contribution to population risk. NATAs only focus on risks resulting from direct inhalation of air toxics rather than evaluating other indirect pathways, such as ingestion. Cancer and noncancer health effects are estimated from breathing air toxics at a constant concentration over a lifetime (i.e., 70 years). Results from the NATA are used in prioritizing pollutants and emission sources. The 2014 NATA results reported that cancer risk due to inhalation of MSATs was 12.43% of the national cancer risk by source groups [32], and this is equal to “4 in 1 million people”. This percentage does not include the cumulative cancer risk due to indirect exposure of MSATs, nor other noncancer health impacts such as damage to the central nervous system or cardiovascular disease. A NATA is not appropriate for identifying local-scale air toxics “hot spots”, nor is it appropriate for identifying localized risks or individual risks from air toxics from near-roadway-related exposures. Furthermore, NATAs do not include risks due to non-inhalation exposure pathways.

The Regional Air Impact Modeling Initiative (RAIMI) included the development of a suite of integrated software tools and reports, developed under contract for the EPA Region 6 Compliance Assurance and Enforcement Division [45]. The RAIMI program and tools were developed to conduct community-wide cumulative air dispersion and human health risk assessment modeling for hundreds of air toxics from thousands of air pollution sources. Human health risk assessment modeling involves evaluating chronic cancer risks, noncancer hazards, and short-term acute exposure and is conducted following the methodologies published in the Human Health Risk Assessment Protocol (HHRAP) for Hazardous Waste Combustion Facilities [37]. Exposure pathways include direct inhalation and multiple indirect pathways, such as water, plant, and animal tissue ingestion. The first RAIMI pilot study was conducted in Harris County, Texas [46], including the Houston Ship Channel industrial corridor.

The most significant obstacle to the implementation of the RAIMI program was source parameterization (i.e., characterizing physical source parameters required to support air dispersion modeling). After preliminary modeling was completed, it was determined that many sources driving adverse human health risk were in fact fugitive sources, which required the development of necessary physical source parameters to model as fugitive sources instead of point sources as originally reported in the Texas Commission on Environmental Quality (TCEQ) emissions inventory database. The RAIMI was the first regional study to integrate all the necessary tools and peer-reviewed guidance required to conduct large-scale cumulative human health risk assessments. The project outcome resulted in the identification of multiple risk hotspots, where the EPA was able to utilize its regulatory authority to enforce existing permit limits and, where necessary, modify permits to ensure that emissions control or process changes were made to protect cumulative health impacts to the communities affected.

The dispersion of emissions from on-road sources cannot be accurately estimated using a single point source. This chapter employs non-point sources to estimate the dispersion of pollution from a road network. Another weakness of the RAIMI program is the emissions inventory inputs. The RAIMI emissions inventories tend to follow the EPA's AP-42 emission factors, and these emission factors are not up to date with the current science, such as improved fuel formulation with less sulfur content or efficient internal combustion engines equipped with advanced emission control technologies. The on-road mobile emissions inventory in the proposed methodology is prepared using the EPA's state-of-the-science maintained Vehicle Emission Simulator called MOVES.

The Indiana Department of Environmental Management's RAIMI study [28] demonstrated that the incremental cancer risk from exposure to MSATs was "18.6 in 1 million people", and the cancer risk drivers were formaldehyde and benzene. Moreover, the highest noncancer risk was also due to MSAT exposure, with the noncancer risk driver being acrolein. Acrolein is a common air toxic, found in urban settings, that is emitted from motor vehicles [48].

The Minnesota Pollution Control Agency (MPCA) conducted a statewide screening-level human health risk assessment study of all inventoried emission sources, including on-road mobile sources, non-road mobile sources, area (non-point sources), and permitted point sources located in Minnesota [47]. The MPCA conducted a cumulative risk study according to the methodologies and science in the HHRAP [37]. The statewide screening-level human health risk assessment was conducted using the Minnesota statewide risk screening (MNRISKS) tool [47]. MNRISKS automates the process

of compiling the emissions inventory by incorporating data for point, area, and mobile sources, air dispersion modeling, and risk assessment.

MNRISKS incorporates the AERMOD air dispersion model (American Meteorological Society (AMS)/United States Environmental Protection Agency (EPA) Regulatory Model) and risk assessment protocols outlined in the HHRAP to predict cancer risk and noncancer hazard indices.

The cumulative human risk study is updated for the entire state every three years, with a comprehensive emissions inventory of all source categories, including more than 12,271 industrial point source emissions processes, on-road and non-road mobile sources, and 18 subcategories of area sources. More than 250 pollutants from all source categories are included in the study.

Following the study by MPCA, a collection of peer-reviewed articles was published [27, 29, 49], confirming the necessity of evaluating emissions from on-road sources and their contribution to adverse health effects resulting from acute and chronic exposure.

Numerous articles include the results from the two most recently published MNRISKS studies based on emissions for 2008 [27] and 2011 [29]. Using these studies, Pratt et al. [27] showed that on-road mobile sources contributed to the highest cancer risks within the 2008 MNRISKS study, with a cancer risk of $3.70\text{E-}04$, which is equal to an increase in cancer incidence of “370 in 1 million people”. Additionally, exposure to MSATs contributed to the highest noncancer risk.

Pratt et al. [29] compared different source groups and concluded that nontraditional sources, such as on-road mobile sources, were important sources of human health risk. Furthermore, on-road mobile source contribution to cancer and noncancer endpoints was higher than point sources. The analysis of Pratt et al. [29] showed that formaldehyde, acrolein, and diesel particulate matter (DPM) were the inhalation cancer risk drivers, and these air toxics are found in motor vehicle exhaust. The maximum DPM inhalation cancer risk modeled using MNRISKS-AERMOD was equal to an increase in cancer incidence of “900 in 1 million people”.

One area for improving the MNRISKS study is the better characterization of emissions and health impacts from mobile sources by introducing spatiotemporal considerations, i.e., changes in space over time. An existing limitation in current air toxics risk analyses is the practice of only employing annual averages to classify motor vehicle emissions in space but with time averaged out. Using annual averages can overlook important pathways for mobile toxic emissions. By adding hourly

spatiotemporal capabilities to existing mobile source simulations, a better ability to predict specific area and time health impacts is achievable.

Lastly, all reviewed literature employs deterministic risk models. These deterministic models include average exposure assumptions, such as an adult body weight value of 70 kg, meaning the results do not capture the range of variability in cancer or noncancer health risk.

The literature reviewed in this section is summarized in Table 2-1.

Table 2-1: Literature review summary

Risk Program	Summary of Key Literature
NATA	A U.S. nationwide risk assessment conducted every three years, evaluating only direct inhalation risk. Not suitable for identifying local-scale near-roadway air toxics "hot-spot" risks. The 2014 NATA reports that on-road mobile sources contributed 12.43% of national cancer risk.
RAIMI	A multi-pathway risk assessment program evaluating chronic cancer risks, noncancer hazards, and short-term acute exposure. Uses methodologies from the Human Health Risk Assessment Protocol. The first pilot study focused on Harris County, Texas, including the Houston Ship Channel.
MNRISKS	A Minnesota statewide cumulative risk program updated every three years, assessing direct inhalation and indirect risks (e.g., produce and fish consumption). Covers all source categories, including over 12,271 industrial sources and mobile emissions.

2.2 Materials and Methods

This work presents a novel and verified methodology to quantify the myriad health risks associated with exposure to on-road mobile emissions. Methodology verification is based on validated air dispersion results and observed trends being as expected. The methodology is summarized as follows:

1. Estimate emissions from on-road vehicles using current U.S. regulatory models such as MOVES or similar vehicle emissions simulators.
2. Conduct air dispersion modeling using current U.S. regulatory air dispersion models, such as AERMOD or similar, to predict Mobile Source Air Toxics (MSATs) air concentrations and deposition to media, including soil and water.
3. Determine critical air concentrations for each MSAT of interest.
4. Identify through a multi-pathway fate-and-transport analysis the concentration of the MSATs in various exposure media, to evaluate indirect exposure pathways (e.g., deposition from air to soil, water, sediment, plants, and animal tissue) at sensitive locations of potential high impact.
5. At each sensitive location, determine the total dose to humans from all pathways, i.e., direct inhalation and scenario-relevant indirect pathways.
6. Conduct cumulative human health risk assessment to determine the total cancer and noncancer health effects for all-pathways based on the air toxics doses at each sensitive location (including acute and chronic exposure and direct and indirect exposure pathways).

Figure 2-1 shows the flowchart of the methodology.

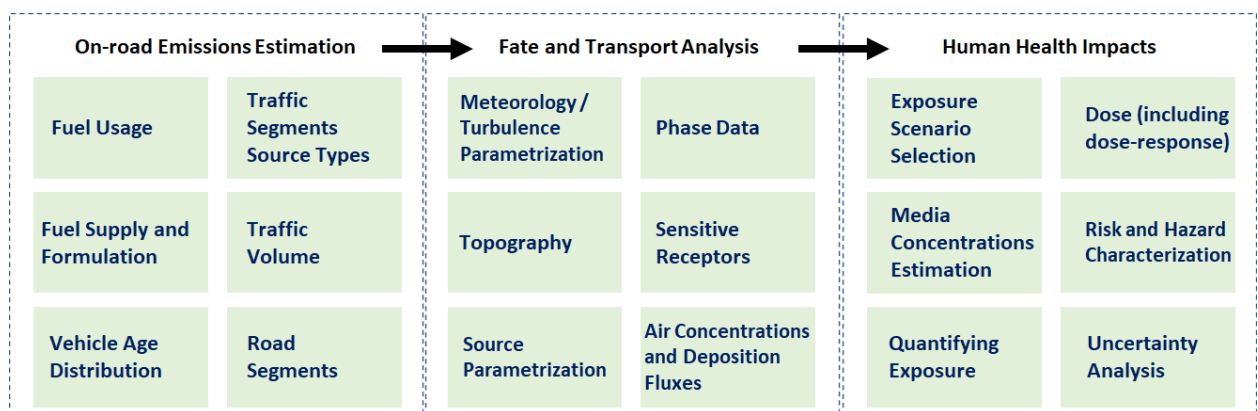


Figure 2-1: Flowchart of the proposed methodology

Three MSATs, namely benzene, formaldehyde, and benzo(a)pyrene, were selected for this study. Though this is a very small fraction of the MSATs, these three chemicals were selected due to their potency and the abundance of cancer and noncancer assessments in the literature.

Under the guidelines for carcinogen risk assessment, benzene is classified as a known human carcinogen [50]. Moreover, the International Agency for Research on Cancer (IARC) has determined that exposure to benzene causes acute myeloid leukemia [51, 6].

Formaldehyde is classified as a human carcinogen as per the findings published by IARC [52], and more recently, the United States Department of Health and Human Services (HHS) has categorized formaldehyde as a known human carcinogen [53]. IARC and EPA both classify benzo(a)pyrene (BaP) as carcinogenic to humans [54, 55].

Due to the proximity of human activity near busy and congested roads, humans are exposed to high ambient concentrations of MSATs such as benzene and other potent Polycyclic Aromatic Hydrocarbons (PAHs) [1] for extended periods.

Furthermore, traffic emissions are particularly rich in ultrafine and nanoparticles that can easily enter the bloodstream and affect multiple organ systems. They can even directly penetrate the brain via the olfactory bulb [56].

2.2.1 On-Road Vehicle Emissions Inventory

In this work, the vehicle emissions inventory is prepared using the EPA's state-of-the-science Motor Vehicle Emission Simulator called MOVES [57]. The version used in this work is MOVES2014b, embedded in the TRAQS system [58].

MOVES2014b estimates the emission factors and emission inventories for the following pollutants: particulate matter (PM10 and PM2.5), nitrous oxides (NOx), sulfur dioxide (SO2), carbon monoxide (CO), greenhouse gases (GHGs), metals, and MSATs, which are the focus of this work. MOVES2014b is capable of modeling continuous releases of MSATs with over 50 different exhaust and evaporative species. Moreover, the simulator accounts for multiple fuel types (e.g., diesel, gasoline, E85) used by various mobile sources, such as cars, buses, motorcycles, and haul trucks.

Furthermore, the simulator 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. It is important to note that the availability and completeness of an on-road emissions inventory are crucial to assess the deleterious effects of MSATs.

Vehicle emissions for this work were generated at the road-segment level. MOVES2014b requires data on road segments as well as vehicle classes, including motorcycles, light-duty vehicles, buses, single-unit trucks, and combination trucks. Additionally, the simulator requires information such as fuel specifications and usage, vehicle age distribution, traffic volume, and meteorology.

2.2.2 Roadway Geometry Characterization

One major impediment in quantifying health impacts from on-road emissions is roadway geometry characterization. MOVES2014b is geospatially unaware and does not require geographic coordinates such as the latitude and longitude to define the road segments in the real world. However, coordinates are required to perform air dispersion analysis of MSATs as they move away from roads toward human receptors. Air dispersion models like AERMOD require precise coordinates to characterize terrain, receptors, and roads, which are considered the emission source rather than the individual vehicles.

The Transportation Air Quality System (TRAQS) [58] is used in this methodology to address roadway geometry characterization. TRAQS addresses the roadway geometry characterization limitation by enabling the user to define the roadway segments within a geographic information system (GIS), automatically saving the precise coordinates in preparation for air dispersion modeling.

Figure 2-2 shows the TRAQS graphical user interface (GUI) and the road segments analyzed in this work.

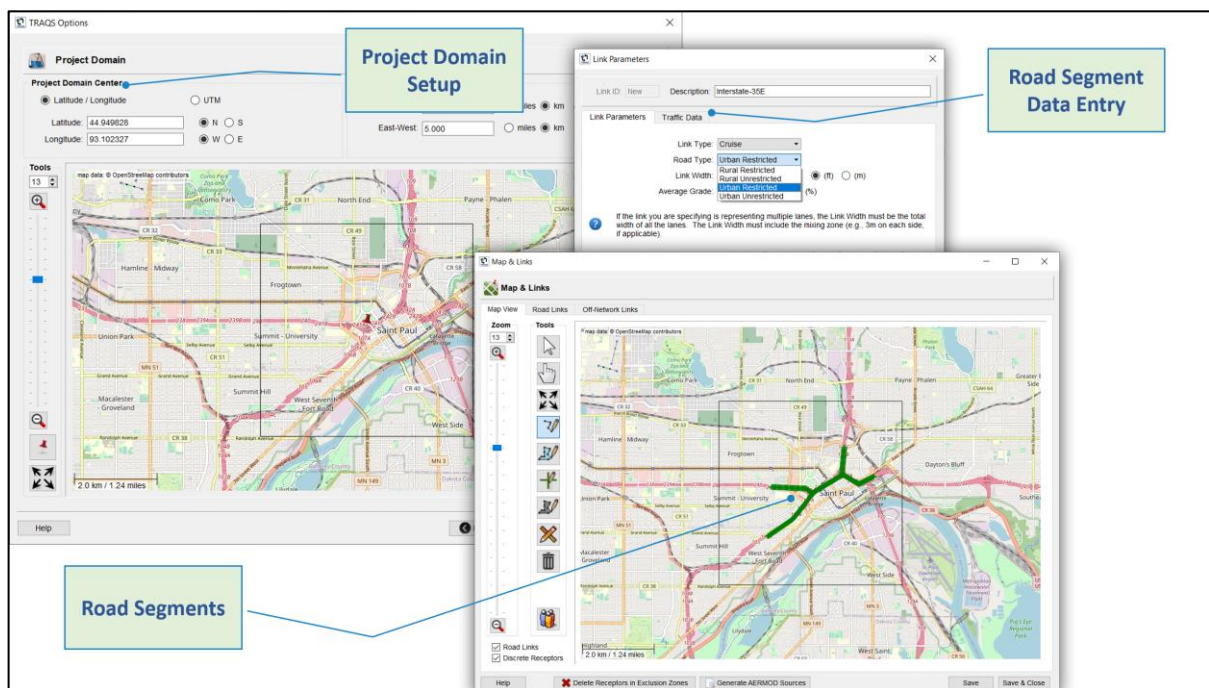


Figure 2-2: Transportation Air Quality System (TRAQS) graphical user interface

2.2.3 Air Dispersion and Deposition Modeling

The air dispersion and deposition modeling subsection is composed of two parts. The first part describes the air dispersion and deposition modeling of MSATs. In the second part, a description of the meteorological files and how they were prepared is presented.

2.2.3.1 Air Dispersion and Deposition Modeling

Air dispersion modeling is defined as the mathematical description of pollutant transport in the atmosphere. Air dispersion modeling is performed to simulate the transport, diffusion, and deposition of MSATs in the ambient air once emitted by the various emissions processes, such as running exhaust, extended idle exhaust, crankcase running exhaust, or fuel vapor venting.

Air dispersion models are used to predict the downwind concentrations of an emission source, provided the model is fed with all necessary data, such as source parameters like coordinates; physical characteristics like stack height, exit temperature, exit velocity, and diameter; and project-specific data like meteorological conditions, geophysical data layers (e.g., terrain, land use), and receptor information (including sensitive population and ambient air monitor locations).

Air dispersion modeling is needed to:

1. Predict air concentrations and deposition on vegetation, land, and water.
2. Determine on-road mobile source emission contributions to ambient air quality.
3. Better understand how MSATs amplify the deleterious effects of air pollution.
4. Aid in the selection of ambient air monitoring stations, such as identifying target road segments for near-road monitoring.

In this chapter, MSATs dispersion and deposition are simulated by an extensively validated Gaussian EPA model, AERMOD (American Meteorological Society (AMS)/United States Environmental Protection Agency (EPA) Regulatory Model). AERMOD is the EPA's preferred dispersion model for regulatory applications [59]. In this work, a commercialized version of AERMOD called AERMOD View [60] is used to calculate the hourly and annual air concentrations and deposition fluxes resulting from on-road emissions. AERMOD View utilizes EPA's AERMOD model version 19191.

The proposed methodology is flexible in incorporating more sophisticated air dispersion models, such as non-steady-state puff models, which simulate three-dimensional complex wind fields and derive meteorology from a combination of upper-air launches and point-based surface observations to estimate ambient air concentrations and deposition.

2.2.3.2 Meteorological Files

The Weather Research and Forecasting (WRF) model [61] was used to generate the AERMOD meteorological files. WRF is a prognostic meteorology model developed in a collaborative partnership between the U.S. National Center for Atmospheric Research (NCAR), the National Centers for Environmental Prediction (NCEP), the Naval Research Laboratory, and others. The WRF model is a limited-area, non-hydrostatic, terrain-following sigma-coordinate model designed to simulate or predict mesoscale and regional-scale atmospheric circulation.

The air dispersion meteorological files were generated by processing the AERMET-Ready data files output by the Mesoscale Model Interface Program [62] through the most recent version of the U.S. EPA's AERMET meteorological pre-processor executable (Version 19191). This includes the use of the MMIF-generated AERSURFACE output file for Stage 3 surface characteristics.

The two generated meteorological data files are: surface data and profile data. The surface data file contains the hourly boundary layer parameter estimates, and the profile data file contains multiple-level observations of wind speed, wind direction, temperature, and standard deviation of the fluctuating wind components.

A wind rose for the study area is shown later in this chapter in the ("Case Study") subsection.

2.2.4 Human Health Risk Assessment

Human health risk assessment is the scientific process that estimates the probability that adverse health effects may occur in humans resulting from exposure to environmental stressors, now or in the future. The human health risk assessment process includes the completion of four steps: (1) hazard or stressor identification, (2) exposure assessment, (3) dose-response assessment, and (4) risk characterization. Risk is an estimate of the "chance" that the exposure will cause or contribute to harmful human health effects.

Risk characterization is the process whereby human health risk estimates are used to establish the presence or absence of risk and quantification of expected outcomes. In addition, risk characterization strives to identify the key factors contributing to risk in such a way that regulators, risk analysts, or air quality engineers are armed with actionable information to guide decisions regarding what actions, if any, should be taken to mitigate risk. Risk characterization includes calculating the incremental lifetime cancer risks and noncancer hazards by combining the toxicity benchmarks and exposure quantities [63].

Cancer risk is defined as the probability of contracting cancer based on a unique set of exposure and toxicity assumptions throughout a lifetime, which is assumed to be 70 years for this research. For example, a risk of $5.0\text{E-}05$ is interpreted to mean that an individual has up to a five in 100,000 increased chance of developing cancer during their lifetime from the evaluated exposure. Future research will employ a probabilistic approach for the lifetime variable (e.g., lifetime probability distribution).

Noncancer hazard is an estimate of the likelihood that a human will experience noncancer health effects (e.g., neurological, cardiovascular, reproductive, respiratory, etc.) as a result of exposure to MSATs through the scenario-specific exposure pathways and routes. A hazard is calculated as a ratio of the receptor's potential exposure relative to a standard exposure level such as a reference dose or reference concentration.

There are two primary hazard classifications based on the length of the exposure duration: acute for short-term exposure and chronic for long-term exposure.

The multi-pathway and cumulative human health risk assessment in this chapter was performed following the U.S. EPA 2005 Final Human Health Risk Assessment Protocol (HHRAP) [37]. The HHRAP provides guidance for completing a human health risk assessment study from modeled air concentrations and deposition fluxes. HHRAP was developed for evaluating human health risks from hazardous waste combustion facilities such as incineration plants or energy generation plants.

This research extended the HHRAP methodology by incorporating source characterization for mobile sources to evaluate health risks associated with MSAT exposure. The chronic and acute dose-response values were obtained from the following sources: (1) U.S. EPA's Integrated Risk Information System [64], (2) California's EPA (CalEPA) chemical database [65], and (3) CDC's Agency for Toxic Substances and Disease Registry Toxic Substances Portal [66]. For MSATs with multiple dose-response values from the above sources, the more conservative value was selected.

To quantify the potential health risks associated with exposure to MSATs, 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 HHRAP does not define target risk levels, as this responsibility is left to local and state regulatory agencies, who are better equipped to consider other factors such as background concentrations.

However, the U.S. EPA, through its Region 6 Office [67], has established target risk levels within the Agency, which range as follows: (1) for carcinogenic risks, a low and likely negligible risk is described as an incremental lifetime cancer risk of $1.0\text{E}-06$ to less than $1.0\text{E}-05$; (2) a potentially elevated risk is an incremental lifetime cancer risk greater than $1.0\text{E}-04$; (3) for noncancer hazards, a low and likely negligible risk is defined as a hazard quotient ranging between 0 and 0.25.

In this work, the following target levels are used to interpret the magnitude of cancer risks and noncancer hazards: a cancer risk not to exceed a target risk level of $1.0\text{E}-05$ and a hazard quotient not to exceed a target risk level of 0.25.

The target risk levels are not a discrete indicator of observed harmful effects. If the modeled risk values fall within the accepted target risk levels, it might be concluded, without further analysis, that a proposed project does not present an unacceptable risk. A risk calculation that exceeds the target values triggers further consideration of the underlying scientific basis and uncertainties associated with the risk calculation.

The general equations used to calculate the cancer risk and noncancer risk in this work are:

$$\text{Cancer Risk} = \text{dose} \times \text{toxicity} \quad (2-1)$$

$$\text{Noncancer Risk} = \frac{\text{average daily dose}}{\text{reference dose}} \quad (2-2)$$

$$\text{Noncancer Risk}_{inh} = \frac{\text{exposure air concentration}}{\text{reference concentration}} \quad (2-3)$$

$$\begin{aligned} \text{Acute Hazard Quotient}_{inh} \\ = \frac{\text{acute air concentration}}{\text{acute inhalation exposure criteria}} \end{aligned} \quad (2-4)$$

Equation (2-2) is used to calculate the noncancer risk from oral exposure, whereas Equation (2-3) evaluates the noncancer risk from inhalation exposure. Equation (2-4) is used to calculate the acute (short-term) inhalation hazard quotient.

2.2.4.1 Cumulative Risk

Humans may be exposed to multiple carcinogenic air toxics via a single exposure pathway such as inhalation. The total cancer risk for a single pathway is calculated from the sum of cancer risks for each Mobile Source Air Toxic (MSAT) (e.g., benzene, formaldehyde, and benzo(a)pyrene).

In addition, exposure may come via multiple pathways (e.g., inhalation, produce, animal tissues, water); therefore, the cumulative cancer risk is calculated from the sum of total cancer risks for each exposure pathway.

2.2.4.2 Risk Modeling

Due to the complex nature of MSAT fate and transport in different media such as air, water, soil, and atmospheric deposition, more than 78 fate, transport, and exposure equations are used to compute media concentrations, potential cancer risk, and noncancer health effects.

Many of these equations are non-linear, such as the calculation of the cumulative soil concentration, calculation of MSAT loss due to runoff, or the calculation of aboveground produce concentration due to direct deposition of particle-phase MSATs such as benzo(a)pyrene or diesel particulate matter.

A hybrid modeling approach was used to calculate the cancer and noncancer risks and cumulative risk. The hybrid model consists of a computer program known as IRAP-h View [68] and a custom Python program developed for this chapter. IRAP-h View follows the methodologies published in the HHRAP.

IRAP-h View, also known as the risk engine or risk modeling platform in this work, was used to calculate the cancer and noncancer risks encapsulating all the guidance and procedures contained in HHRAP. The cumulative risk was calculated using the Python code.

In addition to modeled ground-level air concentrations, the proposed methodology is flexible in utilizing in situ measured concentrations from near-road monitoring and air monitoring networks to estimate risk resulting from exposure to MSATs through the inhalation exposure pathway.

This chapter fills gaps in the mobile emissions health impact literature, as described below:

1. **Mobile Emissions Inventory Data Quality:** This work generates the on-road vehicle emissions inventory using a state-of-the-science simulator instead of using outdated emission factors. Additionally, the methodology presented in this chapter is flexible in incorporating spatiotemporal considerations to improve the characterization of emissions and health impacts.
2. **Spatial Accuracy:** The Vehicle Emission Simulator (MOVES) lacks spatial information. To fill this gap, the present work geospatially links the emissions results to air dispersion and deposition models, air toxics fate and transport analysis, and a risk modeling platform to estimate the impacts of MSATs on humans.
3. **Allocation of Source Impacts:** Previous work accounted for a lumped air concentration and could not apportion sources to specific human impacts. This work conducts a multi-pathway fate-and-transport analysis of MSATs, source by source. This allows allocating the contribution of each source's deleterious effects from on-road mobile sources, revealing the full extent of exposure to MSATs.

2.3 Results

2.3.1 Case Study

A case study for high-traffic roadway segments located in the city of Saint Paul in the U.S. state of Minnesota is presented. The roadway segments are within the Interstate-35E, Interstate-94, and US-10 junction in Saint Paul, and the study area is in Ramsey County. Figure 2-3 shows the location of the high-traffic roadway.

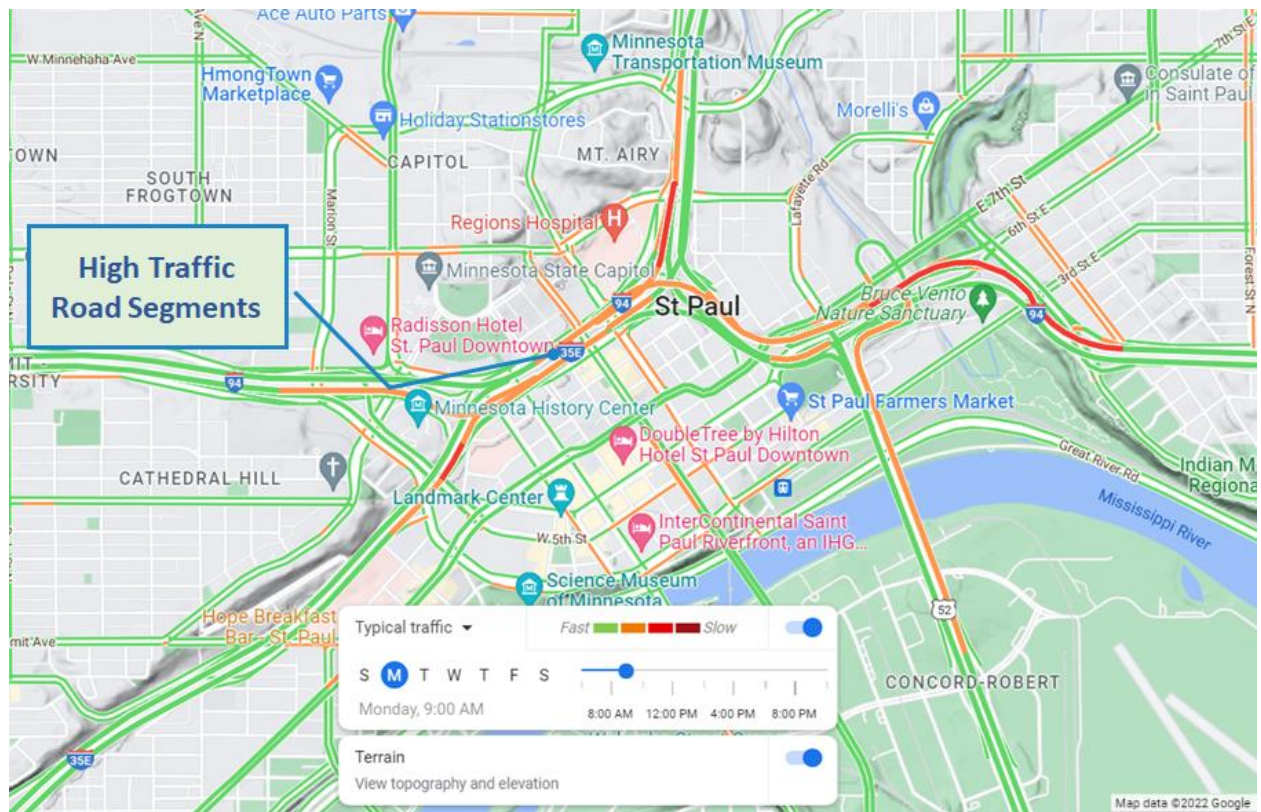


Figure 2-3: Location of high-traffic road segments on Interstate 35E. Map data: Google Maps

This study area was selected because it contains roadway segments with some of the highest annual average daily traffic (AADT) in Ramsey County [69] and because there is a near-road monitoring site (Saint Paul—Ramsey Health Center with AQS Site ID: 27-123-0868) within the study area. The Saint Paul—Ramsey Health Center monitoring site is located at 44°57'2.50" N, 93°5'54.60" W and was chosen to be the project center.

Table 2-2 shows the AADT at the nearest point to the project center for the following years: 2011, 2017, 2018, 2019, and 2020.

Table 2-2: Project center in Saint Paul annual average daily traffic

Year	AADT
2011	183,000
2017	197,000
2018	203,000
2019	203,000
2020	160,000

The datasets used for model input and validation in this case study are from 2011. The 2017, 2018, and 2019 AADT were included in Table 2-2 to demonstrate the upward trend of vehicle volume growth from 2011. However, the AADT in 2020 dropped from 2019 due to the COVID-19 pandemic.

The following subsections contain information on the air dispersion model setup, meteorological data for the study area, sensitive receptors, developed exposure scenarios, and target risk levels.

2.3.1.1 Air Dispersion Model Setup

The high-traffic roadway segments were modeled as a series of area sources in AERMOD View. Area sources were selected because they are one of the preferred ways to represent vehicle emissions based on guidance from the EPA [70, 71], and these types of sources create a uniform emission characterization of a roadway segment.

In addition, the area source algorithm does not contain distance restrictions for model calculations like point and volume sources.

Ground-level concentrations of the MSATs were modeled using AERMOD with its non-default regulatory model options. These options include the use of elevated terrain, Fast Area Sources

(FASTAREA), optimized Area Source Plume Depletion (AREADPLT), and the use of processing routines to handle calm and missing meteorological data.

The annual and 1-hour averaging times were selected, and the following control pathway specifications were included: concentration, dry deposition, wet deposition, and total deposition. In addition, the dry and wet depletion options were selected to account for plume depletion due to dry and wet removal mechanisms.

The air dispersion modeling was conducted using the unit emission rate concept, where the entire area source is emitting at a rate of 1 g per second divided by the source area. This modeling approach eliminates multiple air dispersion modeling runs for the vast number of MSATs. Scaling of the unitized concentrations by the appropriate MSAT emission rate is applied in the risk modeling platform.

2.3.1.2 Prognostic Weather Setup

The Weather Research and Forecasting (WRF) model was used to generate the air dispersion meteorological files for the project area for 2011. A 4-km refined grid cell resolution was selected to depict the meteorological characteristics of the project area adequately. The wind rose for the project center, generated using WRPLOT View [72], is shown in Figure 2-4.

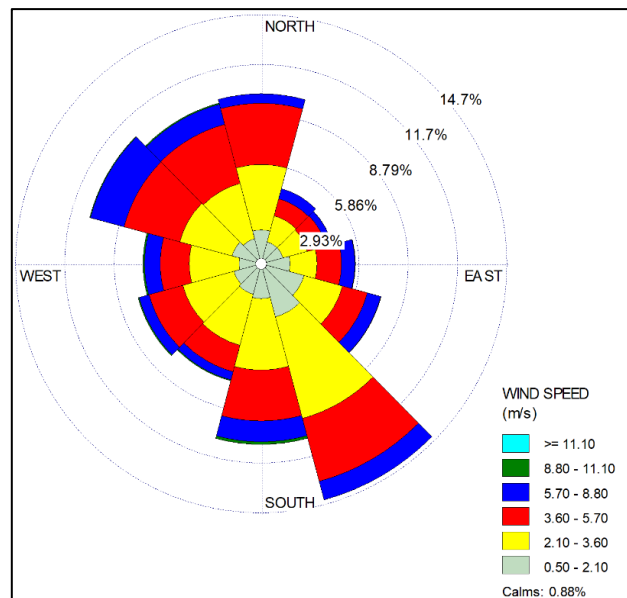


Figure 2-4: Wind rose plot showing the wind patterns at the project center for 2011

Following the successful execution of WRF, three meteorological variables (i.e., wind direction, wind speed, and temperature) were extracted and analyzed against measurements from two meteorological stations (Saint Paul Downtown Airport and Minneapolis-Saint Paul International Airport), both located within 13 km of the WRF center point.

Operational analysis of these variables was conducted following standard recommendations for statistical analysis procedures used to validate meteorological model performance. The metrics used to evaluate model performance are bias, mean absolute error, root mean square error, and index of agreement. These metrics were compared against established model performance benchmarks for meteorological models [73]. For example, common benchmarks for the index of agreement include a value greater than or equal to 0.8 [74]. Further information is provided in Appendix A.

The WRF data were compared to surface weather observations at two meteorological stations, showing good agreement, while statistical error measurements indicated satisfactory agreement between the WRF model and observations. Based on these comparisons, it is suggested that the WRF model adequately represents the weather conditions for Saint Paul, Minnesota, and its surrounding areas at the centermost grid cell.

In 2011, the prevailing winds blew from the southeast 15% of the time, with an average speed of 3.41 m/s. Statistical analysis of meteorological data, such as frequency distributions and counts, is important for understanding the probability of downwind impacts. In addition, wind speed and direction variability heavily influence the transport of traffic-related air toxics and thus exposure.

Studies found that the average concentrations of MSATs in a neighborhood decreased as wind speeds increased. The decrease in concentrations is mainly due to horizontal dilution and vertical dilution, which is a function of mixing layer height [75, 76, 77, 78].

2.3.1.3 Sensitive Receptors

Sensitive receptors are locations where people are more susceptible to adverse effects of exposure to mobile source air toxics (e.g., schools, hospitals, places of worship, etc.). Three sensitive receptors were included in the multi-pathway human health risk assessment. The names and locations of these receptors are summarized in Table 2-3.

Table 2-3: Assessed sensitive receptor locations

Sensitive Receptor	Sensitive Receptor Name	Latitude and Longitude
Receptor 1	Saint Paul—Ramsey Health Center	44°57'2.50" N, 93°5'54.60" W
Receptor 2	Anderson Office Building	44°57'3.90" N, 93°5'48.59" W
Receptor 3	Minnesota State Capitol	44°57'18.71" N, 93°6'8.21" W

The criteria to identify and set up these sensitive receptors included the following steps:

1. Identify the project grid nodes with the maximum modeled values for each air parameter within a defined area. These air parameters include unitized hourly air concentrations from vapor, particle, and particle-bound phases and unitized annual average air concentrations from vapor, particle, and particle-bound phases.
2. Ignore grid nodes located in the middle of the roadway, as evaluating risk at locations where people are exposed intermittently is not the focus of this chapter.
3. Select the applicable exposure scenarios (discussed below).

2.3.1.4 Multi-Pathway Exposure Scenarios

The multi-pathway human health risk assessment was conducted for the following four exposure scenarios: Farmer Adult, Farmer Child, Urban Resident Adult, and Urban Resident Child. For each of the three sensitive receptors, the evaluated exposure pathways are summarized in Table 2-4. Future work will incorporate additional exposure scenarios such as Fisher Adult and Fisher Child and unique scenarios such as hunter (e.g., deer meat consumption).

Table 2-4: Evaluated exposure pathways

Exposure Pathways	Applicable Exposure Scenario
Direct Inhalation of Vapors and Particles	Urban Residents and Farmers
Incidental Ingestion of Soil	Urban Residents and Farmers
Ingestion of Drinking Water from Surface Water Sources	Urban Residents and Farmers
Ingestion of Homegrown Produce	Urban Residents and Farmers
Ingestion of Homegrown Beef	Applies only to Farmers
Ingestion of Milk from Homegrown Cows	Applies only to Farmers
Ingestion of Homegrown Chicken	Urban Residents and Farmers
Ingestion of Eggs from Homegrown Chicken	Urban Residents and Farmers
Ingestion of Homegrown Pork	Applies only to Farmers

This subsection describes a few key exposure parameters, including duration, frequency, averaging time, and rates of ingestion and inhalation. These factors are evaluated in human health risk assessments to quantify exposure through different exposure routes such as ingestion, inhalation, and dermal exposure.

Exposure duration is the length of time that a receptor (individual or population) is exposed to the MSATs being evaluated. Exposure frequency is defined as the frequency with which the exposure occurs. This exposure parameter is provided in number of days per year. A typical conservative value of 350 days per year is used based on the protective assumption that a receptor (e.g., an individual) spends a maximum of two weeks away from the exposure source.

The averaging time is the period over which exposure is averaged, and this parameter varies with the type of MSATs. For example, for carcinogenic MSATs, the averaging time equals 25,550 days (cumulative intake is prorated over a lifetime of 70 years). In contrast, the averaging time for noncancerous MSATs equals the exposure duration multiplied by 365 days.

The inhalation rate is described as the amount of air inhaled over a specified period. This value should be representative of the population being evaluated when conducting human health risk assessments. An inhalation rate of 0.83 m³/h is recommended for an adult, and 0.3 m³/h for a child [37]. These values correspond to 20 m³/day and 7 m³/day, respectively.

The ingestion rate is defined as the amount of water, food (including produce, fish, fruits, meat, etc.), or soil containing the MSATs that a receptor (e.g., individual or population) ingests during a specified period. The ingestion rate parameter is expressed in units of mass per unit time. As mentioned above, a representative ingestion rate value should be used when estimating human health risk associated with exposure to MSATs.

2.3.1.5 Target Risk Levels

The target risk levels for evaluating exposure to MSATs are narrowed to increase the level of conservatism and to account for exposure to the study area's background concentrations and account for different lifestyles. These targets are used in this chapter to interpret the magnitude of cancer risk and noncancer health effects. The target risk levels are summarized in Table 2-5.

Table 2-5: Target risk levels

Description	Value	Normalized Value
Cancer Risk Threshold	1.00E-05	1.0E-05/1.0E-05 = 1
Hazard Quotient Threshold	0.25	0.25/0.25 = 1

2.3.2 Air Dispersion and Deposition Model Results

This subsection presents the air dispersion and deposition model results, including ground-level air dispersion patterns (contour plots) for each MSAT and the deposition term for each MSAT at the three sensitive receptors.

The annual average concentrations generated by the air dispersion simulation were used to evaluate potential adverse health impacts from chronic exposure to MSATs. Additionally, the maximum 1-hour concentrations were used to evaluate the potential for adverse health effects from acute exposure. The contours shown in this subsection were generated from the air dispersion model's

maximum hourly and annual average results. The concentration values presented represent unitized values expressed in $\mu\text{g-s/g-m}^3$.

2.3.2.1 Benzene

The fraction of benzene's air concentration in the vapor phase (F_v) equals 1 [37]; therefore, only the vapor phase was modeled. Figure 2-5 shows the benzene unitized maximum hourly air concentrations (in $\mu\text{g-s/g-m}^3$) from the vapor phase contour plot, and Figure 2-6 shows the unitized annual average air concentrations (in $\mu\text{g-s/g-m}^3$) from the vapor phase.

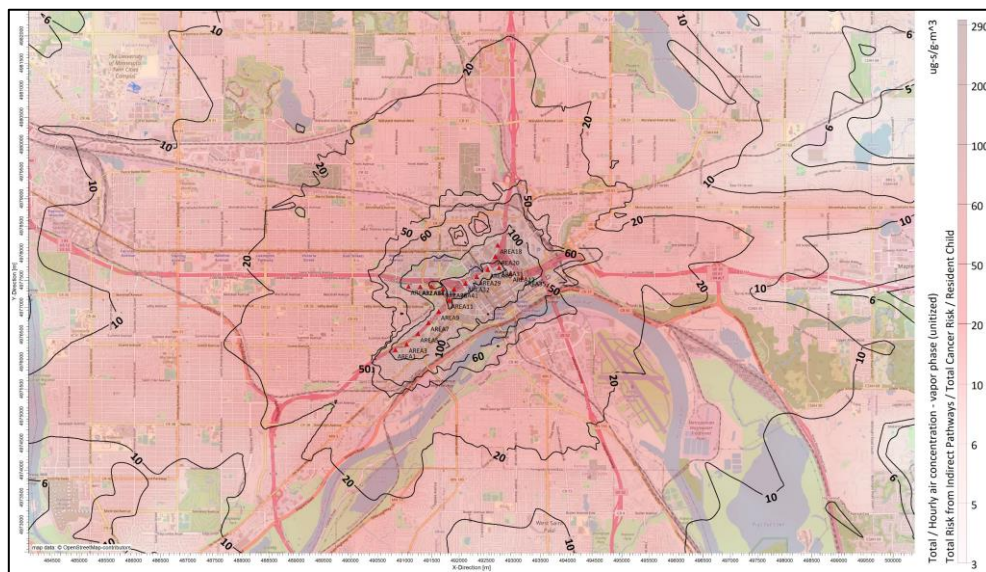


Figure 2-5: Benzene hourly air concentration—vapor phase (unitized) contour plot overlaid on OpenStreetMap satellite imagery (OpenStreetMap, n.d.). Unit used in contour is $\mu\text{g-s/g-m}^3$

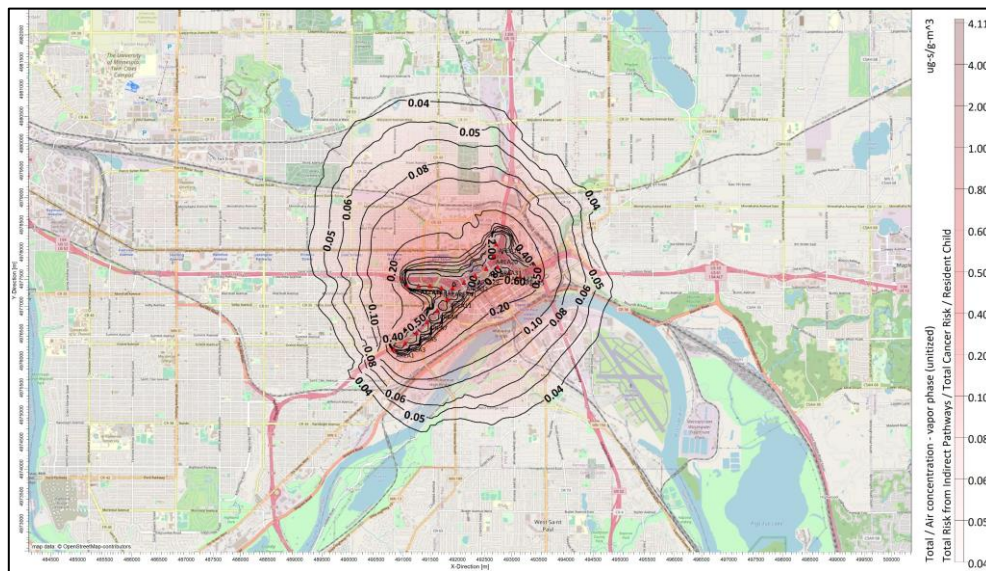


Figure 2-6: Benzene annual air concentration—vapor phase (unitized) contour plot overlaid on OpenStreetMap satellite imagery (OpenStreetMap, n.d.). Unit used in contour is $\mu\text{g-s/g-m}^3$

The generated contours are based on the distribution of the modeled results, and the modeled sources are represented by the red triangles in Figure 2-5 and Figure 2-6.

In Figure 2-5, the maximum concentration observed within the modeled domain is $289.7 \mu\text{g-s/g-m}^3$. Areas of high concentration values are clustered around the project's center, and the maximum value's location is $44^\circ 56' 59.4'' \text{ N}$, $93^\circ 6' 8.35'' \text{ W}$ ($491,928.63 \text{ m E}$, $4,977,382.64 \text{ m N}$, Zone 15 in UTM Coordinate System).

In Figure 2-6, the annual maximum concentration value dropped to $4.1 \mu\text{g-s/g-m}^3$ due to the annual averaging. The annual averaging is the arithmetic mean of the 8,760 hourly concentrations minus calm and missing hours. The unitized benzene maximum concentration (in $\mu\text{g-s/g-m}^3$) values and their locations are shown in Table 2-6.

Table 2-6: Benzene maximum concentration values and their respective locations

Air Parameter	Maximum (Max) Concentration	Location of Max in Degrees and UTM
Unitized hourly air concentration from vapor phase	289.7 $\mu\text{g-s/g-m}^3$	44°56'59.4" N, 93°6'8.35" W (491,928.63 m E, 4,977,382.64 m N, Zone 15)
Unitized annual average air concentration from vapor phase	4.1 $\mu\text{g-s/g-m}^3$	

2.3.2.2 Formaldehyde

Like benzene, the fraction of formaldehyde's air concentration in the vapor phase equals 1 [37]; therefore, formaldehyde is assumed to only occur in the vapor phase. Figure 2-7 shows the formaldehyde unitized maximum hourly air concentrations from the vapor phase contour plot.

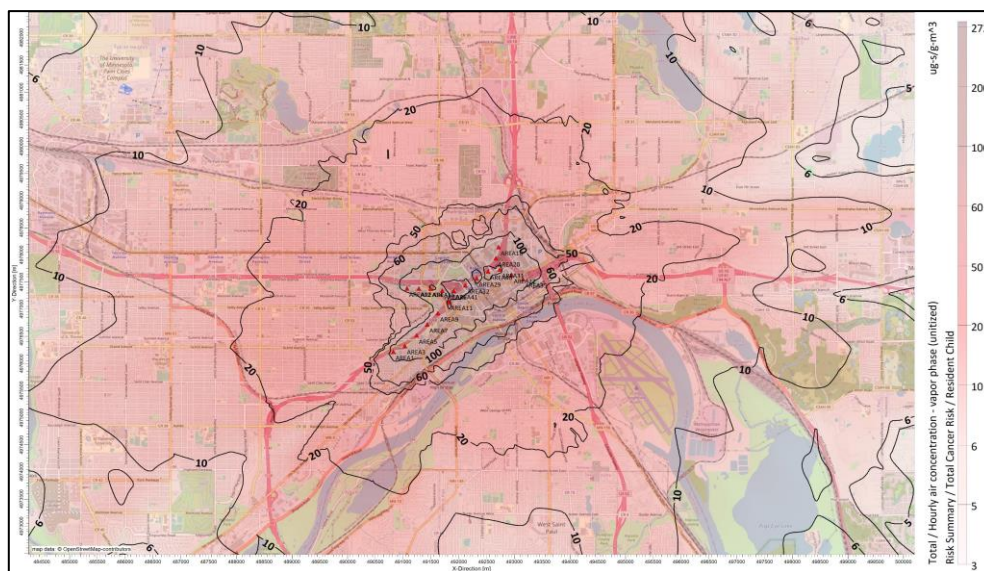


Figure 2-7: Formaldehyde hourly air concentration—vapor phase (unitized) contour plot overlaid on OpenStreetMap satellite imagery (OpenStreetMap, n.d.). Unit used in contour is $\mu\text{g-s/g-m}^3$

In Figure 2-7, the maximum air concentration value is 271.8 $\mu\text{g-s/g-m}^3$. Similar to benzene, high concentrations were located near the project's center. The unitized annual average air concentrations from the vapor phase ground-level air dispersion pattern are shown in Figure 2-8.

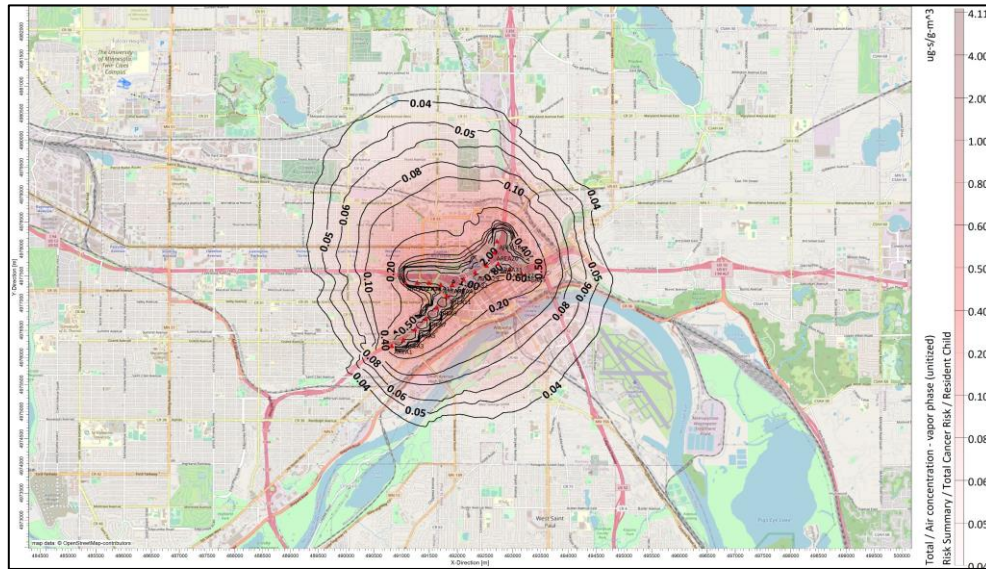


Figure 2-8: Formaldehyde annual air concentration—vapor phase (unitized) contour plot overlaid on OpenStreetMap satellite imagery (OpenStreetMap, n.d.). Unit used in contour is $\mu\text{g-s/g-m}^3$

A summary of the unitized formaldehyde maximum concentration values and their locations is provided in Table 2-7.

Table 2-7: Formaldehyde maximum concentration values and their respective locations

Air Parameter	Maximum (Max) Concentration	Location of Max in Degrees and UTM
Unitized hourly air concentration from vapor phase	271.8 $\mu\text{g-s/g-m}^3$	44°56'59.4" N, 93°6'8.35" W (491,928.63 m E, 4,977,382.64 m N, Zone 15)
Unitized annual average air concentration from vapor phase	4.07 $\mu\text{g-s/g-m}^3$	

The observed similarity in the shapes of the ground-level air dispersion patterns (contour plots) presented in Figure 2-5, Figure 2-6, Figure 2-7, and Figure 2-8 is explained by the likeness of the benzene and formaldehyde emission rate values.

The slight differences in the concentration results presented in Table 2-6 and Table 2-7 are based solely on the differences in gas deposition input parameters for benzene and formaldehyde. The gas deposition parameters are pollutant diffusivity in air, pollutant diffusivity in water, cuticular resistance, and Henry's law constant.

2.3.2.3 Benzo(a)pyrene (BaP)

The fraction of BaP's air concentration in the vapor phase (F_v) is 0.294, and the balance (0.706) is emitted as condensed in a particle-bound portion [37]. Particle-bound modeling requires the provision of particle size distributions for particles originating from on-road sources.

The fate and transport of these particles is determined by the size of the particle, and larger particles deposit closer to the source, whereas smaller particles remain suspended in the air for prolonged periods [79]. In other words, BaP can be transported over long distances and deposited farther away from the source [80].

Figure 2-9 shows the BaP unitized maximum hourly air concentrations from the vapor phase contour plot; the unitized maximum hourly air concentrations from the particle-bound phase contour plot are shown in Figure 2-10.

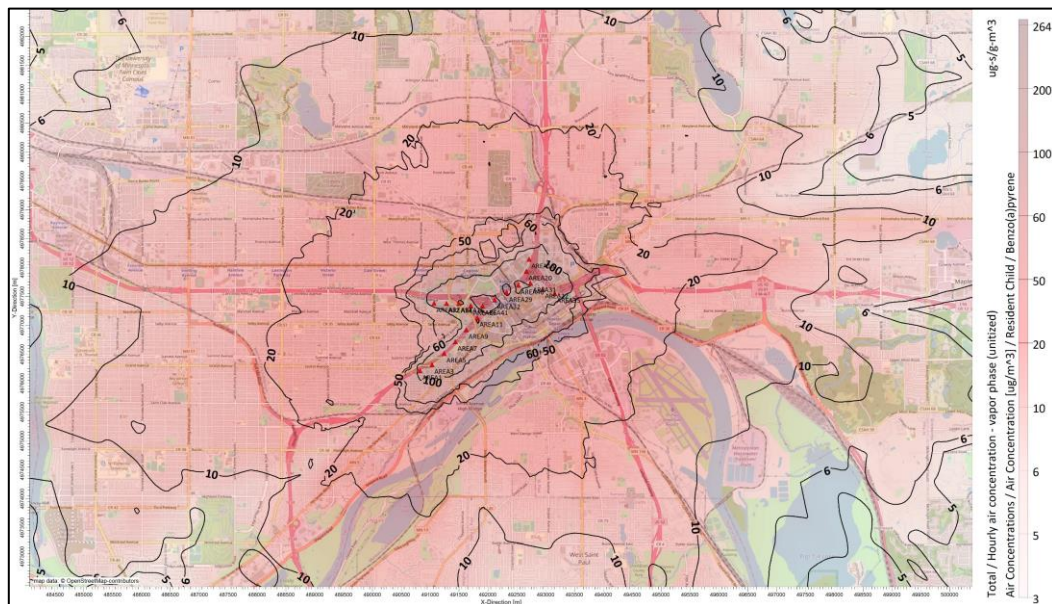


Figure 2-9: BaP hourly air concentration—vapor phase (unitized) contour plot overlaid on OpenStreetMap satellite imagery (OpenStreetMap, n.d.). Unit used in contour is $\mu\text{g-s/g-m}^3$

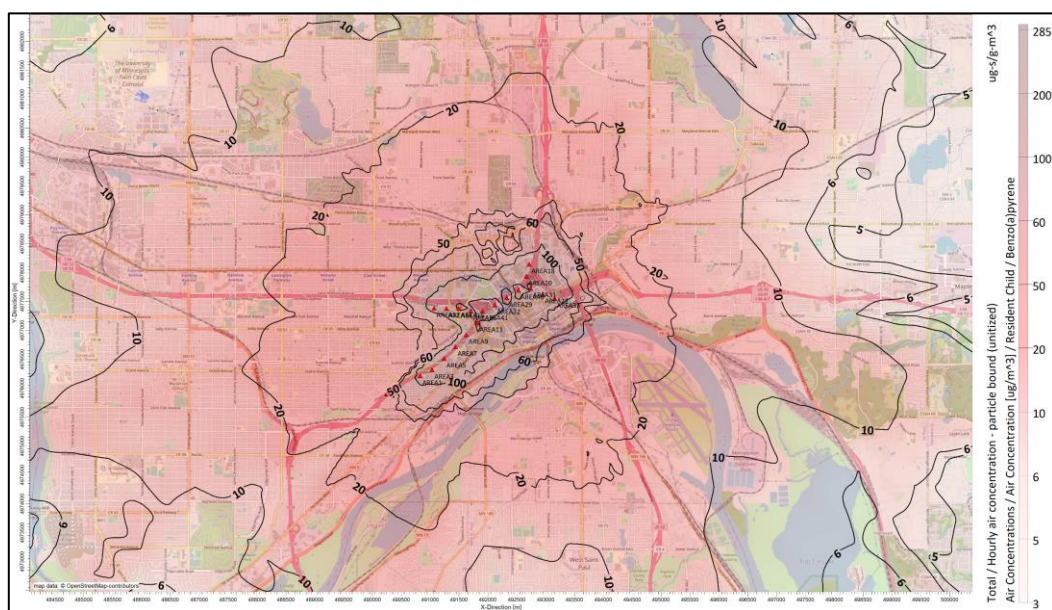


Figure 2-10: BaP hourly air concentration—particle-bound phase (unitized) contour plot overlaid on OpenStreetMap satellite imagery (OpenStreetMap, n.d.). Unit used in contour is $\mu\text{g-s/g-m}^3$

The calculated unitized maximum hourly air concentration from the vapor phase is 263.7 $\mu\text{g-s/g-m}^3$, and the unitized maximum hourly air concentration for the particle-bound phase is 283.7 $\mu\text{g-s/g-m}^3$. Both maximum hourly concentrations occurred near the on-road emission sources.

Figure 2-11 and Figure 2-12 show the unitized annual average air concentrations from the vapor and particle-bound phase contour plots, respectively.

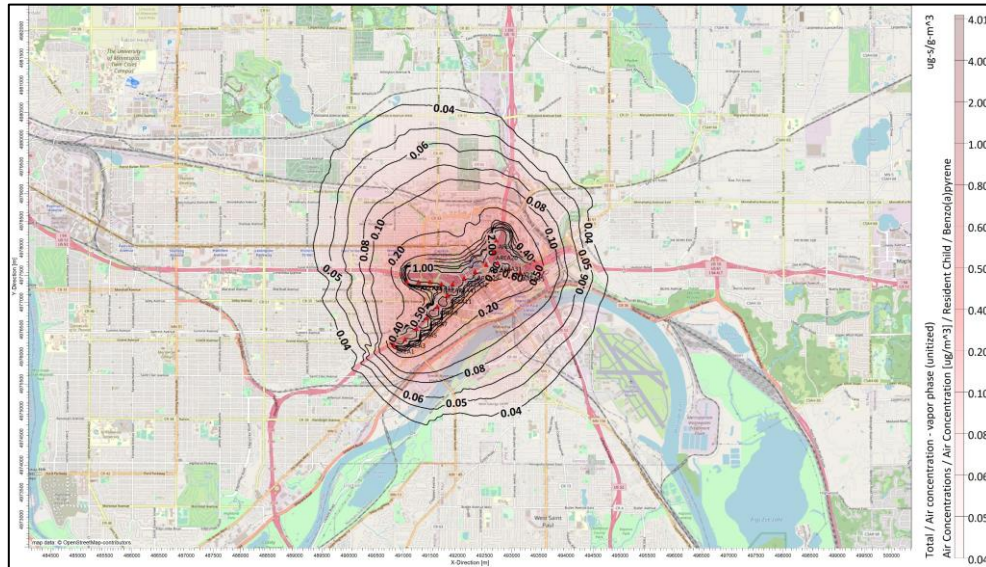


Figure 2-11: BaP annual air concentration—vapor phase (unitized) contour plot overlaid on OpenStreetMap satellite imagery (OpenStreetMap, n.d.). Unit used in contour is $\mu\text{g-s/g-m}^3$

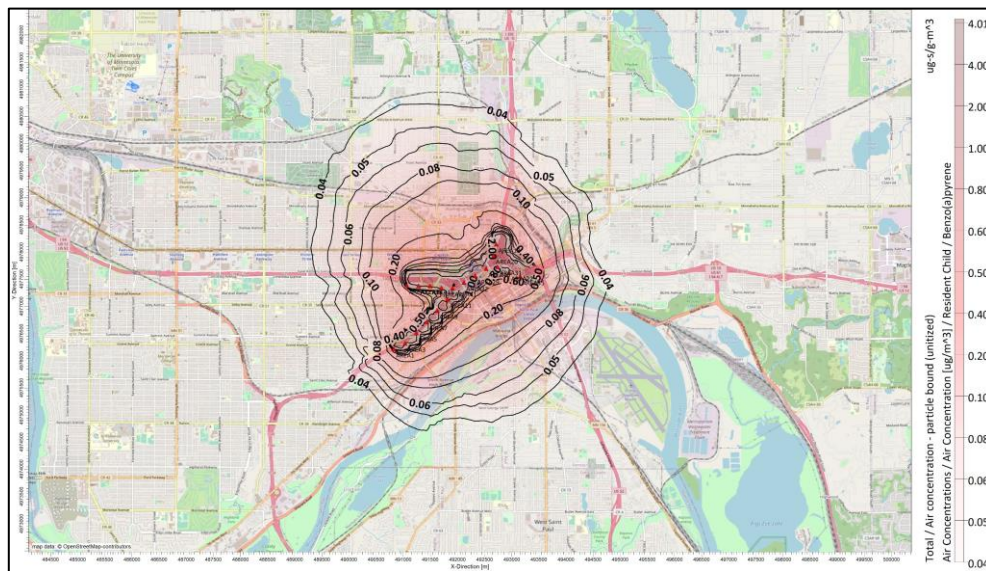


Figure 2-12: BaP annual air concentration—particle-bound phase (unitized) contour plot overlaid on OpenStreetMap satellite imagery (OpenStreetMap, n.d.). Unit used in contour is $\mu\text{g-s/g-m}^3$

The contour plots in both Figure 2-11 and Figure 2-12 show that the maximum values have decreased compared to the hourly air concentrations. The unitized maximum concentration values and their respective locations are shown in Table 2-8.

Table 2-8: Benzo(a)pyrene maximum concentration values and their respective locations

Air Parameter	Maximum (Max) Concentration	Location of Max in Degrees and UTM
Unitized hourly air concentration from vapor phase	263.7 $\mu\text{g-s/g-m}^3$	44°56'59.38" N, 93°6'8.38" W (491,927.97 m E, 4,977,382.02 m N, Zone 15)
Unitized hourly air concentration from particle bound	283.7 $\mu\text{g-s/g-m}^3$	
Unitized annual average air concentration from vapor phase	4.01 $\mu\text{g-s/g-m}^3$	
Unitized annual average air concentration from particle bound	4.06 $\mu\text{g-s/g-m}^3$	

2.3.2.4 Deposition Term

The deposition term represents the rate at which particles and vapor-phase MSATs are directly deposited from air to soil, water, and above-ground produce. The deposition term is the predominant driver of MSAT concentrations in the indirect exposure pathways. A summary of the results at the three sensitive receptors is shown in Table 2-9.

Table 2-9: Deposition term calculated values at the three receptor locations

Sensitive Receptor	Deposition Term [milligram MSATs/kg soil]		
	Benzene	Formaldehyde	Benzo(a)pyrene
Saint Paul—Ramsey Health Center	1.60E-03	1.75E-01	4.30E-04
Anderson Office Building	6.32E-04	7.48E-02	1.84E-04
Minnesota State Capitol	7.49E-05	1.24E-02	2.59E-05

The MSAT deposition rates per unit area of soil are calculated from the unitized annual average dry and wet deposition fluxes from the vapor phase. The dry and wet deposition fluxes are calculated from the ambient air quality modeling conducted for benzene, formaldehyde, and benzo(a)pyrene.

The highest deposition term value (italicized) was calculated at the Saint Paul—Ramsey Health Center for benzo(a)pyrene. The maximum deposition term value associated with BaP comes as no surprise due to the physicochemical properties of this aromatic hydrocarbon [80, 81], particle size distribution, and the phases it is emitted in (e.g., vapor phase and particle-bound phase).

2.3.3 Risk Results

This subsection summarizes the results of the comprehensive multi-pathway human health risk assessment, and the following results (standard and normalized) are included:

- Total individual (i.e., MSAT-specific) cancer risks and noncancer hazards.
- Acute Hazard Quotient.
- Cumulative excess lifetime cancer risks (ELCRs) and chronic noncancer hazards.

The normalized risk values were computed by dividing the calculated risk value, cancer or noncancer, by the respective target risk level (e.g., cancer values were divided by 1.0E-05, and hazard values were divided by 0.25). Italicized risk values indicate a value exceeding the Target Risk Levels shown in Table 2-5.

2.3.3.1 Total Individual Cancer Risks and Noncancer Hazards

Table 2-10, Table 2-11, and Table 2-12 summarize the total individual cancer risks and noncancer hazards for each MSAT (e.g., benzene, formaldehyde, and benzo(a)pyrene) across all pathways for four exposure scenarios at three different locations (sensitive receptors) within the Interstate-35E, Interstate-94, and US-10 junction in the Saint Paul study area. Italicized risk values indicate a value exceeding the Target Risk Levels.

Table 2-10: Total individual benzene cancer risks and noncancer hazards

Saint Paul—Ramsey Health Center				
Risk	Urban Resident Adult	Urban Resident Child	Farmer Adult	Farmer Child
Cancer	5.30E-07	1.06E-07	7.06E-07	1.06E-07
Hazard	5.28E-03	5.28E-03	5.28E-03	5.28E-03
Normalized Results (Cancer Values Divided by 1.0E-05, Hazard Values Divided by 0.25)				
Risk	Urban Resident Adult	Urban Resident Child	Farmer Adult	Farmer Child
Cancer	5.30E-02	1.06E-02	7.06E-02	1.06E-02
Hazard	2.11E-02	2.11E-02	2.11E-02	2.11E-02
Anderson Office Building				
Risk	Urban Resident Adult	Urban Resident Child	Farmer Adult	Farmer Child
Cancer	2.37E-07	4.74E-08	3.16E-07	4.74E-08
Hazard	2.37E-03	2.37E-03	2.37E-03	2.37E-03
Normalized Results				
Risk	Urban Resident Adult	Urban Resident Child	Farmer Adult	Farmer Child
Cancer	2.37E-02	4.74E-03	3.16E-02	4.74E-03
Hazard	9.46E-03	9.46E-03	9.46E-03	9.46E-03
Minnesota State Capitol				
Risk	Urban Resident Adult	Urban Resident Child	Farmer Adult	Farmer Child
Cancer	3.81E-08	7.63E-09	5.08E-08	7.63E-09
Hazard	3.80E-04	3.80E-04	3.80E-04	3.80E-04
Normalized Results				
Risk	Urban Resident Adult	Urban Resident Child	Farmer Adult	Farmer Child
Cancer	3.81E-03	7.63E-04	5.08E-03	7.63E-04
Hazard	1.52E-03	1.52E-03	1.52E-03	1.52E-03

Table 2-11: Total individual formaldehyde cancer risks and noncancer hazards

Saint Paul—Ramsey Health Center				
Risk	Urban Resident Adult	Urban Resident Child	Farmer Adult	Farmer Child
Cancer	9.43E-07	1.89E-07	1.26E-06	1.89E-07
Hazard	1.75E-02	1.76E-02	1.75E-02	1.77E-02
Normalized Results (Cancer Values Divided by 1.0E-05, Hazard Values Divided by 0.25)				
Risk	Urban Resident Adult	Urban Resident Child	Farmer Adult	Farmer Child
Cancer	9.43E-02	1.89E-02	1.26E-01	1.89E-02
Hazard	6.99E-02	7.05E-02	7.00E-02	7.08E-02
Anderson Office Building				
Risk	Urban Resident Adult	Urban Resident Child	Farmer Adult	Farmer Child
Cancer	4.21E-07	8.42E-08	5.61E-07	8.42E-08
Hazard	7.79E-03	7.86E-03	7.81E-03	7.89E-03
Normalized Results				
Risk	Urban Resident Adult	Urban Resident Child	Farmer Adult	Farmer Child
Cancer	4.21E-02	8.42E-03	5.61E-02	8.42E-03
Hazard	3.12E-02	3.14E-02	3.12E-02	3.16E-02
Minnesota State Capitol				
Risk	Urban Resident Adult	Urban Resident Child	Farmer Adult	Farmer Child
Cancer	6.73E-08	1.35E-08	8.97E-08	1.35E-08
Hazard	1.25E-03	1.26E-03	1.25E-03	1.26E-03
Normalized Results				
Risk	Urban Resident Adult	Urban Resident Child	Farmer Adult	Farmer Child
Cancer	6.73E-03	1.35E-03	8.97E-03	1.35E-03
Hazard	4.99E-03	5.03E-03	5.00E-03	5.05E-03

Table 2-12: Total individual benzo(a)pyrene cancer risks and noncancer hazards

Saint Paul—Ramsey Health Center				
Risk	Urban Resident Adult	Urban Resident Child	Farmer Adult	Farmer Child
Cancer	1.86E-07	8.41E-08	<i>4.59E-05</i>	9.87E-06
Hazard	7.18E-02	7.21E-02	1.08E-01	1.24E-01
Normalized Results (Cancer Values Divided by 1.0E-05, Hazard Values Divided by 0.25)				
Risk	Urban Resident Adult	Urban Resident Child	Farmer Adult	Farmer Child
Cancer	1.86E-02	8.41E-03	<i>4.59E+00</i>	9.87E-01
Hazard	2.87E-01	2.88E-01	4.34E-01	4.97E-01
Anderson Office Building				
Risk	Urban Resident Adult	Urban Resident Child	Farmer Adult	Farmer Child
Cancer	8.00E-08	3.61E-08	<i>2.02E-05</i>	4.34E-06
Hazard	3.20E-02	3.21E-02	4.81E-02	5.50E-02
Normalized Results				
Risk	Urban Resident Adult	Urban Resident Child	Farmer Adult	Farmer Child
Cancer	8.00E-03	3.61E-03	<i>2.02E+00</i>	4.34E-01
Hazard	1.28E-01	1.28E-01	1.92E-01	2.20E-01
Minnesota State Capitol				
Risk	Urban Resident Adult	Urban Resident Child	Farmer Adult	Farmer Child
Cancer	1.16E-08	5.16E-09	3.12E-06	6.70E-07
Hazard	5.11E-03	5.12E-03	7.59E-03	8.67E-03
Normalized Results				
Risk	Urban Resident Adult	Urban Resident Child	Farmer Adult	Farmer Child
Cancer	1.16E-03	5.16E-04	3.12E-01	6.70E-02
Hazard	2.04E-02	2.05E-02	3.04E-02	3.47E-02

From the three analyzed MSATs, the calculated human health risks resulting from exposure to benzene and formaldehyde fall below the target level of $1.0\text{E-}05$ for cancer and 0.25 for noncancer hazard risks; however, the calculated risk from benzo(a)pyrene exposure exceeds the target risk level for cancer.

From Table 2-12, the calculated cancer risk for the Farmer Adult scenario at the Saint Paul—Ramsey Health Center receptor exceeds the published target levels, with a risk value of $4.59\text{E-}05$, which is equal to “45.9 in 1 million people.” Similarly, the calculated cancer risk for the Farmer Adult scenario at the Anderson Office Building receptor exceeded the target levels. The calculated risk value at the Anderson Office Building receptor is $2.02\text{E-}05$, which is equal to “20.2 in 1 million people.”

The elevated cancer risk results at the Saint Paul—Ramsey Health Center were expected because that location had the maximum calculated air concentration and the maximum deposition term value. These values are $1.49\text{E-}04 \mu\text{g}/\text{m}^3$ and $4.30\text{E-}04$ milligram benzo(a)pyrene per kg soil, respectively.

2.3.3.2 Acute Hazard Quotient-Inhalation (AHQ_{inh})

Table 2-13 summarizes the acute hazard quotient-inhalation (AHQ_{inh}) at the study area’s three different sensitive receptors. The acute inhalation risk is evaluated by comparing the maximum 1-hour (acute) air concentrations estimated by the air dispersion model to the acute inhalation exposure criteria (AIEC).

The calculated acute hazard quotients for the three analyzed MSATs, which are used to evaluate short-term inhalation exposures to estimate noncancer health effects, all fall within the range of target levels. Therefore, actual short-term exposure to benzene, formaldehyde, or benzo(a)pyrene resulting from mobile emissions is believed to be well within acceptable risk levels.

Table 2-13 summarizes the maximum hourly air concentrations, the respective acute hazard quotient at the three sensitive receptor locations, and the normalized acute hazard quotient.

Table 2-13: Maximum hourly air concentrations and acute hazard quotient AHQ_{inh} results

Saint Paul—Ramsey Health Center			
MSATs	Max Hourly Conc. ($\mu\text{g}/\text{m}^3$)	AHQ_{inh}	AHQ_{inh} (Normalized)
Benzene	7.96E+00	6.12E-03	2.45E-02
Formaldehyde	8.25E+00	8.77E-02	3.51E-01
Benzo(a)pyrene	7.04E-03	1.17E-05	4.69E-05
Anderson Office Building			
MSATs	Max Hourly Conc. ($\mu\text{g}/\text{m}^3$)	AHQ_{inh}	AHQ_{inh} (Normalized)
Benzene	8.99E+00	6.91E-03	2.76E-02
Formaldehyde	9.33E+00	9.92E-02	3.97E-01
Benzo(a)pyrene	7.98E-03	1.33E-05	5.32E-05
Minnesota State Capitol			
MSATs	Max Hourly Conc. ($\mu\text{g}/\text{m}^3$)	AHQ_{inh}	AHQ_{inh} (Normalized)
Benzene	3.04E+00	2.34E-03	9.37E-03
Formaldehyde	3.05E+00	3.25E-02	1.30E-01
Benzo(a)pyrene	2.66E-03	4.44E-06	1.77E-05

2.3.3.3 Cumulative Excess Lifetime Cancer Risks and Chronic Noncancer Hazards

The normalized cumulative excess lifetime cancer risks (ELCRs) and noncancer hazards across all pathways for four exposure scenarios at three sensitive receptors are presented in Table 2-14. The estimated cumulative ELCRs were calculated by summing up the total individual (i.e., MSAT-specific) cancer risks for each exposure pathway and scenario.

Table 2-14: Normalized cumulative excess lifetime cancer risks and chronic noncancer hazards

Excess Lifetime Cancer Risks (ELCRs) (Across All Pathways)				
Sensitive Receptor/Exposure Scenarios	Urban Resident		Farmer	
	Adult	Child	Adult	Child
Saint Paul—Ramsey Health Center	1.66E-01	3.79E-02	<u>4.79E+00</u>	1.02E+00
Anderson Office Building	7.38E-02	1.68E-02	2.11E+00	4.47E-01
Minnesota State Capitol	1.17E-02	2.63E-03	3.26E-01	6.91E-02
Noncancer Hazard Indices (Across All Pathways)				
Sensitive Receptor/Exposure Scenarios	Urban Resident		Farmer	
	Adult	Child	Adult	Child
Saint Paul—Ramsey Health Center	3.78E-01	3.80E-01	5.25E-01	5.89E-01
Anderson Office Building	1.69E-01	1.69E-01	2.33E-01	2.61E-01
Minnesota State Capitol	2.69E-02	2.70E-02	3.69E-02	4.12E-02

Likewise, the cumulative noncancer hazards were calculated by adding the MSAT individual hazard indices. The cited method for noncancer risk is conservative, whereas a non-conservative approach would separate each hazard index by its toxic endpoint and categorize noncancer hazards separately.

The ELCRs for the Farmer Adult and Farmer Child scenarios are above the cancer risk threshold at the Saint Paul—Ramsey Health Center. Similarly, the Farmer Adult scenario ELCRs exceed the U.S. EPA’s acceptable range at the Anderson Office Building. However, all the other exposure scenarios are within the acceptable risk levels.

The potential for adverse noncancer health effects from exposure to on-road mobile emissions was not expected at all three sensitive receptors because all the noncancer risk results were deemed acceptable, as shown in Table 2-14.

The highest computed ELCR value (italicized and underlined in Table 2-14) was for the Farmer Adult scenario at the Saint Paul—Ramsey Health Center, whereas the highest computed noncancer hazard was for the Farmer Child at the same location (italicized in Table 2-14).

2.4 Discussion

This section discusses the results of the comprehensive multi-pathway human health risk assessment and examines the validation of the methodology and model performance. It then evaluates the strengths and limitations of the methodology and the likelihood of the scenario being realistic. Finally, variability and uncertainty associated with the analysis are assessed.

2.4.1 Comprehensive Multi-Pathway Human Health Risk Assessment

The cumulative human health impact results based on the evaluation of modeled ambient air concentrations and deposition fluxes at the three sensitive receptor locations indicate cancer risks in excess of the target risk level $1.0\text{E-}05$ (Normalized value = 1.00). A summary of the comprehensive multi-pathway human health risk assessment is presented in Table 2-15.

Table 2-15: Summary of the comprehensive multi-pathway human health risk assessment

Risk Type	Summary
Total Individual Cancer Risk	Cancer risks exceeding the target risk level ($1.0\text{E-}05$) are associated with Benzo(a)pyrene at Saint Paul—Ramsey Health Center and Anderson Office Building for the Farmer Adult scenario (see Table 2-12).
Individual Noncancer Risk	Noncancer risks for all receptor populations are well below the U.S. EPA's acceptable range of 0.25.
Acute Hazard Quotient - Inhalation	The acute hazard quotient for all receptor populations evaluated is below the U.S. EPA's acceptable range of 0.25.
Cumulative Excess Lifetime Cancer Risk (ELCR)	- Cumulative cancer risks above the target risk level ($1.0\text{E-}05$) occur in the following scenarios (see Table 2-14): a. Farmer Adult and Farmer Child at Saint Paul—Ramsey Health Center b. Farmer Adult at Anderson Office Building - The cumulative noncancer risks for all scenarios remain within the U.S. EPA's acceptable range.

It is important to note that there are uncertainties associated with computing and interpreting the cumulative ELCRs and noncancer risks. The cumulative risk results represent a high-level overview of the potential for cancer risk (gross cancer levels) and noncancer hazards from cumulative exposure to MSATs.

These uncertainties in the cumulative risk computations stem from the relationship between exposure, critical effect systems, and the development of malignant or benign tumors, as each MSAT targets different organs within the human body. For example, the critical effect of benzene exposure is observed in the immune system [82], whereas formaldehyde targets the gastrointestinal tract [83], and benzo(a)pyrene affects the developmental, immune, and reproductive systems [55].

2.4.2 Validation of Methodology

The near-road air monitoring site used to validate the air dispersion model is the Saint Paul—Ramsey Health Center. The Saint Paul—Ramsey Health Center monitoring site is located at 44°57'2.50" N, 93°5'54.60" W and was chosen to be the project center. The station base elevation is 251 m above sea level, and the monitors are positioned on the north side of the building, approximately 60 m south of the Interstate-94 corridor and its interchange with Interstate-35E.

The station measures the following pollutants: PM₁₀, PM_{2.5}, metals, CO, carbonyls, O₃, SO₂, NO_x, asbestos, and volatile organic compounds (VOCs) [84].

This monitoring site was chosen to validate the air dispersion model because of the following:

1. Proximity to the modeled high-traffic roadway segments.
2. Absence of other sources within the project area, increasing confidence in attributing the concentrations to the high-traffic roadway segments.
3. Availability of model-to-monitor ratio data for MSATs.

Figure 2-13a shows the location of the Saint Paul—Ramsey Health Center monitoring site. The monitoring equipment is displayed in Figure 2-13b.

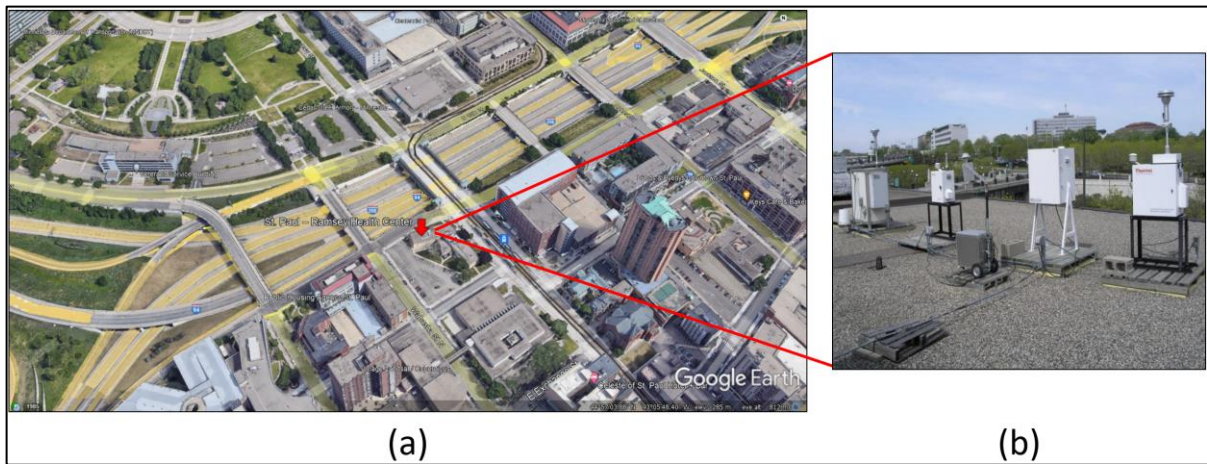


**Figure 2-13: (a) Saint Paul—Ramsey Health Center monitoring site. Map data: Google Earth.
(b) Monitoring equipment. Photo: Minnesota Pollution Control Agency**

The MSATs included in the validation study are benzene and formaldehyde. After completing the air dispersion modeling, the modeled results were compared with the monitored concentrations at the near-road monitoring site.

Comparing modeled and monitored concentrations is critical in evaluating air dispersion model performance. Using data from air monitoring stations to validate air dispersion model performance is known as model validation or calibration. Calibrating the model is an iterative process used to improve model performance and reduce uncertainty.

Table 2-16 summarizes modeled on-road air concentrations in comparison to annual mean monitored concentrations at the Saint Paul—Ramsey Health Center monitoring site.

Table 2-16: Modeled on-road air concentrations compared to annual mean monitored concentrations at the Saint Paul—Ramsey Health Center monitoring site

Benzene—Annual Values		
Modeled (On-road)	1.11	µg/m ³
MPCA Monitored	0.71	µg/m ³
MPCA Modeled	1.90	µg/m ³
On-road Modeled Source Contribution	72%	-
MPCA Modeled (On-road)	1.37	µg/m ³
MPCA Monitored—Assuming On-road Contribution is 72.0%	0.51	µg/m ³
MPCA Modeled/MPCA Monitored (Ratio)	2.68	-
Modeled (On-road)/MPCA Monitored (Ratio)	2.17	-
Formaldehyde—Annual values		
Modeled (On-road)	1.10	µg/m ³
MPCA Monitored	2.20	µg/m ³
MPCA Modeled	2.60	µg/m ³
On-road Modeled Source Contribution	36%	-
MPCA Modeled (On-road)	0.94	µg/m ³
MPCA Monitored—Assuming On-road Contribution is 36.0%	0.79	µg/m ³
MPCA Modeled/MPCA Monitored (Ratio)	1.18	-
Modeled (On-road)/MPCA Monitored (Ratio)	1.39	-

The ratios presented in Table 2-16 conform with the often-quoted factor of two accuracy, a ratio recognized in air dispersion modeling. The validation of the air dispersion model demonstrated reasonable agreement between the modeled concentrations and the available measurements at the near-

road air monitoring site. However, results varied between benzene and formaldehyde, and the modeled concentrations were higher than the measured values.

The MPCA monitored concentrations and source contribution percentages were obtained from MPCA's MNRISKS Model-Monitor Tool [85].

2.4.3 Air Dispersion Model Performance

In the Validation of Methodology subsection, it was determined that the air dispersion model's performance is deemed acceptable because the annual modeled concentrations of benzene and formaldehyde are within a factor of two [86] of the corresponding observed concentrations.

Additionally, two statistical indicators were calculated to assess the model's performance: fractional bias (FB) and normalized mean square error (NMSE) [87]. FB and NMSE were calculated using the equations shown below, and the values of these statistical indicators are summarized in Table 2-17.

$$FB = 2 \left(\frac{\overline{C_0} - \overline{C_p}}{\overline{C_0} + \overline{C_p}} \right) \quad (2-5)$$

$$NMSE = \frac{\overline{(C_0 - C_p)^2}}{\overline{C_0 C_p}} \quad (2-6)$$

where C_0 represents the observed concentration, and C_p represents the modeled concentration.

Table 2-17: Air dispersion model performance statistics

Statistical Indicator	Formaldehyde	Benzene
Fractional Bias	-0.33	-0.74
Normalized Mean Square Error	0.11	0.64

An ideal air dispersion model would have both fractional bias and normalized mean square error equal to zero [87]. The fractional bias was selected as a performance measure because it is

symmetrical and bounded, as referenced in [88]. A negative value of fractional bias indicates model overprediction.

2.4.4 Strengths and Limitations of the Methodology

This subsection outlines the strengths and limitations of the research methodology applied in this study. Key strengths of the methodology include:

1. The methodology integrates three modeling approaches: on-road vehicle emissions inventory, air dispersion, and risk estimation. This allows for a comprehensive assessment of cumulative human health impacts from multiple exposure routes, including inhalation and ingestion. It evaluates both chronic (cancer and noncancer) and acute health effects of MSATs.
2. Spatiotemporal factors are incorporated to improve the real-world representation of emissions and exposure patterns.

It is worth noting that the methodology presented has certain limitations, which are summarized below:

1. The approach relies on average exposure parameters, such as body weight and inhalation rate, which may not fully reflect population variability. Ongoing research is focused on developing a stochastic risk model to assess risks across different demographic groups, including vulnerable populations.
2. Non-mobile-source air toxics, such as indoor emissions and the formation of secondary air toxics, are not included in the current exposure assessment. Future research could expand to cover these aspects.

2.4.5 Determining the Existence of Exposure Scenarios in the Study Area

Human health impacts from MSAT exposure are calculated and analyzed for multiple developed scenarios in this chapter. The analyzed scenarios include Farmer Adult, Farmer Child, Urban Resident Adult, and Urban Resident Child. In this subsection, the likelihood of a developed scenario being realistic is discussed.

As revealed in the Risk Results section, the farmer scenario drives the total individual cancer risk in the case of benzo(a)pyrene and the cumulative cancer risk at the Saint Paul—Ramsey Health Center and the Anderson Office Building sensitive receptors. However, both sensitive receptors (e.g., Saint Paul—Ramsey Health Center and Anderson Office Building) are in highly developed,

commercially zoned urban areas, meaning the likelihood of the farmer exposure scenario driving the risk at these locations is very low.

In addition, the exposure pathways for a farmer scenario are not likely to occur because the following exposure pathways are improbable in a highly developed urban area:

1. Incidental ingestion (e.g., from hand-to-mouth contact) of soil.
2. Ingestion of homegrown produce such as lettuce, potatoes, onions, or tomatoes.
3. Ingestion of homegrown beef, chicken, eggs, or home-reared pork.

It is important in the human health risk assessment process to take a common-sense approach in analyzing the developed scenarios within a project domain and describing the probability of that scenario occurring. If the exposure scenario does not occur in the study area, the relevant exposure pathways that represent the exposure scenario do not exist, and therefore, no risk occurs.

In this chapter, the farmer scenario was included to demonstrate the different scenarios that can be applied and analyzed within the proposed methodology. Furthermore, the farmer scenario can be easily applied in other exposure settings, such as evaluating human health risks in a project area situated in agricultural land with major heavy diesel commercial motor vehicle operations, such as the Interstate 5 and California 99 intersection in the San Joaquin Valley, the United States' most productive farming area.

2.4.6 Variability and Uncertainty Discussion

Several exposure parameters used in the risk and hazard characterization were represented by average values corresponding to the 50th percentile of the exposure factor distribution, such as body weight and metabolic rates. These values misrepresent the natural variability in a population and, therefore, introduce uncertainty in the risk characterization and influence the results.

Three dominant exposure parameters, along with their associated uncertainties, are described in this subsection: body weight, inhalation rate, and consumption rate of animal tissue. These parameters are used in quantitatively estimating cancer risk and noncancer hazards.

2.4.6.1 Body Weight

The body weight (BW) parameter is used to calculate MSAT intake via the ingestion exposure pathway [37], and the ratio of the consumption rate to body weight is termed the dose.

The U.S. EPA's recommended default BW values in deterministic human health risk assessments are 70 kg for adults and 15 kg for children [37], and these values were used in the case study presented in this chapter.

The values for this parameter are project-specific and may be higher or lower depending on the project location and the assessed population. Therefore, the default BW values might not accurately reflect the population's actual body weights due to intra-population variability. However, the degree of variation of BW is not expected to significantly impact the risk results because, in the case of adults, the default value is within 25% of the true value for most adults [89].

2.4.6.2 Inhalation Rate

The inhalation rate parameter is used to estimate the average daily MSAT intake via the inhalation exposure pathway. The U.S. EPA's recommended default values are 0.83 m³/h for adults and 0.3 m³/h for children [37].

The uncertainties associated with this parameter include varying inhalation rates among the population due to many factors, such as age, activity level, genetics, and lifestyle. For example, a farmer or fisher exposure scenario may have an increased respiratory rate due to the strenuous activities involved in their jobs, such as heavy carrying and lifting. Moreover, farmers may have prolonged exposures to MSATs due to the proximity of their homes to their workplaces (e.g., farms).

2.4.6.3 Consumption Rate of Animal Tissue

The consumption rate of animal tissue is a critical parameter in estimating the daily intake of MSATs from the ingestion of animal tissue. This parameter represents the ingestion of pork, beef, milk, poultry, or eggs and is expressed in kg/kg-day on a fresh weight (FW) basis.

The recommended default consumption rate values [37] used in this study are summarized in Table 2-18.

Table 2-18: Recommended default animal tissue consumption rate values

Animal Tissue (Homegrown)	Farmer [kg/kg-day FW]	
	Adult	Child
Beef	0.00122	0.00075
Milk	0.01367	0.02268
Poultry	0.00066	0.00045
Eggs	0.00075	0.00054
Pork	0.00055	0.00042

The uncertainties associated with this parameter include employing default average values that are not representative of project-specific conditions. Moreover, these default values do not consider the substantial inter-person variability in dietary intake [90]. The variability associated with this parameter may underestimate or overestimate the studied population's consumption rate of animal tissue and thus impact exposure calculations.

As described in this subsection, significant sources of uncertainty in human health risk assessment include using default average values for dominant exposure parameters to represent the population without considering the natural variability in a studied population. As a result, default average values provide inadequate information about variability surrounding the risk from exposure.

Variability can be addressed using a stochastic model to improve analysis by reducing data uncertainty. Moreover, reducing and characterizing population variability can assist in focusing the analysis on segments of the population at higher risk from environmental exposure. Future research is needed to address variability issues by developing a new stochastic analysis-based risk characterization to better describe the risk posed to human health and the surrounding ecosystem.

2.5 Conclusions

This chapter presents a novel and validated methodology for quantifying human health risks from exposure to MSATs emitted by on-road sources. It addresses key gaps in existing literature by integrating emissions estimation, air dispersion and deposition modeling, multi-pathway exposure assessment, and risk quantification to comprehensively assess MSAT-related risks. This approach overcomes critical limitations, including mismatches in roadway geometry between models, reliance on average emissions for health impact estimates, and the exclusion of non-inhalation pathways.

Validation against monitored air concentrations at the Saint Paul–Ramsey Health Center showed reasonable agreement, supporting the methodology’s reliability. Risk results indicate that benzo(a)pyrene may pose elevated cancer risks, especially under prolonged exposure scenarios. For the Farmer Adult scenario, the total benzo(a)pyrene cancer risk reached $4.95\text{E-}05$ (49.5 in 1 million) at Saint Paul–Ramsey Health Center and $2.02\text{E-}05$ (20.2 in 1 million) at the Anderson Office Building—both exceeding the $1.0\text{E-}05$ target cancer risk level.

On-road mobile sources pose significant health risks due to their proximity to human receptors. Individuals near high-traffic corridors face elevated exposures, as demonstrated by risk levels exceeding established thresholds. Non-inhalation risks often surpass inhalation risks, amplifying cumulative exposures. Further, exposure estimates may be underestimated due to default assumptions—such as standard body weights, inhalation rates, and dietary intake—that may not reflect real-world variability. These findings highlight the urgent need for refined exposure assessments and mitigation strategies in transportation planning and regulatory policy.

Future research should prioritize stochastic modeling to better capture variability in exposure factors and expand assessments to include ecological health impacts of MSATs. Exploring risk drivers such as endocrine disruption and DNA methylation may reveal long-term health consequences and underrecognized exposure scenarios. The deterministic framework presented here serves as a foundation for future probabilistic modeling efforts aimed at improving MSAT exposure assessments.

Chapter 3. Extending Multi-Pathway Human Health Risk Assessment from Regional to Country-Wide—A Case Study on Kuwait

Chapter 2 established a deterministic risk assessment for MSATs, focusing on mobile sources and localized exposure. However, stationary sources such as industrial facilities, oil and gas operations, and wastewater treatment plants also contribute significantly to cumulative health risks. Chapter 3 expands the scope by implementing a country-wide cumulative risk assessment, identifying dominant chemical risk drivers and key exposure pathways at sensitive receptors. Using Kuwait as a case study, this research extends EPA methodologies, originally developed for hazardous waste combustion facilities, to industrial, wastewater, and oil and gas operations sources. Applying risk assessment to a region seldom studied enhances global understanding of air toxics health risk variability across different regions (e.g., coastal versus inland) while supporting long-term goals of targeted risk reduction and mitigation resource allocation.

3.1 Introduction

Kuwait, a country in the Middle East, has been selected as the country of study, rather than, for example, the United States, due to the unfragmented nature of the data and the smaller size of the country. Kuwait is situated in the northeastern region of the Arabian Peninsula, bordered by Saudi Arabia to the south and Iraq to the north. It spans an area of approximately 17,818 square kilometers, slightly smaller than New Jersey in land area, and is located at the geographical coordinates of 29.3759° N, 47.9774° E [91].

Kuwait is a country that heavily relies on its abundant oil and gas resources, which have led to the development of various industries such as petrochemical production, refining, power generation, crude steel production using electric arc furnaces, and desalination using natural gas and heavy oil.

As a result of various oil, gas, and industrial activities, numerous pollutants are emitted from different emission source types. These include point sources, such as stacks associated with compressors, boilers, turbines, incinerators, flares, and exhaust vents related to glycol dehydration processes.

In addition, area and volume sources represent other categories of emission sources. Area sources encompass fugitive emissions characterized by non-buoyant releases with negligible vertical extent, such as equipment leaks or landfill emissions. In contrast, volume sources involve non-point emissions with an initial vertical extent, including prescribed burns and marine vessel emissions.

Additionally, on-road and non-road mobile sources contribute to pollutant emissions. On-road mobile sources comprise motorcycles, passenger cars, light-duty trucks, buses, and heavy-duty trucks. Non-road mobile sources include agricultural equipment, aircraft jets, forklifts, and construction equipment, such as graders and backhoes.

Due to various industrial activities, oil and gas operations, traffic congestion, and frequent dust storms in Kuwait throughout the year [92, 93], these factors lead to poor ambient air quality. Recent research indicates a decline in air quality across both urban and rural areas of Kuwait [94, 95, 96], which has subsequently been associated with increased mortality and morbidity rates [97, 98, 99].

Kuwait has three distinct air quality zones (AQZs): coastal, inland, and production, which are further divided into eleven subzones [100]. Each air quality zone and subzone presents a unique combination of emission source types, pollutants, and exposure scenarios that must be considered. The proposed approach employs the validated U.S. Environmental Protection Agency (EPA) Human Health Risk Assessment Protocol (HHRAP) risk assessment methodology [37] established in the US and Canada.

3.1.1 Literature Review

Exposure to hazardous air pollutants (HAPs), also known as air toxics, is associated with various health impacts [3], leading to short-term and long-term health problems. The specific health effects experienced by the population depend on the type, concentration, and mixture of air toxics, as well as the duration and frequency of exposure. For instance, respiratory issues may arise from exposure to methyl mercaptan, which can aggravate respiratory conditions [101], resulting in increased coughing, wheezing, and shortness of breath. Moreover, cardiovascular diseases, including atherosclerosis, have been linked to exposure to polycyclic aromatic hydrocarbons (PAHs) such as benzo(a)pyrene [9, 102, 103, 104].

Neurological disorders may also be associated with exposure to air toxics, as pollutants like mercury and lead can cause cognitive impairments and memory loss [105], and in severe cases, conditions such as convulsions and coma [106, 107, 108, 109]. Additionally, reproductive issues, including fertility problems [110], hormonal imbalances [111, 112], and congenital disorders [113], have been associated with exposure to certain air toxics, such as dioxins and some PAHs.

Lastly, long-term exposure to HAPs, such as benzene, formaldehyde, and 1,3-butadiene, can increase the risk of developing several types of cancer, including lung cancer [114, 115, 116, 117, 118, 8] and lymphohematopoietic cancers [119]. It is important to note that vulnerable populations, including children, the elderly, and those with pre-existing health conditions, may be more susceptible to the adverse effects of air toxics. The HAPs summarized in Table 3-1 represent only a small fraction of air toxics released into the ambient environment from various industrial processes. These chemicals have been selected for their potency and the extensive availability of both cancer and noncancer assessments in the literature.

Table 3-1: Human health effects of selected hazardous air pollutants

HAPs	Source Type	Exposure Route	Target Organ/System	Ref.
Methyl mercaptan	Point, area, volume	Inhalation	Respiratory	[50]
PAHs	Point, area, volume, on-road, and non-road mobile sources	Inhalation; oral	Cardiovascular	[9, 102, 103, 104]
Mercury	Point, area, volume	Inhalation; oral	Central nervous and peripheral nervous systems	[105]
Lead	Point, area, volume	Inhalation; oral	Central nervous and peripheral nervous systems	[106, 107, 108]
Dioxins	Point, area, volume, on-road, and non-road mobile sources	Oral	Reproductive	[110, 111, 112, 113]
Benzene	Point, area, volume, on-road, and non-road mobile sources	Inhalation; oral	Hematological and respiratory	[114, 115, 116, 117, 118]
Formaldehyde	Point, area, volume, on-road, and non-road mobile sources	Inhalation; oral	Hematological	[8]
1,3-Butadiene	Point, area, volume, on-road, and non-road mobile sources	Inhalation	Hematological and immune	[119]

To date, no country-wide studies have addressed the cumulative human health risks associated with exposure to hazardous air pollutants. Furthermore, no studies have yet considered multiple exposure (i.e., direct inhalation and scenario-relevant indirect pathways) in combination with numerous different sources (e.g., oil and gas operations; wastewater treatment plants; etc.). See Table 3-2 for a summary of the types of human health risk assessments that have and have not been conducted.

Table 3-2: Summary of human health risk assessments in literature

Multi-Source	Multi-Pathway	Cumulative	Ref.
Yes	No	No	[120] 2020; [121] 2015; [122] 2012
No	Yes	No	[123] 2022; [124] 2019; [125] 2009
Yes	No	Yes	[126] 2022; [127] 2019; [27] 2015
No	Yes	Yes	[3] 2023; [128] 2014; [129] 2010; [130] 2006
Yes	Yes	Yes	This Thesis

3.1.2 Objectives

The specific objectives of this research, using Kuwait as a case study, are as follows:

1. To implement a country-wide cumulative human health risk assessment incorporating multi-source and multi-pathway exposures.
2. To compare country-wide human health risk variability by region, for example, coastal versus inland.
3. To identify the chemical risk driver (defined here as the dominant chemical of potential concern) for a particular sensitive receptor.
4. To identify if the direct or indirect pathway is the dominant pathway of risk for a particular sensitive receptor.

Though beyond the scope of this chapter, these objectives support the long-term goals of targeted risk reduction and targeted mitigation resource allocations.

3.1.3 Additional Contributions

1. This research extends and adapts EPA methodologies initially designed for hazardous waste combustion facilities to additional emission sources, including wastewater treatment plants and glycol dehydration units, but excludes fugitive emissions and mobile sources.
2. Additionally, this research provides a case study for a region of the world that has seldom been subjected to comprehensive human health risk assessments.

Figure 3-1 presents the methodology framework used in this study.

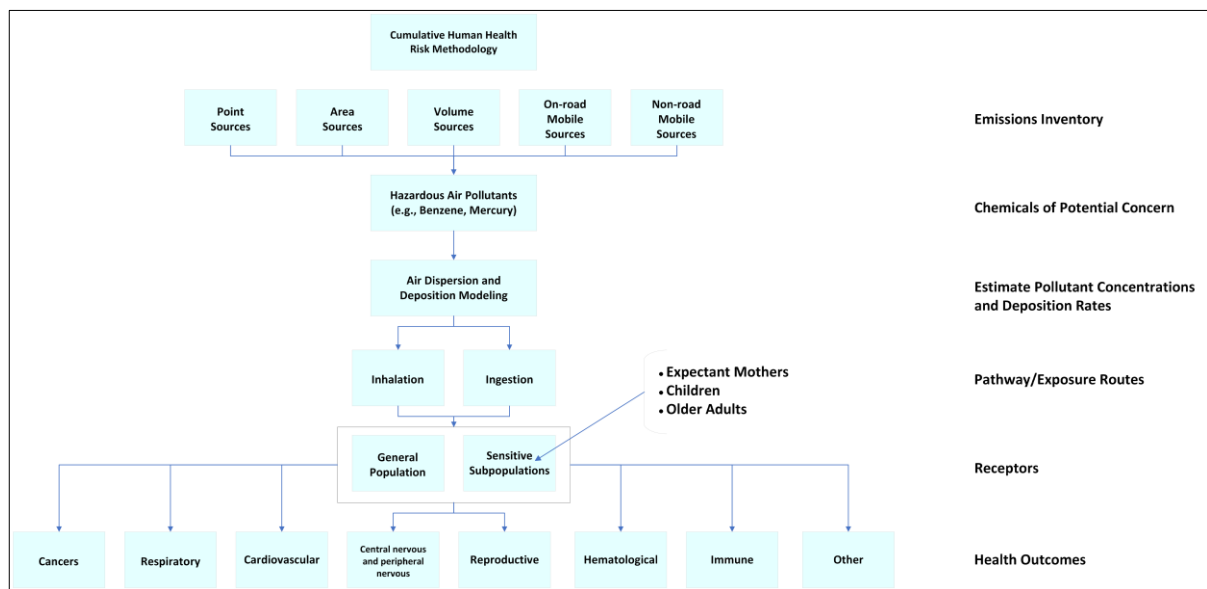


Figure 3-1: Flow diagram illustrating the application of a multi-pathway health risk assessment methodology in Kuwait's air quality zones

3.2 Materials and Methods

The methodology consists of the following sequential steps:

1. Develop an emissions inventory (or augment an existing inventory) for each air quality zone.
2. Conduct air dispersion modeling using current US regulatory air dispersion models, such as AERMOD or similar, to estimate pollutant concentrations in the air and their deposition rates in various exposure media.
3. Estimate the concentrations of pollutants at the point-of-contact for receptor populations by conducting an environmental transport-and-fate analysis.
4. Identify realistic exposure scenarios to estimate the types and magnitudes of human exposure to pollutants.
5. Assess the levels, frequencies, and durations of contact between humans and pollutants.
6. Calculate multi-pathway and cumulative cancer risks and noncancer hazards for each air quality zone (risk characterization).
7. Examine the contributing factors and underlying drivers of unacceptable risks (risk drivers).

3.2.1 Emissions Inventory

An emissions inventory plays a vital role in managing air quality by enabling decision-makers to:

1. Measure the contribution of each sector's emissions.
2. Analyze emission trends both retrospectively and prospectively.
3. Provide guidance and assistance to policymakers and industry in regulating emissions and establishing achievable targets.
4. Supply the necessary data for accurate assessment of current or future emissions using air quality modeling tools, with an accurate emissions inventory being a prerequisite for air quality modeling.
5. Identify locations for monitoring hazardous air pollutant hotspots.

In this study, emissions were calculated using the methods and algorithms summarized in Table 3-3 below.

Table 3-3: Overview of EPA and industry methods/algorithms for estimating emissions from various sources

Method/Algorithm	Objective	Ref.
EPA ¹ AP-42 emission factors	Estimate emissions from various source categories, including point sources (e.g., industrial stacks)	[131]
AP-42, Fifth Edition, Volume I, Chapter 7: Liquid Storage Tanks	Calculate volatile organic compound (VOC) and hazardous air pollutant (HAP) emissions from floating- and fixed-roof storage tanks	[131]
EPA WATER9	Estimate emissions from wastewater treatment plants	[132]
EPA LandGEM	Calculate emissions from landfills, utilizing the methane generation rate and potential methane generation capacity parameters	[133]
GRI-GLYCalc	Estimate emissions from glycol dehydration units	[134]
API ² AMINECalc	Estimate emissions from amine gas treatment plants	[135]

¹ U.S. Environmental Protection Agency. ² American Petroleum Institute.

Table 3-3 lists methods chosen for their applicability, accuracy in emissions estimations, and broad acceptance within the scientific, regulatory, and industrial community. These validated methods were developed by reputable organizations such as the U.S. Environmental Protection Agency (EPA) and American Petroleum Institute (API), based on research, empirical data, and engineering principles.

For each air quality zone, unique identification codes were assigned to each emission unit and to each process based on the fuel type consumed. These codes facilitated the tracking of results and thorough examination of emissions from individual units and processes across facilities and sectors. This coding procedure was instrumental in the study's quality assurance process, enabling the review

of assumptions, calculations, procedures, and the evaluation of the quality and representativeness of the inventory data [136].

It is important to note that emissions calculated using AP-42 emission factors were based on normal, steady-state conditions and did not take into account worst-case scenario conditions, such as during process start-up and shut-down, as well as process upsets. While these abnormal operating conditions have a relatively short time span, resulting emissions can easily exceed relevant emission limits and potentially cause ground-level concentrations that exceed relevant ambient air quality standards (e.g., National Ambient Air Quality Standards).

According to Obaid et al. [137], various industry sectors, including power and/or heat generation, nuclear power generation, petrochemical production, energy-from-waste generation, and sulfuric acid production, exhibit different characteristics for start-up and shut-down emissions compared to steady-state conditions, and these characteristics differ for different source types and pollutants. The same authors note that attempts to characterize emissions during these conditions face numerous challenges, including the unavailability of manufacturers' design data and the complex relationship between different dynamic process functions and timing of events.

In addition to these challenges, other uncertainties associated with the calculation of criteria pollutants and HAPs such as benzene, toluene, ethylbenzene, and xylene (BTEX) emissions include data gaps, data quality, and inconsistencies across different power plants and sometimes even different emission units within the same power plant.

The general formula for estimating emissions is [131]:

$$Emissions = A \times EF \times \left(1 - \frac{ER}{100}\right) \quad (3-1)$$

where A represents the activity rate, EF is the emission factor, and ER is the total percentage (%) of emission reduction efficiency.

It is important to note that the availability and completeness of an emissions inventory are critical factors in evaluating the risks to human health associated with exposure to hazardous air pollutants.

3.2.2 Atmospheric Dispersion and Deposition Modeling

Air dispersion modeling is defined as the mathematical representation of pollutant transport in the atmosphere, along with the quantification of deposition rates in various exposure media [3]. Air dispersion modeling is conducted to characterize the transport, magnitude, and spatial distribution of pollutant concentrations in the atmosphere. It is also used to compare modeled and monitored air concentrations to evaluate the air dispersion model performance and representativeness of the emissions inventory. Moreover, air dispersion and deposition modeling is a prerequisite for conducting a multi-pathway human health risk assessment, as such modeling is used to estimate pollutant concentrations in the air, which are crucial for calculating inhalation cancer risks, and to estimate deposition rates, which are critical for assessing indirect exposure pathways.

In this study, air dispersion and deposition modeling was conducted using the current EPA-preferred regulatory model, American Meteorological Society/United States Environmental Protection Agency Regulatory Model (AERMOD) [59], employing version 22112.

AERMOD, an extensively validated steady-state Gaussian plume model, integrates air dispersion principles based on planetary boundary layer turbulence structure and scaling concepts. It addresses surface and elevated emission sources and accommodates simple and complex terrain. Since 2005, the EPA has approved AERMOD as its regulatory model, and it is widely used in various applications, including permitting, air quality assessments, and human health risk evaluations [59].

Designed as a near-field model for distances up to 50 km from the source, AERMOD exhibits robust performance across diverse terrains and under a wide array of meteorological conditions. AERMOD calculates concentrations using mathematical principles, such as plume rise, atmospheric dispersion of buoyant (or neutrally buoyant) effluent, turbulence theory, and Gaussian distribution [138].

The AERMOD modeling system does not utilize stability classes, which were used in the previous Industrial Source Complex Short Term 3 (ISCST3) model but employs a more advanced turbulence scaling parameterization scheme. The model predominantly relies on surface weather observations to generate vertical profiles of variables, including temperature, temperature gradient, wind speed, wind direction, and turbulent velocities within the atmospheric boundary layer. The EPA's AERMET model is the meteorological preprocessor responsible for producing AERMOD meteorological data files.

While validation of the AERMOD model is a desirable aspect of air dispersion modeling to ensure result credibility for a specific study area, such an evaluation was not conducted due to the absence of available measured air pollutant concentrations.

3.2.2.1 Unitized Emission Approach

The unitized emission approach in air dispersion modeling is a highly efficient method for evaluating the impact of multiple pollutants emitted from a single source [139]. This technique relies on the principle that air concentration levels and deposition fluxes of pollutants are linearly related to the source's emission rate, meaning that a tenfold increase in emissions would result in a tenfold increase in receptor concentrations. By employing a standardized emission rate of 1 g per second (g/s) for the source, this approach streamlines the modeling process and significantly reduces computation time.

Using the unitized emission rate, a single air dispersion modeling run can provide concentration and deposition data for all emitted pollutants. This is achieved by multiplying the unit emission results by the actual emission rate of each individual pollutant. Consequently, the need for running the model separately for each pollutant is eliminated, enhancing the method's efficiency.

The output of this approach includes unitized annual average and maximum 1-hour average air concentrations and deposition rates for the vapor, particle, and particle-bound phases. These unitized values can be further used as inputs for risk modeling, wherein media-specific concentrations and deposition of each contaminant are calculated by scaling the unitized concentrations by the appropriate pollutant emission rate.

The unitized emission approach is particularly beneficial for modeling a vast number of pollutants, as it minimizes calculation imprecision due to low emission values and simplifies the process by avoiding multiple runs for each modeled pollutant.

3.2.2.2 Meteorological Data

The Weather Research and Forecasting (WRF) model [3, 61] was used to generate two AERMOD meteorological data files (surface and profile files) for each of the three air quality zones in Kuwait (i.e., coastal, inland, and production). To ensure adequate coverage across the country, three pseudo-meteorological stations (pseudo-stations) were strategically positioned. Pseudo-stations define the locations for which WRF data are generated, with meteorological parameters that are representative of the selected location.

3.2.2.3 Air Dispersion Model General Options

The modeling options and settings used in AERMOD for the analysis are provided in Table 3-4.

Table 3-4: AERMOD model options and settings used in the modeling analysis

Model Option	Setting
AERMOD Executable	Version 22112
Dispersion Options	Non-default regulatory option selected
	Fast all sources (FASTALL)
	Flat (FLAT)
Calculation Type	Unitized (unit emission rate concept representing the $\mu\text{g}/\text{m}^3$ impact per 1 g/s of emissions; refer to 3.2.2.1)
Output	Concentration; total, dry, and wet deposition
Dispersion Coefficient	Rural
Pollutants	Benzene, formaldehyde, toluene, and benzo(a)pyrene
Averaging Periods	1-hour and annual
Source Types	Point
Receptors	Uniform Cartesian grid and discrete Cartesian receptors (sensitive receptors)
Terrain	Terrain in Kuwait can be approximated as flat; however, terrain files were included for completeness.
Meteorological Data Files	2017 hourly meteorological data, contained in surface and profile files, for Kuwait's three air quality zones, generated using the WRF model

3.2.2.4 Receptors (Calculation Points)

Receptors are specific locations with designated coordinates where pollutant concentrations are calculated. These receptors can represent human populations, ecosystems, or other areas sensitive to air pollution. AERMOD uses meteorological data, emission source parameterizations, and terrain datasets, such as the National Aeronautics and Space Administration (NASA) Shuttle Radar Topography Mission (SRTM) digital files, to predict the dispersion and concentration of pollutants at these receptors, thereby helping to evaluate air quality and assess the potential impact on health or the environment.

In each of the three air quality zones, two uniform Cartesian receptor grids were established centered around the facility of interest. These grids were configured with both fine (100 m spaced grid from the centroid of the emission sources out to a radius of 3 km) and coarse (500 m spaced grid extending from 3 km to 10 km) settings to cover all areas where emissions may have a significant impact. The selection of receptor locations typically depends on regulatory requirements and the specific objectives of the air quality assessment, as different projects may have varying goals and criteria for receptor placement.

Additionally, sensitive receptors, defined as locations where people are more susceptible to adverse effects from exposure to hazardous air pollutants (such as schools, hospitals, residential areas, daycare facilities, care facilities, and places of worship), were included in the project. Further information about these sensitive receptors can be found in the (“Case Study”) section of this chapter.

3.2.3 Transport, Fate, Exposure, and Risk Characterization (TFER)

3.2.3.1 Transport-and-Fate Modeling

This step examines the behavior and movement of HAPs following their release into the atmosphere, which is critical for determining their potential impact on human health and the environment. Transport refers to the physical movement of HAPs as they travel through the environment, while fate pertains to their ultimate destination within environmental media (e.g., soil, food, water). This analysis aims to calculate the concentrations of HAPs in air, soil, produce, milk, meats, eggs, fish, and drinking water.

The focus is on ensuring accuracy in assessing the presence of HAPs in different environmental media, which is vital for understanding their potential impact on human health and the environment. The media concentrations were estimated using over forty mathematical equations [37], including a system of non-linear equations, such as the cumulative soil concentration for carcinogens and non-carcinogens, and the hazardous air pollutants' loss constant due to runoff. The modeling approach accounted for numerous factors, including meteorological conditions and the physicochemical properties of the pollutants. It also considered environmental characteristics of the study area, such as annual evapotranspiration, annual irrigation, annual precipitation, and annual runoff.

It is important to acknowledge that the modeling process may involve certain assumptions, limitations, or uncertainties that should be considered when interpreting the results. To facilitate transport-and-fate modeling, the computer application IRAP-h View [68] was used, which provides outputs such as the aboveground exposed produce concentration due to direct deposition [mg/kg].

3.2.3.2 Exposure Quantification

Exposure evaluation is the process of assessing the frequency, duration, and extent of human exposure to air pollutants, focusing on hazardous air pollutants in this study. Exposure can be estimated using one of three approaches [140]: direct measurement, estimation, or exposure reconstruction. The proposed methodology utilizes the estimation approach.

In addition to the exposure scenarios and pathways, it is essential to consider the demographic and lifestyle factors that may influence exposure. These factors include age, gender, occupation, socioeconomic status, dietary habits, and cultural practices. Understanding these factors can help identify susceptible populations and inform targeted risk reduction strategies.

This section describes the equations used for estimating exposure, as well as exposure scenarios and locations and scenario-relevant exposure pathways. It is important to note that separate equations are used for inhalation and ingestion exposure to account for differences in exposure pathways, mechanisms, and potential health effects.

Exposure was estimated using Equations (2) and (3) [140]:

$$Exposure\ (inhalation) = ADD\ (inhalation) = \frac{C_{air} \times InhR \times ET \times EF \times ED}{BW \times AT} \quad (3-2)$$

where ADD represents the average daily dose (mg/kg-day), C_{air} is the concentration of hazardous air pollutant(s) in the air (mg/m³), $InhR$ is the inhalation rate (m³/h), ET is the exposure time (h/day), EF is the exposure frequency (days/year), ED is the exposure duration (years), BW is the body weight (kg), and AT is the averaging time (days).

$$Exposure\ (ingestion) = ADD\ (ingestion) = \frac{C_{medium} \times IngR \times EF \times ED}{BW \times AT} \quad (3-3)$$

where ADD represents the average daily potential dose (mg/kg-day), C_{medium} is the concentration of hazardous air pollutant(s) in the medium (e.g., mg/L, mg/g), $IngR$ is the ingestion rate (e.g., L/day, g/day), EF is the exposure frequency (days/year), ED is the exposure duration (years), BW is the body weight (kg), and AT is the averaging time (days).

Exposure scenarios define the combination of exposure pathways and exposure parameters applied to complete the risk and hazard calculations described in the risk characterization section. For the purposes of this study, exposure scenarios include the resident adult and resident child scenarios [37], which differ in factors such as exposure duration, frequency, and body weight. The included exposure pathways are summarized in Table 3-5.

Table 3-5: Exposure pathways used in multi-pathway exposure assessments

Exposure Pathways
Direct inhalation of vapors and particles
Incidental ingestion of soil
Ingestion of drinking water from treated surface water sources
Ingestion of homegrown produce

Exposure scenario locations refer to the physical places (i.e., receptors) within the study area chosen for evaluating residential adult and child exposure scenarios. To ensure thorough evaluation of all potential residential exposure locations throughout Kuwait, air dispersion modeling results were generated using two uniform Cartesian receptor grids and sensitive receptors. For more information on receptor grids and sensitive receptors, refer to Section 3.2.2.4. This approach is consistent with established best practices in air quality assessment [37].

Figure 3-2 illustrates the relationship between human exposure to HAPs through multiple pathways. HAPs exist in various environmental media, such as air, water, and soil, and can enter the human body via different exposure routes, including ingestion and inhalation. HAPs are deposited into environmental media, such as water and soil, through atmospheric deposition.

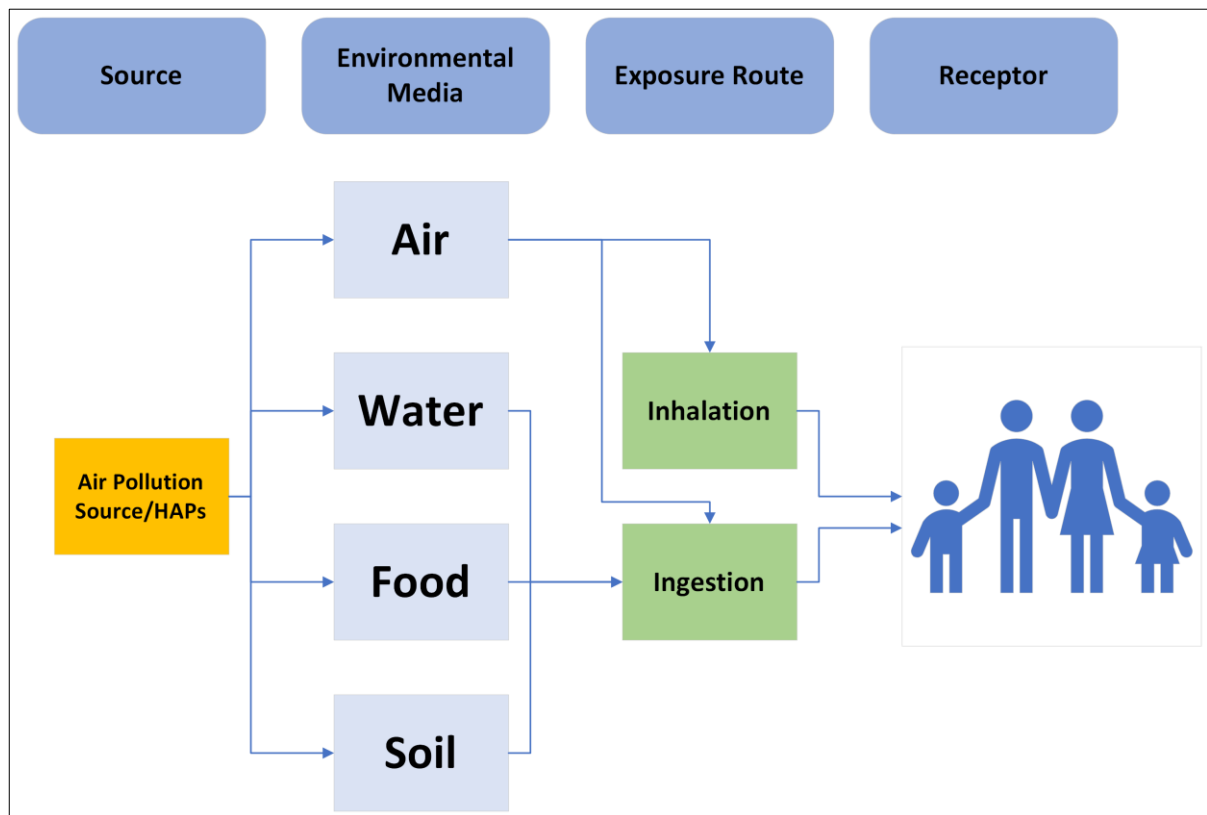


Figure 3-2: A visual representation of the multi-pathway human exposure to hazardous air pollutants

3.2.3.3 Quantitative Estimation of Cancer Risk and Noncancer Hazard

Risk characterization is a crucial component in human health risk assessments. It synthesizes information from preceding assessment steps, such as transport-and-fate modeling (Section 3.2.3.1) and exposure quantification (Section 3.2.3.2), to determine the presence or absence of risk and quantify expected outcomes. Furthermore, risk characterization identifies key factors contributing to risk, enabling regulators, engineers, analysts, and stakeholders to make informed decisions about appropriate actions for risk reduction.

It is essential to recognize the uncertainties and limitations inherent in the risk characterization process, which may stem from factors like incomplete emissions data, variability in exposed populations, and assumptions made during the assessment to account for temporal variability in emissions. Acknowledging these uncertainties provides context for the results and informs future research or decision-making.

Cancer risk is the incremental probability of an individual developing cancer over a lifetime due to exposure to a specific carcinogenic hazardous air pollutant or a mixture of carcinogenic hazardous air pollutants [37]. The predicted risk represents the additional risk of cancer from the exposure being analyzed, which is supplementary to other risks resulting from numerous factors (e.g., smoking). This risk is expressed as a probability ranging from zero (no risk) to one (certainty of developing cancer), without any units or dimensions.

Cancer risk is represented as a numerical value, such as “1 in 100,000” or “ 1×10^{-5} ”, indicating that one individual in a population of 100,000 is expected to develop cancer due to exposure to the carcinogenic hazardous air pollutant over a lifetime. The calculation of cancer risk considers several factors, such as the concentration of the carcinogenic hazardous air pollutant in the environment, exposure duration, frequency, and exposure route (e.g., ingestion or inhalation), as well as the hazardous air pollutant’s cancer potency factor or slope factor.

For inhalation exposures, chronic cancer risks for individual pollutants are estimated by multiplying the long-term exposure concentration estimate by the corresponding pollutant-specific inhalation unit risk. This calculation determines the potential incremental cancer risk, as illustrated in Equation (4):

$$CancerRisk(inhalation) = Inhalation\ Exposure\ Concentration \times Inhalation\ Unit\ Risk \quad (3-4)$$

For each ingestion exposure pathway being evaluated, chronic cancer risks for individual pollutants are estimated by multiplying the lifetime average daily dose estimate for each ingestion exposure pathway by the pollutant-specific cancer slope factor. This calculation estimates potential incremental cancer risk, as illustrated in Equation (5):

$$Cancer\ Risk\ (ingestion) = Lifetime\ Average\ Daily\ Dose \times Cancer\ Slope\ Factor \quad (3-5)$$

Noncancer hazard refers to potential adverse noncancer health effects, such as respiratory issues, neurological complications, and neurotoxicity, resulting from exposure to a hazardous air pollutant or a mixture of hazardous air pollutants. Exposure duration plays a significant role in hazard classification, with two primary categories: acute hazards associated with short-term exposure and chronic hazards linked to long-term exposure.

Evaluating noncancer hazards involves comparing the estimated exposure level of a substance to its reference dose or reference concentration, which represents levels deemed safe without appreciable risk of adverse health effects throughout a lifetime of exposure [141]. The noncancer hazard is calculated using Equation (6):

$$Hazard\ Quotient = \frac{Estimated\ Exposure\ Level}{Reference\ Concentration\ (or\ Reference\ Dose)} \quad (3-6)$$

The hazard index, a conservative measure, is commonly used in human health risk assessments involving multiple hazardous air pollutants or exposure pathways. This index represents the sum of the individual hazard quotients for each hazardous air pollutant or pathway.

In this research, cumulative risk (cancer or noncancer) is defined as the risk resulting from simultaneous exposure to multiple hazardous air pollutants from multiple sources through one or more pathways or exposure routes, such as inhalation or ingestion. The total cumulative cancer risk was calculated using Equation (7):

$$R_{total} = \sum(R_{i,j}) + \sum(R_{i,inhalation}) \quad (3-7)$$

where R_{total} represents the cumulative cancer risk estimate, considering both ingestion and inhalation pathways, $R_{i,j}$ represents the individual risk estimate for hazardous air pollutant i and ingestion pathway j , $R_{i,inhalation}$ represents the individual risk estimate for hazardous air pollutant i through the inhalation pathway, i denotes the different hazardous air pollutants (e.g., hazardous air pollutant 1, hazardous air pollutant 2, hazardous air pollutant 3, etc.), and j denotes the different ingestion pathways (e.g., vegetable ingestion, fish ingestion, egg ingestion, beef ingestion, etc.). On the other hand, the total cumulative noncancer risk (hazard index) was estimated using Equation (8):

$$Hazard\ Index_{total} = \sum(HQ_{i,j}) + \sum(HQ_{i,inhalation}) \quad (3-8)$$

where $Hazard\ Index_{total}$ represents the total hazard index, considering both ingestion and inhalation pathways, $HQ_{i,j}$ represents the hazard quotient for hazardous air pollutant i and ingestion pathway j , $HQ_{i,inhalation}$ represents the hazard quotient for hazardous air pollutant i through the inhalation pathway, i denotes the different hazardous air pollutants (e.g., hazardous air pollutant 1, hazardous air pollutant 2, hazardous air pollutant 3, etc.), and j denotes the different ingestion pathways (e.g., vegetable ingestion, fish ingestion, egg ingestion, beef ingestion, etc.).

Lastly, the acute inhalation hazard quotient was calculated using Equation (9):

$$Acute\ Inhalation\ Hazard\ Quotient = \frac{acute\ air\ concentration}{acute\ inhalation\ exposure\ criteria} \quad (3-9)$$

Regulatory agencies establish target risk levels to assess potential health risks from hazardous air pollutant exposure and ensure public safety. Carcinogenic risk levels typically range from 1×10^{-4} to 1×10^{-6} , indicating a negligible increase in cancer risk due to exposure. For noncancer health effects, a hazard quotient or hazard index of 1 or lower indicates no expected adverse effects, while values above 1 suggest potential risks.

In this study, narrower target risk levels for hazardous air pollutant exposure were adopted based on the U.S. EPA Region 6 HHRAP Addendum [67], to account for conservatism and background

concentration exposure within the study area. Consequently, cancer risk levels should not exceed 1×10^{-5} , and the hazard quotient/index should not exceed 0.25.

It is essential to note that target risk levels are not definitive indicators of adverse effects, as risk estimations inherently have uncertainties. These uncertainties can arise from exposure variability, limited toxicological data, temporal emissions' variability, and assessment assumptions.

If calculated risks are within target values, no further investigation is needed, and the evaluated conditions are not considered to present unacceptable risks. However, when target values are exceeded, the methodology entails further review by risk managers, risk analysts, or engineers. This review process involves examining the scientific basis and uncertainties associated with the calculation, including toxicity factors and uncertainty factors addressing study limitations and data quality issues, as well as investigating the contributing factors and underlying drivers of unacceptable risks.

3.2.3.4 Risk Driver Analysis: Forensic Analysis of Human Health Risks

Risk driver analysis is a systematic, project-specific process that addresses the fundamental question, "Where does the risk come from?". It is important to identify and understand the primary factors contributing to unacceptable cancer and noncancer risks associated with hazardous air pollutant exposure in a specific project setting. This stage involves an examination of various exposure elements, including scenarios, such as farmers, residents, local fish consumers, construction workers, and outdoor workers, as well as pathways like air, water, soil, plants, and animals.

The analysis assesses the significance of specific exposure pathways, such as inhalation, ingestion, and dermal contact, to pinpoint the most critical factors leading to elevated risk levels. A vital aspect of this process is tracing back to the sources or chemicals contributing to the increased risks. Additionally, the analysis investigates the potential for bioaccumulation of hazardous air pollutants in tissues or agricultural produce to enhance understanding of the risks associated with human health in the project area. For instance, methylmercury is a lipophilic substance that tends to bioaccumulate in the aquatic food chain, leading to human exposure primarily through fish consumption [142, 143].

The project-specific risk assessment is re-evaluated upon completing the analysis to ensure accuracy and guide the development of targeted risk reduction strategies. These strategies may include emission controls, exposure reduction measures, or regulatory actions aimed at addressing the identified

risk drivers and protecting public health within the project's scope. Figure 3-3 illustrates a simplified example of a risk driver analysis. As shown in the example, the risk driver analysis employs a systematic approach to investigate the various components of human health risk assessment, identifying the elements contributing to elevated risk and tracing them back to the emission sources or hazardous air pollutants driving the risk.

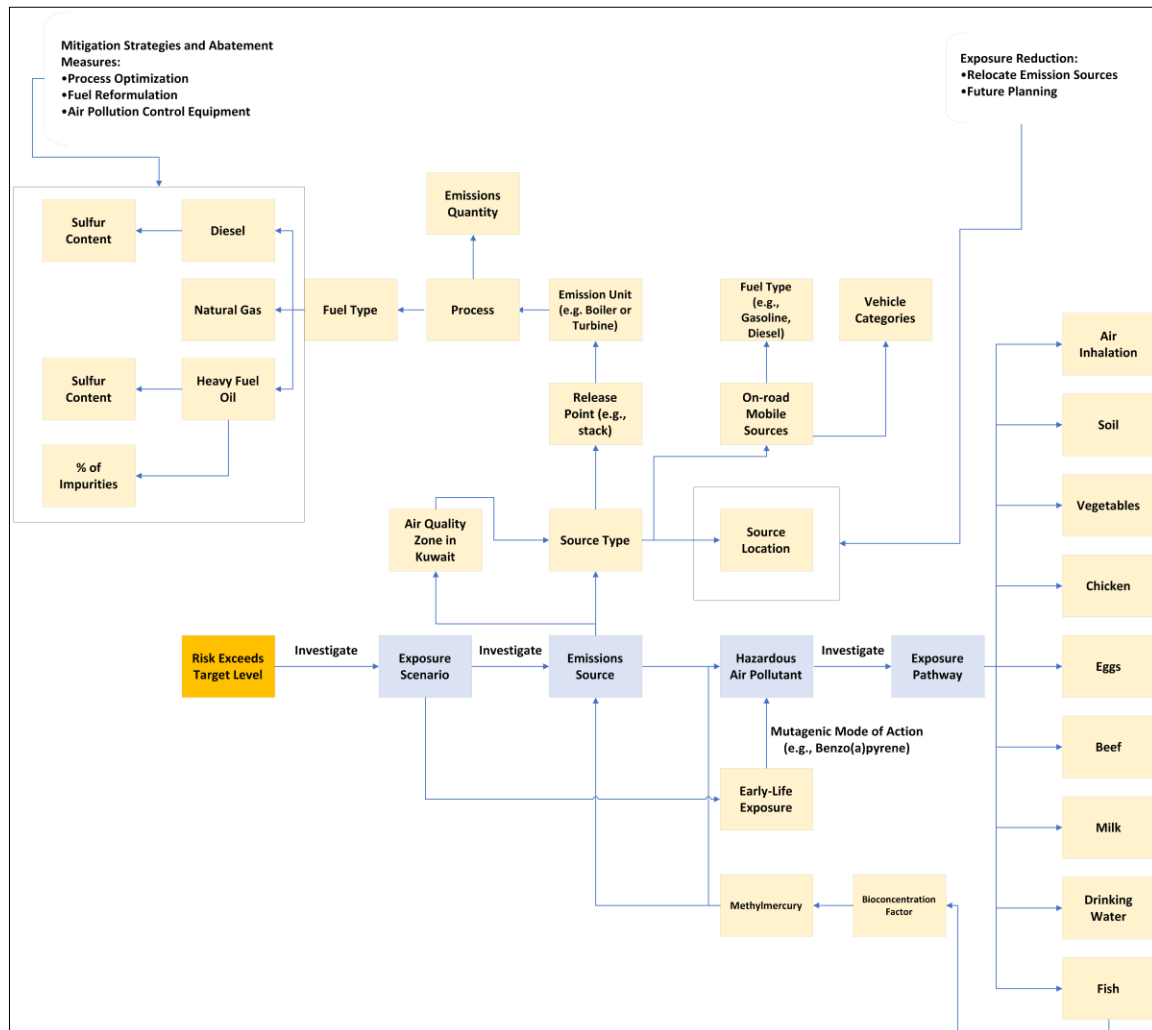


Figure 3-3: Simplified example of a risk driver analysis

This methodology was implemented to evaluate human health risks associated with exposure to hazardous air pollutants in Kuwait. The methodology includes developing emissions inventories for

each air quality zone, conducting air dispersion modeling using AERMOD, estimating pollutant concentrations at the point of contact, identifying exposure scenarios, and assessing the levels and frequencies of contact between humans and pollutants.

Through the process of calculating multi-pathway and cumulative risks, this assessment considered both direct and indirect exposure pathways. This approach allowed for the examination of contributing factors and underlying drivers of unacceptable risks in each air quality zone, ultimately providing a consistent and robust risk characterization upon which risk mitigation strategies can be developed to protect human health in Kuwait.

3.3 Results

3.3.1 Case Study

This section presents the application of the human health risk assessment methodology outlined above to a focused case study in Kuwait, using data from the year 2017. The analysis concentrates on hazardous air pollutants, emphasizing the impact of direct emissions from industrial sources across Kuwait's three air quality zones. Although mobile sources and fugitive emissions are significant contributors to localized hazardous air pollutants [3, 139, 29], they were intentionally excluded from this case study. This approach enables a focused examination of the effects of industrial emissions, providing a clear baseline for the analysis. In addition, industrial sources are regulated, which provides a legal framework upon which emissions reduction actions can be developed to address specific issues identified through the health risk analysis process.

Emission sources, representative of Kuwait's distinctive pollution landscape, were selected to ensure an accurate depiction. The selection process employed a multi-criteria approach, factoring in not only the type of industry, geographical location within air quality zones, and the intensity of emissions but also the unique contribution of each source to Kuwait's pollution landscape. The unique contribution refers to how certain industries in Kuwait are characterized by specialized processes and materials, resulting in distinct emissions profiles and pollution effects that differ from other regions. In addition, the selection criteria were further enriched by considering the proximity to populated areas, meteorological conditions, and land-use characteristics (inland versus shoreline), acknowledging their potential impact on the dispersion and intensity of pollutants. This multi-faceted selection process

ensures that the study remains comprehensive, representative, and aligned with the actual pollution landscape in Kuwait.

The results of the assessments for each air quality zone, to be presented later in this chapter, provide an understanding of the prevailing air quality conditions and associated health risks in Kuwait. This case study effectively underscores the practical applicability and potential impact of the proposed methodology in human health risk assessment. A map of the study region is provided in Figure 3-4 for a clearer geographical context.

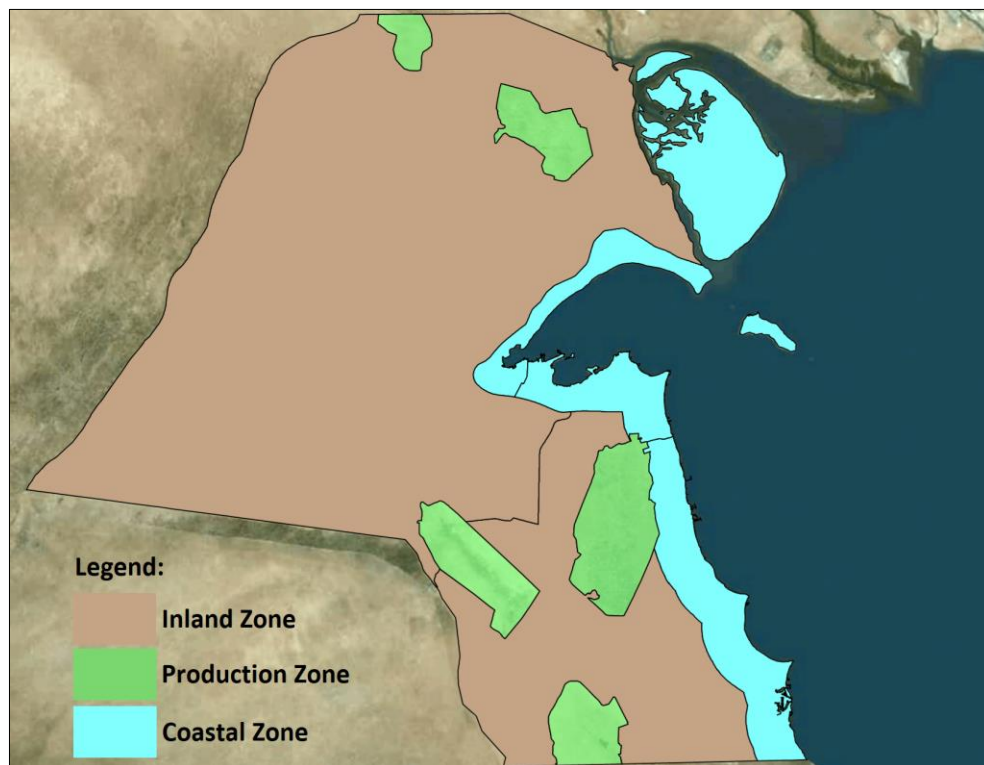


Figure 3-4: Kuwait air quality zones. Map data: Lakes Satellite

In an effort to accurately represent Kuwait's unique pollution landscape, several facilities of interest were selected across the three air quality zones, each characterized by different types of emission sources. Table 3-6 presents a summary of the number of modeled facilities in each air quality zone, along with the number of their corresponding emission sources. Additionally, Table 3-7 lists the hazardous air pollutants that were considered in this study.

Table 3-6: Summary of the number of modeled facilities and corresponding number of emission sources in the air quality zones of Kuwait

Air Quality Zone	Number of Modeled Facilities	Number of Emission Sources within Modeled Facilities
Inland	4	21
Production	7	21
Coastal	10	47

Table 3-7: Hazardous air pollutants considered in this chapter

Hazardous Air Pollutants
Benzene
Formaldehyde
Toluene
Benzo(a)pyrene

For this study, a small, representative subset of hazardous air pollutants was selected based on their known adverse impacts on human health and their common presence in industrial emissions. These pollutants, drawn from a larger emissions inventory, were chosen due to their potency, prevalence in industrial emissions, and the extensive availability of both cancer and noncancer health risk assessments in the literature. Furthermore, a subset of pollutants is all that is required in order to demonstrate that by using a normalized pollutant, the human health impacts of any number of hazardous air pollutants can be easily estimated, meaning only one calculation of the time-consuming air dispersion modeling is required.

By limiting the pollutants analyzed, this chapter does not provide a comprehensive picture of air pollutant health risks in Kuwait; such a comprehensive impact assessment is outside the focus and scope of this chapter. Table 3-1 (refer to Section 3.1.1) details the adverse health effects associated with these selected pollutants.

3.3.2 Cumulative Risk Results

This subsection presents the cumulative health risks from all pathways evaluated within the human health risk assessment study. This includes the analysis of cancer, noncancer (hazard index), and acute noncancer health effects within Kuwait's three distinctive air quality zones: inland, production, and coastal. For a detailed definition of cancer risk and noncancer risk, refer to Section 3.2.3.3 of this chapter. In the tables presented below, numerical data are provided to illustrate the calculated health risks for each receptor, under specific exposure scenarios, in the inland, production, and coastal air quality zones. Each value represents a statistical estimate of the risk posed to a "receptor", which refers to an individual, either an adult or a child, residing in the area. These risk estimates are calculated based on potential exposure to various air pollutants, considering factors such as pollutant concentrations, exposure duration, frequency, and the inherent toxicity of the pollutants.

3.3.2.1 Inland

Table 3-8, Table 3-9 and Table 3-10 provide a summary of the cumulative cancer risk, cumulative noncancer risk, and cumulative acute noncancer risk for the inland air quality zone. The sensitive receptors for this zone were identified based on the grid nodes, which exhibited the maximum air concentration values. This approach ensures the analysis focuses on the areas with the highest potential exposure.

Table 3-8: Cumulative cancer risk (inland)

Sensitive Receptor/Exposure Scenarios	Resident Adult	Resident Child
Receptor 1	4.79×10^{-11}	9.63×10^{-12}
Receptor 2	4.74×10^{-9}	9.55×10^{-10}
Receptor 3	1.02×10^{-8}	2.05×10^{-9}
Receptor 4	3.77×10^{-11}	1.76×10^{-11}

Table 3-9: Cumulative noncancer risk (inland)

Sensitive Receptor/Exposure Scenarios	Resident Adult	Resident Child
Receptor 1	8.83×10^{-7}	8.94×10^{-7}
Receptor 2	9.30×10^{-5}	9.86×10^{-5}
Receptor 3	2.02×10^{-4}	2.16×10^{-4}
Receptor 4	3.45×10^{-8}	8.93×10^{-8}

Table 3-10: Cumulative acute noncancer risk (inland)

Sensitive Receptor/Exposure Scenarios	Resident Adult/Child
Receptor 1	4.47×10^{-5}
Receptor 2	2.21×10^{-4}
Receptor 3	3.00×10^{-4}
Receptor 4	8.61×10^{-10}

The inland zone demonstrates a low health risk profile, with all sensitive receptors displaying risks below the established thresholds, as outlined in Section 3.2.3.3, for cancer, noncancer, and acute noncancer effects. The identified pollutants do not appear to pose significant health hazards for either adults or children within this air quality zone, based on the selected receptors.

3.3.2.2 Production

Table 3-11, Table 3-12 and Table 3-13 provide a summary of the cumulative cancer risk, cumulative noncancer risk, and cumulative acute noncancer risk for the production air quality zone. Consistent with the inland air quality zone, the sensitive receptors for this zone were determined based on the grid nodes with the calculated maximum air concentration values. Though these receptors share the same designations as those in the inland zone, they represent distinct locations within the production air quality zone. This method of selection maintains the focus of the analysis on areas with the highest potential exposure.

Table 3-11: Cumulative cancer risk (production)

Sensitive Receptor/Exposure Scenarios	Resident Adult	Resident Child
Receptor 1	3.11×10^{-11}	6.26×10^{-12}
Receptor 2	2.34×10^{-9}	4.73×10^{-10}
Receptor 3	1.20×10^{-8}	2.41×10^{-9}
Receptor 4	3.83×10^{-11}	1.76×10^{-11}

Table 3-12: Cumulative noncancer risk (production)

Sensitive Receptor/Exposure Scenarios	Resident Adult	Resident Child
Receptor 1	5.76×10^{-7}	5.86×10^{-7}
Receptor 2	4.45×10^{-5}	4.64×10^{-5}
Receptor 3	2.38×10^{-4}	2.55×10^{-4}
Receptor 4	3.39×10^{-8}	8.85×10^{-8}

Table 3-13: Cumulative acute noncancer risk (production)

Sensitive Receptor/Exposure Scenarios	Resident Adult/Child
Receptor 1	2.49×10^{-5}
Receptor 2	2.71×10^{-4}
Receptor 3	2.71×10^{-4}
Receptor 4	8.78×10^{-10}

The study findings for the production zone align closely with those from the inland zone. No significant health risks were identified, as all cumulative risks across different population segments remained below the specified thresholds. This finding indicates that the current levels of selected pollutants may not contribute significantly to health impacts within the production zone.

3.3.2.3 Coastal

Table 3-14, Table 3-15 and Table 3-16 present a summary of the cumulative cancer risk, cumulative noncancer risk, and cumulative acute noncancer risk within the coastal air quality zone. The sensitive receptors identified in this zone have been selected due to their strategic placement within densely populated areas and locations where populations are likely to be more susceptible to adverse effects from exposure to hazardous air pollutants. Such locations include critical community hubs such as hospitals, schools, resorts, and places of worship. The chosen receptors are of particular interest due to the intersecting factors of high potential pollutant concentrations and significant population presence, which jointly contribute to an increased potential for exposure.

Table 3-14: Cumulative cancer risk (coastal)

Sensitive Receptor/Exposure Scenarios	Resident Adult	Resident Child
General Ahmadi Hospital	6.12×10^{-6}	2.31×10^{-6}
Fatima Bint Asad High School for Girls	7.56×10^{-6}	2.87×10^{-6}
Ahmadi Hospital	1.09×10^{-5}	4.33×10^{-6}
Ahmadi Zoo	7.92×10^{-6}	3.04×10^{-6}
Adan Hospital	4.05×10^{-6}	1.52×10^{-6}
Hilton Kuwait Resort	3.63×10^{-6}	1.31×10^{-6}
Mosque (North Ahmadi)	5.79×10^{-6}	2.23×10^{-6}

Table 3-15: Cumulative noncancer risk (coastal)

Sensitive Receptor/Exposure Scenarios	Resident Adult	Resident Child
General Ahmadi Hospital	9.67×10^{-3}	1.56×10^{-2}
Fatima Bint Asad High School for Girls	1.19×10^{-2}	1.93×10^{-2}
Ahmadi Hospital	1.26×10^{-2}	2.43×10^{-2}
Ahmadi Zoo	1.13×10^{-2}	1.92×10^{-2}
Adan Hospital	6.02×10^{-3}	9.92×10^{-3}
Hilton Kuwait Resort	6.84×10^{-3}	1.00×10^{-2}
Mosque (North Ahmadi)	7.76×10^{-3}	1.36×10^{-2}

Table 3-16: Cumulative acute noncancer risk (coastal)

Sensitive Receptor/Exposure Scenarios	Resident Adult/Child
General Ahmadi Hospital	1.19×10^{-1}
Fatima Bint Asad High School for Girls	1.16×10^{-1}
Ahmadi Hospital	9.69×10^{-2}
Ahmadi Zoo	1.05×10^{-1}
Adan Hospital	6.81×10^{-2}
Hilton Kuwait Resort	9.65×10^{-2}
Mosque (North Ahmadi)	8.29×10^{-2}

The cumulative health risks in the coastal air quality zone are within acceptable limits; however, they are notably higher when compared to the findings for the inland and production zones. For cancer risks, only the Ahmadi Hospital, highlighted in boldface in Table 3-14, with a cancer risk of 1.09×10^{-5} for the resident adult exposure scenario, slightly exceeds the specified threshold of 1×10^{-5} .

To provide a practical understanding of the cancer risk value above, consider it in terms of population-scale risk. If 100,000 adults were exposed to the same level of carcinogenic hazardous air pollutants—such as benzene, formaldehyde, toluene, and benzo(a)pyrene—from multiple emission sources over a lifetime, approximately 1.09 individuals in this group could be expected to develop cancer due to this exposure. However, this is a statistical estimate and does not guarantee that any individual in this group will indeed develop cancer from this exposure.

To further contextualize, the cancer risk at the Ahmadi Hospital sensitive receptor can be roughly compared to the risk of dying from an encounter with a hornet, wasp, or bee sting, which is 1 in 54,516 [144]. This comparison serves to convey that, while non-zero, the additional risk posed by exposure to these pollutants is relatively manageable when viewed alongside everyday risks. However, it should be remembered that these risk assessments rely on models and certain assumptions, including constant, long-term exposure. Actual risk may vary depending on factors such as genetic predispositions, lifestyle choices, and actual exposure patterns.

For the noncancer and acute noncancer risks, all sensitive receptors displayed values below the defined thresholds of 0.25. This suggests that, despite higher pollutant levels in the coastal zone, the risk remains below thresholds of significant health concern. However, the risk is more pronounced in this zone compared to the inland and production zones.

3.4 Discussion

In this section, key risk drivers are analyzed, including specific pollutants, their sources, and the main exposure pathways. Subsequently, the strengths and limitations of the methodology are considered. Finally, the section concludes with an examination of the variability and uncertainty associated with the analysis.

3.4.1 Risk Driver Analysis

The risk driver analysis conducted in this assessment identified a singular case of cumulative cancer risk exceeding the target risk levels in the coastal air quality zone. This elevated risk was specifically noted at Ahmadi Hospital for the resident adult exposure scenario. A systematic examination of exposure scenarios, emission sources, and exposure pathways indicated that the primary risk driver was benzo(a)pyrene. Moreover, the analysis suggested that the risk is primarily associated with indirect exposure pathways.

This finding implies that the origin of this pollutant may be localized emissions within the coastal zone, potentially originating from industrial activities or combustion processes. Specific source information cannot be disclosed due to confidentiality restrictions. However, it is worth noting that the likely culprits include industrial flares and other combustion sources. Consequently, the insights provided by the applied methodology serve as a key tool for policymakers and regulators. These findings can guide the consideration and implementation of targeted risk reduction strategies, such as emission control measures at identified sources or various mitigation strategies. The ultimate goal of these strategies, informed by this research, would be to uphold public health standards within the project's scope while reducing the cancer risk to acceptable levels.

3.4.2 Strengths and Limitations

In this section, the strengths and limitations of the research methodology employed in this case study are examined. A key strength is the integration of previously validated methods to quantify the country-wide cumulative multi-pathway human health impacts associated with exposure to hazardous air pollutants in Kuwait. While these methods have been previously validated, the present research combines them in a unique manner to address the specific challenges of assessing cumulative, multi-pathway risks in a region with complex industrial activities.

Additionally, the proposed methodology is flexible in incorporating more sophisticated multi-layer, multi-species air dispersion models, such as non-steady-state puff models like CALPUFF or SCIPUFF. The advantage of non-steady-state puff models over steady-state plume models lies in their capacity to capture the temporal and spatial variability in meteorological conditions, leading to a more accurate representation of pollutant dispersion patterns, resulting in more reliable exposure and risk estimates.

However, certain limitations of the approach must also be acknowledged. First, building downwash effects were not evaluated due to a lack of available parameterization. Second, the study did not account for long-range transport of pollutants from areas outside of Kuwait.

3.4.3 Variability and Uncertainty

Variability and uncertainty in human health risk assessments originate from biological differences, external environmental factors, and the presence of gaps in both data availability and scientific knowledge. Variability refers to the differences or range of responses to environmental exposure among

individuals or populations and can be attributed to factors such as genetics, age, lifestyle, and pre-existing health conditions. Environmental factors, such as the duration and route of exposure, as well as spatial–temporal variations in hazardous air pollutant concentrations, further contribute to this variability.

On the other hand, uncertainty refers to the lack or incompleteness of data regarding the input parameters for the methods employed to evaluate human health impacts. This form of uncertainty is inherent at every stage of the assessment process, from limitations in data availability to the extrapolation of results from animal studies to humans. Enhancing the reliability of risk assessments requires effectively addressing both variability and uncertainty. This can be accomplished by employing tools such as stochastic modeling and sensitivity analysis and by emphasizing the necessity for continuous research, monitoring, and data collection to reduce uncertainty over time.

3.5 Conclusions

This chapter presents an innovative approach to quantifying country-wide cumulative risk (cancer, noncancer, and acute) from simultaneous exposure to multiple hazardous air pollutants originating from various sources through one or more exposure pathways, such as direct inhalation and indirect ingestion. This chapter also introduces a method for identifying dominant chemical and pathway risk drivers, providing insight into health risks and a foundation for targeted mitigation. The conclusions drawn from this chapter are as follows:

1. The overall health risk profile across the inland, production, and coastal air quality zones of Kuwait is low to moderate, as most risk values lie beneath the established risk thresholds. This suggests that the current levels of pollutants quantified in this case study do not likely pose significant health threats to the adult and child residential population.
2. The coastal air quality zone has a higher risk profile compared to the inland and production zones, particularly for cancer risks. However, these values are mostly within acceptable limits. An exception is the Ahmadi Hospital for the resident adult exposure scenario, where the cancer risk slightly exceeds the target level.
3. The risk driver analysis identified benzo(a)pyrene as the primary risk driver contributing to the elevated cancer risk at the Ahmadi Hospital in the coastal zone, calculated to be 1.09×10^{-5} , with the likely sources being local industrial emissions or combustion processes. However, the

resident adult exposure scenario assumes continuous, lifetime exposure at the hospital, which may overstate actual risk.

4. This study acknowledges inherent variability and uncertainty in the risk estimates, emphasizing the importance of careful interpretation and the need for further research such as the application of stochastic modeling.

In summary, this research improves the understanding of potential health impacts from exposure to hazardous air pollutants in this region, providing valuable insights for future environmental management strategies and policy decisions. For example, the ability to forecast health impacts can support regulatory permitting, public education, emergency response, and healthcare planning that accounts for air quality's effect on public health in Kuwait.

3.6 Future Work

The methodology presented in this chapter lays the foundation for future work involving stochastic modeling, as further developed in Chapter 5. A probabilistic approach will incorporate distributions for key variables, such as body weight, inhalation and ingestion rates, and metabolic activity, to better capture real-world variability and uncertainty in human health risk estimates.

Additionally, the fate and transport modeling component (Step 3) may be extended to support ecological risk screening at a national scale. This includes estimating ecological screening quotients and evaluating indirect pathways through which ecological degradation may impact human health—such as through disruptions to food chains, water quality, or biodiversity.

These future directions aim to integrate ecological and human health perspectives to support more holistic environmental decision-making.

Chapter 4. Country-Wide Ecological Health Assessment Methodology for Air Toxics: Bridging Gaps in Ecosystem Impact Understanding and Policy Foundations

Chapter 4 transitions from human health to ecological impacts by introducing the Ecological Health Assessment Methodology (EHAM), developed to assess air toxics risk to terrestrial and aquatic ecosystems. Building on the transport and fate modeling framework, this chapter evaluates bioaccumulation and trophic transfer of hazardous air pollutants across environmental media. Applying EHAM at the country-wide scale addresses critical gaps in ecosystem risk understanding, enabling integration of ecological integrity into air quality policy and extending U.S. EPA's methodology beyond hazardous waste facilities to diverse emission sources.

4.1 Introduction

Previous research investigated the adverse effects of air toxics on human health [18]. During that work, a noticeable gap was identified in the broader literature concerning the ecological impact of air toxics on terrestrial and aquatic ecosystems.

That study [18] also recommended a country-wide ecological risk assessment to fill existing gaps. As a consequence, this chapter introduces the Ecological Health Assessment Methodology (EHAM). EHAM is based on the United States Environmental Protection Agency's (US EPA's) Screening Level Ecological Risk Assessment Protocol (SLERAP) for Hazardous Waste Combustion Facilities [145], expanding its application to a wider range of emission sources. This chapter focuses on estimating ecological risks from industrial sources, and EHAM could include other significant emission sources, such as fugitive emissions or mobile sources if such data are available.

The general procedure established by EHAM offers a flexible framework that can easily be adapted to integrate these additional sources, thereby broadening its applicability.

Air toxics emissions can be transported over vast areas, and food webs often span multiple ecological zones. Therefore, improving understanding of the sources contributing to cumulative ecological (and health [146]) risk requires applying EHAM over a broad area that captures the sources of interest.

Furthermore, by applying EHAM over a vast area, more informed policy decisions can be made, such as determining where best to situate a new industrial emission source or air quality monitoring station. Ideally, the vast area would be worldwide, but this is impractical for computation, data availability, and political reasons, with data availability being the major technical barrier.

In North America, even applying EHAM state- or province-wide remains difficult due to a lack of emissions data. To emphasize the desirability of a wide-scale application, this research applied EHAM at the country-wide scale. Kuwait was selected, as it was the only country for which the necessary, sufficiently large-scale (>10%), country-wide industrial air toxics emissions data were available.

Due to data confidentiality restrictions, specific information identifying the sources cannot be disclosed. This is not a problem for this chapter, which focuses on presenting the method to implement EHAM; however, using EHAM for policy decisions may have to confront source confidentiality considerations. Kuwait has two major ecosystems that are particularly vulnerable to air toxics: desert and coastal ecosystems.

In the present study, air modeling and deposition simulations originally conducted for human health risk assessments were adapted to estimate ecological risks associated with air toxics.

While the case study concentrates on Kuwait's unique desert and coastal ecosystems, the methodologies implemented and the insights gained hold broader implications.

The fate and transport of air toxics within ecological settings exhibit characteristics similar to those impacts observed in human health assessments, as extensively detailed in [147]. This similarity highlights the interconnectedness of human and ecological health, broadening the overall understanding of the implications of air pollution.

Ecosystems, fundamental in maintaining global biodiversity and health, play distinct roles. Wetlands, often referred to as the “kidneys of the Earth” [148], not only purify water but, like other ecosystems, also suffer from infiltration by air toxics.

Similarly, grasslands and forests maintain soil fertility, with their root systems acting as defenders against erosion [149, 150]. Although it is common to think of wetlands, grasslands, and forests when speaking of ecosystems, and to think of deserts as barren, desert ecosystems such as those that dominate Kuwait do play a crucial environmental role as carbon sequestration sinks due to their vast size [151].

Furthermore, Kuwait has coastal ecosystems, characterized by their unique interface between land and sea, that are vital for biodiversity conservation and that provide crucial services like storm protection and nursery grounds for many marine species [152].

Coastal ecosystems are particularly vulnerable to air toxics that can accumulate in the coastal food web, impacting both terrestrial and marine life. For example, airborne mercury, primarily from industrial sources, can be deposited in water bodies, converting to methylmercury in aquatic systems.

This toxic form then bioaccumulates in fish, ultimately impacting birds and mammals that rely on these fish for food. Furthermore, the influence of air toxics on one ecosystem can have ripple effects, altering nutrient cycles and energy flow in adjacent ecosystems.

This intricate web of interactions necessitates a comprehensive, multi-ecosystem-based approach to manage and mitigate the effects of air pollution.

A summary of the entry mechanisms and affected trophic levels of air toxics in both terrestrial and aquatic ecosystems is provided in Table 4-1.

Table 4-1: Comparison of entry mechanisms and affected trophic levels for air toxics in terrestrial and aquatic ecosystems

	Terrestrial Ecosystems (Ref.)		Aquatic Ecosystems (Ref.)	
Entry Mechanisms	Dry deposition	[153, 154]	Rainfall	[164, 165, 166]
	Wet deposition	[153, 154]	Tidal activities	[167, 168, 169]
	Direct inhalation	[155, 156]	Direct uptake from water	[170, 171, 172]
	Plant–soil interactions	[157, 158]	Watershed loading (surface runoff, point source discharge, atmospheric deposition, and riverine input)	[173, 174, 175]
Affected Trophic Levels	Herbivores	[159, 160]	Benthic organisms	[176, 177, 178]
	Omnivores	[159, 160]	Intermediate consumers	[179, 180, 181]
	Predators (via bioaccumulation)	[159, 160]	Larger aquatic predators (via bioaccumulation)	[159, 160, 167]
Inter-ecosystem Interactions	Watershed loading affects adjacent aquatic ecosystems	[161, 162, 163]	Watershed loading affects adjacent terrestrial ecosystems	[161, 162, 163]

To complement and clarify the data in Table 4-1, Figure 4-1 shows the multi-dimensional ecological ramifications of air toxics across both terrestrial and aquatic ecosystems.

Figure 4-1 highlights the impacts of air toxics on diverse aquatic organisms, from benthic invertebrates and vegetation to higher-order predators like fish and amphibians. Similarly, in terrestrial ecosystems, the figure demonstrates how soil-deposited pollutants can have cascading effects, influencing a range of organisms from vegetation to herbivores like deer and rabbits, as well as predators such as owls and red foxes.

Table 4-1 and Figure 4-1 provide an easily interpretable overview of the ecological risks associated with air toxics. These elements give readers both the data and the conceptual framework needed to understand the broader implications.

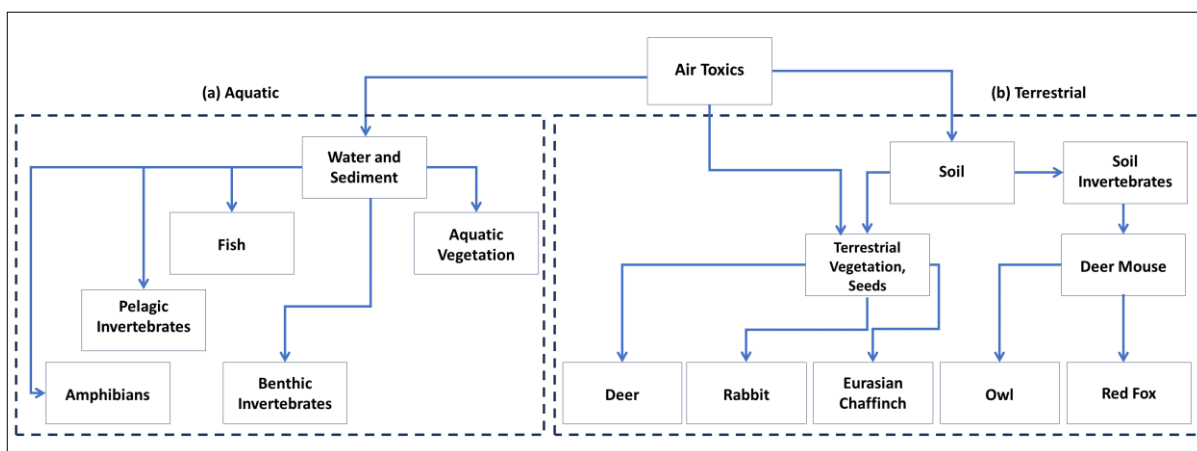


Figure 4-1: Example of transport pathways and fate of air toxics into (a) aquatic and (b) terrestrial ecosystems

In summary, EHAM as described here provides a structured and adaptable framework for assessing the ecological risks posed by air toxics at a country-wide level. Although this study employs the methodology within the specific environmental context of Kuwait, the principles and approaches are adaptable to other ecosystems. This adaptability enhances the methodology's generalizability, offering a valuable tool in the global endeavor to mitigate ecological harm from air toxics.

4.1.1 Literature Review, Objectives, and Additional Contributions

This section provides a literature review focused on the effects of air toxics on ecological health and highlights that past ecological risk assessments have primarily concentrated on chemicals other than air toxics. Additionally, this section outlines the research objectives and the contributions of the current study.

Examples of significant impacts from air toxics on select organisms are given in Table 4-2. Note that these impacts are all determined by exposing an organism to varying concentrations of a toxic and may be used to generate toxicity reference values (TRVs) used by EHAM, as discussed in Section 4.2.8. That is, these impacts are not ecological risk determinations, but rather organism risk determinations. The varied examples in Table 4-2 also show the diverse impacts that air toxics can have on ecological receptors and help to contextualize the broader ecological implications of air pollution.

Table 4-2: Examples of documented health impacts of air toxics on selected organisms

Ecological Health Impact	Ref.
Amphibian (Common frog): Increased embryonic mortality rate	[182]
Bird (Pied flycatchers): Habitat degradation; impaired reproductive success	[183, 184]
Fish (African catfish): Decreased sperm motility	[185]
Fish (Common carp): Increased lipid peroxidation; DNA damage; elevated micronuclei frequency in blood, gill, and liver	[186]
Fish (European perch): Decline in sperm performance; sperm structural damage (flagella, plasma membrane, axoneme); decreased ATP content; damage to sperm midpiece and mitochondria; disrupted activation due to membrane damage	[187]
Fish (Lake trout, largemouth bass, northern pike, walleye): Behavioral alterations; changes in gene expression; impacts on growth	[188]
Fish (Norwegian waters): Induction of phase-I enzymes; DNA adduct formation; neoplastic lesions	[189]
Fish (Peacock blenny): Circulatory issues in gills; liver damage (reduced cell size, cellular integrity loss, structural disruption)	[190]

Continued next page

Ecological Health Impact	Ref.
Fish (Trahira): Electrophysiological changes in retinal horizontal cells	[191]
Fish (Zebrafish): Changes in lipid metabolism and cellular transport; gonadal damage; oxidative stress in testes; altered sex hormones affecting reproduction	[192, 193]
Mammal (Mink): Reproductive failure	[194]
Mesozooplankton: Accumulation of air toxics in tissues, fecal pellets, and eggs; transfer to higher trophic levels	[195]
Phytoplankton: Disrupted growth and photosynthesis; oxidative stress induction	[196, 197]

In addition to the direct impacts mentioned earlier, bioaccumulation (gradual accumulation of air toxics within organisms over time) and biomagnification (increase in concentration of air toxics as they move through different trophic levels) contribute to the amplification of ecological impacts. Notably, phytoplankton and mesozooplankton, which form the base of the food chain, play a critical role in transferring the effects of air toxics upward within the food web. This cascading effect ultimately influences aquatic and terrestrial apex predators. The widespread consequences of air toxic contamination in complex ecosystems thus become evident.

While the primary focus of this research investigates the impacts of air toxics on ecosystems, it is also important to acknowledge that these effects can indirectly impact wildlife through the consumption of contaminated plants, fish, or meats. This pattern of contamination and exposure follows a trajectory similar to human exposure to air toxics' fate and transport, as extensively detailed in prior work [3]. The intake of such contaminated food escalates the risks of exposure for animals, emphasizing the necessity of robust air quality standards in controlling toxic emissions and mitigating ecosystem contamination.

Table 4-3 reveals how the current work contributes to the literature on the effects of toxics on ecosystems, and although the list of literature in Table 4-3 is not all-inclusive, it does reflect the scarcity of studies into the impact of air toxics on ecosystems. The distinguishing features used to characterize the literature presented in Table 4-3 follow the Source, Pathway, and Receiver model, which parallels the workflow of EHAM. The Source, Pathway, and Receiver model [198] is a framework used to understand and assess pollution's impacts. It traces the journey of an air toxic from its origin ("Source"), through the routes it takes in the environment ("Pathway"), to the entities that may be affected or harmed ("Receiver"). This model offers a perspective on how air toxics interact with and impact ecosystems, providing insights for risk assessments.

Table 4-3: Examples of past ecological risk assessments, including the present work, with source, pathway, and receiver characterization

#	Literature (Year)	Source	Pathway	Receiver	Ref.
1	A Procedure for Performing Population-Level Ecological Risk Assessments (2000)	Land/ water	Land to local ecosystems	Quail, shrew, fish	[199]
2	Potential Long-Term Ecological Impacts Caused by Disturbance of Contaminated Sediments: A Case Study (2002)	Water	Release from disturbed sediments to water column	Mummichog, blue crab, polychaete, striped bass	[200]
3	An Ecological Risk Assessment for Spinosad Use on Cotton (2002)	Land	Terrestrial insecticide application to cotton crops	Groundwater, mourning dove, field sparrow, blue tit, bees, bluegill, rainbow trout, carp, grass shrimp, eastern oyster, freshwater diatom, Sharkey soil	[201]
4	Ecological Risk Assessment of Contaminated Soils Through Direct Toxicity Assessment (2005)	Land	Leaching of contaminants from soil	Plants, earthworm, Daphnia, algae, fish	[202]

#	Literature (Year)	Source	Pathway	Receiver	Ref.
5	Probabilistic Ecological Risk Assessment of 1,2,4-Trichlorobenzene at a Former Industrial Contaminated Site (2005)	Land	Leaching from soil to groundwater	Aquatic invertebrates, microbial soil population, plants	[203]
6	A Screening-Level Assessment of Lead, Cadmium, and Zinc in Fish and Crayfish from Northeastern Oklahoma, USA (2006)	Water	Runoff from land to water bodies	Humans, carp, catfish, bass, crappie, carnivorous wildlife, crayfish	[204]
7	Anthropogenic Input of Selected Heavy Metals in the Aquatic Sediments of Hochiminh City, Vietnam (2006)	Water	Industry and agricultural runoff	Aquatic sediments in rivers and canals	[205]
8	Potential Importance of Inhalation Exposures for Wildlife Using Screening-Level Ecological Risk Assessment (2006)	Air	Airborne contaminants inhaled by wildlife	Small mammals	[206]
9	Environmental Risk Assessment of Pharmaceutical Residues in Wastewater Effluents, Surface Waters, and Sediments (2006)	Water	Wastewater and surface water contamination	Aquatic organisms	[207]
10	Bioconcentration of Dioxins and Furans in Vegetation (2007)	Land	Soil-to-vegetation contamination	Vegetation including grass and clover	[208]

#	Literature (Year)	Source	Pathway	Receiver	Ref.
11	Predicting Pesticide Environmental Risk in Intensive Agricultural Areas. II: Screening Level Risk Assessment of Complex Mixtures in Surface Waters (2009)	Land	Pesticide drift and runoff	Algae, Daphnia, fish	[209]
12	Evidence of impacts from DDT in pelican, cormorant, stork, and egret eggs from KwaZulu-Natal, South Africa (2019)	Land	Aerial transport and aquatic ecosystems	African openbill, pelican, molluscan, piscivorous birds, pink-backed pelican, egrets, white-breasted cormorant	[210]
13	DSS-OSM: An Integrated Decision Support System for Offshore Oil Spill Management (2021)	Water	Spread of oil in marine environments	Marine organisms	[211]
14	This Thesis	Air	Atmospheric dispersion and deposition of air toxics	Terrestrial and aquatic species across various trophic levels	

Most prior ecological risk assessments have focused on chemicals other than air toxics, such as pesticides. Regulatory frameworks like the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA) [212] have propelled research chiefly toward pesticides. Similarly, federal laws such as the American Clean Water Act [213] and the Endangered Species Act [214] have mainly directed attention toward waterborne toxics. In Europe, the European Food Safety Authority (EFSA) has established risk assessment frameworks for human health, animal health, and ecological areas [215] that are similar to the EPA protocols. To date, the EFSA frameworks have primarily been applied to plant protection products, i.e., pesticides, insecticides, and fungicides [216]. These assessments, as substantiated by the literature reviewed in Table 4-3, are invaluable in their targeted scope but frequently limited in geographic extent and seldom include air toxics. This narrow approach calls for a more comprehensive

evaluation that considers air toxics and encompasses both terrestrial and aquatic ecosystems on a country-wide scale.

In addition to air toxics, persistent organic pollutants (POPs) are noteworthy due to their environmental persistence and potential for bioaccumulation [217]. While POPs are not the primary focus of this study, their prospective integration into the EHAM presents a significant research avenue.

The primary objective of this work is twofold: first, to introduce the Ecological Health Assessment Methodology, a novel and comprehensive approach for ecological risk assessment of air toxics that transcends traditional methods; and second, while EHAM is adept for country-wide evaluations, its design also facilitates detailed ecological risk analyses across various regions within a country, allowing for comparative assessments at multiple geographical scales.

In addition to the primary objectives, this study offers several contributions that enhance the scope and applicability of the EHAM framework:

1. Developing unique food web models tailored to local ecosystems to support country-wide ecological risk assessment. These models incorporate region-specific trophic interactions, allowing for more accurate and localized evaluations.
2. Extending US EPA methodologies originally designed for hazardous waste combustion facilities to include other emission sources, such as wastewater treatment plants and glycol dehydration units.
3. Estimating cumulative country-wide ecological risk from simultaneous exposure to multiple air toxics, including polycyclic aromatic hydrocarbons originating from diverse industrial sources.

4.2 Materials and Methods

In alignment with the stated objectives, this section outlines the methodology devised to fill the identified gaps in prior ecological risk assessments. The methodology was validated using accepted emissions estimation methods and algorithms developed by reputable organizations, such as the US EPA and the American Petroleum Institute (API) [18]. Furthermore, air dispersion modeling was performed using AERMOD, a steady-state Gaussian plume model, which is the most extensively validated model for the present purpose and is currently the US EPA's preferred regulatory model [59].

The Ecological Health Assessment Methodology (EHAM) is outlined in the following sequential steps:

1. Develop a country-wide emissions inventory by identifying and quantifying the sources of air toxics;
2. Perform air dispersion and deposition modeling using current US regulatory models. This step aims to estimate the atmospheric pollutant concentrations and their annual deposition rates, focusing on habitat-specific scenario locations within the defined project area (e.g., country);
3. Characterize exposure settings for affected habitats within the project area;
4. Develop habitat-specific food webs based on exposure data and receptor interactions;
5. Select assessment endpoints within each trophic level of the habitat-specific food web;
6. Identify measurement receptors to assess air-toxics-related measures of effect, such as toxicity values or receptor-specific chronic no-observed-adverse-effect levels (NOAELs);
7. Assess exposure via direct uptake and ingestion, targeting both lower- and higher-trophic-level receptors based on their interaction with air toxics;
8. Evaluate the toxicity of air toxics by identifying toxicity reference values;
9. Estimate the country-wide cumulative ecological risk from simultaneous exposure to numerous air toxics from multiple industrial sources.

4.2.1 Emissions Inventory

In the first step of the methodology, a country-wide emissions inventory is developed to identify and quantify the sources of air toxics. For this study, Kuwait served as the project area and was divided into three distinct air quality zones.

In areas with varying wind patterns and urban pollution sources, further considerations may be needed, such as subdividing the area based on similar air quality, meteorological conditions, and pollution source types.

It is important to acknowledge the uncertainties in emission estimates, which include variability in emission factors, inaccuracies in activity data, and challenges posed by temporal and spatial

resolution. Additionally, differences in emissions during start-up and shut-down phases compared to steady-state conditions must be considered.

This step establishes the base data layer for ecological risk assessment. The completeness and availability of the emissions inventory are essential for accurately evaluating the ecological risks associated with exposure to air toxics.

The existing emissions inventory, while beyond the scope of this chapter, can be expanded to include on-road vehicle emissions. Detailed estimation methods are available in Chapter 2.

For additional details on the methods and algorithms employed in emissions estimation, readers are referred to Section 3.2.1 of Chapter 3 [18].

4.2.2 Air Dispersion and Deposition Modeling

The air dispersion and deposition modeling for this study was performed using AERMOD, which is the current regulatory model preferred by the US EPA, employing its version 22112 [59]. Further details can be found in the study presented in [18], specifically in Section 3.2.2.

4.2.3 Exposure Setting Characterization

In this step, the focus is on assessing the potential cumulative ecological impact due to the release of air toxics from multiple facilities throughout the country. This involves two primary processes: the identification of ecological receptors and the selection of habitats.

For the selection of habitats, environments potentially affected by facility emissions across the diverse landscapes of the country are identified by correlating them with the air dispersion modeling results. These results consist of the unitized output of concentration and deposition rates. Habitats are characterized by their biotic and abiotic elements and typically fall into two main categories: terrestrial and aquatic. Tools like land use land cover (LULC) maps, topographic maps, and aerial photographs are used to delineate these habitats accurately.

Within the exposure setting characterization, terrestrial habitats across the country are chosen based on air parameter values from AERMOD. The process starts with defining the habitats to be evaluated and then identifying specific receptor grid nodes with the highest yearly average air concentration and deposition values. The proximity of several water bodies, which influence receptor intake values in exposure equations, is also considered.

For aquatic habitats, the distinction between a range of water bodies and non-submerged areas across the country is crucial. The selection process mirrors terrestrial methods but includes an additional consideration of air toxics loading from watersheds. For example, an oil and gas facility located upstream might release air toxics such as PAHs, which are particle-bound, like benzo(a)pyrene. When deposited, these air toxics could travel through the watershed and accumulate in downstream aquatic habitats. This downstream location, even if distant from the emission source, might experience increased levels of these air toxics due to runoff and water flow, thereby emphasizing the need to evaluate not just direct air deposition, but also the indirect introduction of air toxics through the watershed.

The selection process involves defining the aquatic habitats and pinpointing receptor grid nodes, particularly those with the highest annual average concentrations. Special attention is given to large water bodies, focusing closely on the most impacted areas and employing additional or nearest receptor grid nodes when needed.

The process of identifying ecological receptors ensures a representation of both plant and animal communities within a habitat. Ecological receptors are selected based on their known or projected sensitivities to air toxics, their ecological importance as keystone or indicator species, and their direct relevance to human health (e.g., species consumed by local populations).

This identification underscores all potential exposure pathways, including the food chain. The food web specific to potentially impacted habitats is explicitly defined to ensure that complete exposure pathways for both plant and animal communities are ascertained.

This research employed distinct techniques depending on the habitat's nature, whether terrestrial or aquatic. For instance, analysis of aquatic habitats might involve the use of hydrological models to estimate concentrations of air toxics in different water layers, while that of terrestrial habitats may utilize soil sampling and bioaccumulation models to gauge the levels of air toxics in the soil and its subsequent impact on plant and animal life. Analysis of aquatic habitats delves deeper into water quality metrics, whereas that of terrestrial habitats emphasizes soil integrity, vegetation cover, and air-toxic sensitivities. This methodology encompasses defining boundaries using the air dispersion model's receptor grid nodes, evaluating the highest concentrations of air toxics, and understanding the impact on water bodies and watersheds across the country.

4.2.4 Habitat-Specific Food Web Development

Using data from the exposure setting characterization, habitat-specific food webs are constructed to represent guilds and communities of receptors exposed to air toxics emissions from various facilities throughout the country.

In this context, a guild refers to a group of species that function at a specific trophic level and exploit a common resource base in a similar way [218]. To organize the structure of these food webs, trophic levels are employed. The first trophic level (TL1) includes primary producers like plants. The second trophic level (TL2) consists of primary consumers, such as herbivores and detritivores. Higher trophic levels include omnivores and carnivores, classified as the third trophic level (TL3) and fourth trophic level (TL4), respectively. TL3 and TL4 organisms typically consume a variety of food sources, including those from the lower trophic levels.

Exposure pathways in these habitat-specific food webs are categorized into two primary types: direct and indirect exposure pathways. In this study, a direct exposure pathway refers to the immediate transfer of air toxics to an ecological receptor through avenues such as respiration or direct contact with contaminated media (e.g., soil, water). Conversely, an indirect exposure pathway involves a more complex transfer mechanism, typically through the food web itself. For example, air toxics may be absorbed by primary producers (TL1) and subsequently bioaccumulate up the trophic levels, affecting primary consumers (TL2), omnivores, and carnivores (TL3 and TL4). The criteria for categorizing these pathways are based on the number of intermediary steps between the release of air toxics and their contact with the ecological receptor, as well as the mechanisms of transfer (e.g., ingestion, respiration, dermal absorption).

Food webs show how energy, originating from primary sources like plants, is transferred through different organisms. The effectiveness of these webs in revealing the exposure pathways hinges on factors such as the dietary habits of organisms, the specific entities within the chain, and aspects like the availability and breakdown of the assessed air toxics. These habitat-specific food webs are important in highlighting both direct and indirect exposure pathways. Furthermore, they facilitate the establishment of mathematical relationships between guilds (e.g., quantification of the daily dose of air toxics ingested by a measurement receptor) and provide a detailed exposure analysis for different ecological receptors.

Utilizing the community approach, a prevalent framework in the field [145], the following steps are integral to food web development and embody the core elements of this approach:

1. Within a habitat, potential receptors are identified and grouped based on their feeding habits or guilds;
2. The food web structure is organized by trophic levels, such as plants and primary consumers (herbivores);
3. An important component involves delineating the feeding relationships among distinct guilds and communities.

The completion of these steps, according to the community approach, results in a thorough food web for each assessed habitat, designed for utilization in subsequent risk characterization.

4.2.5 Habitat-Specific Trophic Level Assessment Endpoint Selection

An assessment endpoint serves as an explicit representation of an ecological attribute requiring protection [219], particularly in the context of food web structure and ecosystem function.

These assessment endpoints are identified on a granular level, specific to each community and guild within individual trophic layers of habitat-specific food webs. Guild-specific examples include the roles of seed dispersers and decomposers, while community-specific examples focus on attributes like species richness and productivity.

The careful selection of these endpoints aims to ensure the overall ecological integrity of both guilds and communities, thereby safeguarding the structure and function of the habitat-specific food webs they comprise.

4.2.6 Measurement Receptor Identification for Measures of Effect Assessment

Identifying measurement receptors is critical when evaluating the ecological risk associated with air toxics emitted from multiple facilities.

Criteria for selection are based on factors such as ecological relevance—for instance, a population of phytoplankton might serve as a foundational element in aquatic food webs; exposure potential, as exemplified by soil invertebrates with higher metabolic rates selected for their elevated ingestion rates relative to body weight; and sensitivity to these toxics, like certain benthic invertebrates that may be sensitive to heavy metals.

These criteria serve to identify receptors that are most representative of the assessment endpoints for each community and class-specific guild. Consequently, the identified receptors serve as the key factors based upon which measures of effect assessments are conducted, utilizing toxicity benchmarks such as ambient water quality criteria and receptor-specific no-observed-adverse-effect levels.

For example, receptors for soil communities may include soil invertebrates and terrestrial plant communities. For surface water communities, receptors for surface water may extend to water invertebrates, phytoplankton, zooplankton, and fish communities.

4.2.7 Trophic-Level-Specific Exposure Assessment for Air Toxics

In trophic-level-specific exposure assessment for air toxics, the evaluation of exposure and toxicity varies depending on the ecological receptor's position in the food chain. For lower-trophic-level organisms, exposure is assessed through direct uptake pathways from environmental media like soil, water, and sediment. In contrast, higher-trophic-level organisms are evaluated based on the ingestion of different organisms along with media contaminated with air toxics. These approaches, tailored for lower and higher trophic levels, respectively, make use of TRVs, which are either media-specific for lower trophic levels or dose-specific for higher trophic levels. The TRVs used in this assessment are derived from the peer-reviewed literature and validated databases, ensuring a rigorous and reliable basis for risk characterization.

This section describes the equations used for assessing exposure including:

4.2.7.1 Daily Dose (DD) of Air Toxics Ingested by a Measurement Receptor

The calculation of this dose is key for assessing health risks across various trophic levels and can be determined using the following generic equation [145]:

$$DD = \sum IR_F \times C_i \times P_i \times F_i + (IR_M \times C_M \times P_M) \quad (4-10)$$

where DD represents the dose of air toxics ingested (mg air toxics/kg body weight-day), IR_F is the measurement receptor animal or plant food item ingestion rate (kg/kg body weight-day), C_i is the air toxics concentration in the i th animal or plant item (mg air toxics/kg), P_i is the proportion of the i th food item that is contaminated (unitless), F_i is the fraction of the diet consisting of animal or plant item i (unitless), IR_M is the measurement receptor media ingestion rate (kg/kg body weight-day (bed sediment or soil) or L/kg body weight-day (water)), C_M is the air toxics concentration in the medium (mg/kg (bed sediment or soil) or mg/L (water)), and P_M is the proportion of ingested media that is contaminated (unitless).

The calculation of this dose is adapted from methodologies developed by the US EPA, which are extended in this study to incorporate additional emission sources.

In calculating the daily dose within class-specific guilds, two distinct dietary scenarios are considered: Equal Diet and Exclusive Diet. In the Equal Diet, it is assumed that the measurement receptor consumes food items from different groups in equal proportions. The fraction of the diet consisting of each animal or plant item F_i is thus equal to one divided by the total number of food item groups ingested. In contrast, the Exclusive Diet proposes that the measurement receptor's diet is entirely composed of a single food item group at a time. Here, F_i is set to one for the food item group, while F_i values for all other food items are set to zero. This scenario is iteratively applied to each food item group, generating a range of potential exposure values based on exclusive diets.

4.2.7.2 Bioconcentration Factor (BCF)

The concentrations of air toxics in phytoplankton, invertebrates, and rooted aquatic plants can be calculated from the steady-state bioconcentration factor (BCF), thereby enabling the evaluation of the dose ingested by the measurement receptor.

The BCF is defined as the ratio of the concentration of air toxics in a biological food item to their concentration in the environmental media and can be calculated using Equation (2) [145]:

$$BCF = \frac{C_i}{C_M} \quad (4-11)$$

where BCF is the bioconcentration factor ((sediment, soil), or L/kg (water)) (unitless), C_i is the air toxics concentration in i th animal or plant item (mg of air toxics/kg), and C_m is the air toxics concentration in media (mg/kg (sediment, soil), or mg/L (water)).

4.2.7.3 Food-Chain Multiplier (FCM)

A multiplier is applied to the bioconcentration factor (BCF) of air toxics to estimate the bioaccumulation factor (BAF), thereby accounting for the chemical's accumulation up the food chain through predator-prey interactions.

Air toxics with the greatest potential to biomagnify (defined here as the increase in concentration of air toxics as they move upward through the different trophic levels) are highly lipophilic, exhibit low water solubility, and are resistant to metabolic breakdown [220].

The octanol–water partition coefficient serves as an important metric for understanding the bioaccumulative tendencies of air toxics, providing valuable insights into their transport, fate, and potential ecotoxicological impacts. The bioaccumulation factor can be calculated using Equations (3) and (4) [145]:

$$FCM = \frac{BAF_l}{K_{ow}} \quad (4-12)$$

$$FCM = \frac{BAF_l}{BCF_l} \quad (4-13)$$

where FCM is the food-chain multiplier, BAF_l , lipid-normalized, represents the concentration of air toxics in aquatic organisms per unit volume of free chemical in the water, measured in units of L/kg, K_{ow} is the octanol–water partition coefficient (L/kg), and BCF_l , lipid-normalized, quantifies the ratio of the air toxics concentration in aquatic organisms to their freely available concentration in the water, expressed in L/kg.

The FCM is used to estimate biomagnification and is calculated as the ratio of the FCM for the measurement receptor to that of its predator.

Table 4-4 provides a summary of key references where ecological risk assessors or researchers can find essential parameters for exposure assessment.

Table 4-4: Summary of key references for parameters used in exposure assessment

Parameter	Source	Remarks
Ingestion rate for measurement receptors	U.S. EPA Wildlife Exposure Factors Handbook [221]	Data on ingestion rates across species.
Food-chain multiplier	Great Lakes Water Quality Initiative Technical Support Document for the Procedure to Determine Bioaccumulation Factors [222]	Provides standardized FCM values.
Bioaccumulation Factor Model	Gobas Model [223]	Models for calculating BAF.

4.2.8 Toxicity Assessment for Air Toxics

The toxicity of air toxics is assessed through the identification of TRVs that are specific both to the air toxics in question and to the measurement receptor under examination. The toxicity database used for these TRVs is the Ecotoxicology (ECOTOX) knowledgebase [224], which contains information on over 12,837 chemicals and 13,895 species. A TRV serves as a benchmark, specifically indicating a concentration or dose of air toxics that is not expected to cause observed adverse effects on ecologically relevant endpoints during chronic (long-term) exposure [145].

In ecological risk characterization, these values, tailored for lower-trophic-level communities, are specific to the media with which these organisms interact. For upper-trophic-level, class-specific guilds, the benchmarks are provided in terms of the dose ingested.

To illustrate the application of TRVs, consider the case of a mallard duck exposed to polycyclic aromatic hydrocarbons [225]. According to laboratory data, benchmarks indicating increased embryonic mortality were found at concentrations of 0.2 and 2.0 mg/kg egg. These concentrations serve as thresholds beyond which an increased risk of adverse effects on mallard duck embryos can be anticipated. This metric thus becomes crucial for evaluating the potential ecological risk posed by polycyclic aromatic hydrocarbons in avian communities.

TRVs serve as indispensable tools for systematically assessing and managing the potential risks that air toxics pose to both lower- and upper-trophic-level organisms, thereby contributing to the goal of ecological risk mitigation.

4.2.9 Ecological Risk Characterization

Ecological risk characterization encompasses both risk estimation and risk description. The quantification of risk estimation employs the quotient method, which yields an Ecological Screening Quotient (ESQ).

This ESQ is calculated as a ratio wherein the estimated exposure level (EEL) of air toxics is divided by the TRV that is specific to both the air toxics and the measurement receptor. The formula is expressed as:

$$ESQ = \frac{EEL}{TRV} \quad (4-14)$$

where *ESQ* is the ecological screening quotient (unitless), *EEL* is the estimated exposure level to air toxics (mass air toxics/mass media (communities) or mass daily dose air toxics ingested/mass body weight-day (class-specific guilds)), and *TRV* is the air toxics' toxicity reference value (mass air toxics/mass media (communities) or mass daily dose air toxics ingested/mass body weight-day (class-specific guilds)).

This equation is adapted from methodologies developed by the US EPA [145].

In this study, the methodology is extended to incorporate additional emission sources such as glycol dehydration units and wastewater treatment plants. Furthermore, it is expanded to estimate the country-wide cumulative ecological risk from simultaneous exposure to air toxics, such as polycyclic aromatic hydrocarbons originating from multiple industrial sources.

To evaluate the potential cumulative risk posed by simultaneous exposure of a measurement receptor to multiple air toxics at a specific location, the air toxics-specific ESQ values should be aggregated to determine a cumulative ESQ, as presented in Equation (6):

$$Cumulative\ ESQ = \sum_{i=1}^n ESQ_i \quad (4-15)$$

where *Cumulative ESQ* is the total ecological screening quotient at a specific location (unitless), n is the number of air toxics under consideration, and ESQ_i is the ecological screening quotient for the i th air toxic (unitless).

Following the estimation of risk, risk description articulates the scope and nature of potential ecological impacts, providing a critical interpretive context for ESQ values relative to regulatory benchmarks and ecological receptors.

For example, the ESQ for fish continuously exposed to methylmercury in water bodies receiving industrial emissions is calculated at 35. Methylmercury is a bioaccumulative form of mercury that poses significant risks to aquatic life, particularly predatory fish, due to biomagnification through the food chain. The presence of methylmercury is attributed to the methylation of inorganic mercury emissions from industrial processes.

Additionally, there is inherent uncertainty in assuming that fish diets consist solely of contaminated food sources, as fish typically consume a variety of aquatic organisms and detritus. Nonetheless, given methylmercury's potential to bioaccumulate and the sensitivity of fish to its toxic effects, exceedance of the target risk level warrants careful consideration. If necessary, further investigation should be conducted to confirm mercury levels in the aquatic environment and assess the actual risk to aquatic biota.

4.3 Analysis of Results

4.3.1 Case Study

Leveraging the methodology detailed above, this section presents the application of EHAM for a country-wide ecological health assessment in a case study of Kuwait, using data from 2017. Central to this assessment was the development of unique food web models, tailored to local ecosystems. These food webs, incorporating region-specific trophic interactions, enable a more accurate and localized risk assessment, important for understanding the ecological dynamics of Kuwait's environment.

This study considers Kuwait's distinct air quality zones, as shown in Figure 4-2, each with unique ecological characteristics and sensitivities. For example, coastal zones are integral to marine and shoreline ecosystems, while inland zones are crucial for desert flora and fauna. Further details pertaining to Kuwait's air quality zones, the selection of air toxics emission sources for human health

risk assessment, and data on modeled facilities and emission sources can be found in Section 3.3.1 (“Case Study”) of Chapter 3.

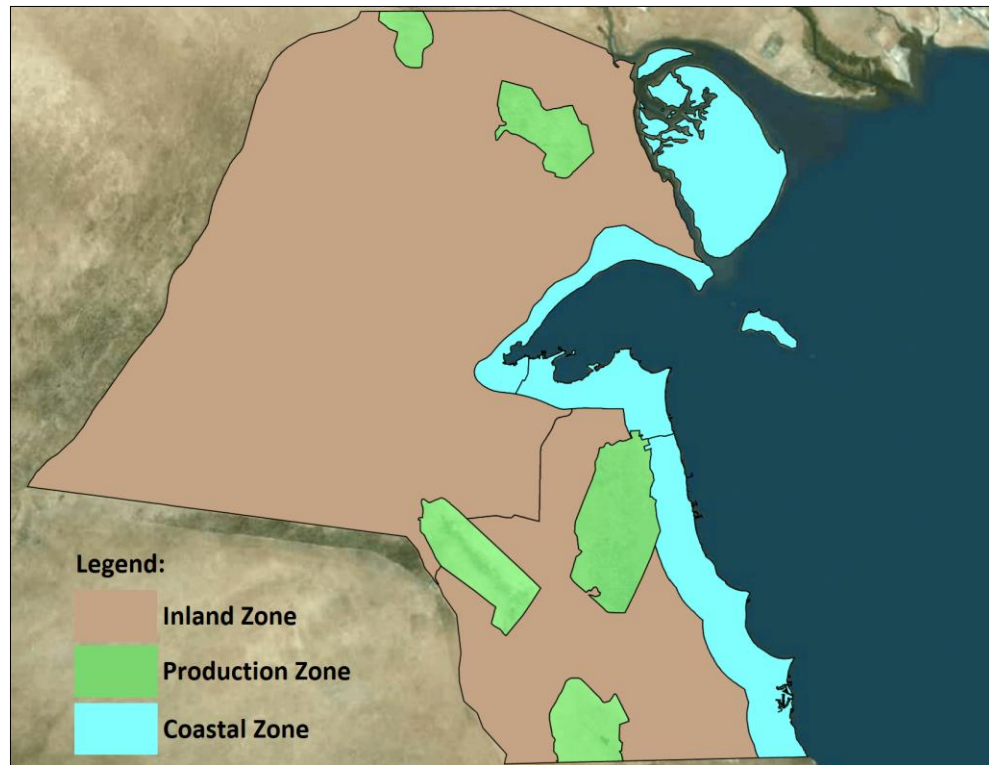


Figure 4-2: Kuwait air quality zones. Map data: Lakes Satellite

The same four air toxics, benzene, formaldehyde, toluene, and benzo(a)pyrene, investigated in [18], are also assessed in this chapter due to their common occurrence and well-documented potency. A wide variety of emission sources, including combustion sources, glycol dehydrators, and wastewater treatment plant operations, were modeled. These sources were selected due to their notable contributions of air toxics, including the four air toxics assessed in this chapter. For details on the entry mechanisms and affected trophic levels of these air toxics, readers are referred to Table 4-1. This approach supports an integrated perspective that facilitates a thorough assessment of ecological risks, thereby enhancing the understanding of air quality impacts in a wider ecological framework.

For this research, unique food web models were developed, tailored to local ecosystems to achieve a country-wide ecological risk assessment for Kuwait.

These models intricately mapped the interactions among species, organized by trophic levels and guilds to reflect the ecological structure of the region. Incorporating region-specific trophic interactions, they provided a more accurate and localized risk assessment. Drawing on the literature [226, 227, 228] to identify these biota, terrestrial species such as the red fox and the gerbil, and desert birds like the hoopoe, along with coastal species including the Indo-Pacific king mackerel, the greasy grouper, the crab plover, and the mudskipper, were grouped based on their feeding habits and roles within these ecosystems.

This organization by trophic level—from primary producers like the *Rhazotrypa* epapposum plant to primary consumers and higher-level predators—enabled a detailed understanding of the ecological dynamics. These tailored models also employed food-chain multipliers, crucial for quantifying the biomagnification of air toxics such as benzo(a)pyrene across different trophic levels. By capturing both direct and indirect pathways of air toxics exposure, the models provided an in-depth understanding of the ecological risks associated with environmental contaminants in Kuwait.

4.3.2 Cumulative Risk Results

This section outlines the established target risk levels and presents findings related to the country-wide cumulative ecological risk, with a specific focus on community and guild-based measurement receptors within Kuwait's three distinctive air quality zones: coastal, inland, and production.

Within this assessment, an ESQ value of less than 1 is interpreted as indicating negligible ecological risk, suggesting that the level of air toxics exposure is below the threshold likely to cause adverse effects on local biota. Conversely, an ESQ of 1 or greater signals a potential risk, warranting further detailed investigation to ascertain the extent and implications of ecological impacts due to air toxics [145].

4.3.2.1 Coastal Air Quality Zone

The cumulative ESQ analysis in the coastal zone reveals a range of ecological risks among different species, determined according to their specific diets and trophic interactions.

Table 4-5 summarizes the cumulative ESQ values for seven receptor groups, providing insights into the variable ecological risks within this ecologically sensitive area.

Table 4-5: Summary of cumulative ESQ values for measurement receptors in the coastal zone

#	Receptor Group	Diet	Cumulative ESQ (Unitless)	ESQ Above 1.0 (Y = Yes/N = No)
1	Carnivorous Bird	Equal	3.02×10^2	Y
		Exclusive (Detailed Breakdown Below)	-	-
		Carnivorous Fish	6.06×10^1	Y
		Herbivorous Bird	1.15×10^1	Y
		Herbivorous Mammal	1.18×10^1	Y
		Omnivorous Bird	1.01×10^3	Y
		Omnivorous Fish	4.15×10^1	Y
		Omnivorous Mammal	6.77×10^2	Y
2	Carnivorous Mammal	Equal	2.66×10	Y
		Exclusive (Detailed Breakdown Below)	-	-
		Carnivorous Fish	4.65×10^{-1}	N
		Herbivorous Bird	1.84×10^{-2}	N
		Herbivorous Mammal	2.11×10^{-2}	N
		Omnivorous Bird	9.07×10	Y
		Omnivorous Fish	2.91×10^{-1}	N
		Omnivorous Mammal	6.06×10	Y
3	Carnivorous Shorebird	Equal	9.04×10^2	Y
		Exclusive (Detailed Breakdown Below)	-	-
		Benthic Invertebrates	1.08×10^3	Y

		Omnivorous Bird	3.12×10^3	Y
		Omnivorous Fish	1.40×10^2	Y
		Planktivore Fish	5.59×10^1	Y
		Water Invertebrates	1.27×10^2	Y
		Benthic Invertebrates	1.08×10^3	Y
4	Herbivorous Bird	Equal	1.87×10^1	Y
		Exclusive (Detailed Breakdown Below)	-	-
		Algae	3.30×10^1	Y
		Aquatic Vegetation	4.39×10	Y
5	Herbivorous Mammal	Equal	6.42×10^{-1}	N
		Exclusive (Detailed Breakdown Below)	-	-
		Algae	1.17×10	Y
		Aquatic Vegetation	1.11×10^{-1}	N
6	Omnivorous Bird	Equal	5.15×10^2	Y
		Exclusive (Detailed Breakdown Below)	-	-
		Algae	1.66×10^2	Y
		Aquatic Vegetation	3.32×10^1	Y
		Benthic Invertebrates	1.71×10^3	Y
		Water Invertebrates	1.51×10^2	Y
7	Omnivorous Mammal	Equal	1.59×10	Y
		Exclusive (Detailed Breakdown Below)	-	-
		Algae	7.11×10^{-1}	N
		Aquatic Vegetation	8.03×10^{-2}	N

	Benthic Invertebrates	8.04×10	Y
	Herbivorous Bird	2.93×10^{-2}	N
	Herbivorous Mammal	3.66×10^{-2}	N
	Water Invertebrates	6.38×10^{-1}	N

In Table 4-5, when the diet type is listed as “Equal,” the ESQ is calculated based on an equal diet dose [145], meaning that the measurement receptor consumes food items from different groups in equal proportions. Conversely, when the diet type is anything other than “Equal,” it implies an exclusive diet, focusing on one particular food source. For instance, the ESQ value of 6.06×10^1 for carnivorous birds under a diet of “Carnivorous Fish” indicates the risk assessment based on an exclusive diet of these fish.

Significantly, the data indicate a trend where measurement receptors at higher trophic levels tend to exhibit higher ESQ values compared to those at lower levels, likely due to the process of biomagnification. Carnivorous birds, for example, consistently show high ESQ values across different diets, suggesting considerable risk, particularly when their diet includes omnivorous birds and mammals. This pattern aligns with ecological principles where contaminants can accumulate in higher concentrations as one moves up the food chain, thus posing greater risks to predators at the top.

Carnivorous shorebirds also demonstrate elevated ESQ values, particularly when consuming benthic invertebrates and omnivorous birds, further indicating their vulnerability in the coastal ecosystem due to biomagnification. Conversely, carnivorous mammals exhibit a mixed risk profile, with varied ESQ values depending on their diet. Herbivorous birds and mammals, as well as omnivorous species, show variable ESQ values, reflecting a complex web of ecological interactions and risks associated with their specific feeding habits.

These findings highlight the need for policy measures aimed at reducing air toxics emissions, particularly those that can bioaccumulate. An example policy could involve stricter regulations on industrial emissions, coupled with enhanced monitoring and enforcement mechanisms. Such proactive measures would be important in safeguarding the coastal ecosystem and its diverse inhabitants from the escalating risks of biomagnification.

4.3.2.2 Inland Air Quality Zone

This subsection examines the cumulative ecological risk in Kuwait's inland air quality zone, with a specific focus on the assessment of ESQ values across various terrestrial species representative of this zone. Table 4-6 summarizes these findings, presenting the cumulative ESQ values for six receptor groups.

Table 4-6: Summary of cumulative ESQ values for measurement receptors in the inland zone

#	Receptor Group	Diet	Cumulative ESQ (Unitless)	ESQ Above 1.0 (Y = Yes/N = No)
1	Carnivorous Bird	Equal	4.55×10^{-6}	N
		Exclusive (Detailed Breakdown Below)	-	-
		Herbivorous Bird	1.49×10^{-7}	N
		Herbivorous Mammal	1.92×10^{-7}	N
		Omnivorous Bird	1.16×10^{-5}	N
		Omnivorous Mammal	6.25×10^{-6}	N
2	Carnivorous Mammal	Equal	3.91×10^{-8}	N
		Exclusive (Detailed Breakdown Below)	-	-
		Herbivorous Bird	2.60×10^{-9}	N
		Herbivorous Mammal	2.96×10^{-9}	N
		Omnivorous Bird	9.77×10^{-8}	N
		Omnivorous Mammal	5.31×10^{-8}	N
3	Herbivorous Bird	Equal	4.28×10^{-6}	N
		Exclusive (Detailed Breakdown Below)	-	-
		Terrestrial Plant	4.28×10^{-6}	N
4		Equal	7.00×10^{-8}	N

	Herbivorous Mammal	Exclusive (Detailed Breakdown Below)	-	-
		Terrestrial Plant	7.00×10^{-8}	N
5	Omnivorous Bird	Equal	3.68×10^{-6}	N
		Exclusive (Detailed Breakdown Below)	-	-
		Terrestrial Invertebrates	2.71×10^{-6}	N
		Terrestrial Plant	4.65×10^{-6}	N
6	Omnivorous Mammal	Equal	2.82×10^{-8}	N
		Exclusive (Detailed Breakdown Below)	-	-
		Herbivorous Bird	2.71×10^{-9}	N
		Herbivorous Mammal	3.37×10^{-9}	N
		Terrestrial Invertebrates	3.41×10^{-8}	N
		Terrestrial Plant	7.26×10^{-8}	N

The analysis of Kuwait's inland air quality zone presents a stark contrast to the coastal zone, with Table 4-6 showing all six receptor groups having cumulative ESQ values below 1.0. This uniformity indicates negligible ecological risk from air toxics across various species, including carnivorous, herbivorous, and omnivorous groups, irrespective of their trophic levels.

Notably, carnivorous birds and mammals exhibit extremely low ESQ values, suggesting minimal biomagnification impact. Similarly, herbivorous and omnivorous species display low ESQ values, highlighting the reduced influence of air toxics in this zone.

These findings suggest that current air quality standards in the inland zone are effectively protecting the terrestrial biota from air toxics exposure. The results emphasize the necessity of not only maintaining but also potentially strengthening these standards. Moreover, regular ecological risk assessments are recommended to ensure these standards continue to safeguard ecological health, adaptively managing and responding to evolving environmental and industrial challenges. This approach is important for the long-term protection and sustainability of these ecosystems.

4.3.2.3 Production Air Quality Zone

This subsection analyzes the cumulative ecological risk within Kuwait's production air quality zone. Table 4-7 presents these findings, summarizing the cumulative ESQ values for six receptor groups.

Table 4-7: Summary of cumulative ESQ values for measurement receptors in the production zone

#	Receptor Group	Diet	Cumulative ESQ (Unitless)	ESQ Above 1.0 (Y = Yes/N = No)
1	Carnivorous Bird	Equal	7.09×10^{-7}	N
		Exclusive (Detailed Breakdown Below)	-	-
		Herbivorous Bird	2.37×10^{-8}	N
		Herbivorous Mammal	3.11×10^{-8}	N
		Omnivorous Bird	1.81×10^{-6}	N
		Omnivorous Mammal	9.76×10^{-7}	N
2	Carnivorous Mammal	Equal	6.09×10^{-9}	N
		Exclusive (Detailed Breakdown Below)	-	-
		Herbivorous Bird	4.09×10^{-10}	N
		Herbivorous Mammal	4.70×10^{-10}	N
		Omnivorous Bird	1.52×10^{-8}	N
		Omnivorous Mammal	8.30×10^{-9}	N
3	Herbivorous Bird	Equal	7.17×10^{-7}	N
		Exclusive (Detailed Breakdown Below)	-	-
		Terrestrial Plant	7.17×10^{-7}	N
4	Herbivorous Mammal	Equal	1.18×10^{-8}	N
		Exclusive (Detailed Breakdown Below)	-	-

		Terrestrial Plant	1.18×10^{-8}	N
5	Omnivorous Bird	Equal	5.97×10^{-7}	N
		Exclusive (Detailed Breakdown Below)	-	-
		Terrestrial Invertebrates	4.20×10^{-7}	N
		Terrestrial Plant	7.75×10^{-7}	N
6	Omnivorous Mammal	Equal	2.82×10^{-8}	N
		Exclusive (Detailed Breakdown Below)	-	-
		Herbivorous Bird	4.64×10^{-9}	N
		Herbivorous Mammal	4.30×10^{-10}	N
		Terrestrial Invertebrates	5.43×10^{-10}	N
		Terrestrial Plant	5.31×10^{-9}	N

In Kuwait's production air quality zone, Table 4-7 shows a consistent pattern of low ecological risk, with all six receptor groups exhibiting cumulative ESQ values well below 1.0. This uniformity across carnivorous, herbivorous, and omnivorous species indicates negligible risk from air toxics, a significant finding considering the industrial nature of this zone. Notably, both higher- and lower-trophic-level species show minimal biomagnification impact, suggesting effective air quality control in the production area.

These encouraging results reinforce the effectiveness of current air quality regulations in this zone, emphasizing the importance of sustained and potentially enhanced environmental policies. Regular ecological risk assessments are vital to continually monitor and adapt these standards, ensuring that they respond effectively to the dynamic nature of industrial activities and environmental changes. This proactive approach to environmental management is crucial for maintaining ecological health and guiding future policy development in similar industrial contexts.

To provide a practical understanding of ecological risk from air toxics exposure, it is important to interpret the ESQ correctly. This quotient, derived from the ratio of EEL to TRV, as explained earlier in this chapter, is not a probabilistic estimate. For example, an ESQ of 0.01 signifies that the exposure

level is 100 times lower than the TRV, implying substantially reduced ecological risk, not a 1 in 100 chance of an adverse effect. An ESQ of 1.0 means that the exposure level is equal to the TRV, while an ESQ of 10 indicates an exposure level that is ten times higher than the TRV.

For ESQ values greater than 1.0, a deeper examination into the specific toxicological profiles of the air toxics is necessary. This involves understanding the acute and chronic effects of these pollutants, their bioaccumulative potential across different species, and the extent of their impact on key ecological functions, such as nutrient cycling and predator–prey dynamics. An ESQ above 1.0 suggests potential ecological risk. However, higher ESQ values should not be interpreted linearly with risk magnitude—they indicate an increasing qualitative likelihood or severity of ecological risk from air toxics.

Understanding these ESQ values is crucial for risk management, decision making, and the development of informed environmental policies. For instance, ESQs significantly greater than 1.0 could prompt further investigation, immediate mitigation efforts, or the adjustment of existing policies, whereas lower ESQs might indicate areas where current management and policy strategies are effective. Thus, these interpretations guide actions to proactively mitigate and manage ecological risks associated with air toxics, while also serving as a foundation for policy formulation and refinement.

4.3.3 Ecological Risk Driver Analysis

In the country-wide ecological risk assessment of Kuwait's air quality zones, benzo(a)pyrene was identified as the risk driver in the coastal zone.

This outcome aligns with findings from a companion human health risk study [18]. Benzo(a)pyrene's notably higher bioconcentration factors, in comparison to other air toxics, signal its significant potential for bioaccumulation and subsequent ecological impact. This is particularly evident in the elevated cumulative ESQ values for carnivorous birds, calculated at 3.02×10^2 for those with an equal diet and a cumulative ESQ reaching 1.01×10^3 for those predominantly feeding on omnivorous birds. Both figures significantly exceed the established target risk levels.

These values demonstrate benzo(a)pyrene's capacity for bioaccumulation across different trophic levels within the food chain. This bioaccumulation highlights the substance's ecological threat in the coastal zone, a habitat characterized by intricate interactions between marine and terrestrial species. Therefore, this analysis emphasizes the importance of assessing specific toxicological profiles

in ecological risk assessments, especially for air toxics like benzo(a)pyrene that exhibit high bioconcentration factors.

4.3.4 Assumptions and Limitations of EHAM in the Kuwait Case Study

The country-wide ecological risk assessment of air toxics is based on several key assumptions and limitations, which are outlined as follows:

1. **Bioavailability of Air Toxics:** This study presumes that air toxics in food items and environmental media are fully bioavailable to ecological receptors. “Bioavailable” here means the fraction of a substance that is readily absorbed and can influence biological processes;
2. **Presence of Sensitive Life Stages:** This study assumes that the most sensitive life stages of measurement receptors are present within the assessment area, resulting in a more protective ecological health risk assessment;
3. **Conservative Estimates:** This study utilizes conservative estimates for body weights and food ingestion rates of measurement receptors;
4. **Equal Exposure Across Species:** This study operates under the assumption that each species within any given ecological guild or community is equally exposed to the air toxics;
5. **Contaminated Food and Media:** This study assumes that 100 percent of the food items and media ingested by receptors are contaminated, implying exclusive feeding within the assessment area;
6. **Cross-Interaction of Air Toxics:** The current assessment assumes independent effects of each air toxic, without considering potential cross-interactions, such as synergistic, antagonistic, or additive effects, that might occur when multiple pollutants coexist.

4.4 Conclusions

This chapter introduces the Ecological Health Assessment Methodology (EHAM). To illustrate EHAM, a country-wide ecological risk assessment from air toxics is shown to be possible using Kuwait as the case study, revealing that it is not restricted to small regions. The key conclusions from this study are:

1. Substantial Ecological Risk in the Coastal Zone: This study demonstrates significant ecological risks in Kuwait's coastal zone, particularly for carnivorous birds, with cumulative ESQ values reaching 3.12×10^3 . This highlights the substantial impact of air toxics in this ecologically sensitive area, driven largely by the process of biomagnification.
2. Negligible Risks in the Inland and Production Zones: In contrast, both the inland and production air quality zones exhibit negligible ecological risks, as indicated by the consistently low ESQ values across various species. This suggests the effective control of air toxics levels in these zones, protecting the ecological health of diverse species.
3. Benzo(a)pyrene as a Primary Ecological Risk Driver: Similar to findings in human health risk, benzo(a)pyrene is identified as a key risk driver in the coastal zone due to its strong bioaccumulative potential, underlining its significant ecological threat. This analysis highlights the importance of assessing specific toxicological profiles in ecological risk assessments, especially for air toxics like benzo(a)pyrene that exhibit elevated bioconcentration.
4. Development of Tailored Food Web Models for Local Ecosystems: This study's unique models, customized for Kuwait's ecosystems, offered a representative ecological risk assessment.
5. Importance of Diet Type and Trophic Level in Ecological Risk Assessments: This analysis highlights the importance of considering both the diet type and trophic level in assessing ecological risks due to air toxics exposure.
6. Need for Regular Ecological Risk Assessments: This study underscores the importance of conducting regular ecological risk assessments to adapt to environmental and industrial changes, ensuring that air quality standards continue to protect ecological health.
7. Informing Policy and Management Decisions: The findings and methodologies developed in this study offer valuable insights for environmental management strategies and policy decisions. These can guide actions to mitigate ecological risks, such as adjusting existing policies or implementing targeted mitigation efforts in high-risk areas.

4.5 Future Work

Future work will expand the EHAM analysis to include additional emission sources, such as fugitive and mobile sources, thereby broadening its ecological risk assessment scope.

This expansion will be followed by a transition to a probabilistic approach, characterized by the development of a stochastic model incorporating distributions for variables like food ingestion rates of measurement receptors and fractions of diet. This evolution addresses the inherent variability and uncertainty in ecological risk assessments, enabling a more accurate representation of risk by capturing the range of possible outcomes in ecological systems and exposure scenarios.

Continuing to explore the interactions and potential synergistic effects of different air toxics when multiple pollutants coexist, future studies will provide a more comprehensive understanding of their cumulative ecological impact. These advancements in EHAM will significantly contribute to more effective environmental management strategies and the ongoing development of ecological risk assessment methods.

Chapter 5. Advancing Human Health Risk Assessment Through a Stochastic Methodology for Mobile Source Air Toxics

Chapter 5 presents a new stochastic human health risk assessment methodology for mobile source air toxics, using Monte Carlo simulations to quantify variability among individuals and uncertainty in exposure and risk estimates. This chapter builds upon the deterministic framework in Chapter 2 and addresses Objectives 4 and 5 of this thesis. The stochastic approach improves risk characterization by identifying high-exposure subpopulations, addressing uncertainty more effectively, and informing cost-effective, data-driven decisions for resource allocation.

5.1 Introduction

Mobile source emissions from roadways near human populations often result in greater health impacts than emissions from industrial facilities in North America [2, 3], primarily due to their proximity to densely populated areas and the high exposure to pollutants such as mobile source air toxics (MSATs).

There is a problem with the traditional use of worst-case or average exposure values in human health risk assessments for MSATs [3], which results in overlooking the considerable variability among individuals in a population, the specific vulnerabilities of sensitive subpopulations, and the inability to capture episodic high-exposure events that considerably impact risk assessment outcomes.

Stochastic methods address these challenges by capturing variability and uncertainty for a more comprehensive risk assessment. Deterministic methods provide baseline exposure–response relationships, while stochastic methods account for the range of possible exposures in different individuals, reflecting real-world complexities. A new stochastic method for MSATs is proposed to provide a more accurate and comprehensive assessment of health risks, paving the way for more effective and equitable risk management strategies.

5.1.1 Mobile Source Air Toxics (MSATs)

MSATs, a subset of hazardous air pollutants, are emitted from on-road mobile sources such as trucks, passenger cars, buses, and motorcycles, as well as from non-road sources, including marine vessels, airplanes, and agricultural equipment [3].

This research focuses on emissions from on-road mobile sources due to their proximity to human populations and their direct impact on urban air quality. The United States Environmental Protection Agency (EPA) has identified more than 1160 compounds in the exhaust and evaporative emissions from on-road and non-road mobile sources [13, 14], including non-exhaust emissions like gasoline vapor, fluid leaks (including washer fluids and deicing chemicals), particulate matter from brake dust and tire wear, and re-entrained road dust.

Key MSATs include benzene, 1,3-butadiene, formaldehyde, acetaldehyde, acrolein, polycyclic aromatic hydrocarbons (PAHs), naphthalene, hexavalent chromium, ethylbenzene, and diesel exhaust. These toxic emissions significantly contribute to urban air pollution, posing risks of cancer and noncancer health effects in humans, as summarized in Table 5-1. Additionally, they have been linked to health impacts in animals [17]. Table 5-1 includes examples of MSATs and their associated health impacts, showing the range of potential effects, although the selection is not comprehensive.

Table 5-1: Summary of health effects associated with MSATs exposure

Mobile Source Air Toxics	Health Effect(s)	Ref.
Acetaldehyde	Nose cancer	[5]
Benzene	Leukemia	[5]
	Childhood leukemia *	[6]
1,3-Butadiene	Leukemia	[5]
Diesel Exhaust	Lung cancer	[7]
Formaldehyde	Nose and throat cancer	[8]
Hexavalent Chromium (VI)	Lung cancer	[5]
Naphthalene	Nose cancer	[5]
PAHs	Cardiovascular diseases	[9]

* Research indicates that even acute exposure to certain MSATs in high-traffic areas ($\geq 20,000$ vehicles per day) can increase the odds of childhood leukemia, with an odds ratio for leukemia of 8.28 (95% Confidence Interval [CI]: 2.09–32.80) [6].

Despite the critical nature of MSATs, the regulatory practices for monitoring and controlling their full range are currently limited, with fewer than 70 of the 1160 identified MSATs actively monitored by regulatory agencies [15, 16].

This shortfall in regulatory monitoring, combined with the proximity of mobile emission sources to human populations [229, 10, 12, 33] and the increased exposure to elevated MSATs concentrations, such as benzene and PAHs near busy and congested roads [1], highlights the need for a detailed health risk assessment.

5.1.2 Deterministic Human Health Risk Assessments

Human health risk assessment estimates the probability and magnitude of adverse health effects resulting from exposure to environmental stressors. It involves four key steps: hazard identification, exposure assessment, dose–response assessment, and risk characterization.

Risk characterization uses health risk estimates to determine the presence of risk, quantify outcomes, and identify contributing factors [3]. This information guides informed decisions on mitigation strategies and the design or improvement of pollution control systems, including those for vehicles.

Cancer risk is defined as the incremental probability (unitless) of an individual developing cancer over their lifetime, typically assumed to be 70 years [3], based on specific exposure and toxicity assumptions. For example, a risk of $5.0\text{E-}05$ indicates that an individual has up to a 5 in 100,000 increased chance of developing cancer from the evaluated exposure.

The noncancer hazard is an estimate of the likelihood of experiencing health effects such as neurological, cardiovascular, reproductive, or respiratory issues due to MSATs exposure through various pathways and routes. Hazards are calculated as a ratio of exposure to reference doses or concentrations and are classified as acute or chronic [3].

Traditional risk assessments typically rely on deterministic methods, using worst-case or average exposure scenarios [3]. For example, deterministic models often assume an average adult body weight of 70 kg when calculating MSATs intake. This assumption overlooks individual variability, leading to oversimplifications that misrepresent actual risk levels across different population segments.

To address these limitations, this research employs stochastic methods, which capture variability and uncertainty in real-world exposure scenarios, enabling a more accurate and equitable assessment of health risks.

5.1.3 Literature Review

Large-scale assessments like the Air Toxics Screening Assessment (AirToxScreen) [2], a successor to the National Air Toxics Assessment starting in 2017, and the Regional Air Impact Modeling Initiative (RAIMI) [46] have contributed to understanding MSATs' impact.

AirToxScreen is a deterministic model that focuses on direct inhalation risks but does not account for other exposure pathways like ingestion, nor does it address local-scale air toxics “hot spots” and individual risks from near-roadway exposures.

RAIMI, while comprehensive in assessing cumulative air dispersion and human health risks, faced challenges in source parameterization. The RAIMI study in Harris County, Texas, identified risk hotspots but highlighted the need for improved emission source characterizations.

Furthermore, statewide screening assessments like the Minnesota Pollution Control Agency's MNRISKs [230] study provided extensive inventories of emissions, including on-road and non-road sources.

These studies, including AirToxScreen, RAIMI, and MNRISKs, though insightful, primarily used deterministic models based on average exposure assumptions, like a standard adult body weight of 70 kg and inhalation rates of 0.83 m³/h. This approach fails to capture the variability in cancer or noncancer health risks among different population segments.

Additionally, the annual averages used in these models overlook significant exposure pathways and spatiotemporal dynamics, limiting the ability to predict health impacts for specific areas and times accurately.

The literature consistently highlights the need for improved methodologies that account for variability and uncertainty, emphasizing the importance of employing stochastic methods in MSATs risk assessments.

For an expanded review of the literature on deterministic evaluations of the human health risks of MSATs, readers are referred to the “Literature Review” section of Chapter 2.

Table 5-2 summarizes previous probabilistic methods in risk assessment, including their scope, methodologies, and consideration of multiple pathways and cumulative risks.

Table 5-2: Examples of probabilistic methods in risk assessment literature

Study/Ref.	Pollutant/Emissions Source	Methodology	Multi-Pathway ¹	Cumulative ²
Bruce et al. (2007)/ [231]	PAHs from industrial contaminated sites	Comparative analysis of deterministic and probabilistic risk assessment approaches for PAHs, incorporating Monte Carlo simulations to account for variability in exposure factors and sample heterogeneity	No	Yes
Iqbal et al. (2019)/ [124]	Nitrogen Oxides (NOx) from vehicles	Probabilistic emissions modeling using Monte Carlo simulation for NOx emissions	No	No
Widiana et al. (2019)/ [232]	Ambient volatile organic compounds (VOCs) in primary schools	Source apportionment of VOCs via a principal component analysis and health risk assessment, enhanced by Monte Carlo simulations for uncertainty and sensitivity analyses	No	Yes
Jiménez-Oyola et al. (2021a)/ [233]	Heavy metal(loid)s (As, Cd, Cr, Cu, Ni, Pb, and Zn) from gold mining activities	Bayesian probabilistic approach to multi-pathway human health risk assessment	No	Yes
Ramesh et al. (2021)/ [234]	Uranium in groundwater	Evaluation of human health risks from uranium-contaminated groundwater via deterministic and probabilistic approaches (Monte Carlo simulations) with sensitivity analysis	No	No
Jiménez-Oyola et al. (2021b)/ [235]	Potentially toxic elements in polluted rivers/Ecuadorian Amazon	Evaluating multi-pathway human exposure to potentially toxic elements through deterministic and probabilistic methods, incorporating Monte Carlo simulation and sensitivity analysis for hazard index and total cancer risk assessment	No ³	Yes

Study/Ref.	Pollutant/Emissions Source	Methodology	Multi-Pathway ¹	Cumulative ²
Redmon et al. (2021)/ [120]	Trace metals in oilfield-produced water used for irrigating crops in California's Central Valley	Probabilistic assessment of health risks from consuming crops irrigated with oilfield-produced water via Monte Carlo simulation of metal concentrations, crop uptake, and human exposure	No	Yes
Zhang et al. (2022)/ [236]	Seven carcinogenic PAHs from fossil fuel usage	Evaluation of carcinogenic risks from seven polycyclic aromatic hydrocarbons using deterministic and probabilistic approaches with Monte Carlo simulations and a multimedia multi-pathway model	Yes	Yes
Khoshakhlagh et al. (2022)/ [237]	Inhalational exposure to 1,3-butadiene and styrene among workers in carpet factories	Human health risk assessment based on inhalational exposure to 1,3-butadiene and styrene using Monte Carlo simulation for calculating carcinogenic risk and non-carcinogenic risk	No	Yes
Panqing et al. (2023)/ [238]	Heavy metals contamination in soil, focusing on Zn, Cu, Cr, Pb, and Hg from traffic, natural sources, coal, and industrial sources	Quantitative health risk assessment of soil heavy metals for different populations using the Unmix model and Monte Carlo simulation for source apportionment and risk evaluation	No	Yes
This Thesis	Air toxics from on-road mobile sources	Stochastic risk assessment method for MSATs, incorporating spatiotemporal dynamics and variability analysis	Yes	Yes

¹ Multi-Pathway: Indicates if multiple exposure pathways (e.g., inhalation, ingestion, dermal contact) were considered in the study. ² Cumulative: Denotes whether the study conducted a cumulative risk assessment of multiple pollutants or sources. ³ The inhalation exposure pathway was not evaluated in this study.

No peer-reviewed studies have yet employed stochastic modeling to assess the health risks from air toxics emitted by on-road mobile sources.

This significant gap in the literature underscores the limitations of deterministic methods, which fail to account for the variability inherent in population exposures.

By incorporating randomness and variability through probabilistic frameworks, stochastic models offer an essential and advanced approach for evaluating complex and dynamic exposure scenarios, particularly in environments heavily influenced by on-road mobile sources.

5.1.4 Research Objectives and Contributions

The primary objective of this research is to make effective use of the benefits of stochastic risk modeling, which include the identification of high-risk groups, an enhanced understanding of uncertainty and variability, improved risk management, support for policy and regulatory decisions, and cost-effective resource allocation.

These benefits are achieved by assessing human health risks from MSATs exposure through a newly tailored stochastic modeling method aimed at the following:

1. Evaluating the (i) cumulative and (ii) multi-pathway risks of MSATs exposure. This includes the combined effects of multiple pollutants and exposure routes, often overlooked in traditional assessments. The primary exposure pathways modeled in this study are inhalation and ingestion.
2. Accounting for the overall population risk while identifying vulnerable subpopulations, high-risk groups, and exposure hotspots.
3. Comparing the outcomes of stochastic and deterministic models to demonstrate the advantages of probabilistic methods for assessing MSATs health risks.

Towards these objectives, this chapter contributes the following:

1. Introduces a stochastic model for environmental health risk assessments of MSATs. This approach helps identify and quantify risks for subpopulations that are more vulnerable due to higher exposure or increased susceptibility. Although it is primarily focused on MSATs, this research lays the groundwork for extending this methodology to other chemicals, such as per- and polyfluorinated substances, by incorporating new relevant exposure pathways. This promotes broader application and future research.

2. Performs a sensitivity analysis to identify dominant variables affecting the risk results, ensuring a focused and resource-efficient stochastic risk assessment.
3. Incorporates spatiotemporal dynamics to enhance emission characterization by integrating hourly data on wind speeds, traffic patterns, and emissions, addressing the limitations of annual averages and enabling dynamic exposure assessments for targeted interventions.
4. Identifies MSATs cancer risk drivers to inform targeted control measures to reduce public health risks.

5.2 Methodology

Succinct summary of methodology steps:

1. Utilize current U.S. regulatory models, such as the MOtor Vehicle Emission Simulator (MOVES) or the International Vehicle Emissions Simulator, to estimate the emissions from on-road vehicles;
2. Employ air dispersion models like the American Meteorological Society/EPA Regulatory Model (AERMOD) to predict MSATs air concentrations and deposition;
3. Identify realistic exposure scenarios to estimate the types and magnitudes of human exposure to MSATs;
4. Conduct a multi-pathway fate-and-transport analysis to determine MSATs concentrations in various media, assessing both direct and indirect exposures at sensitive locations;
5. Quantify exposure levels to determine cumulative and multi-pathway cancer risks (deterministic MSATs risk characterization);
6. Identify dominant variables that affect MSATs exposure and risk outcomes and implement Monte Carlo simulations to explore variability in exposure and enhance the understanding of risk to the population as a whole (stochastic MSATs risk characterization);
7. Verify Monte Carlo simulations to ensure precision and reliability by analyzing and plotting outputs (e.g., exposure distributions) from realizations, comparing key statistical metrics to initial parameters, and conducting manual calculations to confirm cancer risk percentiles and their statistical consistency.

Highlights:

This methodology extends and adapts stochastic modeling techniques, specifically Monte Carlo simulations, to assess human health risks from MSATs.

By tailoring these probabilistic methods to on-road mobile source emissions, it addresses a critical gap in human health risk assessment and enables a comprehensive evaluation of variability in MSATs exposures, which deterministic methods cannot achieve.

Using 100,000 realizations for each scenario (adult and child), this research examines complex exposure scenarios and captures a wide range of possible outcomes (including worst-case scenarios).

This approach refines and improves cumulative and multi-pathway risk assessments by providing detailed quantification of probabilities for various risk outcomes, especially for MSATs exposures.

Detailed Methodology Sections

5.2.1 On-Road Vehicle Emissions Inventory

In this research, a refined emissions inventory was developed utilizing the EPA's state-of-the-art MOVES [239].

MOVES calculates the emissions for pollutants including particulate matter (PM₁₀ and PM_{2.5}), nitrous oxide (N₂O), sulfur dioxide (SO₂), carbon monoxide (CO), greenhouse gases (GHGs), metals, and MSATs. MSATs are the primary focus of this research.

The simulator models releases of MSATs, covering over 50 exhaust and evaporative species, and supports different fuel types, such as diesel, gasoline, and E85 (a blend of 85% ethanol and 15% gasoline), for various mobile sources, including cars, buses, motorcycles, and trucks.

MOVES requires data on several parameters to estimate emissions. These include road segments, vehicle classes, fuel types and usage, vehicle age distribution, traffic volume, and meteorological data.

The integration of MOVES with the Transportation Air Quality System (TRAQS) [3] enables this methodology to achieve the following:

1. Address MOVES's lack of spatial awareness by utilizing TRAQS to overlay the emissions outputs onto high-resolution geographic grids. This enhances the emissions data's integration with air dispersion models and improves the spatial accuracy of the emissions inventory.
2. Incorporate hourly spatiotemporal data to improve emissions characterization and population exposure assessments. This approach models traffic patterns, weather conditions, and time-specific factors, enabling the identification of high-emission periods, such as peak traffic hours, and low-emission periods, such as nighttime. By capturing time-dependent exposures, this methodology surpasses traditional approaches that rely on annual averages, offering dynamic insights for refined risk assessments.

For further details on the vehicle emissions inventory, see Chapter 2 [3], where this methodology was first introduced and applied.

5.2.2 Air Dispersion and Deposition Modeling

Air dispersion modeling is defined as the mathematical description of pollutant transport in the atmosphere.

This process is conducted to simulate the transport, diffusion, and deposition of MSATs once they are emitted from mobile sources. The goal is to determine air concentrations, deposition on various media, and pollutant distributions both near and far from urban roads. This modeling facilitates the following:

1. Determination of the relative impact of on-road mobile source emissions on ambient air quality compared to other sources, such as power plants or refineries;
2. Understanding the contributions of MSATs to overall air pollution;
3. Identification of roads with significant pollution impacts for targeted monitoring and mitigation efforts.

In this study, the EPA's preferred regulatory model, AERMOD [240], is employed for air dispersion and deposition modeling. AERMOD is suited to this research due to its ability to accurately simulate pollutant behavior over various terrains and near-road environments.

As a validated steady-state Gaussian plume model in urban settings and near roadways, AERMOD uses dispersion principles based on planetary boundary-layer turbulence and scaling concepts, including plume rise and a Gaussian distribution [138].

However, AERMOD does not account for atmospheric chemical reactions. The proposed method is flexible and can incorporate other air dispersion models, such as CALPUFF, which can handle atmospheric chemical reactions.

In this study, AERMOD's outputs include annual average air concentrations for vapor-phase, particle-phase, and particle-bound pollutants [139].

For more detailed information on the air dispersion model, see Section 2.3.1.1 in Chapter 2 [3].
Meteorological Data for AERMOD

The Weather Research and Forecasting (WRF) model [61] was used to generate AERMOD meteorological files due to its advanced capabilities in simulating atmospheric conditions. WRF's high-resolution data improves the precision of air quality predictions by providing a detailed representation of the atmospheric state. The WRF model outputs two meteorological data files: surface data (.SFC) and profile data (.PFL).

The surface data file, which contains hourly boundary-layer parameter estimates, is important for understanding dispersion patterns at ground level.

The profile data file, containing multi-level observations of wind speed, wind direction, temperature, and the standard deviation of fluctuating wind components, provides a vertical atmospheric profile that is essential for assessing pollutant transport and diffusion across different altitudes.

5.2.3 Exposure Scenario Identification

This step identifies high-risk areas and vulnerable populations by characterizing exposure scenarios based on geographical and demographic factors. An exposure scenario involves various “exposure pathways” through which MSATs affect a “receptor”.

An exposure “pathway” refers to the journey MSATs take from their source to the person being exposed, such as through the inhalation of vapors and particulates or the ingestion of contaminated fish. The “route” of exposure specifies how these substances enter the body.

Sensitive receptors include schools, hospitals, and places of worship. These are prioritized due to the increased vulnerability of their occupants to the adverse effects of MSATs. The criteria for identifying high-exposure scenarios at sensitive receptors include the following:

1. Selecting grid nodes with the highest modeled air parameter values within a specific area;
2. Excluding grid nodes located in roadways, as intermittent exposure locations are not the focus of this study;
3. Evaluating scenarios relevant to the study area, such as urban residents, farmers, fishers, or nursing infants.

In human health risk assessments, a common-sense approach is important for analyzing scenarios within a project domain and evaluating their likelihood. If a scenario is not applicable to the study area, the corresponding pathways and risks do not exist. For example, a child playing in a park adjacent to heavy traffic is a realistic exposure scenario, unlike a less probable situation, such as a farmer working in a highly urbanized area devoid of agriculture.

The inclusion of all recommended exposure pathways, including water and soil ingestion, follows the U.S. EPA’s Human Health Risk Assessment Protocol (HHRAP) [37]. As the contribution of certain pathways may vary by location or study, their inclusion supports an adaptable risk assessment and minimizes the risk of overlooking pathways that could be significant under varying environmental or socio-economic conditions.

In this study, the evaluated exposure scenarios include urban resident adults and children, with their associated exposure pathways shown in Table 5-3 and Figure 5-1.

Table 5-3: Evaluated exposure pathways associated with urban resident adult and urban resident child scenarios

Exposure Pathways
Inhalation of vapors and particles
Incidental ingestion of soil
Ingestion of drinking water from treated surface water sources

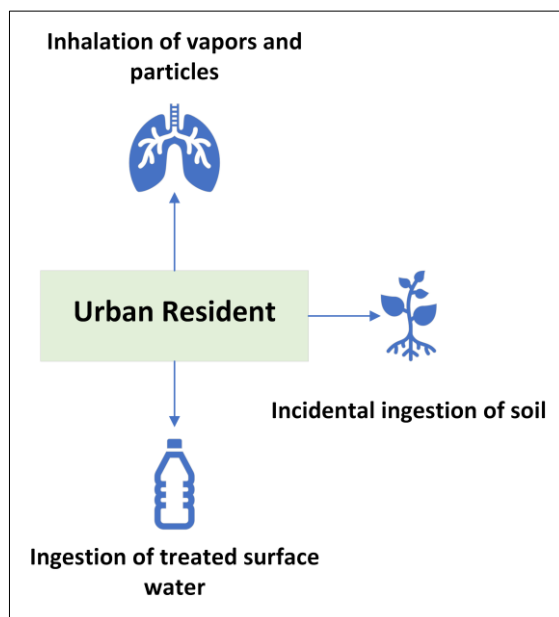


Figure 5-1: Urban resident exposure scenarios and associated exposure pathways

5.2.4 Multi-Pathway Fate-and-Transport Analysis

This analysis evaluates how MSATs move through the environment (“Transport”) and what happens to them after their release (“Fate”).

The process estimates MSAT concentrations in air, soil, water, and food at points of human contact, which is critical for assessing both direct and indirect risks.

Importantly, a lack of transport and fate data should not be interpreted as an absence of potential exposure and risk.

In this research, the medium concentrations were estimated using mathematical equations published in the HHRAP [37].

The outputs from this analysis include estimates of MSAT concentrations in air for direct inhalation risk evaluations, concentrations in soil, and concentrations of MSATs in produce due to direct deposition. These are essential for a comprehensive human health risk assessment.

5.2.5 Deterministic MSATs Risk Characterization

A deterministic human health risk assessment methodology for MSATs was previously developed [3], extending EPA methodologies originally designed for hazardous waste combustion facilities, as detailed in the HHRAP [37], to on-road mobile sources.

This section revisits the key elements of this deterministic risk characterization approach for MSATs.

The deterministic methodology quantifies MSATs exposure by evaluating contact frequency, duration, receptor body weight, and exposure magnitude.

Deterministic models were then applied to calculate cumulative and multi-pathway cancer risks from MSATs exposure.

The outcomes of this risk characterization are presented numerically, with explanatory text to facilitate risk interpretation (see Section 5.1.2).

Inhalation and ingestion exposures were calculated using Equations (1) and (2) [37]:

$$Exposure (inhalation) = ADD (inhalation) = \frac{C_{air} \times EF \times ED}{AT \times 365} \quad (5-16)$$

where ADD represents the average daily dose ($\mu\text{g}/\text{m}^3$), C_{air} is the concentration of MSATs in the air ($\mu\text{g}/\text{m}^3$), EF is the exposure frequency (days/year), ED is the exposure duration (years), AT is the averaging time (year), and 365 is a conversion factor (days/year).

$$Exposure (ingestion) = ADD (ingestion) = \frac{C_{medium} \times Ingr \times EF \times ED}{BW \times AT} \quad (5-17)$$

where ADD represents the average daily dose (mg/kg-day), C_{medium} is the concentration of MSATs in the medium (e.g., mg/L, mg/g), $Ingr$ is the ingestion rate (e.g., L/day, g/day), EF is the exposure frequency (days/year), ED is the exposure duration (years), BW is the body weight (kg), and AT is the averaging time (days).

Table 5-4 [37] summarizes the variables used in the deterministic risk assessments conducted in this research.

Table 5-4: Exposure variables used in deterministic risk characterization

Variable (Unit)	Point Estimate *
Exposure Frequency, Adult and Child (day/year)	350
Body Weight, Adult (kg)	70
Body Weight, Child (kg)	15
Exposure Duration, Adult (year)	30
Exposure Duration, Child (year)	6
Consumption Rate of Soil, Adult (kg/day)	1×10^{-4}
Consumption Rate of Soil, Child (kg/day)	2×10^{-4}
Drinking Water Consumption Rate, Adult (L/day)	1.4
Drinking Water Consumption Rate, Child (L/day)	0.67

* All variable values in this table were adopted from the EPA's HHRAP, "Appendix C: Risk Characterization Equations," which provides recommended defaults for use in risk assessments when site-specific data are unavailable.

The deterministic risk characterization equations used are summarized below:

$$\begin{aligned} \text{Incremental Cancer Risk (inhalation)} = & \text{Exposure Concentration} \times \text{MSATs Unit Risk Factor} \end{aligned} \quad \begin{matrix} (5- \\ 18) \end{matrix}$$

$$\begin{aligned} \text{Incremental Cancer Risk (ingestion)} = & \text{Quantified Exposure} \times \text{MSATs Cancer Slope Factor} \end{aligned} \quad \begin{matrix} (5- \\ 19) \end{matrix}$$

The cancer risk calculations in this study used peer-reviewed toxicity values, including unit risk factors (URFs) for inhalation exposure and cancer slope factors (CSFs) for ingestion pathways. The toxicity values for the MSATs modeled in this study are summarized in Table 5-5.

Table 5-5: URFs and CSFs used for cancer risk calculations of MSATs modeled in this study

MSATs	URF ($\mu\text{g}/\text{m}^3$) ⁻¹	CSF (mg/kg-day) ⁻¹	Reference
Benzene	7.8×10^{-6}	5.5×10^{-2}	EPA's HHRAP Companion Database [37]
Formaldehyde	1.3×10^{-5}	1.3×10^{-5}	
Benzo(a)pyrene	1.1×10^{-3}	7.3	

Next, the cumulative and multi-pathway risks from MSATs exposure represent the total risk from exposure to multiple MSATs through various pathways, including inhalation and ingestion, from different sources.

This risk is quantified using Equation (5) [18]:

$$R_{total} = \sum(R_{i,j}) + \sum(R_{i,inhaltation}) \quad (5-20)$$

where R_{total} represents the cumulative cancer risk estimate, considering both ingestion and inhalation pathways. $R_{i,j}$ represents the individual risk estimate for MSAT compound i and ingestion pathway j ; and $R_{i,inhaltation}$ is the individual risk estimate for MSAT compound i via the inhalation pathway. The index i refers to specific MSAT compounds (e.g., benzene, 1,3-butadiene, formaldehyde, etc.), and j denotes the various ingestion pathways (e.g., vegetable ingestion, fish ingestion, egg ingestion, beef ingestion, etc.).

Aligning the calculated risks with regulatory target levels is crucial for public health and compliance. Regulatory agencies typically establish cancer risk target levels between 1×10^{-4} and 1×10^{-6} [67], indicating minimal increases in risk. This study applies a stricter target level, adopting a maximum cancer risk value of 1×10^{-5} . This stricter target level accounts for background exposure and area-specific risks, serving as reference points for evaluating whether the risks fall within acceptable boundaries.

When the risks exceed these thresholds, an additional analysis is warranted, potentially involving refined exposure assessments or advanced traffic and emissions modeling (as proposed in this research) to better understand MSATs contributions and the associated health risks.

5.2.6 Stochastic MSATs Risk Characterization

This step advances beyond deterministic methods to probabilistic approaches. Step 6 utilizes Monte Carlo simulations to capture the variability and uncertainty in environmental exposures to MSATs.

Unlike traditional deterministic approaches that often oversimplify complex exposure scenarios, the use of stochastic methods introduces a higher level of precision in risk estimates by accounting for a broader range of possible outcomes.

This approach employs probability distributions to describe the range and likelihood of possible risk values.

A key benefit of stochastic analysis is its ability to handle real-world variability and uncertainty, a common limitation of deterministic models.

This methodology enhances decision-making by identifying risk hotspots and enabling sensitivity analyses, focusing efforts on high-impact areas and variables, which directly inform risk management strategies and interventions.

By integrating probability distributions for dominant variables into risk assessment models, this methodology captures a spectrum of potential health outcomes, reflecting the complexity of real-world MSAT exposures.

The application of probability density functions (PDFs) and cumulative distribution functions within this framework provides a nuanced and detailed view of risk characterization for MSAT exposures.

5.2.6.1 Sensitivity Analysis

A sensitivity analysis (contribution 2 in Section 5.1.4) was conducted using a custom Python script to evaluate the impact of key exposure variables before full stochastic modeling. This analysis prioritized the most influential parameters to optimize resources (e.g., simulation times).

Steps in the Sensitivity Analysis:

1. Variable selection: Five key exposure variables were analyzed: body weight, exposure frequency, exposure duration, drinking water consumption rate, and soil consumption rate. These were chosen based on their relevance to risk calculations for both adult and child populations.
2. Variation application: The Python script systematically varied each variable by $\pm 20\%$, a threshold determined through trial and error. Smaller and larger ranges were tested, and $\pm 20\%$ was found to effectively capture realistic variability while maintaining the interpretability of the results. Additionally, other studies have employed similar variation percentages in their sensitivity analyses [241, 242].
3. Evaluation of results: The script compared risk outcomes for each variation, focusing on identifying variables with the greatest impact on calculated risks.

Key Observations:

1. Body weight: While showing lower sensitivity compared to other variables, body weight remains important, especially in the children scenario, due to its inverse relationship with exposure calculations.
2. Exposure frequency and duration: These variables emerged as the most influential factors, highlighting their importance in stochastic modeling and the need to accurately capture exposure patterns.
3. Drinking water consumption: Moderate sensitivity was observed, suggesting its relevance under certain scenarios.

5.2.6.2 Selecting Distributions for Key Exposure Variables

The sensitivity analysis results directly informed the prioritization and selection of key variables for Monte Carlo simulations by identifying parameters with the greatest influence on risk outcomes.

For these prioritized variables, probability distributions were selected based on a review of peer-reviewed literature [231, 235, 243, 244] to ensure they reflect the variability of each variable.

Table 5-6 lists the key exposure variables employed in the probabilistic model, along with their selected distributions and parameters.

These variables and their empirically supported distributions form the basis for the stochastic risk characterization in this study, enabling a detailed and realistic representation of variability in human exposure to MSATs.

Table 5-6: Distributions and parameters for key exposure variables in stochastic risk assessment

Variable	Distribution	Parameters
Exposure Frequency, Adult and Child (day/year)	Triangular	Minimum = 180, Maximum = 365, Likeliest = 345
Body Weight, Adult (kg)	Normal	Mean = 72, Standard Deviation = 15.9

Variable	Distribution	Parameters
Body Weight, Child (kg)	Normal	Mean = 15.6, Standard Deviation = 3.7
Exposure Duration, Adult (year)	Lognormal	Mean = 11.36, Standard Deviation = 13.72
Exposure Duration, Child (year)	Uniform	Minimum = 1, Maximum = 6
Consumption Rate of Soil, Adult (kg/day)	Lognormal	Central Tendency * = 1×10^{-5} , Upper Percentile * = 5×10^{-5}
Consumption Rate of Soil, Child (kg/day)	Lognormal	Central Tendency = 3×10^{-5} , Upper Percentile = 9×10^{-5}
Drinking Water Consumption Rate, Adult (L/day)	Lognormal	Median = 0.5020, Upper Percentile = 2.906
Drinking Water Consumption Rate, Child (L/day)	Lognormal	Median = 0.1110, Upper Percentile = 1.069

* Note: “Central Tendency” refers to the 50th percentile, and “Upper Percentile” refers to the 95th percentile.

5.2.6.3 Monte Carlo Simulations

All Monte Carlo simulations in this study use 100,000 realizations for each scenario. This number of realizations ensures statistical reliability of the risk estimates, minimizing the influence of random fluctuations and supporting the convergence of results. Preliminary tests using 10,000 and 50,000 realizations showed that the mean, standard deviation, and 5th–95th percentiles stabilized at 100,000 simulations, confirming this number’s adequacy for this analysis.

The simulations quantitatively characterize uncertainty and variability in risk estimates by sampling from the input probability distributions of key exposure variables.

Each simulation involves random sampling from the input distributions of exposure factors and constructing a composite uncertainty distribution by combining these sampled values with the model

equations. This iterative process is repeated for 100,000 realizations, resulting in a distribution of cancer risk estimates.

The results from these realizations are tallied to produce histograms, which illustrate the distribution of potential risk outcomes.

The 95th percentile of the overall risk distribution is used as the benchmark for identifying high-risk scenarios, aligning with common regulatory standards [245].

5.2.6.4 Verification of the Monte Carlo Simulation

The verification process for the Monte Carlo simulations employed in this study involved analyzing and plotting the outputs from the 100,000 realizations and comparing the resulting statistics for each distribution listed in Table 5-6 against the initial input parameters.

This comparison demonstrated that the calculated values align closely with the input specifications, with discrepancies falling within acceptable limits for high-precision simulations.

To quantify the precision of the sampling process, the Standard Errors of the Mean (SEMs) were calculated. For the adult scenario, the SEM for soil consumption was 6.51×10^{-8} kg/day, and for water consumption, it was 4.12×10^{-3} L/day.

These low SEM values confirm that random fluctuations were minimized, highlighting the reliability of the Monte Carlo sampling process in reproducing the intended input characteristics with high precision.

An expanded example of the verification process is provided using the “Body Weight-Adult (kg)” variable. Figure 5-2 shows the output distribution, where the input mean (72 kg) aligns closely with the calculated mean (~72.02 kg).

The calculated standard deviation (15.91 kg) also closely matches the input standard deviation (15.9 kg).

The standard error of the mean, calculated as the standard deviation of the sample divided by the square root of the number of realizations, is minimal at 0.050 kg.

These statistics, alongside the low SEM, reinforce the accuracy of the sampling process for this parameter.

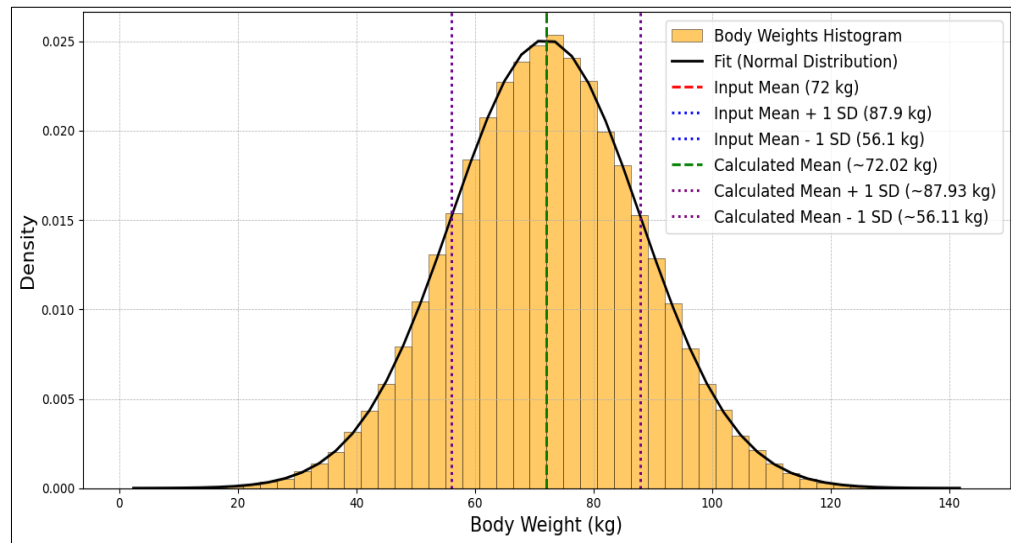


Figure 5-2: Distribution of body weights, showing close alignment between the input mean (72 kg) and the calculated mean (~72.02 kg), along with their respective standard deviations (± 1 standard deviation, SD)

In addition to Figure 5-2, Figure 5-3 shows a quantile–quantile (Q-Q) plot for body weights, demonstrating the alignment of the simulation data with the theoretical quantiles.

This visual confirmation supports the goodness-of-fit for the assumed normal distribution of the “Body Weight-Adult (kg)” variable and further verifies the accuracy of the Monte Carlo sampling process for this input parameter.

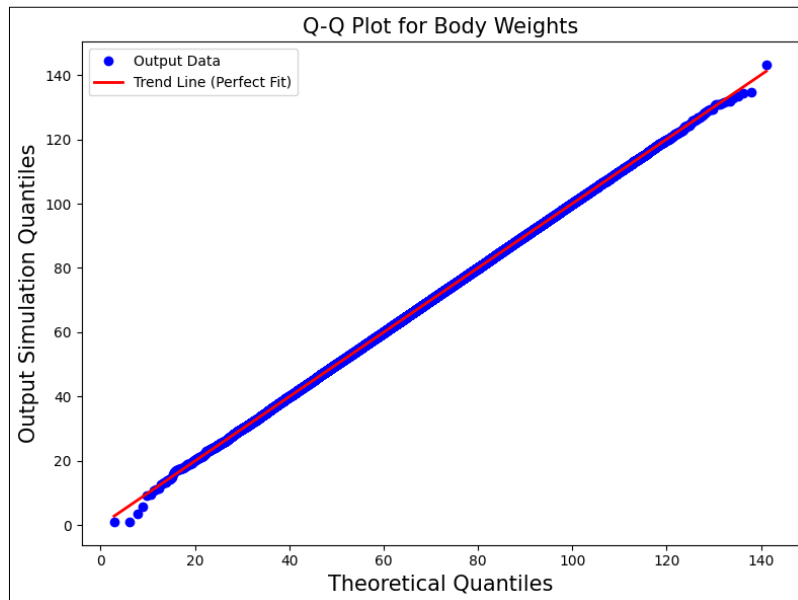


Figure 5-3: Q-Q plot for body weights, showing the alignment of the simulation data with the theoretical quantiles. The red trend line represents the ideal fit for a theoretical normal distribution

Another example of verification is provided for the “Exposure Duration (years)” parameter in the child scenario. Figure 5-4 shows the output distribution, where the input minimum (1 year) and maximum (6 years) match the calculated values.

Figure 5-5 presents a Q-Q plot, demonstrating a strong linear alignment between the simulated data and theoretical quantiles.

Together, these figures confirm that the Monte Carlo simulation reliably reproduces the characteristics of the uniform distribution.

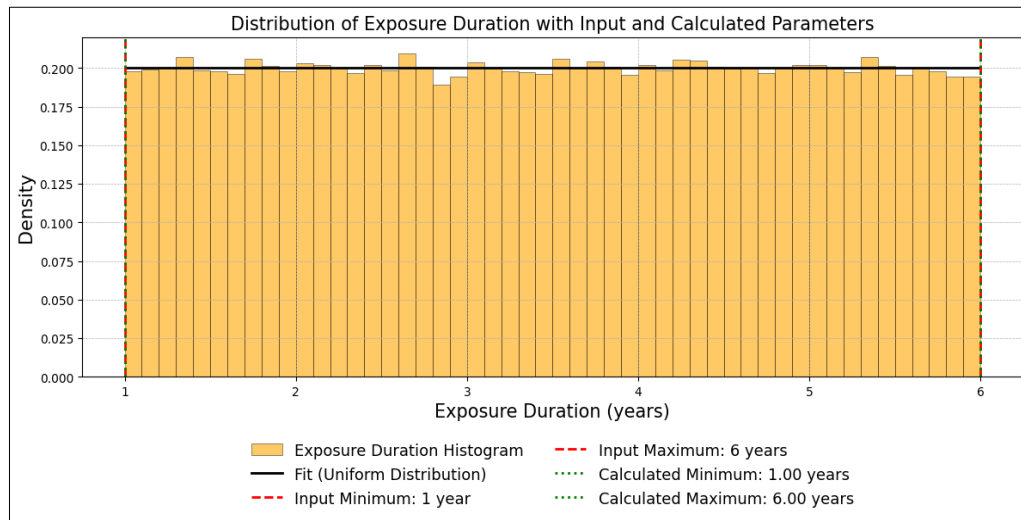


Figure 5-4: Distribution of exposure duration (years) for the child scenario, showing alignment between the input minimum (1 year) and maximum (6 years) and their respective calculated values

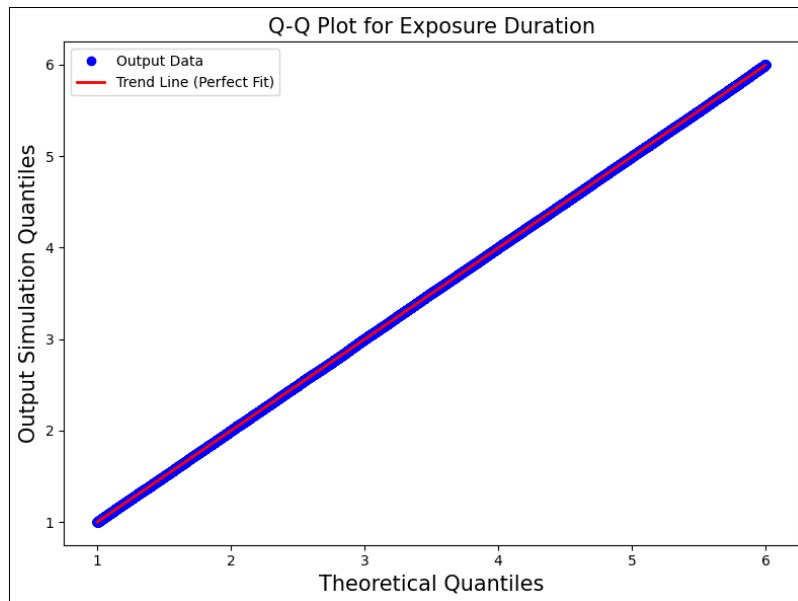


Figure 5-5: Q-Q plot for exposure duration (years) for the child scenario, showing the alignment of the simulation data with theoretical quantiles. The red trend line represents the ideal fit for a uniform distribution

The Monte Carlo simulations were also verified by comparing the model outputs with independent calculations performed in a spreadsheet.

Intermediate results for specific percentiles, such as the maximum cumulative risk, were printed from the Monte Carlo simulation code and cross-validated with manually derived results in the spreadsheet.

For example, the maximum cumulative risk calculated using the simulation matched the manual calculation down to a negligible difference ($\Delta = -1.11 \times 10^{-14}$), which is consistent with round-off errors inherent in numerical computations.

Each calculated risk, whether from benzene, formaldehyde, or benzo(a)pyrene exposure via different pathways (air, water, soil), showed no difference between the simulated and manually calculated risks, highlighting the exactness of the simulation's algorithm in processing and integrating complex exposure data.

This verification process establishes the reliability and precision of the Monte Carlo simulations, demonstrating their suitability for academic research and practical applications in environmental health risk assessments. By confirming alignment with input parameters and manual calculations, the model provides a robust foundation for conducting comprehensive risk analyses.

This ensures that the simulation outputs can reliably inform public health policy and intervention strategies, supporting efforts to mitigate exposure risks and prioritize vulnerable populations.

Figure 5-6 provides a visual breakdown of the stochastic risk characterization process, illustrating the progression from sensitivity analysis and variable distributions through random sampling, iterative calculations, and results aggregation, leading to verification and final decision-making.

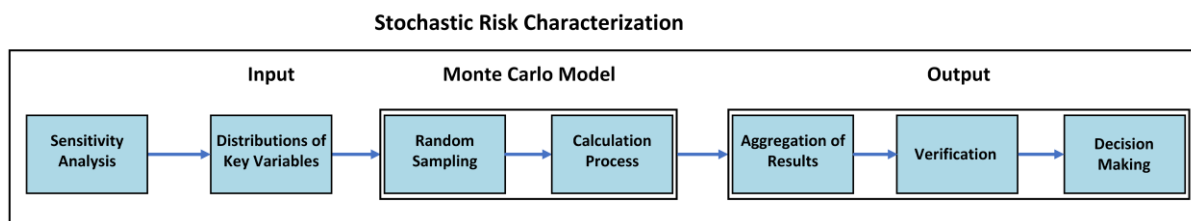


Figure 5-6: Visual breakdown of the stochastic risk characterization process

5.3 Results

5.3.1 Case Study

This case study demonstrates the application of the stochastic risk modeling method in assessing the health risks from MSATs near a high-traffic roadway.

This case study was conducted on the high-traffic roadway segments located at the junction of Interstate-35E, Interstate-94, and US-10 in Saint Paul, MN, USA. This location was selected due to the availability of data and past work [3].

These segments, identified by sequence number 11508 in the Traffic Mapping Application [43], provided 2019 traffic data for this analysis.

The study area, centered at coordinates 44°57'2.50" N, 93°5'54.60" W, is known for having annual average daily traffic (AADT) volumes among the top 5% in Ramsey County, indicating a high traffic flow that may impact local air quality. Figure 5-7 and Figure 5-8 display the location of these roadways and the AADT data, respectively.

Figure 5-8 shows the AADT data from 1998 to 2022 [246], with a 3-year moving average applied to capture the long-term trends while minimizing short-term fluctuations.

The Traffic Mapping Application, which integrates geographic information systems with traffic volume records, was utilized to identify the high-traffic roadway segments and provided all the traffic data for this analysis.

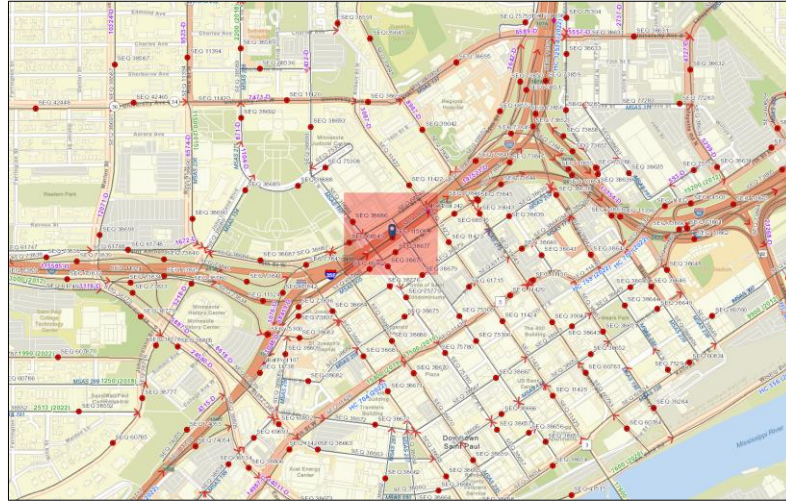


Figure 5-7: Map of the study area showing high-traffic roadways in Saint Paul, Minnesota. Red circles indicate traffic count locations; the blue marker highlights sequence number 11508, and the red box shows its position. Map data: Esri Community Maps contributors, County of Ramsey, Metropolitan Council, MetroGIS, © OpenStreetMap, Microsoft, Esri, TomTom, Garmin, SafeGraph, GeoTechnologies, Inc., METI/NASA, USGS, EPA, NPS, US Census Bureau, USDA, USFWS

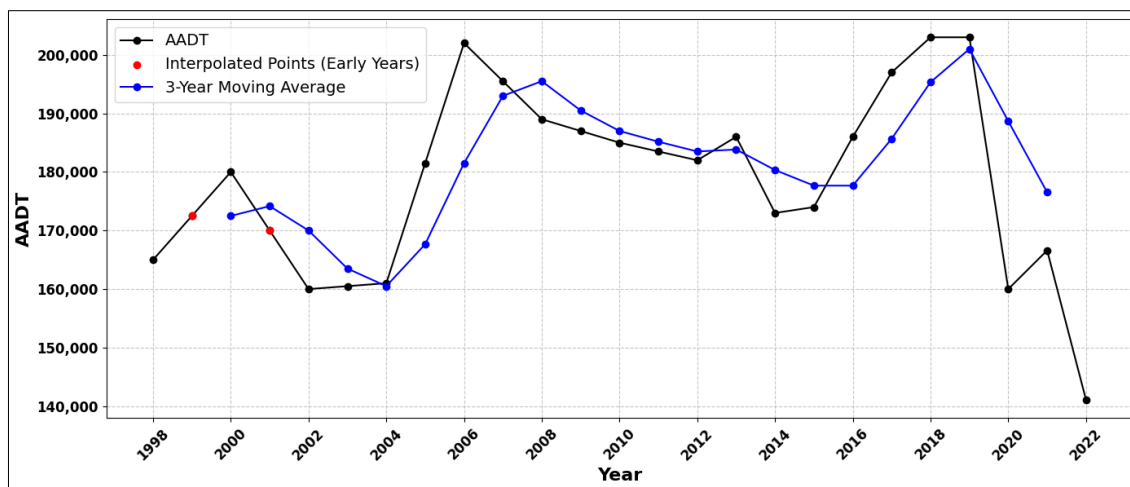


Figure 5-8: AADT at the study site with 3-year moving average. The 3-year moving average excludes the data points at the beginning (1998) and end (2022) due to insufficient data for a complete 3-year window. Linear interpolation was applied to estimate the missing data points in the early years (e.g., 1999 and 2001) to enable the calculation of a valid 3-year moving average

These traffic levels, which rank among the top 5% of AADT volumes in Ramsey County, lead to increased emissions of MSATs such as benzene, formaldehyde, and benzo(a)pyrene, all of which have been linked to serious health risks, including leukemia and cardiovascular diseases, as summarized in Table 5-1.

These three MSATs were selected for modeling in this study due to their severe health impacts, their prevalence in the study area, and their notable contribution to air pollution near high-traffic roadways [3, 5, 6, 8, 9].

While these pollutants represent only a fraction of the total MSATs, they pose substantial health risks, making them critical targets for risk assessment and regulatory actions.

Increased concentrations of these MSATs in urban environments result in measurable public health burdens [5, 6, 8, 9], particularly for vulnerable populations, including children, the elderly, and individuals with pre-existing health conditions, who are more susceptible to the adverse effects of air pollutants.

This case study provides a foundation for stochastic investigations into environmental exposures to MSATs, enabling a more precise quantification of the health risks and the identification of high-risk groups.

Additionally, it enhances the understanding of the variability and uncertainty in exposure data, which can inform risk management strategies in public health interventions and policy decisions.

These efforts are important in high-traffic urban areas, where the cumulative impacts of air pollution are most pronounced and require targeted mitigation efforts.

5.3.2 Deterministic Risk Results

The deterministic cumulative and multi-pathway cancer risk estimates for MSATs exposure in both adults and children are presented in this section.

These results, calculated from the deterministic model described in the Methodology section, focus on benzene, formaldehyde, and benzo(a)pyrene due to their significant health impacts and prevalence in the study area.

Table 5-7 and Table 5-8 provide a breakdown of the cancer risk by exposure pathway (inhalation, water ingestion, and soil ingestion) for each MSAT.

Any cases where the risk values exceed the regulatory thresholds, as discussed in Section 5.2.5, are indicated with “Yes” (exceeds) or “No” (does not exceed) in the respective tables.

Table 5-7: Cancer risk estimates for adults at the study site in Saint Paul, Minnesota, based on 2019 model results, with regulatory threshold exceedance (values represent unitless lifetime cancer risk)

MSATs	Risk from Exposure Pathways			Multi-Pathway Risk	Exceeds
	Inhalation	Water Ingestion	Soil Ingestion		
Benzene	1.30E-05	4.53E-04	3.23E-08	4.66E-04	Yes
Formaldehyde	2.18E-05	1.07E-07	7.64E-12	2.19E-05	Yes
Benzo(a)pyrene	1.81E-03	6.01E-02	4.29E-06	6.19E-02	Yes
Cumulative Risk	-	-	-	6.24E-02	Yes

Table 5-8: Cancer risk estimates for children at the study site in Saint Paul, Minnesota, based on 2019 model results, with regulatory threshold exceedance (values represent unitless lifetime cancer risk)

MSATs	Risk from Exposure Pathways			Multi-Pathway Risk	Exceeds
	Inhalation	Water Ingestion	Soil Ingestion		
Benzene	2.63E-06	1.69E-04	7.77E-08	1.72E-04	Yes
Formaldehyde	4.35E-06	3.99E-08	1.83E-11	4.39E-06	Yes
Benzo(a)pyrene	3.63E-04	2.24E-02	1.03E-05	2.28E-02	Yes
Cumulative Risk	-	-	-	2.30E-02	Yes

Both the cumulative and multi-pathway cancer risks, $6.24\text{E-}02$ for adults and $2.30\text{E-}02$ for children, exceed the established regulatory thresholds at the study site in Saint Paul, Minnesota, based on the 2019 data.

To further contextualize these cancer risk estimates, the adult cancer risk of $6.24\text{E-}02$ (approximately 1 in 16) is elevated relative to regulatory thresholds but remains lower than the lifetime odds of dying from heart disease, which is 1 in 6.

For children, the cancer risk estimate of $2.30\text{E-}02$ (roughly 1 in 43) is comparable to the odds of dying from an accidental opioid overdose (1 in 55).

These comparisons are based on the lifetime odds of death from selected causes in the United States [144].

5.3.3 Stochastic Risk Results

This section presents the stochastic cumulative and multi-pathway cancer risk estimates for MSAT exposure in both adults and children, derived from Monte Carlo simulations.

Unlike the deterministic approach, which provides a single-point estimate, the stochastic analysis captures the variability and uncertainty inherent in the risk assessment process.

The results include probabilistic ranges for individual MSATs and cumulative multi-pathway risks for adults and children, offering a more comprehensive characterization of key drivers and risk distributions.

Table 5-9 shows the multi-pathway stochastic cancer risk estimates for individual MSATs (benzene, formaldehyde, and benzo(a)pyrene) in adults and children.

The table displays key percentiles (p5, p50, p95) from the cumulative and multi-pathway cancer risk distributions.

The fifth percentile (p5) represents the lower bound, indicating where only 5% of the simulated risk values fall below this level.

The median (p50) denotes the central tendency of the distribution, while the 95th percentile (p95) reflects the upper bound, often used as a conservative estimate of high-risk scenarios.

Table 5-9: Multi-pathway stochastic cancer risk estimates (unitless) for adults and children from individual MSATs at the study site in Saint Paul, Minnesota, based on 2019 model results and 100,000 realizations

MSATs/ Scenario	Adults			Children		
	Percentile (p5)	Median (p50)	Percentile (p95)	Percentile (p5)	Median (p50)	Percentile (p95)
Benzene	4.04E-06	3.60E-05	3.72E-04	2.16E-06	1.60E-05	1.70E-04
Formaldehyde	9.04E-07	4.44E-06	2.13E-05	7.55E-07	2.11E-06	3.78E-06
Benzo(a)pyrene	5.41E-04	4.79E-03	4.94E-02	2.92E-04	2.13E-03	2.26E-02

Based on the individual MSAT estimates, Table 5-10 presents the cumulative and multi-pathway stochastic cancer risk estimates for adults and children, showing the combined impact of MSAT exposure.

Cumulative risk is the total cancer risk from exposure to multiple MSATs and emission sources, whereas multi-pathway risk accounts for contributions from different exposure routes, such as inhalation and ingestion.

Table 5-10: Stochastic cancer risk estimates (unitless) for adults and children at the study site in Saint Paul, Minnesota, based on 2019 model results. All values represent estimates of cumulative and multi-pathway cancer risk from MSAT exposure

Scenario	Percentile (p5)	Median (p50)	Percentile (p95)
Adults	5.47E-04	4.84E-03	4.98E-02
Children	2.95E-04	2.14E-03	2.28E-02

The stochastic risk estimates provide a range of potential risk outcomes, in contrast to the point estimates from the deterministic approach.

For adults, the 95th percentile cumulative risk is 4.98E-02, suggesting a potential upper bound of risk that is substantial and close to the deterministic estimate of 6.24E-02.

Similarly, for children, the 95th percentile risk is 2.28E-02, reflecting variability while confirming the high-risk level indicated by the deterministic estimate of 2.30E-02.

Notably, the median (p50) stochastic estimates are lower than the deterministic values for both adults and children, reflecting the conservative nature of deterministic methods, which rely on upper-bound assumptions [37].

To represent the spread and variability in the cumulative risk estimates, Figure 5-9 and Figure 5-10 show the relative frequency density (as an empirical approximation of the PDF) alongside the cumulative probability distribution for both adult and child scenarios.

These figures demonstrate how the stochastic risk distributions extend beyond deterministic estimates, providing a more comprehensive view of the potential health impacts of exposures to MSATs.

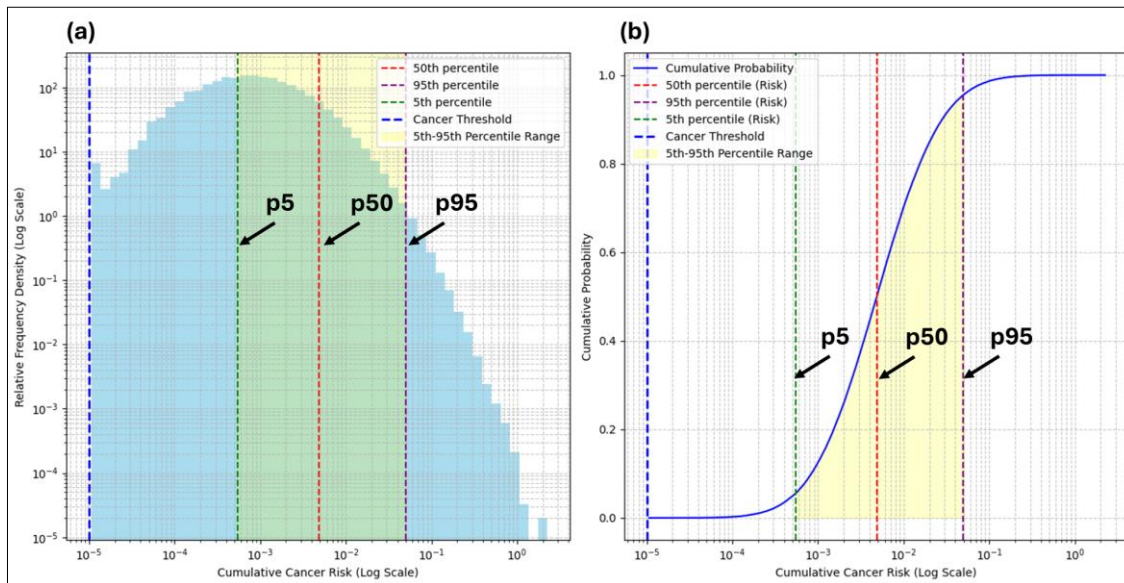


Figure 5-9: Relative frequency density plot (a) and cumulative probability plot (b) for adult cumulative cancer risk estimates, showing a positively skewed distribution, distributional spread, and risk thresholds

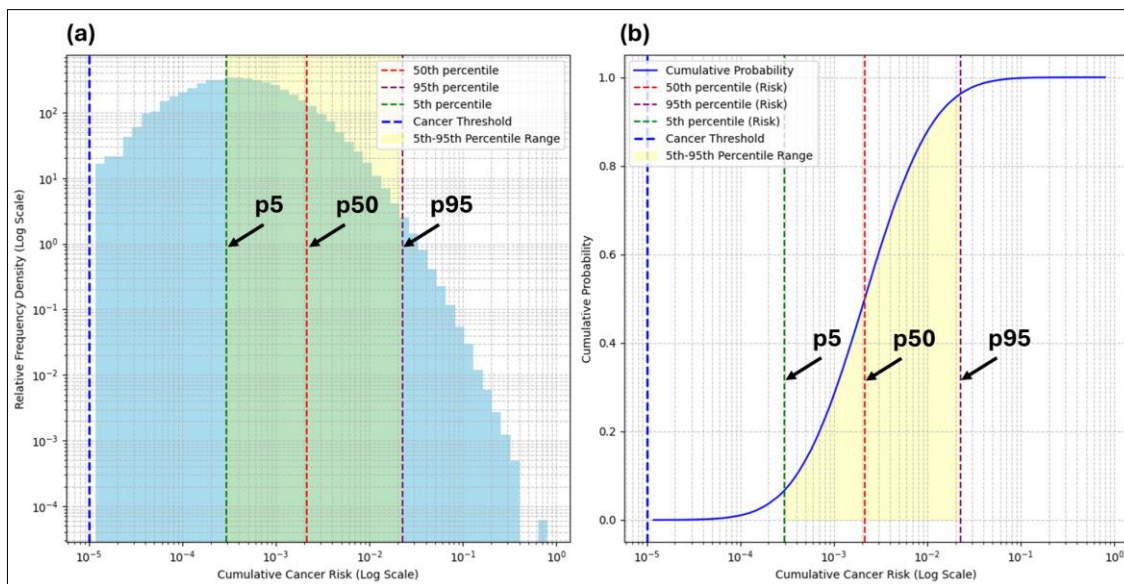


Figure 5-10: Relative frequency density plot (a) and cumulative probability plot (b) for child cumulative cancer risk estimates, showing a positively skewed distribution, distributional spread, and risk thresholds

The relative frequency density plots for both the adult and child scenarios in Figure 5-9 and Figure 5-10 show the spread and variability in the cumulative cancer risk estimates. The shaded regions between the dashed 5th and 95th percentile lines visually emphasize the range where the majority of the simulated risks fall, while the dashed lines themselves mark precise thresholds.

Together, these elements illustrate both the variability and provide key reference points critical for interpreting the stochastic risk estimates.

The inclusion of the percentile lines (5th, 50th, and 95th) aligns with the risk estimates in Table 5-9 and Table 5-10. The 95th percentiles are emphasized as they represent the upper bound of the risk scenarios, which are critical for regulatory benchmarks and health risk assessments, especially in identifying high-risk subpopulations.

The cumulative probability plots complement the relative frequency density plots by showing the likelihood of the risk estimates falling below various thresholds, with the cancer threshold line marking the key decision-making points.

The log scale used in both plots accommodates the broad range of simulated risk values, allowing for clearer distinctions between the lower and higher-risk levels that might otherwise be compressed on a linear scale.

The drop-off at the upper end of the cumulative probability plots occurs because the probability becomes low for higher risk values, even with 100,000 realizations.

This drop-off behavior reflects the reduced likelihood of extreme outcomes in stochastic modeling while capturing the full range of variability.

5.4 Discussion

5.4.1 Cancer Risk Drivers

The cancer risk estimates reveal that benzo(a)pyrene is the dominant risk driver (contribution 4 in Section 5.1.4) for both adults and children.

Its significantly higher CSF of $7.3 \text{ (mg/kg-day)}^{-1}$ (see Table 5-5 for the pollutants' CSF and URF values), along with its environmental persistence and bioaccumulation potential [247], leads to elevated multi-pathway risks, particularly from the water ingestion pathway.

Water ingestion is the dominant contributor to risk for both benzo(a)pyrene and benzene. Inhalation serves as a secondary contributor to risk for these compounds. For formaldehyde, inhalation remains the major risk contributor. Its contribution through ingestion pathways is minimal due to its lower CSF of $1.3 \times 10^{-5} \text{ (mg/kg-day)}^{-1}$.

Soil ingestion contributes minimally to the overall cancer risk for all of the modeled compounds. However, in children, its contribution is slightly higher due to behaviors such as hand-to-mouth activity and their lower body weight, which increases the relative dose.

5.4.2 Comparison of Adults to Children

The adult cancer risk is notably higher than the child cancer risk, primarily due to the longer exposure duration assumed for adults (30 years versus 6 years for children).

Despite this, benzo(a)pyrene remains the primary contributor to elevated risks in both populations, driven by its potent carcinogenicity and contributions from both inhalation and water ingestion pathways.

5.4.3 Necessity of a Cumulative Risk Assessment

A cumulative risk assessment is essential to understanding the combined exposure burden from multiple MSATs across various pathways.

While the cancer risk from individual MSATs like benzene, formaldehyde, and benzo(a)pyrene may fall below the regulatory thresholds when they are evaluated separately, their combined exposure often exceeds these thresholds, as demonstrated by the cumulative risk estimates in Table 5-10.

For example, the 50th percentile stochastic risk for formaldehyde (children scenario) is 2.11×10^{-6} , below the regulatory threshold of 1×10^{-5} , but the cumulative risks for both adults and children exceed these limits, highlighting the additive burden of combined exposures.

Children are particularly vulnerable due to their developing physiology, increased exposure rates, and behaviors such as outdoor play, which elevate their contact with pollutants.

As shown in Table 5-10, the cumulative cancer risks for children exceed the regulatory thresholds, emphasizing the need to address health disparities among subgroups with heightened susceptibility.

By capturing the additive impacts of exposures across pollutants and pathways, cumulative risk assessments provide a more realistic representation of health risks.

Traditional assessments focusing on individual pollutants fail to account for these combined effects, potentially underestimating the true public health burden.

This study demonstrates that stochastic modeling enhances the cumulative risk characterization by incorporating variability, providing actionable insights for targeted interventions, particularly for high-risk groups like children and the elderly in high-traffic urban areas.

5.4.4 Evaluation of Realistic and Unrealistic Exposure Pathways

The water ingestion pathway significantly contributes to the calculated cancer risks but does not reflect the realistic conditions in the urban, commercially zoned case study area.

Its intentional inclusion demonstrates the methodology's flexibility and adaptability in evaluating diverse exposure pathways, as recommended by the HHRAP.

Despite influencing the overall calculated cancer risk estimates derived from the model, the water ingestion pathway serves as a methodological example rather than a real-world impact.

If the pathway does not exist in the study area, the associated risk does not occur, emphasizing the need to interpret risks within the context of realistic exposure scenarios and localized environmental conditions for accurate and actionable conclusions.

5.4.5 Comparing Stochastic and Deterministic Models: Outcomes, Pros, and Cons

This section compares deterministic and stochastic models, highlighting their key differences, advantages, and limitations in risk assessment, as summarized in Table 5-11.

Table 5-11: Differences between stochastic and deterministic risk models

Criterion	Deterministic Model	Stochastic Model
Output	Single-point risk estimate	Probabilistic range of risk estimates
Variability Captured	No, uses fixed parameter values	Yes, reflects real-world variability
Analytical Scope	Worst-case or average exposure scenarios	Explores the full range of variability and identifies high-risk scenarios (e.g., 95th percentile)
Ease of Interpretation	Simple to interpret for practitioners	Accessible to practitioners (e.g., air quality engineers, risk analysts, regulators, and policymakers) through visualizations like cumulative distribution functions
Computational Needs	Limited computational resources required	Requires considerable computational resources
Decision-Making	Limited insights for vulnerable populations or worst-case scenarios	Supports targeted interventions and effective decision-making

Stochastic modeling for MSATs addresses the limitations of the deterministic methods by generating probabilistic outputs that reflect the variability across populations and exposure scenarios.

These outputs, such as cumulative distribution functions, facilitate the identification of high-risk scenarios and support effective interventions, including emission control strategies, urban zoning policies, and tailored public health initiatives for vulnerable populations.

Though stochastic modeling requires more computational resources, advances in parallel computing and cloud-based platforms mitigate this challenge, making it a viable option for comprehensive risk assessments.

By providing a detailed characterization of variability and uncertainty, this approach equips practitioners with actionable insights to develop data-driven strategies for mitigating MSATs exposure risks.

5.5 Conclusions

This research addresses critical limitations of deterministic methods in human health risk assessments for MSATs by developing a stochastic method tailored to on-road mobile sources.

Deterministic methods, reliant on point estimates, often overlook the variability among individuals and the vulnerabilities of sensitive subpopulations.

In contrast, stochastic modeling provides a probabilistic framework that captures variability, enabling a comprehensive evaluation of health risks.

The key findings from this case study demonstrate that the stochastic modeling approach provides actionable insights.

The deterministic method estimated cumulative cancer risks of $6.24\text{E-}02$ for adults and $2.30\text{E-}02$ for children. In contrast, the stochastic method provided a range of risk estimates, with the 95th percentile equal to $4.98\text{E-}02$ for adults and $2.28\text{E-}02$ for children.

These findings demonstrate that the stochastic method for MSATs captures variability by simulating a distribution of cancer risk estimates, enabling consideration of worst-case scenarios and providing a realistic depiction of the variability.

The key contributions of this research are as follows:

1. **Development of a Stochastic Method for Mobile Sources:** This research introduces the first stochastic method extended and adapted to on-road mobile source emissions, which fills a critical gap in environmental health risk assessment by accounting for the variability in the MSAT exposures across population groups.
2. **Incorporation of Spatiotemporal Dynamics for Enhanced Emission Characterization:** This research incorporates hourly data on wind speeds, traffic patterns, and emissions to address the episodic and localized nature of mobile source exposures, improving upon the limitations of annual averages with dynamic and refined risk assessments.
3. **Advancement of Public Health Strategies for Vulnerable Populations:** This work provides actionable insights for public health policies, facilitating the identification of high-risk zones and enabling targeted interventions to reduce MSAT exposure risks in urban environments.

In conclusion, the stochastic method for MSATs advances human health risk assessment by addressing variability and offering a refined framework, as detailed in this section.

5.6 Future Work and Applications

Building on this research, several avenues for future studies and practical applications are recommended to improve public health outcomes and enhance air quality:

1. Development of health index metrics: Create composite health indices to integrate the impacts of multiple MSATs and exposure pathways, providing a holistic measure of risk.
2. Exploration of indoor–outdoor air interactions: Investigate how outdoor air quality influences the indoor pollutant levels in sensitive environments such as homes, schools, and workplaces. Understanding the interaction between outdoor infiltration and indoor emissions is essential for addressing complex exposure patterns and refining risk assessments.
3. Characterization of indoor pollutant sources: Use stochastic methods to analyze indoor emissions and evaluate cumulative exposures. Understanding these is vital for total health risk assessment, as individuals spend most of their time indoors.

4. Ecological applications: Expand stochastic modeling to evaluate the ecological risks from mobile emissions, improving predictions of environmental outcomes and supporting ecosystem management strategies.

Chapter 6. Conclusions and Future Work

6.1 Conclusions

This dissertation advances air toxics risk assessment through four published, peer-reviewed studies, each presenting a validated or demonstrated methodology applied to a real-world case study. Together, these contributions form a suite of approaches that improve fidelity (how accurately models represent real-world conditions), scalability (applicability to country- or region-wide scales), and interpretability (clarity and usability of model outputs for decision-making and communication) of both human and ecological health risk characterizations.

The research addresses critical gaps in existing literature and practice, including reliance on deterministic models, limited integration of multiple exposure pathways, and insufficient ecological analysis. It introduces methodological advancements in emissions inventory development, air dispersion modeling, multi-source and multi-pathway cumulative exposure assessment, and stochastic risk quantification. These methods are applied to mobile and stationary sources, enabling a more comprehensive and realistic understanding of population-level and ecosystem-scale risks.

Chapter 2 presents the first deterministic methodology for cumulative, multi-pathway human health risk assessment specific to MSATs. It integrates mobile-source emissions inventories (using MOVES), air dispersion modeling (AERMOD), and exposure estimation across inhalation and ingestion routes. This framework supports source attribution and spatial prioritization for mitigation and establishes a foundation for subsequent probabilistic modeling in Chapter 5.

In a Saint Paul, Minnesota case study, benzo(a)pyrene cancer risks exceeded the target level for carcinogens at several urban receptor sites, with significant contributions from non-inhalation exposure.

Chapter 3 presents a methodology for country-wide assessment of cumulative health risks from stationary sources, including glycol dehydration units, wastewater treatment plants, and oil and gas operations. The evaluation of emissions across distinct air quality zones demonstrates the feasibility of multi-source, multi-pathway modeling at national and regional scales and enables cross-zone risk comparisons.

This approach revealed zone-specific risk patterns that localized assessments would likely overlook. While cumulative cancer and noncancer risks were generally within acceptable levels, the Ahmadi Hospital site exceeded the target cancer risk level for adult residents due to benzo(a)pyrene. These findings support the application of regional-scale modeling to guide national planning and targeted mitigation.

Chapter 4 introduces the new Ecological Health Assessment Methodology (EHAM), a significant advancement in ecological risk analysis for air toxics. It adapts and extends the U.S. EPA's SLERAP framework, develops habitat-specific food webs, and quantifies exposure and bioaccumulation across terrestrial and aquatic ecosystems. A country-wide application revealed elevated risks in Kuwait's coastal zone, particularly for carnivorous shorebirds, due to benzo(a)pyrene biomagnification. These findings reinforce its role as a key risk driver and highlight the need for ecosystem-specific models and targeted policy responses.

A key contribution of this research is the development of the first stochastic human health risk assessment methodology specifically tailored for MSATs. Presented in Chapter 5, this verified method uses Monte Carlo simulations to account for inter-individual variability in key exposure parameters such as body weight, inhalation rate, exposure duration, and dietary intake. This probabilistic framework addresses longstanding limitations in MSAT risk modeling, particularly the inability to represent variability and uncertainty in individual exposures.

In the Saint Paul, Minnesota case study, the model computed full cancer risk distributions for adults and children, including the 5th, 50th (median), and 95th percentiles. The 95th percentile revealed high-risk exposure scenarios and highlighted vulnerable subpopulations. Because MSAT exposures fluctuate with location, time of day, and population activity patterns, deterministic models may oversimplify risk and mask such variability. This stochastic approach provides a more accurate representation of real-world exposure dynamics and enhances the scientific credibility and regulatory utility of MSAT risk estimates.

The benefits of the stochastic methodology developed in this research include:

1. Captures inter-individual variability in exposure and dose by using probabilistic inputs for key exposure factors such as inhalation rate, body weight, and exposure duration. This enables more representative population-level risk estimates.
2. Quantifies uncertainty in risk estimates through full probability distributions, enabling percentile-based outputs (e.g., 95th percentile) that deterministic models cannot provide. This strengthens confidence in risk communication and regulatory thresholds.
3. Identifies vulnerable subpopulations by disaggregating risk by age, location, and behavior. This enables targeted health protection strategies and supports environmental justice assessments.
4. Generates cumulative, multi-pathway cancer risk distributions instead of fixed values. This improves interpretation of total exposure across inhalation and ingestion routes.
5. Supports scenario-based sensitivity and uncertainty analysis to prioritize influential parameters. This guides efficient data collection and resource allocation in risk management.
6. Provides a mobile-source-specific stochastic framework with public health relevance, enabling identification of urban MSAT hotspots, characterization of population-level risk variability, and support for equity-focused mitigation. This strengthens its utility for exposure management and urban risk reduction.

These findings confirm that all five research objectives were met, which in turn led to the contributions listed on the following page. The methodologies developed across the four articles directly address the key gaps in MSATs and air toxics risk assessment identified in Chapter 1.

The academic contributions of this thesis are as follows:

1. Introduce the first deterministic methodology for cumulative, multi-pathway human health risk assessment of MSATs.
2. Improve emissions inventory quality using validated methods and high-resolution spatiotemporal inputs, recognizing that accurate and detailed emissions data are fundamental to credible air toxics risk assessment.
3. Refine air dispersion and deposition modeling using unitized emissions, receptor-specific concentrations, and spatiotemporally resolved emissions estimates, improving exposure resolution for both human and ecological assessments.
4. Enable detailed cumulative and multi-pathway risk assessments from mobile and stationary sources by integrating inhalation and ingestion pathways, and allocating the contribution of each source to overall health risk.
5. Identify cancer risk drivers to inform targeted control measures, with benzo(a)pyrene consistently emerging across all case studies as a candidate for prioritized mitigation.
6. Advance ecological risk assessment through the development of habitat-specific food web models and the application of EHAM, enabling cumulative, trophic-level-based screening of air toxics across terrestrial and aquatic ecosystems to support ecological risk management.
7. Perform a sensitivity analysis to identify dominant variables affecting the risk results, ensuring a focused and resource-efficient stochastic risk assessment.
8. Introduce probabilistic methods to characterize variability and uncertainty in MSATs exposure, supporting more informative and defensible risk decisions.
9. Support future application to other classes of chemicals by enabling the integration of additional exposure pathways into the stochastic framework, laying the groundwork for broader use in emerging contaminant assessments.

This integrated risk architecture provides a foundation for engineers, researchers, and policymakers to assess and manage air toxics risks more effectively across sources, pathways, and receptors, supporting comprehensive and equitable decision-making.

6.2 Future Work

Following the technical advancements introduced in this research, several directions for future work are recommended.

- **Technical model refinements**

1. Expand stochastic modeling to ecological exposure parameters, including variability in food ingestion rates and dietary composition of key ecological receptors, to support future development of a probabilistic ecological risk framework.
2. Streamline the full modeling workflow into an open-source or modular platform. Support this with interactive dashboards and visualizations (e.g., cumulative distribution functions) to make Monte Carlo outputs more accessible to air quality engineers, risk analysts, regulators, and policymakers. This would enhance usability, transparency, and the integration of stochastic results into real-world planning and policy.
3. Refine stochastic human health scenarios by moving beyond the adult–child split to incorporate finer age bins (e.g., infants, toddlers, adolescents, older adults) for more detailed exposure and risk estimates.

- **Application to additional sources and contaminants**

1. Extend human health and ecological risk models to include emerging airborne contaminants (e.g., ultrafine particles, airborne endocrine-disrupting compounds, microplastics), which are increasingly recognized as relevant to human and ecological exposure but remain largely unregulated.
2. Account for overlooked non-tailpipe VOC sources such as washer fluids and deicing chemicals, particularly in electric vehicles, to improve inventory completeness and exposure assessments. This is critical as fleet electrification shifts the VOC emissions landscape toward non-combustion-related sources.

- **Policy and regulatory integration**

1. Embed stochastic methods into permitting processes, cumulative impact frameworks, and environmental justice assessments.

2. Develop composite health indices to integrate the cumulative impacts of multiple MSATs and exposure pathways, supporting holistic risk characterization and regulatory prioritization.
- **Scenario-based risk forecasting**
 1. Couple risk models with land-use change, transportation development, and vehicle electrification scenarios to assess how future infrastructure and regulatory shifts may alter air toxics exposures.

6.3 Research Application

The methodologies and frameworks developed in this research are applicable across multiple stakeholder domains:

1. Environmental regulators can apply the refined emissions inventory, air dispersion modeling, and multi-pathway risk assessment tools for air toxics permitting, compliance evaluation, and cumulative impact screening. The stochastic framework allows for risk ranges and exceedance probabilities, improving decision defensibility.
2. Urban planners and transportation engineers can incorporate near-road exposure risk data into transportation design and mitigation strategies to reduce population exposure, particularly in high-traffic urban areas and sensitive land-use zones.
3. Public health researchers and exposure scientists can link stochastic risk outputs to health burden models or integrate them with biomonitoring and epidemiologic datasets to improve attribution of health outcomes to mobile-source pollution.
4. Industry and environmental advocates can use the multi-scale modeling tools in community risk assessments, stakeholder engagement, and environmental impact studies. The ability to disaggregate risk by source, pathway, and receptor supports transparent and participatory decision-making.

These applications highlight the practicality and transferability of the proposed methodologies. The research offers a technical foundation that connects emissions data, exposure science, and health or ecological endpoints, advancing academic understanding and enabling the practical implementation of evidence-based environmental management.

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Appendices

Appendix A

This appendix provides supporting information for the analysis in the Prognostic Weather Setup section of Chapter 2. After WRF and MMIF were successfully executed, wind speed, wind direction, and ambient temperature were extracted from the AERMOD-ready surface files and compared to data from two nearby weather stations. To validate the performance of the WRF model, these variables were assessed using statistical metrics such as bias, mean absolute error, root mean square error, and index of agreement. These metrics are standard tools for assessing the accuracy of meteorological models and are defined below.

- Bias (b): Average difference between modeled and observed values. Positive values indicate model overprediction, while negative values indicate underprediction.
- Mean Absolute Error (MAE): Mean of the absolute differences between modeled and observed values, providing an average magnitude of error without regard to direction.
- Root Mean Square Error (RMSE): Square root of the mean of squared differences between modeled and observed values, giving greater weight to larger errors.
- Index of Agreement (IOA): A dimensionless measure (0 to 1) of agreement between modeled and observed values, where 1 represents perfect agreement.

The statistical metrics for the WRF model were compared against established performance benchmarks for meteorological models, as presented in Table A-1.

Table A-1: Statistical benchmarks for evaluating model performance

Meteorological Variable	b	MAE	RMSE	IOA
Wind Direction (deg)	$\leq \pm 10$	≤ 30	-	-
Wind Speed (m/s)	$\leq \pm 0.5$	-	≤ 2	≥ 0.6
Temperature (°C)	$\leq \pm 0.5$	≤ 2	-	≥ 0.8

Tables A-2 and A-3 present statistical metrics for the WRF model compared to observations from the weather stations at Saint Paul Downtown Airport and Minneapolis–Saint Paul International Airport, respectively.

Table A-2: 2011 statistics for WRF extracted points compared to observations at Saint Paul Downtown Airport

Meteorological Variable	b	MAE	RMSE	IOA
Wind Direction (deg)	−6.18	32.54	-	-
Wind Speed (m/s)	0.49	-	1.48	0.84
Temperature (°C)	0.46	2.25	-	0.98

Table A-3: 2011 statistics for WRF extracted points compared to observations at Minneapolis–Saint Paul International Airport

Meteorological Variable	b	MAE	RMSE	IOA
Wind Direction (deg)	−7.74	28.6	-	-
Wind Speed (m/s)	0.68	-	1.43	0.86
Temperature (°C)	0.90	2.25	-	0.99