PARKLAND REFINING (B.C.) LTD.

AIR EMISSIONS HUMAN HEALTH RISK ASSESSMENT

JANUARY 31, 2022

VERSION 1.0

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PARKLAND REFINING (B.C.) LTD.

PROJECT NO.: 211-04808-00
DATE: JANUARY 31, 2022
VERSION: 1.0

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January 31, 2022

PARKLAND REFINING (B.C.) LTD.
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Attention: Vicki Bowman, Manager, Environmental

Dear Madam:

Subject: Parkland Burnaby Refinery Air Emissions Human Health Risk Assessment

WSP Canada Inc. was retained by Parkland Refining (B.C.) Ltd. ("Parkland") to prepare a Human Health Risk Assessment ("HHRA") for the Burnaby Refinery and tank farm ("the refinery"). The HHRA was completed in support of Parkland's planned application to amend the Air Quality Management Permit for the refinery to incorporate the results of Parkland's Refined Technology Assessment.

Please find attached for your review and comment, the HHRA, including tables, figures, and appendices.

Sincerely,

Francis Ries, B.Sc., P.Eng.
Senior Air Quality Engineer

Theresa Repaso-Subang, HBSc., DABT, ERT, QPRA
Senior Technical Lead & Team Lead – Toxicology & Risk Assessment

FJR/trs
Encl.
cc:
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SIGNATURES

Theresa Repaso-Subang, H.BSc., DABT, ERT, QPRA
Senior Technical Lead & Team Lead – Toxicology & Risk Assessment

Francis Ries, B.Sc., P.Eng.
Senior Air Quality Engineer

EXECUTIVE SUMMARY

Parkland Refining (B.C.) Ltd. ("Parkland") operates the Burnaby refinery and tank farm ("the refinery") under the authorization of Metro Vancouver Regional District ("MVRD") air discharge permit GVA0117, which is set to expire on July 31, 2022. With the expiration date approaching, Parkland is well underway in the application process to renew and amend the existing permit to align with Parkland's commitment to investing in new technologies and infrastructure to reduce air emissions. As part of the permit application process, Parkland retained WSP Canada Limited (WSP) to prepare this Human Health Risk Assessment ("HHRA"), which builds on an Air Quality Dispersion Assessment ("AQA") that WSP has already completed (WSP, 2021).

A HHRA is a scientific process that predicts the nature and likelihood of harmful health effects that may occur should people be exposed to chemicals in the environment. The current HHRA was formally requested by Fraser Health Authority ("FHA") in December 2020 in anticipation of Parkland's permit amendment application. Pursuant to this request, a HHRA workplan describing the proposed approach and methodology for completing the HHRA was submitted to MVRD, the FHA, and the First Nations Health Authority for review on June 22, 2021 and was accepted by all of these agencies on or before August 20, 2021. Following the accepted workplan, WSP has conducted the HHRA, incorporating comments and feedback received from consultation with the aforementioned agencies, the Parkland Community Advisory Panel ("CAP"), members of the general public, the City of Burnaby, Vancouver Coastal Health, and Tsleil-Waututh Nation.

PROBLEM FORMULATION

The HHRA problem formulation defines the "where, what, when, who, how" of the assessment. Two previous health risk assessments for the refinery completed in 2002 and 2013 provided important guidance in establishing the following problem formulation for the current assessment:

- **Where**: a 10-kilometre ("km") x 10 km study area centered on the refinery, which encompasses all locations predicted by the AQA to potentially experience elevated levels of refinery-source contaminants of potential concern ("COPC"). This assessment area is significantly larger than previous assessments, which focused more narrowly on the North Burnaby area near the refinery.
- **What:** COPCs included in this assessment include sulphur dioxide ("SO_2"), nitrogen dioxide ("NO_2"), fine particulate matter ("$PM_{2.5}$"), the three pollutants for which the permit amendment is requesting reductions in emissions limits. Volatile Organic Compounds ("VOC") benzene and 1,3-butadiene are also included. The previous 2002 and 2013 assessments identified SO_2, benzene and 1,3-butadiene as key COPC.
- **When**: 2017-2019 air quality monitoring data from MVRD stations surrounding the refinery were utilized for one of the 4 HHRA scenarios (more detail in "Exposure Assessment" below), and AQA dispersion modelling output data based on meteorological input from May 1, 2018 through April 30, 2019 were utilized for the other 3 modelled scenarios.
- **Who:** Receptor groups included in the study were residents of the study area including seniors in long term care, attendees of daycares, schools and hospitals in the study area, adults who work near the refinery, visitors/recreational users of areas near the refinery, and Tsleil-Waututh Nation members using their lands for cultural practices.
- **How:** The exposure pathways identified for the assessment included inhalation of COPC emitted into ambient air, and skin contact / ingestion of COPC deposited onto assessment area soils. A screening assessment of the deposition rates of refinery emissions indicated that deposition levels were below applicable thresholds, and as such, skin contact and ingestion were not assessed further.

EXPOSURE ASSESSMENT

The exposure assessment estimates levels of COPC that receptors throughout the study area are exposed to. Four exposure assessment scenarios were defined to identify potential changes in health impacts associated with Parkland's permit amendment application:

- **Scenario 1 – 2017-2019 Ambient Monitoring**: Exposure data from MVRD network stations located within the HHRA Study Area. The most recent three years of validated monitoring data (2017-2019) for SO_2, NO_2, $PM_{2.5}$, benzene and 1,3-butadiene were used to derive exposure concentrations.
- **Scenario 2 – Current Permit Maximum**: Exposure data from dispersion modelling results for maximum emissions authorized under MVRD Permit GVA0117 dated January 27, 2021 for all refinery sources of SO_2, nitrogen oxides (NO_x), $PM_{2.5}$.
- **Scenario 3 – Amended Permit Maximum**: Exposure data from dispersion modelling results for maximum emissions limits requested in Parkland's permit amendment application to MVRD for all refinery sources of SO_2, NO_x, $PM_{2.5}$. These limits incorporate emission reductions associated with Fluid Catalytic Cracker ("FCC") SO_2 and NO_x reduction additives, future installation of the Tail Gas treatment Unit ("TGTU") and CO Boiler flue gas recirculation, and past installation of the FCC Third Stage Separator ("TSS").
- **Scenario 4 – Amended Permit Normal**: Exposure data from dispersion modelling results for expected normal operating levels for all refinery sources of SO_2, NO_x, $PM_{2.5}$, following the implementation of all changes detailed in Scenario 3.

HAZARD ASSESSMENT

The hazard assessment identifies the potential health effects associated with short-term (acute) and long-term (chronic) exposures to COPCs selected for assessment, utilizing health agency databases and academic literature to establish toxicological reference values ("TRV") that are protective of non-cancer and cancer health endpoints. For this assessment, health-protective TRVs were established for acute exposure to SO_2, NO_2, $PM_{2.5}$, benzene and 1,3-butadiene, and for chronic exposure to NO_2, $PM_{2.5}$, benzene and 1,3-butadiene.

RISK CHARACTERIZATION / RESULTS

The risk characterization brings together the results of the Exposure Assessment and Hazard Assessment to estimate the health risks associated with COPC exposures for each receptor group throughout the study area. These risks are expressed as a Hazard Quotient ("HQ") for non-cancer risks, and Incremental Lifetime Cancer Risk ("ILCR") for cancer risks. For both metrics, exceedance of a value of 1.0 indicates risks beyond acceptable levels. **Section 6** of the report provides tables and maps detailing the Risk Characterization results, which are summarized by pollutant below:

- **SO_2:** Air quality monitoring data (Scenario 1) indicate infrequent, limited duration periods with Acute HQ>1.0 for the MVRD Burnaby Capitol Hill monitoring station, with a total of 25 hours ("hrs") exceeding over the three years of monitoring. Dispersion modelling data similarly indicates infrequent, very short periods with Acute HQ>1.0 for receptors near the refinery for Scenario 2 (current permit maximum), with 4 hours per year exceeding for the maximally exposed receptor. These exceedances are eliminated for all but a single recreational receptor very near the refinery for Scenario 3 (amended permit maximum), and completely eliminated for Scenario 4, highlighting the significant positive impact of the 45% SO_2 emission reductions associated with the permit amendments.
- **NO_2:** Air quality monitoring data (Scenario 1) indicate no monitoring stations for which Acute HQ>1.0, and one station for which the long term (chronic) HQ is slightly above 1.0, though the exceeding station is MVRD T9 (Port Moody), which is quite distant from the refinery. Regardless of the station locations and their respective proximity to the refinery, both acute and chronic HQs were near 1.0, indicating high background / non-refinery NO_2 levels. Dispersion modelling data indicates that refinery-only risks do not exceed HQ>1.0 for any identified receptor group for either acute or chronic NO_2 exposures. However, due to the high baseline / non-refinery NO_2 levels (Acute HQ=0.95) indicated above, cumulative refinery + background Acute HQ>1.0 for many receptors in the study area are predicted for 8-15% of the year depending on the modelled scenario. NO_x emission reductions associated with the permit amendment (18%) produce modest reductions in cumulative HQ. Health risks due to exposure to ambient NO_2 within the HHRA study area are driven primarily by high baseline NO_2 levels, which are out of the control of the refinery.
- **$PM_{2.5}$:** Air quality monitoring data (Scenario 1) indicate no monitoring stations for which acute or chronic HQ>1.0. Dispersion modelling data similarly indicate no sensitive receptors for which HQ>1.0 for both refinery-only and cumulative exposures. Emissions reductions associated with the permit amendment (23%) produce modest reductions in cumulative HQ. The results of the HHRA indicate that baseline levels of $PM_{2.5}$ contribute significantly to the cumulative HQ.

- **Benzene & 1,3-Butadiene:** Air quality monitoring data (Scenario 1) indicate no monitoring stations for which Acute HQ>1.0 and Chronic ILCR>1 per 100,000. Review of ambient monitoring data from 1999 to 2019 shows a strong downward trend in both benzene & 1,3-butadiene concentrations.

MITIGATION MEASURES

Based on previous air quality assessments conducted by WSP for Parkland, as well as the results of this HHRA, key mitigation actions are as follows:

- Continued reduction of SO_2 emissions from key sources including the FCC and SRU. The emissions reductions incorporated into Scenarios 3 and 4 (45% reduction for amended permit maximum relative to current permit maximum) in the HHRA will lead to significant reductions in the extent and frequency of elevated SO_2 levels, and their associated respiratory health risks.
- Continued reduction of NO_x emissions from key sources including the FCC and COB. The emissions reductions incorporated into Scenarios 3 and 4 (18% reduction for amended permit maximum relative to current permit maximum) in the HHRA will lead to modest reductions in the extent and frequency of elevated NO_2 levels very near the refinery, along with their associated respiratory health risks.
- Continued reduction of $PM_{2.5}$ emissions from the key refinery source: the FCC. The emissions reductions incorporated into Scenarios 3 and 4 (23% reduction for amended permit maximum relative to current permit maximum) in the HHRA will lead to modest reductions in $PM_{2.5}$ levels very near the refinery, along with their associated health risks.
- Improved monitoring coverage for SO_2, NO_2 and $PM_{2.5}$ throughout the HHRA study area with the addition of a new Parkland-funded permanent MVRD monitoring location on the north shore of Burrard Inlet and addition of monitors to existing MVRD stations. For VOCs, leverage the fenceline VOC monitoring installed in early 2022 to better characterize near-site VOC levels.
- Continued utilization of operation and maintenance programs focused on emissions control, including the SO_X Curtailment Event procedure, FCC sulphur scavenging catalyst inversion event procedure, and VOC leak detection and repair program.
- Ongoing engagement with community stakeholders, including the CAP, on questions and concerns related to refinery air quality and human health impacts.
- Ongoing engagement with Tsleil-Waututh Nation, on questions and concerns related to refinery air quality and human health impacts.

In addition to these mitigation measures, WSP recommends updates of this HHRA in support of future permit amendments for the refinery that result in significant changes to emissions.

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GLOSSARY

Acronym	Definition
AAQC	Ambient Air Quality Criteria
AAQO	Ambient Air Quality Objective
ACS	American Cancer Society
AENV	Alberta Environment
AHR	Airway Hyper-Responsiveness
AML	Acute Myeloid Leukemia
ANL	Acute Nonlymphocytic Leukemia
ARM	Ambient Ratio Method
AQA	Air Quality Assessment
AQG	Air Quality Guideline
AQMG	Air Quality Modelling Guideline
AQO	Air Quality Objective
ATSDR	Agency for Toxic Substances and Disease Registry
BC MoECCS	British Columbia Ministry of Environment and Climate Change Strategy
$BMCL_{ADJ/UF}$	95% Lower Confidence Limit on the Benchmark Concentration (Adjusted/Uncertainty Factor)
BMD	Benchmark Dose
CAAQS	Canadian Ambient Air Quality Standard
Cal OEHHA	California Office of Environmental Health Hazard Assessment
CAP	Community Advisory Panel
CCME	Canadian Council of Ministers of the Environment
CDC	Centers for Disease Control
CO	Carbon Monoxide
COB	Carbon Monoxide Boiler
COPC	Contaminant of Potential Concern
COPD	Chronic Obstructive Pulmonary Disease
CVD	Cardiovascular Disease
DABT	Diplomate of the American Board of Toxicology
ECCC	Environment and Climate Change Canada
EE	Exposure Estimate
ED	Emergency Department
eNO	Exhaled Nitric Oxide
ER	Exposure Ratio
ERT	European Registered Toxicologist
ERV	Emergency Room Visit
ESL	Effects Screening Level
FCC	Fluid Catalytic Cracker
FEV	Forced Expiratory Volume
FGR	Flue Gas Recirculation
FHA	Fraser Health Authority
FOE	Frequency of Exceedance
FVC	Forced Vital Capacity
HA	Hospital Admissions
HC	Health Canada
HHRA	Human Health Risk Assessment
HNO_3	Nitric Acid

Acronym	Definition
HPFS	Health Professionals Follow-Up Study
HQ	Hazard Quotient
Hr	Hour
HRV	Heart Rate Variability
IAAQO	Interim Ambient Air Quality Objective
IARC	International Agency for Research on Cancer
IARL	Indoor Air Reference Level
IgE	Immunoglobulin E
ILCR	Incremental Lifetime Cancer Risk
IPCS	International Program on Chemical Safety
IQR	Interquartile Range
ISA	Integrated Science Assessment
IUR	Inhalation Unit Risk
KCO	Gas Transfer Coefficient
Km	Kilometre
LDAR	Leak Detection and Repair
LOAEC	Lowest Observed Adverse Effect Concentration
LOAEL	Lowest Observed Adverse Effect Level
LTC	Long-Term Care
M	Metre
MPOI	Maximum Point of Impingement
MRL	Minimum Risk Level
MV	Metro Vancouver
MVRD	Metro Vancouver Regional District
NAAQO	National Ambient Air Quality Objective
NAAQS	National Ambient Air Quality Standard
NAPS	National Air Pollution Surveillance
NHS	Nurses' Health Study
NOx	Nitrogen Oxides
NO	Nitric Oxide
NO_2	Nitrogen Dioxide
NOAEL	No Observed Adverse Effect Level
NO_x	Nitrogen Oxides, including nitric oxide (NO) and nitrogen dioxide (NO_2)
NPRI	National Pollutant Release Inventory
O_3	Ozone
ON MECP	Ontario Ministry of Environment, Conservation and Parks
PEFR	Peak Expiratory Flow Rate
PM	Particulate Matter
PM_{10}	Coarse Particulate Matter
$PM_{2.5}$	Fine Particulate Matter
ppb	Parts per Billion
ppm	Parts per Million
RA	Risk Assessment
REL	Reference Exposure Level
ReV	Reference Value
RfC	Reference Concentration
RTA	Refined Technology Assessment
SBR	Styrene Butadiene Rubber

Acronym	Definition
SO_2	Sulphur Dioxide
SO_x	Sulphur Oxides
sR_{aw}	Increased Specific Airway Resistance
SRU	Sulphur Recovery Unit
TCEQ	Texas Commission on Environmental Quality
TGTU	Tail Gas Treatment Unit
TRS	Total Reduced Sulphur
TRV	Toxicity Reference Value
TSS	Third Stage Separator
TWN	Tsleil-Waututh Nation
UF	Uncertainty Factor
$\mu g/m^3$	Microgram per Cubic Metre
URTI	Upper Respiratory Tract Infection
US EPA	United States Environmental Protection Agency
VOC	Volatile Organic Compound
WHO	World Health Organization

1 INTRODUCTION

1.1 BACKGROUND

Parkland Refining (B.C.) Ltd. ("Parkland") owns and operates the Burnaby refinery and tank farm ("the refinery"), located at 5201 Penzance Drive and 355 N Willingdon Avenue, Burnaby, B.C, as shown on **Figure 1-1**. This Human Health Risk Assessment ("HHRA") was prepared pursuant to a request received from Fraser Health Authority ("FHA") in December 2020, in support of Parkland's planned application to amend the Air Quality Management Permit ("the permit amendment") for the refinery to incorporate the results of Parkland's Refined Technology Assessment ("RTA"). Pursuant to the RTA, Parkland plans to implement non-capital operational changes to the Fluid Catalytic Cracker ("FCC") that will reduce both nitrogen oxide ("NO_x") and sulphur dioxide ("SO_2") emissions, and further plans to install a Tail Gas Treatment Unit ("TGTU") on the Sulphur Recovery Unit ("SRU") that will reduce SO_2 emissions, and a Flue Gas Recirculation ("FGR") system on the CO Boiler ("COB") that will reduce NO_x emissions. The past addition of a Third Stage Separator ("TSS") to the FCC has already reduced fine particulate matter ("$PM_{2.5}$"). All of these past and planned emissions reductions will be incorporated into Parkland's permit amendment.

The HHRA relies on air dispersion modelling results from the recently completed RTA for Non-capital Solutions Supporting Air Quality Assessment ("AQA"; WSP, 2021). The model results represent current refinery emission sources operating at maximum permitted emissions levels and proposed (future) maximum and normal operating emission levels following the implementation of selected control technology options. Baseline air quality was also considered in the assessment, utilizing the 2016-2018 SO_2, NO_2, and $PM_{2.5}$ baseline data set used in the AQA. Based on the results of the air quality dispersion modelling, the HHRA evaluates the potential health impact of the refinery emissions. This HHRA therefore represents a key component of the permit amendment process.

Regulatory and stakeholder consultations on the workplan for the HHRA were initiated by Parkland and supported by the WSP project team. A summary of comments received during the workplan consultation process is provided in **Appendix A - Stakeholder Consultations** along with an indication of how the comments were addressed in the HHRA, if applicable.

1.2 OBJECTIVES

The purpose of the HHRA is to assess potential human health risks, if any, associated with ambient concentrations of identified contaminants of potential concern ("COPCs") that may be influenced by emissions from the refinery.

To achieve this purpose, WSP evaluated the source-pathway-receptor linkage based on possible interactions with human receptors within a 10-kilometre ("km") x 10 km box centered on the refinery, as shown on **Figure 1-2** below. This box encompasses the high density (250 metre ["m"]) receptor grid employed by the AQA dispersion modelling, and encompasses all receptors predicted by the AQA to experience exceedances of current Metro Vancouver Regional District ("MVRD") NO_2 and SO_2 air quality objectives. Note that the figure also includes MVRD air quality monitoring stations in or near the HHRA study area, including both permanent monitoring stations, and temporary special study stations (S147/148).

The objectives of the HHRA included the following:

- To assess whether the predicted concentrations of COPCs in ambient air influenced by the refinery pose a public health concern in the HHRA study area for identified human receptors; and,
- Based on the findings of the HHRA, identify controls, mitigation measures, and/or monitoring programs that can be implemented to prevent or address the potential for health effects.

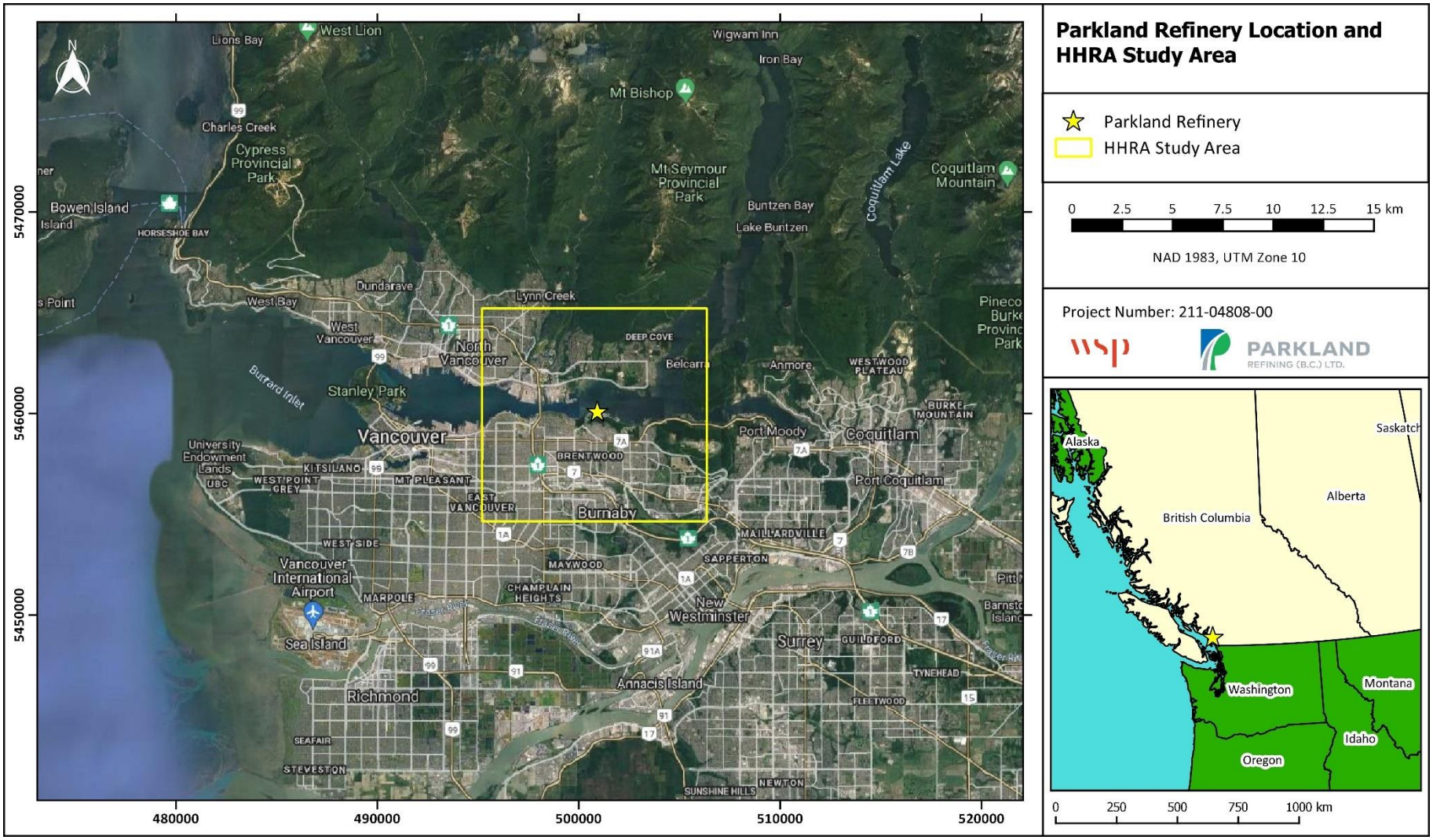


Figure 1-1: Parkland Refinery Location in Metro Vancouver region, with HHRA Study Area Indicated

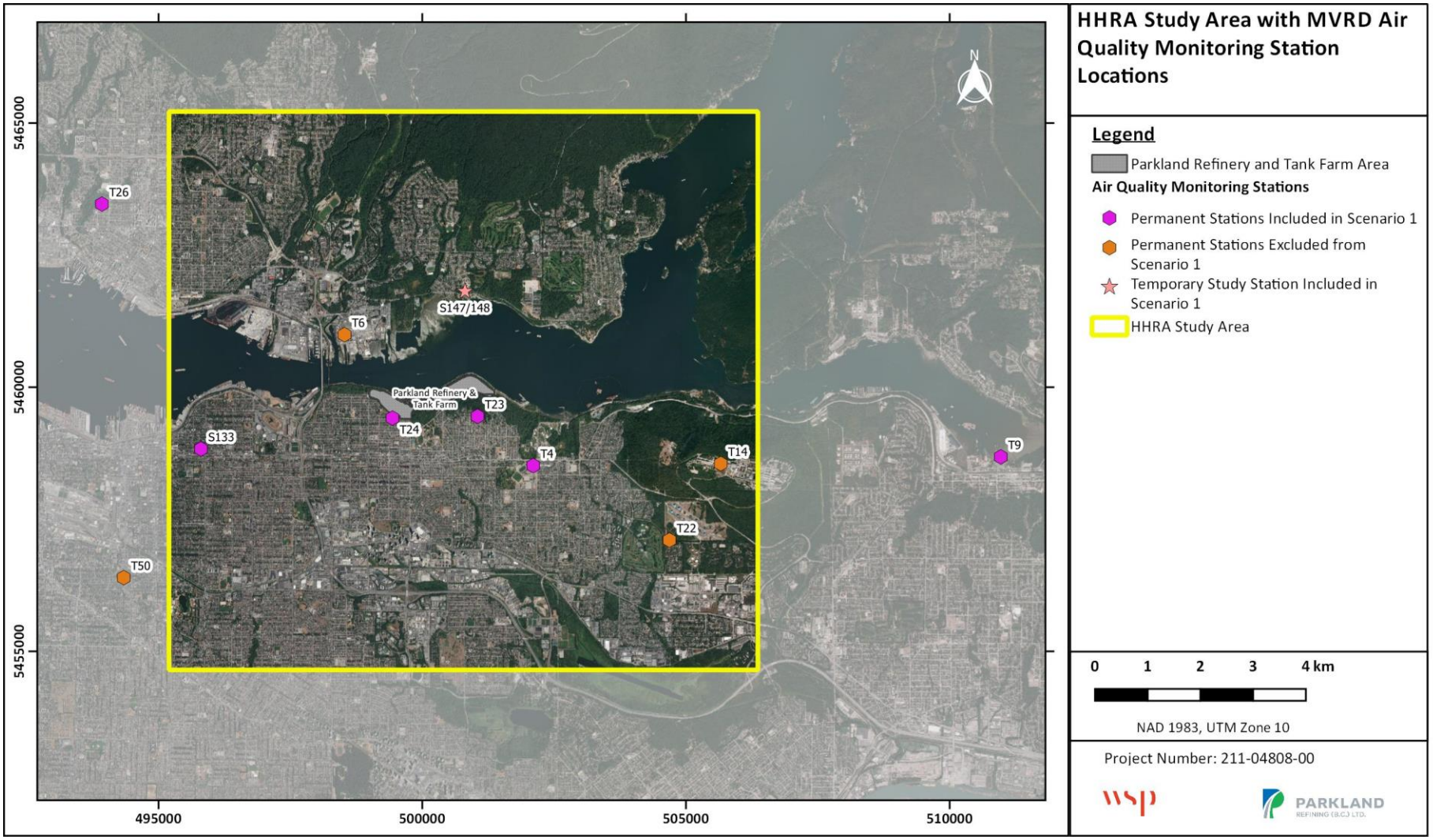


Figure 1-2: HHRA Study Area with MVRD Permanent and Special Study Air Quality Monitoring Stations Indicated

2 PROJECT TEAM MEMBERSHIP

The WSP project team (identified below) has the necessary expertise to complete the HHRA in a manner that meets regulatory and technical requirements. The project team organizational chart is provided in **Figure 2-1** below, and brief summaries of the qualifications and experience of each team member are provided in **Table 2-1** on the following pages.

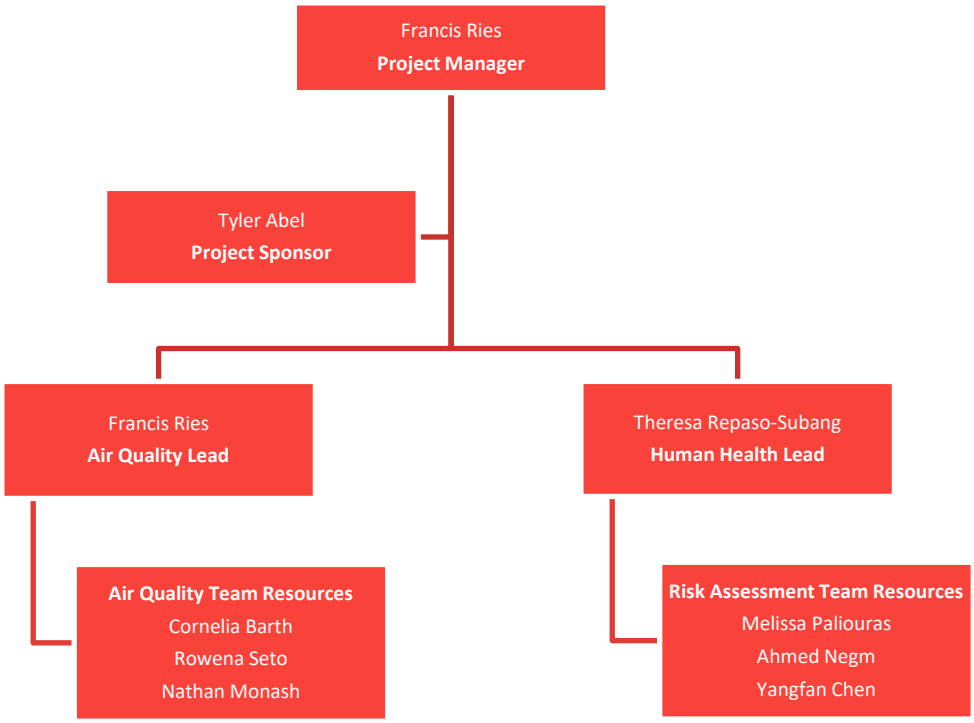


Figure 2-1: WSP Project Team Organizational Structure

Table 2-1 WSP Project Team Qualifications & Experience

Name & Project Role	Education & Professional Affiliations	Description of Qualifications
WSP Air Quality Team		
Francis J. Ries, B.Sc., P.Eng. Project Manager	Professional Engineer, Engineers and Geoscientists BC, 2006 Bachelor of Science in Chemical Engineering, University of Alberta, 2000	Senior Air Quality Engineer in WSP's Vancouver office, with over 20 years of diverse environmental consulting, civil service, industrial and research experience. As a Professional Engineer in British Columbia, Francis is bound by the Engineers and Geoscientists BC professional practice guidelines and code of ethics. Francis has conducted a broad range of projects assessing the air quality and associated human health impacts of emissions from industrial and transportation sources in the Metro Vancouver region and across Canada. He managed the completion of Metro Vancouver's most recent Toxic Air Pollutants Risk Assessment and Emissions Inventory for the Lower Fraser Valley and acted as Metro Vancouver's technical reviewer for the human health risk assessment portions of the Trans Mountain Pipeline Expansion and Roberts Bank Terminal 2 Environmental Assessment applications.
Tyler Abel, M.Sc., Project Sponsor	Master of Science in Atmospheric Science, University of Northern British Columbia, 2008 Bachelor of Science in Biology (Ecology), University of British Columbia, 2002	Manager of WSP's Vancouver Air Quality Group, with 15 years of experience as an Air Quality Specialist providing consulting services and research in the areas of air permitting, environmental assessments and in-field monitoring programs. Tyler has conducted permitting and air quality impact assessments for many industrial clients, including dispersion modelling for a variety of industrial sources to support health studies, permit applications and regulatory compliance.
Cornelia Barth, Ph.D., Air Quality Specialist	Doctor of Philosophy in Hydrology, University of Nevada, Reno, 2013 Diploma in Geography, Freidrich Schiller University Jena, 2005	Air Quality Specialist in WSP's Vancouver Office with over 10 years of interdisciplinary environmental consulting and research experience. Her areas of specialization include air quality dispersion modelling, data analysis, impact assessment and emissions reporting.
Rowena Seto, B.Sc., Air Quality Specialist	Bachelor of Science in Mathematics and Statistics, University of British Columbia, 2018	Air Quality Specialist in WSP's Vancouver Office with 5 years of experience with data analytics, databases, machine learning, statistical modelling, and website development. Her areas of specialization include air quality and meteorological monitoring services, air quality data analysis, air quality dispersion modelling, air permitting support, emission inventories and reporting.
Nathan Monash, B.Sc., Junior Air Quality Specialist	Bachelor of Science in Atmospheric Science, University of British Columbia, 2019	Junior Air Quality Specialist in WSP's Vancouver Office with 2 years of experience in air dispersion modelling, air permitting support, and spatial analysis and mapping.

Name & Project Role	Education & Professional Affiliations	Description of Qualifications
WSP Human Health Risk Assessment Team		
Theresa Repaso-Subang, H.B.Sc., DABT, ERT, QPRA Human Health Lead	European Registered Toxicologist (ERT), United Kingdom Registry of Toxicologists, 2015 Diplomate, American Board of Toxicology (DABT), 2004 Certificate in Risk Analyses and Risk Communications, Harvard School of Public Health, 1995 Bachelor of Science (Honours) in Biomedical Toxicology, University of Guelph, 1990	Senior technical lead for the Toxicology and Risk Assessment group based in WSP's Kitchener, Ontario office, with 30 years of experience in environmental and human health toxicology and risk assessment. As a Diplomate of the American Board of Toxicology and European Registered Toxicologist, Theresa is bound by the codes of conduct of the American Board of Toxicology, Royal Society of Biology and British Toxicology Society, and she has also received certification for the ethical conduct for research involving humans. Theresa is designated as a Qualified Person for Risk Assessments in Ontario and Saskatchewan. Theresa has been involved in the comprehensive reviews of toxicology data to support the development of ambient air quality standards on behalf of Health Canada, Ontario Ministry of Environment, Alberta Environment and Parks and World Health Organization. In support of permit applications, Theresa has been involved in the human health assessment of ambient air concentrations potentially impacted by ongoing and/or proposed infrastructure projects.
Melissa Paliouras, B.Sc., EP, QPRA, Risk Assessment Specialist	Environmental Professional (EP), Canadian Environmental Certification and Approvals Board, 2014 Bachelor of Science in Environmental Monitoring and Analysis, University of Guelph, 2007	Environmental risk assessment specialist based in WSP's Kitchener, Ontario office, with 14 years of experience in human health and ecological risk assessments. Melissa is designated as a Qualified Person for Risk Assessment in Ontario. Melissa's experience in human and ecological risk assessment supports the remediation and redevelopment of contaminated sites, environmental permitting and approvals across North America under a variety of regulatory programs.
Ahmed Negm, M. Env. Sc., Risk Assessment Support	Master of Science in Environmental Science, University of Toronto and York University, 2016. Bachelor of Science in Molecular Biology & Chemistry, University of Guelph, 2015	Risk assessor / environmental scientist based in WSP's Toronto, Ontario office, with over 5 years of experience in environmental management, including risk assessment and environmental site assessments. Ahmed specializes in providing support to human and ecological health risk assessments with responsibilities including data analysis and interpretation, exposure modelling (including vapour intrusion modelling of volatiles), toxicity assessments, risk characterization, development of risk management measures, report writing, and overall project coordination.
Yangfan Chen, M.Sc., Risk Assessment Support	Master of Applied Science in Environmental Engineering, University of Windsor, 2016 Bachelor of Civil Engineering, Fujian University of Technology, 2014	Environmental Risk Assessor based in WSP's Windsor, Ontario office, with 2 years of experience in risk assessment, data management and statistical analysis, exposure modelling and toxicology reviews. As a member of a project funded by the Ontario Ministry of Economic Development, Job Creation and Trade and the Natural Sciences and Engineering Research Council, Yangfan completed a Human Health Risk Assessment of fine particulate matter ($PM_{2.5}$).

3 PROBLEM FORMULATION

The HHRA evaluates the potential acute and chronic health risks associated with air emissions from the refinery. The assessment period for the HHRA is 2017 - 2019, the most recent three-year period for which air quality monitoring data are available, and the period which contains the May 1, 2018 – April 30, 2019 period used for the AQA dispersion modelling studies conducted in support of the RTA (WSP, 2021). This problem formulation takes into consideration the following documents:

- *Kennedy, S.M. et al. (2002) Air Emissions from the Chevron North Burnaby Refinery: Human Health Impact Assessment: Final Report*
- *Fraser Health Authority & Metro Vancouver (2013) Air Quality & Health Impact Assessment Update – Chevron CAP*

3.1 CONTAMINANTS OF POTENTIAL CONCERN

The historical 2002 *Human Health Impact Assessment* performed for the refinery assessed ambient air quality data from MVRD's monitoring stations located both near the refinery and throughout the region. It assessed a broad range of pollutants, including criteria air contaminants such as SO_2, NO_2, and coarse particulate matter ("PM_{10}"), odorous compounds such as total reduced sulphur ("TRS") and over 100 volatile organic compounds ("VOCs"). The key air pollutants identified as posing potential risks to human health in the refinery area included SO_2, benzene, and 1,3-butadiene. The 2013 *Air Quality & Health Impact Assessment Update* focused on these three pollutants and found that benzene and 1,3-butadiene levels had decreased significantly and were consistently below health-based reference concentrations established for the 2013 Update. Concentrations of SO_2 also decreased but infrequent exceedances of 1-hour ("hr") and 24-hr SO_2 air quality objectives were still observed.

Since 2013, ambient concentrations of SO_2, benzene, and 1,3-butadiene have continued to decline both throughout the region and near the refinery (**Appendix B**). Though they were not identified as contaminants of concern in the 2002 or 2013 assessments, focus on NO_2 and $PM_{2.5}$ has increased throughout the region, as there has been significant evolution in understanding of the potential impacts of these pollutants, including the non-threshold nature of their associated health impact. Notably, ambient concentrations of NO_2 and $PM_{2.5}$ show similar downward trends as other pollutants throughout the region and near the refinery (**Appendix B**).

Parkland's past and planned operational and capital upgrades are expected to result in significant reductions in refinery emissions of SO_2 and NO_2 with modest reduction of $PM_{2.5}$ emissions and no expected change in emissions of VOCs including benzene and 1,3-butadiene. Though VOC emissions are not expected to change due to the past and planned operational and capital changes that will be incorporated into Parkland's planned permit amendment, and despite the fact that ambient VOC levels near the refinery and across the region have continued to decline since previous assessments (**Appendix B**), benzene and 1,3-butadiene are included as COPCs in order to provide continuity with previous assessments. Therefore, the COPCs evaluated in the HHRA include: SO_2, NO_2, $PM_{2.5}$, benzene, and 1,3-butadiene.

The 2002 *Human Health Impact Assessment* assessed the risks associated with refinery-area exposure to VOCs other than benzene and 1,3-butadiene to be negligible, and similar to the ambient data shown for benzene and 1,3-butadiene, concentrations of all VOCs measured have declined significantly since 2002 (**Appendix B**). The recently adopted federal regulation titled: *"Reduction in the Release of Volatile Organic Compounds Regulations (Petroleum Sector)"* requires the implementation of fenceline VOC monitoring at refineries across Canada, including Parkland. Fenceline monitoring will start in January 2022 and once measurement results are available, they will provide a large additional data source that could support a reassessment of VOC air quality / health risks very near the refinery fenceline. Based on the significant reduction in VOC emissions since the 2002 assessment, the lack of VOC emissions changes associated with Parkland's planned permit amendment, and the forthcoming availability of the new fenceline VOC monitoring dataset, additional VOCs beyond benzene and 1,3-butadiene are not included as COPCs in the HHRA.

3.2 RECEPTORS OF POTENTIAL CONCERN

The human receptors evaluated in the HHRA were identified based on land use(s) within the project study area. The human receptors associated with the identified land uses are intended to be inclusive of human populations including sensitive subpopulations such as Tsleil-Waututh Nation ("TWN") community members, asthmatics, children, and the elderly. Within the HHRA study area, the following human receptors and receptor locations were identified:

- **Residential Communities** – individuals who live in the residential communities near the refinery, including sensitive receptors such as TWN;
- **Senior's Retirement Facilities** – elderly adults who reside in long-term care ("LTC") facilities;
- **Daycare Facilities** – toddlers and young children who are at a daycare facility for a nine-hour day, five days per week for 50 weeks of the year (i.e., assuming 2 weeks of vacation per year);
- **Schools** – full-time students (children and teenagers) who attend classes and are potentially present for a maximum of nine hours per day, five days per week for 10 months of the year (i.e., length of school year);
- **Hospital Facilities** – adult patients who are receiving care at hospitals whose health is already compromised;
- **Workers** – adults who work near the refinery for a typical eight-hour work shift, five days per week for 50 weeks of the year (i.e., assuming 2 weeks of vacation per year);
- **Visitors** – people who visit the study area for short periods of time, including, but not limited to individuals who use the waterfront areas for recreational purposes;
- **TWN Reserve Lands** – individuals who make use of TWN reserve lands within the HHRA study area for outdoor cultural practices.

The exposure modelling, described in **Section 4**, considered that all of these human receptors may be exposed to ambient concentrations of identified COPCs that may be influenced by emissions from the refinery.

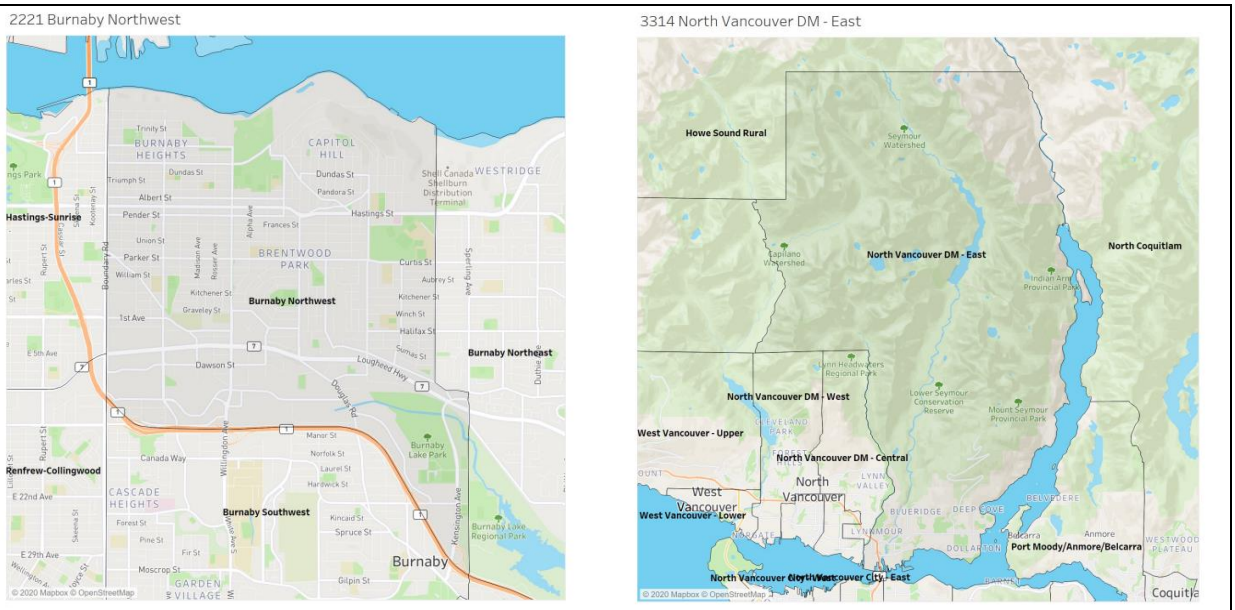
3.2.1 CHARACTERIZATION OF EXISTING COMMUNITY HEALTH

The British Columbia Centre for Disease Control ("BC CDC") and Provincial Health Services Agency provide community health profiles that describe the existing health of communities throughout British Columbia. In order to understand the existing health status of communities surrounding the Parkland Refinery, WSP selected two Community Health Service Areas ("CHSA"):

- 2221 Burnaby Northwest, which is comprised of the neighbourhoods of Burnaby Heights, Capitol Hill, Willingdon Heights, and Brentwood Park.[1]
- 3314 North Vancouver DM – East, which is a community in the District Municipality of North Vancouver and includes the Tsleil-Waututh National reserve lands.[2]

Figure 3-1 below shows the extent of these CHSAs.

[1] http://communityhealth.phsa.ca/CHSAHealthProfiles/PdfGenerator/Burnaby Northwest
[2] http://communityhealth.phsa.ca/CHSAHealthProfiles/PdfGenerator/North Vancouver DM - East



Note: provided by Health Sector Information, Analysis, and Reporting Division, B.C. Ministry of Health

Figure 3-1: Community Health Service Areas Extents Surrounding Parkland Refinery

The health profiles summarize community data including demographics (i.e., population age distribution), diversity, factors that affect overall health namely income, education, employment, physical environments, access to health care services, social support, early childhood development, and personal health practices. In addition, data related to health status and chronic diseases are summarized for each community and compared to provincial data.

The following summarizes key health data for Burnaby Northwest and the District Municipality of North Vancouver-East in comparison to provincial data:

- Based on provincial statistics for 2011 to 2015, men and women live to the age of 84.5 years in Burnaby Northwest CHSA and North Vancouver DM - East CHSA compared to 82.6 years of age for the province as a whole.
- For the Burnaby Northwest CHSA, the age-standardized incidence and prevalence rates for chronic diseases associated with inhalation exposure to identified COPCs including asthma and chronic obstructive pulmonary disease ("COPD") are below provincial averages.
- Similarly, for the North Vancouver DM - East CHSA, the age-standardized incidence and prevalence rates for chronic diseases associated with identified COPCs are also below provincial averages.
- Based on BC Cancer Registry data for 2015-2017, the cancer rates for the selected CHSA and the province overall are shown in **Table 3-1** below.

Table 3-1 Selected CHSA and Provincial Cancer Incidence and Mortality Data (per 100,000)

Crude Incidence / Mortality (per 100,000)	2221 Burnaby Northwest (2015-2017)[1]	3314 District Municipality of North Vancouver – East (2015-2017)[2]	British Columbia (2018)[3]
All Cancers	482.5	516.5	561.3
Female Breast Cancer	144	154	153.1
Leukemia	Not Reported	Not Reported	16.4
Colorectal Cancer	60.6	61.8	64.2
All Cancer Deaths Crude Mortality	165.4	184.5	205.9 [4]
Crude Mortality Death Rate for Leukemia	Not Reported	Not Reported	7.7 [4]

Crude Incidence / Mortality (per 100,000)	2221 Burnaby Northwest (2015-2017)[1]	3314 District Municipality of North Vancouver – East (2015-2017)[2]	British Columbia (2018)[3]

References:

1. http://communityhealth.phsa.ca/CHSAHealthProfiles/PdfGenerator/Burnaby Northwest
2. http://communityhealth.phsa.ca/CHSAHealthProfiles/PdfGenerator/North Vancouver DM - East
3. http://www.bccancer.bc.ca/statistics-and-reports-site/Documents/Crude_Incidence_Rates_Report_2018_20210204.pdf
4. http://www.bccancer.bc.ca/statistics-and-reports-site/Documents/Crude_Mortality_Rates_Report_2018_20210304.pdf

3.3 EXPOSURE PATHWAYS OF CONCERN

A complete exposure pathway requires the following four elements:

- The presence of a chemical substance;
- A migration pathway (environmental transport);
- An exposure point for contact (e.g., air); and,
- An exposure route (e.g., inhalation).

An exposure pathway is not complete unless all four elements are present. If a pathway is incomplete, no significant exposure is anticipated to occur.

As described below, two exposure pathways of concern were identified at the problem formulation stage for human receptors: 1) inhalation of project-related COPCs in ambient air, and 2) direct contact with COPCs as particulates emitted from the refinery via atmospheric deposition (**Figure 3-2**).

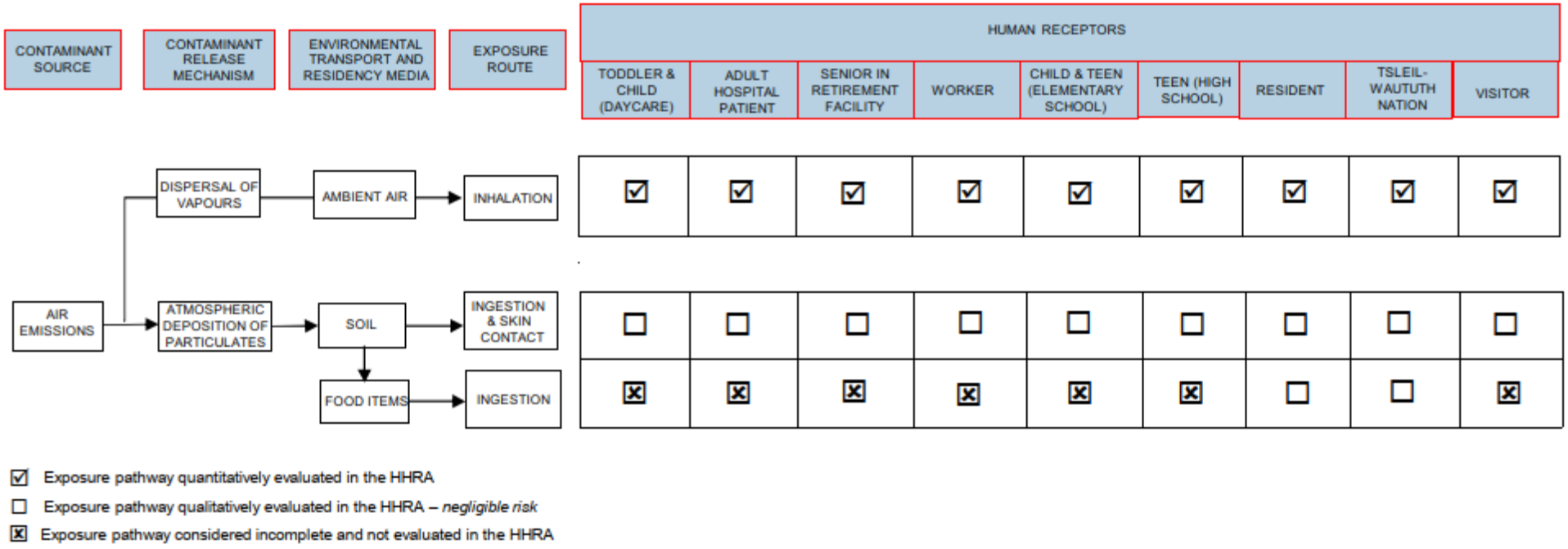


Figure 3-2: HHRA Conceptual Site Model

3.3.1 INHALATION OF AMBIENT AIR

The HHRA quantitatively evaluated potential health effects associated with acute (short-term) and chronic (long-term) inhalation exposures to ambient concentrations of identified COPCs (i.e., SO_2, NO_2, $PM_{2.5}$, benzene, 1,3-butadiene) that may be influenced by emissions from the refinery. These emissions are released into ambient air primarily as vapours and may be subsequently inhaled by human receptors within the HHRA Study Area.

Details of the exposure assessment are provided in **Section 4**.

3.3.2 ATMOSPHERIC DEPOSITION

The dispersion modelling methods used in the AQA (WSP, 2021) were used to analyze deposition for the Current Permit Maximum scenario (i.e., worst case; Scenario 2 described in **Section 4** below). Based on stack test data, the modelling assumed that particulate matter ("PM") was made up of the following size fractions:

- 64% ≤ 2.5 µm ($PM_{2.5}$); and,
- 36% > 2.5 µm and ≤ 10 µm (PM_{10}).

These results include both wet and dry deposition. The results for model Scenario 2 (see **Table 4-1** in **Section 4**) are considered protective of future Scenarios 3 and 4 given that particulate emissions are predicted to decrease under the amended permit. Additional information is provided in the AQA (WSP, 2021).

The rate of deposition from the air quality modelling can be used to estimate changes in future soil concentrations within the HHRA study area. The maximum predicted deposition of particulate matter (i.e., ≤ PM_{10}) from the refinery within the study area under the current permit maximum is 0.0011 mg/dm^2/day, which is less than 1% of the B.C. Ministry of Environment and Climate Change Strategy ("MoECCS") dustfall objective of 1.75 mg/dm^2/day for residential/parkland land use. It is noted that in 2020, B.C. MoECCS released guidance indicating that the dustfall Pollution Control Objectives are no longer relied upon, except in limited circumstances, such as *concerns of an aesthetic or nuisance nature*. Given that the maximum predicted rate of deposition of particulate matter is expected to be several orders of magnitude lower than the standard, it is considered that this provides sufficient evidence that there would be no measurable change in soil quality from depositional contributions via dustfall from the refinery.

In addition, the Ontario Ministry of the Environment, Conservation and Parks ("ON MECP") human toxicology and air standards section of their Standards Development Branch developed a 30-day and annual Ambient Air Quality Criterion ("AAQC") for dustfall on the basis of effects on aesthetics from the deposition of the contaminant (i.e., soiling). The maximum predicted deposition rate of 0.0011 mg/dm^2/day was converted to a daily rate of 0.0011 g/m^2 resulting in a calculated cumulative deposition rate of 0.0033 g/m^2 assuming daily deposition for a 30-day period. This 30-day value (0.0033 g/m^2) is less than 1% of the 30-day Ontario AAQC of 7 g/m^2.

Given that in both cases the maximum predicted rate of particulate matter deposition is shown to be less than 1% of both the B.C. MoECCS dustfall objective and the ON MECP AAQC for dustfall, it is considered that atmospheric deposition of particulate matter would have a *de minimis* impact on the quality of soil and/or food items grown within the HHRA Study Area. No further evaluation of deposition is therefore warranted; the inhalational exposure pathway is the only exposure pathway carried forward for quantitative assessment in the HHRA.

3.4 UNCERTAINTY ANALYSIS

A summary of the major assumptions made in the Problem Formulation stage of the HHRA and resulting uncertainties is provided below:

- For the purposes of exposure modelling, it has been assumed that the predicted concentrations of COPCs in outdoor air are equal to that in indoor air (i.e., established equilibrium). Ambient indoor air concentrations are dependant on a multitude of variables including infiltration rates, indoor decay rates, ventilation system set-ups, and other factors. To maintain a conservative approach, the assumption that equilibrium is established between outdoor and indoor ambient air was applied for this assessment.

- It was considered that all human receptors may be exposed to maximum impacts associated with ambient concentrations of identified COPCs that may be influenced by emissions from the refinery. This approach means the HHRA is conservative in nature.

4 EXPOSURE ASSESSMENT

The exposure assessment step was conducted for each COPC-pathway-receptor combination identified in the problem formulation to estimate the amount of COPCs that human receptors are potentially exposed to. In essence, the exposure scenario is the complete description of the pattern of exposure. For the purposes of the exposure modelling, it was assumed that the predicted concentration of COPCs in outdoor ambient air was equal to that in indoor air (i.e., established equilibrium).

4.1 EXPOSURE ASSESSMENT SCENARIOS

For short-term (acute) and long-term (chronic) exposure durations, concentrations in ambient air used as input into the exposure assessment are either measured from MVRD network stations or predicted based on air quality dispersion modelling scenarios described in **Table 4-1**.

The assessment of COPC exposure concentrations was performed for four (4) scenarios: one (1) using only ambient air quality monitoring data from 2017-2019 to determine COPC exposure concentrations for all identified pollutants for all receptors in the absence of refinery contributions, and three (3) accounting for refinery contributions predicted by air dispersion modelling for various permit scenarios (WSP, 2021). These scenarios are explained in **Table 4-1** below.

Table 4-1 HHRA Exposure Assessment Scenarios

	Scenario 1 - Ambient Monitoring (2017-2019)	**Scenario 2 – Dispersion Modelling – Current Permit Maximum**	**Scenario 3 – Dispersion Modelling – Amended Permit Maximum**	**Scenario 4 – Dispersion Modelling – Amended Permit Normal**
Exposure Data Source	Ambient monitoring data from MVRD network stations located within the HHRA Study Area. The most recent three years of validated ambient data (2017-2019) were used to derive exposure concentrations.	Dispersion modelling results for "current permit[1]" emissions at maximum permitted levels for all refinery sources of SO_2, NO_2, $PM_{2.5}$ for the HHRA Study Area.	Dispersion modelling results for "amended permit" / FCC Non-Capital Solutions + TGTU + COB FGR emissions at maximum permitted levels for all refinery sources of SO_2, NO_2, $PM_{2.5}$ for the HHRA Study Area.	Dispersion modelling results for "amended permit" / FCC Non-Capital Solutions + TGTU + COB FGR emissions at expected normal operating levels for all refinery sources of SO_2, NO_2, $PM_{2.5}$ for the HHRA Study Area.
COPC	SO_2, NO_2, $PM_{2.5}$, benzene, and 1,3-butadiene	SO_2, NO_2, $PM_{2.5}$	SO_2, NO_2, $PM_{2.5}$	SO_2, NO_2, $PM_{2.5}$
Monitoring Stations	T4: SO_2, NO_2, $PM_{2.5}$ T9: SO_2, NO_2, $PM_{2.5}$, benzene, 1,3-butadiene T23: SO_2 T24: SO_2, benzene, 1,3-butadiene T26: SO_2, NO_2, $PM_{2.5}$ S133: SO_2 S147/S148: SO_2, NO_2, $PM_{2.5}$ (** February 2018 – June 2019 only)	Baseline conditions (2016-2018[2]): T4: SO_2, NO_2, $PM_{2.5}$ T9: SO_2, NO_2, $PM_{2.5}$, T24: SO_2 T26: SO_2, NO_2, $PM_{2.5}$	Baseline conditions (2016-2018[2]): T4: SO_2, NO_2, $PM_{2.5}$ T9: SO_2, NO_2, $PM_{2.5}$, T24: SO_2 T26: SO_2, NO_2, $PM_{2.5}$	Baseline conditions (2016-2018[2]): T4: SO_2, NO_2, $PM_{2.5}$ T9: SO_2, NO_2, $PM_{2.5}$, T24: SO_2 T26: SO_2, NO_2, $PM_{2.5}$

	Scenario 1 - Ambient Monitoring (2017-2019)	Scenario 2 – Dispersion Modelling – Current Permit Maximum	Scenario 3 – Dispersion Modelling – Amended Permit Maximum	Scenario 4 – Dispersion Modelling – Amended Permit Normal
Notes: MVRD permanent and special study monitoring station locations are shown on **Figure 1-2**. 1 - Current permit refers to Parkland's most recent MVRD Permit GVA0117 dated January 27, 2021. 2 – Baseline air quality, utilizing the same 2016-2018 SO_2, NO_2, $PM_{2.5}$ baseline data set as the AQA (WSP, 2021).				

This 4-scenario approach was selected for the following reasons:

1) Dispersion modelling in support of the AQA focused only on pollutants which are expected to change as a result of the RTA emission reduction measures. These pollutants include: SO_2, NO_2, and $PM_{2.5}$. As such, no predicted ambient benzene or 1,3-butadiene concentrations are available from the dispersion model output, so health risk associated with exposure to these two COPCs can only be determined from the ambient monitoring data.

2) The dispersion model predicted concentrations of SO_2, NO_2, and $PM_{2.5}$ provide a high level of spatial detail in exposure concentrations, but are by their nature very conservative, and may not align with measured ambient concentrations at MVRD monitoring stations. By estimating the health risks for both ambient monitoring concentrations and dispersion modelled concentrations, predicted health risks attributable to the operations of the refinery can be better explored.

3) The use of "current permit" maximum and "amended permit" maximum and normal dispersion modelling scenarios allows for identification of the potential change in health risks due to the emissions reductions associated with the permit amendment.

4) The use of the "amended permit" normal scenario (Scenario 4) will provide the most representative characterization of the expected future "average" health risks for receptors, while the "amended permit" maximum scenario (Scenario 3) will best characterize "worst case" health risks.

It is important to note that the HHRA Scenario 1 (Ambient Monitoring 2017-2019) utilizes different monitoring stations and years than those used for the AQA baseline (described below in **Section 4.1.2.4**). This difference is due to the fact that the AQA baseline is specifically designed to exclude the influence of the Parkland Refinery to the greatest extent possible, because the dispersion model is used to determine the refinery impact, which is then added to the baseline. For HHRA Scenario 1, the goal is to assess the health risks associated with measured air quality levels, regardless of the level of refinery influence at a given monitoring station. As such, ambient data from Scenario 1 is not used in any way in Scenarios 2-4, and conversely, the ambient baseline values and dispersion model predictions from Scenarios 2-4 are not used as part of the Scenario 1 assessment.

4.1.1 SCENARIO 1 – AMBIENT MONITORING CONCENTRATION DATA

As detailed in **Table 4-1**, COPC ambient concentration data for Scenario 1 were drawn from MVRD monitoring stations. MVRD operates a large and dense network of ambient air quality monitors within the Metro Vancouver region, with **Figure 1-2** providing a detail of the stations in the area near the Parkland Refinery. A brief rationale for inclusion or exclusion of MVRD air quality monitoring stations in the Scenario 1 data is presented below:

- **Stations Included:**
  - **T4** (Burnaby Kensington Park): Located near the Parkland Refinery within the HHRA study area;
  - **T23** (Burnaby Capitol Hill): Located near the Parkland Refinery within the HHRA study area, specifically located to monitor for elevated SO_2 levels associated with refinery operations;
  - **T24** (Burnaby North – McGill Park): Located near the Parkland Refinery within the HHRA study area, specifically located to monitor for refinery VOCs;
  - **S133** (Vancouver Pandora Park): Located within the HHRA study area, specifically located to monitor for elevated SO_2 levels associated with port marine activities along Burrard Inlet;

- **S147/S148** (North Vancouver Special Study): Located within the HHRA study area, specifically located to assess the impact of refinery operations on the north shore of Burrard Inlet;
- **T9** (Port Moody): Located outside of the HHRA study area but was included in past air quality and HHRAs for the Parkland Refinery.
- **T26** (North Vancouver Mahon Park): Located outside of the HHRA study area but provides an additional station in North Vancouver to balance the exclusion of station T6, detailed below.

- **Stations Excluded:**
  - **T6** (North Vancouver Second Narrows): Located within the HHRA study area, but construction activities in 2019 immediately beside the monitoring station compromised the quality of monitoring data from T6. These activities resulted in artificially high NO_2 and $PM_{2.5}$ levels that were not characteristic of broader ambient air contaminant levels in the Burrard Inlet area;
  - **T14** (Burnaby Mountain): Located within the HHRA study area, but due to its location on top of Burnaby Mountain beside Simon Fraser University, measurements are representative of a very specific area that is dissimilar to the rest of the HHRA study area.
  - **T22** (Burnaby Burmount): Located within the HHRA study area, but located immediately beside the Trans Mountain Pipeline tank farm, so concentrations measured here are highly influenced by a specific non-refinery source of VOCs;
  - **T50** (Vancouver-Clark Drive) – Located outside of the HHRA study area, and also located beside a very busy truck route, so concentrations measured here are representative of a very specific near-road micro-environment heavily influenced by traffic emissions.

Monitoring datasets validated to the National Air Pollution Surveillance ("NAPS") quality assurance standard were requested directly from MVRD for the most recent years available at the time of the request (i.e., 2017 – 2019). As a result of the validation requirements, MVRD monitoring data for a given year are not typically available until the following fall, so validated data for 2020 were not yet available. MVRD does not support the use of non-validated data (i.e., 2020 at the time of WSP's data request, or 2021 at the time of report publication) in any third-party analysis, particularly one that is informing MVRD permitting considerations. Further, at MVRD's direction, Parkland installed a meteorological monitoring station at their refinery site in early 2018, and data from this station were used as one of the inputs to the air dispersion model supporting the HHRA (see **Section 4.1.2** below). The HHRA utilized the most recent years for which both validated MVRD air quality and meteorological data and Parkland meteorological data were available.

Monitoring datasets were processed using scripts developed in the R programing language for statistical computing[3], and data were summarized into the acute and chronic averaging periods and statistical forms required for the HHRA. Data processing included a step to remove the effects of wildlife smoke from known forest fire periods. This step involved removing $PM_{2.5}$ data for any days that MVRD had issued an air quality advisory due to wildlife smoke in the region. As such, the decision to exclude these data was based on the advisory determination of MVRD, rather than an algorithm designed by WSP. A list of days and hours excluded from the analysis due to this data processing step is provided in **Appendix C, Table C-3**.

4.1.2 SCENARIOS 2-4 – DISPERSION MODELLING CONCENTRATION DATA

As detailed in **Table 4-1**, COPC ambient air concentration data for Scenarios 2-4 were drawn from air dispersion modelling. Full details of the dispersion modelling approach, configuration and model output are provided in the 2021 AQA report prepared by WSP. A summary of the key details of the dispersion modelling are provided below.

Based on the emissions scenarios described in **Table 4-1** (Current Permit Maximum, Amended Permit Maximum, Amended Permit Normal) ambient concentrations of COPCs from the refinery were predicted from the CALMET/CALPUFF modelling system using methodology consistent with previous dispersion modelling assessments conducted by WSP in support of Parkland submissions to MVRD. All inputs and model switches were

[3] https://www.r-project.org/

determined from the recommendations set out in 2021 BC MoECCS Air Quality Modelling Guideline ("AQMG"), and the assessment was completed following a Modelling Plan approved by MVRD. Modelling was conducted for a 1-year time period from May 1, 2018 through April 30, 2019 to allow for incorporation of on-site meteorological data collected at the Parkland Refinery.

4.1.2.1 *MODELED EMISSIONS*

Predictions of ambient concentrations of COPCs from the dispersion modelling system are driven by refinery air emissions inputs. Refinery SO_2, NO_x and $PM_{2.5}$ emissions were modelled based on Scenarios 2-4 given that emissions of these COPCs will change in Parkland's permit amendment application to MVRD due to capital and non-capital improvements recommend by the RTA. Emission sources included in the modelling include all permitted sources in Parkland's MVRD permit GVA0117 dated January 27, 2021 that emit SO_2, NO_x, or $PM_{2.5}$. Further details of the emissions modelling scenario are provided in the AQA (WSP, 2021).

4.1.2.2 *NO_X CONVERSION*

Emissions of NO_x from refinery sources are predominantly made up of nitric oxide ("NO"), which undergoes conversion to NO_2 once released into the atmosphere. To model this conversion and allow comparison of model results with the NO_2 toxicity reference value ("TRV") selected in **Section 5**, ambient ratio method ("ARM") in the MVRD implementation was employed.

4.1.2.3 *MODEL RECEPTOR GRID*

The CALPUFF receptor grid defines the locations at which the dispersion model predicts COPC concentrations in the model domain. The receptor grid used in this assessment was prepared in accordance with the AQMG and the Dispersion Modelling Plan approved by MVRD; this is described in detail in the AQA (WSP, 2021). A key detail relative to the HHRA is the use of a high-density receptor grid (50 m spacing within 1 km of the refinery fenceline, 250 m spacing within 5 km) throughout the HHRA study area. Locations of sensitive receptors, including daycares, schools, senior and LTC facilities, and hospitals were based on data obtained from the sources listed in **Table 4-2** below. All receptors were set at a flagpole height of 1.5 m to represent an approximate breathing height.

Table 4-2 Sensitive Receptor Location Data Sources

Receptor Location Type	Data Source
Residential Areas	Canada Land Use 2010, Agriculture and Agrifood Canada [1] Metro Vancouver 2011 Generalized Land Use Classification [2]
Daycare Facilities	DataBC: Child Care Map Data [3]
Schools	Data BC: BC Schools - K-12 with Francophone Indicators [4] BC K-12 School and District Contact Information [5]
Seniors Care Facilities	Data BC: Assisted Living Residences [6] Fraser Health - Long term care wait times [7] Vancouver Coastal Health - Choosing a long-term care home [8]
Hospitals	Data BC: Hospitals in BC [9]
Recreational Areas	Visual inspection of area near refinery on Google Earth, consultation comments from members of Community Advisory Panel
Businesses near Parkland Refinery	Visual inspection of area near refinery on Google Earth
Tsleil-Watuth Nation	District of North Vancouver Geoweb [10] Consultation feedback provided by TWN

References:
1. https://open.canada.ca/data/en/dataset/c688b87f-e85f-4842-b0e1-a8f79ebf1133
2. http://www.metrovancouver.org/data
3. https://catalogue.data.gov.bc.ca/dataset/child-care-map-data
4. https://catalogue.data.gov.bc.ca/dataset/bc-schools-k-12-with-francophone-indicators
5. http://www.bced.gov.bc.ca/apps/imcl/imclWeb/Home.do

Receptor Location Type	Data Source
6.	https://catalogue.data.gov.bc.ca/dataset/assisted-living-residences
7.	https://www.fraserhealth.ca/health-topics-a-to-z/long-term-care/long-term-care-map
8.	http://www.vch.ca/your-care/home-community-care/care-options/long-term-care/choosing-a-long-term-care-facility
9.	https://catalogue.data.gov.bc.ca/dataset/hospitals-in-bc
10.	https://geoweb.dnv.org/data/

4.1.2.4 *BASELINE AIR QUALITY*

In dispersion modelling assessments, baseline ambient concentrations of COPCs are determined in order to provide a complete indication of cumulative impacts to air quality. Baseline concentrations are added to model predictions to determine the cumulative impact. In this context, the AQMG states that "baseline" is meant to be the concentrations due to emissions from both natural and anthropogenic sources. In other words, it is intended to be the result of the contribution from all sources except the source(s) being modelled.

It is a typical and acceptable practice to determine the baseline from historical air quality monitoring data. As described in the AQA report (WSP, 2021), monitoring data from four MVRD stations for the 2016-2018 period were used to determine ambient baseline concentrations for all air contaminants considered in the AQA. The stations included in the AQA baseline were:

- **T4** (Burnaby Kensington Park): Located near the Parkland Refinery within the HHRA study area;
- **T24** (Burnaby North - McGill Park): Located near the Parkland Refinery within the HHRA study area, specifically located to monitor for refinery VOCs;
- **T9** (Port Moody): Located outside of the HHRA study area but was included in past air quality and HHRAs for the Parkland Refinery.
- **T26** (North Vancouver Mahon Park): Located outside of the HHRA study area but provides an additional station in North Vancouver to balance the exclusion of station T6.

As described above in **Section 4.1.1**, other monitoring stations in the MVRD monitoring network were excluded from the baseline because they were highly influenced by individual emissions sources such as the Parkland Refinery (T23 – Burnaby Capitol Hill) or other transportation / industrial sources (T6 – North Vancouver Second Narrows), or because they are distant from the refinery, and thus not representative of the refinery area baseline conditions.

It is important to note that the AQA baseline utilizes different monitoring stations and years than those used for the HHRA Scenario 1 (Ambient Monitoring 2017-2019). This difference is due to the fact that the AQA baseline is specifically designed to exclude the influence of the Parkland Refinery to the greatest extent possible, because the dispersion model is used to determine the refinery impact, which is then added to the baseline. For HHRA Scenario 1, the goal is to assess the health risks associated with measured air quality levels, regardless of the level of refinery influence at a given monitoring station. As such, ambient data from Scenario 1 is not used in any way in Scenarios 2-4, and conversely, the ambient baseline values and dispersion model predictions from Scenarios 2-4 are not used as part of the Scenario 1 assessment.

Section 4.3 below provides further discussion of the use of the baseline values to determine cumulative exposures in the HHRA.

4.2 EXPOSURE PARAMETERS BY RECEPTOR GROUP

For short-term (acute) durations the HHRA exposure estimates ("EE") are equivalent to the 1-hour or 24-hour maximum COPC concentrations in ambient air determined for Scenarios 1 through 4 detailed in **Table 4-1**.

For long-term (chronic) durations the EEs are calculated as annual COPC concentrations in ambient air determined for Scenarios 1 through 4, taking into consideration receptor-specific parameters such as exposure frequency and duration. Conservative assumptions were applied in this step of the HHRA to ensure that it is protective of human health, including sensitive subpopulations (e.g., children, elderly, asthmatics).

The B.C. Ministry of Health (2021) guidance document entitled "*British Columbia Guidance for Prospective Human Health Risk Assessment*" indicates that if community specific data on human exposure characteristics are not available, exposure characteristics from Health Canada (2012) should be prioritized. If necessary, other sources can be consulted including the "*Canadian Exposure Factors Handbook*" (Richardson & Stantec Consulting, Ltd., 2013) and the "*Inventory and Analysis of Exposure Factors for Alberta*" (Alberta Health, 2018). Collectively, these references were used to adopt the receptor-specific exposure parameters used for this assessment, as identified in **Table 4-3** to **Table 4-4** below.

The exposure parameters are presented for each of the relevant receptors in the sub-sections below. These exposure parameters are assumptions regarding exposure of a particular study population and provide details of the exposure scenario based on the route of exposure (e.g., inhalation), type of population exposed (e.g., adults or children), and a description of the intervals of exposure (e.g., 9 hours/day, 5 days/week for 50 weeks of the year).

4.2.1 EXPOSURE PARAMETERS FOR RESIDENTS

In this exposure scenario a resident receptor represents various life stages including infant, toddler, child, teenager, and adult. Residents were considered to spend 24 hours/day, 7 days/week for 50 weeks/year (assuming a two-week vacation) within their residential units. This exposure scenario is also applicable to TWN residential communities. The exposure parameters applicable to residents are as follows:

Table 4-3 Exposure Parameters for Residents

Exposure Factor	Units	Infant (0 – 6 Mo.)	Toddler (7 Mo. To 4 Yrs)	Child (5 – 11 Yrs)	Teen (12 – 19 Yrs)	Adult (≥ 20 Yrs)	Reference
EF (exposure frequency for inhalation) = EF_a x EF_b x EF_c	h/yr	8400	8400	8400	8400	8400	Calculated
EF_a (daily exposure frequency)	d/wk	7	7	7	7	7	HC, 2012
EF_b (weekly exposure frequency)	wk/yr	50	50	50	50	50	See note 1
EF_c (hourly exposure frequency)	h/d	24	24	24	24	24	HC, 2012
ED (exposure duration)	yr	0.5	4.5	7	8	60	HC, 2012
AP (averaging period): non-cancer	yr	0.5	4.5	7	8	60	HC, 2012
AP (averaging period): cancer	yr	80	80	80	80	80	HC, 2012

Notes:
h – hour; yr – year; wk – week; d – day; mo – months.
1 - BC Employment Standards Act, Entitlement to Annual Vacation – Act Part 7, Section 57.

4.2.2 EXPOSURE PARAMETERS FOR ELDERLY RESIDENTS IN RETIREMENT FACILITY

In this exposure scenario an elderly resident is considered to be an adult (i.e., 65-80 years old) who spends 24 hours/day, 7 days/week for 50 weeks/year (assuming a two-week vacation period) at a retirement facility/LTC home. The exposure parameters applicable to elderly residents in a retirement facility are equal to those listed below for residents:

Table 4-4 Exposure Parameters for Elderly Residents in a Retirement Facility Setting

Exposure Factor	Units	Adult (65 – 80 Yrs)	Reference
EF (exposure frequency for inhalation) = EF_a x EF_b x EF_c	h/yr	8400	Calculated
EF_a (daily exposure frequency)	d/wk	7	HC, 2012
EF_b (weekly exposure frequency)	wk/yr	50	Site-specific
EF_c (hourly exposure frequency)	h/d	24	HC, 2012
ED (exposure duration)	yr	15	Richardson, 2013
AP (averaging period): non-cancer	yr	15	Richardson, 2013
AP (averaging period): cancer	yr	80	HC, 2012

Exposure Factor	Units	Adult (65 – 80 Yrs)	Reference
Notes: h – hour; yr – year; wk – week; d – day.			

4.2.3 EXPOSURE PARAMETERS FOR YOUNG CHILDREN INCLUDING TODDLERS IN DAYCARE FACILITY

In this exposure scenario a toddler or young child is considered to attend a daycare facility for 9 hours/day, 5 days/week for 50 weeks/year (assuming a two-week vacation period). The exposure parameters applicable to young children and toddlers in a daycare facility are as follows:

Table 4-5 Exposure Factors for Toddlers and Young Children in a Daycare Setting

Exposure Factor	Units	Toddler (1 - 4 Yrs)	Young Child (5 – 6 Yrs)	Reference
EF (exposure frequency for inhalation) = EF_a x EF_b x EF_c	h/yr	2250	2250	Calculated
EF_a (daily exposure frequency)	d/wk	5	5	HC, 2012[1]
EF_b (weekly exposure frequency)	wk/yr	50	50	See note 2
EF_c (hourly exposure frequency)	h/d	9	9	HC, 2012[1]
ED (exposure duration)	yr	4	2	HC, 2012
AP (averaging period): non-cancer	yr	4	2	HC, 2012
AP (averaging period): cancer	yr	80	80	HC, 2012
Notes: h – hour; yr – year; wk – week; d – day. 1 – Based on parent's assumed work day (8 hrs) and commute time (1 hr) 5 days per week. 2 - BC Employment Standards Act, Entitlement to Annual Vacation – Act Part 7, Section 57.				

4.2.4 EXPOSURE PARAMETERS FOR CHILDREN AND TEENS IN ELEMENTARY SCHOOL

In this exposure scenario children and teens are considered to spend 9 hours/day, 5 days/week for 38 weeks/year (i.e., length of school year) at an elementary school. The exposure parameters applicable to children and teenagers in elementary school are as follows:

Table 4-6 Exposure Parameters for School-Aged Children and Teens

Exposure Factor	Units	Child (5 – 11 Yrs)	Teen (12 -13 Yrs)	Reference
EF (exposure frequency for inhalation) = EF_a x EF_b x EF_c	h/yr	1710	1710	Calculated
EF_a (daily exposure frequency)	d/wk	5	5	See note 1
EF_b (weekly exposure frequency)	wk/yr	38	38	See note 1
EF_c (hourly exposure frequency)	h/d	9	9	See note 1
ED (exposure duration)	yr	7	2	HC, 2012
AP (averaging period): non-cancer	yr	7	2	HC, 2012
AP (averaging period): cancer	yr	80	80	HC, 2012
Notes: h – hour; yr – year; wk – week; d – day. 1 – Site-specific. Considers the minimum number of hours of instruction provided in the British Columbia School Calendar Regulation and potential for extra curricular activities at school.				

4.2.5 EXPOSURE PARAMETERS FOR HIGH SCHOOL STUDENTS

In this exposure scenario high school students are considered to be teens (i.e., 13-19 years old) who spend 9 hours/day, 5 days/week for 38 weeks/year (i.e., length of school year) at a high school. The exposure parameters applicable to teenagers in high school are as follows:

Table 4-7 Exposure Parameters for High School Students

Exposure Factor	Units	Teen (13 -19 Yrs)	Reference
EF (exposure frequency for inhalation) = EF_a x EF_b x EF_c	h/yr	1710	Calculated
EF_a (daily exposure frequency)	d/wk	5	See note 1
EF_b (weekly exposure frequency)	wk/yr	38	See note 1
EF_c (hourly exposure frequency)	h/d	9	See note 1
ED (exposure duration)	yr	7	HC, 2012
AP (averaging period): non-cancer	yr	7	HC, 2012
AP (averaging period): cancer	yr	80	HC, 2012

Notes:
h – hour; yr – year; wk – week; d – day.
1 – Site-specific. Considers the minimum number of hours of instruction provided in the British Columbia School Calendar Regulation and potential for extra curricular activities at school.

4.2.6 EXPOSURE PARAMETERS FOR PATIENTS IN HOSPITAL FACILITY

In this exposure scenario a patient is considered to be an adult (i.e., >20 years) who stays in the hospital for 24 hours/day, 7 days/week for 5 weeks/year on an annual basis. The exposure parameters applicable to adult patients in a hospital facility are as follows:

Table 4-8 Exposure Parameters for Adults in a Hospital Setting

Exposure Factor	Units	Adult (≥ 20 Yrs)	Reference
EF (exposure frequency for inhalation) = EF_a x EF_b x EF_c	h/yr	840	Calculated
EF_a (daily exposure frequency)	d/wk	7	HC, 2012[1]
EF_b (weekly exposure frequency)	wk/yr	5	See note 2
EF_c (hourly exposure frequency)	h/d	24	HC, 2012[1]
ED (exposure duration)	yr	60	HC, 2012
AP (averaging period): non-cancer	yr	60	HC, 2012
AP (averaging period): cancer	yr	80	HC, 2012

Notes:
h – hour; yr – year; wk – week; d – day.
1 – Assumed that the hospital is the patient's "residence" for the length of their stay.
2 – Based on the median length of hospital stay (35 days) in Vancouver during COVID-19. *CMAJ* 2020 June 29; 192:E694-701.

4.2.7 EXPOSURE PARAMETERS FOR WORKERS

In this exposure scenario a worker is considered to be an adult (i.e., >20 years) who is employed on a full-time basis and spends 8 hours/day, 5 days/week for 50 weeks/year (assuming a two-week vacation period) at their workplace. The exposure parameters applicable to adult workers in a workplace setting near the refinery are as follows:

Table 4-9 Exposure Parameters for Workers

Exposure Factor	Units	Adult (≥ 20 Yrs)	Reference
EF (exposure frequency for inhalation) = EF_a x EF_b x EF_c	h/yr	2000	Calculated

Exposure Factor	Units	Adult (≥ 20 Yrs)	Reference
EF_a (daily exposure frequency)	d/wk	5	HC, 2012
EF_b (weekly exposure frequency)	wk/yr	50	See note 1
EF_c (hourly exposure frequency)	h/d	8	HC, 2012
ED (exposure duration)	yr	60	HC, 2012
AP (averaging period): non-cancer	yr	60	HC, 2012
AP (averaging period): cancer	yr	80	HC, 2012
Notes: h – hour; yr – year; wk – week; d – day. 1 - BC Employment Standards Act, Entitlement to Annual Vacation – Act Part 7, Section 57.			

4.2.8 EXPOSURE FACTORS FOR VISITORS

A visitor receptor (or recreational receptor) represents various life stages including infant, toddler, child, teen, and adult. The visitor receptor was considered to be a non-resident who frequents the HHRA study area for short trips. The visitor receptor was assumed to spend 2 hours/day, 7 days/week for 50 weeks/year (assuming a two-week vacation period) at recreational areas or otherwise spending short amounts of time within the study area. The exposure parameters applicable to visitors are as follows:

Table 4-10 Exposure Parameters for Visitors

Exposure Factor	Units	Infant (0 – 6 Mo.)	Toddler (7 Mo. To 4 Yrs)	Child (5 – 11 Yrs)	Teen (12 – 19 Yrs)	Adult (≥ 20 Yrs)	Reference
EF (exposure frequency for inhalation) = EF_a x EF_b x EF_c	h/yr	700	700	700	700	700	Calculated
EF_a (daily exposure frequency)	d/wk	7	7	7	7	7	Site-specific
EF_b (weekly exposure frequency)	wk/yr	50	50	50	50	50	See note 1
EF_c (hourly exposure frequency)	h/d	2	2	2	2	2	Site-specific
ED (exposure duration)	yr	0.5	4.5	7	8	60	HC, 2012
AP (averaging period): non-cancer	yr	0.5	4.5	7	8	60	HC, 2012
AP (averaging period): cancer	yr	80	80	80	80	80	HC, 2012
Notes: h – hour; yr – year; wk – week; d – day; mo – months. 1 - BC Employment Standards Act, Entitlement to Annual Vacation – Act Part 7, Section 57.							

4.2.9 EXPOSURE PARAMETERS FOR TSLEIL-WAUTUTH NATION CULTURAL USE

The exposure parameters applicable to TWN members who partake in outdoor cultural activities on TWN Reserve Lands are listed below. These exposure parameters pertain to all life stages including infant, toddler, child, teen, and adult, who are considered to spend 10 hours/day, 1 day/week for 52 weeks/year outdoors at these locations.

Table 4-11 Exposure Parameters for Tsleil-Waututh Nation Cultural Use

Exposure Factor	Units	Infant (0 – 6 Mo.)	Toddler (7 Mo. To 4 Yrs)	Child (5 – 11 Yrs)	Teen (12 – 19 Yrs)	Adult (≥ 20 Yrs)	Reference
EF (exposure frequency for inhalation) = EF_a x EF_b x EF_c	h/yr	520	520	520	520	520	Calculated
EF_a (daily exposure frequency)	d/wk	1	1	1	1	1	Site-specific
EF_b (weekly exposure frequency)	wk/yr	52	52	52	52	52	Site-specific
EF_c (hourly exposure frequency)	h/d	10	10	10	10	10	Site-specific
ED (exposure duration)	yr	0.5	4.5	7	8	60	HC, 2012
AP (averaging period): non-cancer	yr	0.5	4.5	7	8	60	HC, 2012
AP (averaging period): cancer	yr	80	80	80	80	80	HC, 2012

Exposure Factor	Units	Infant (0 – 6 Mo.)	Toddler (7 Mo. To 4 Yrs)	Child (5 – 11 Yrs)	Teen (12 – 19 Yrs)	Adult (≥ 20 Yrs)	Reference
Notes: h – hour; yr – year; wk – week; d – day; mo – months.							

4.3 CUMULATIVE EXPOSURES

As detailed in **Section 4.1.2**, the AQA dispersion model predicted ambient concentrations of COPCs based solely on emissions from the Parkland Refinery. These predictions can then be added to the AQA baseline values for each pollutant to determine the cumulative exposure impact of the refinery together with all other emissions sources contributing to COPC levels within the HHRA study area. Cumulative exposures from two different exposure periods were used in the exposure modelling:

1. Acute Exposures: Maximum 1-hour (SO_2), annual 98th percentile of daily 1-hr maximum (NO_2) or maximum annual 99th percentile 24-hr average 24-hour ($PM_{2.5}$)

2. Chronic Exposures: Maximum annual average of 1-hour concentrations (NO_2, $PM_{2.5}$)

In either case, the applicable baseline ambient concentration is added to the modelled value to derive the maximum cumulative concentration result (i.e., baseline + modelled = cumulative). Note that benzene and 1,3-butadiene cumulative exposures were not assessed for Scenarios 2-4, as these contaminants were not included in the dispersion modelling assessment (see **Table 4-1**). Instead, exposures for these pollutants were assessed only for Scenario 1 (Ambient Monitoring 2017-2019). Note also that chronic exposures for SO2 are not included, as the Hazard Assessment in Section 5 determined that available chronic exposure limits and objectives for SO2 are typically not human health-based, and thus not appropriate for inclusion in an HHRA.

Table 4-12 to **Table 4-14** present the cumulative exposures for Scenarios 2, 3 and 4 respectively. Note that the summary tables show predicted maximum air contaminant concentrations from the refinery at the maximum point of impingement ("MPOI"). Note also that chronic exposures for SO_2 are not included, as the Hazard Assessment in **Section 5** determined that available chronic exposure limits and objectives for SO_2 are typically not human health-based, and thus not appropriate for inclusion in an HHRA.

Table 4-12 Summary of Maximum Modelling Results for COPCs for Scenario 2 (Current Permit Maximum)

COPC	Exposure Period	Baseline Concentration (µg/m³)	Predicted Maximum Concentration[1] (µg/m³)	Maximum Cumulative Concentration (µg/m³)
SO_2	Acute	7.4	488.2	495.6
NO_2	Acute	74.7	105.6	180.3
	Chronic	22.0	3.9	25.8
$PM_{2.5}$	Acute	11.7	8.0	19.7
	Chronic	4.8	0.7	5.5
Notes: Current permit scenario reflects the refinery's MVRD Permit GVA0117 dated January 27, 2021. Note that typical operation of the refinery at fully permitted emission rates for all sources is not viable and would not occur. 1 - Concentrations reflect predicted air contaminant concentrations at the MPOI.				

Table 4-13 Summary of Maximum Modelling Results for COPCs for Scenario 3 (Amended Permit Maximum)

COPC	Exposure Period	Baseline Concentration (µg/m³)	Predicted Maximum Concentration[1] (µg/m³)	Maximum Cumulative Concentration (µg/m³)
SO_2	Acute	7.4	237.2	244.6
NO_2	Acute	74.7	103.1	177.8
	Chronic	22.0	3.1	25.1

COPC	Exposure Period	Baseline Concentration (µg/m^3)	Predicted Maximum Concentration[1] (µg/m^3)	Maximum Cumulative Concentration (µg/m^3)
$PM_{2.5}$	Acute	11.7	6.4	18.1
	Chronic	4.8	0.6	5.4
Notes: Amended permit (maximum) scenario reflects the refinery's permit limit emission releases as proposed in the Permit Application submitted to MVRD on August 20, 2021. Note that typical operation of the refinery at fully permitted emission rates for all sources is not viable and would not occur. 1 - Concentrations reflect predicted air contaminant concentrations at the MPOI.				

Table 4-14 Summary of Maximum Modelling Results for COPCs for Scenario 4 (Amended Permit Normal)

COPC	Exposure Period	Baseline Concentration (µg/m^3)	Predicted Maximum Concentration[1] (µg/m^3)	Maximum Cumulative Concentration (µg/m^3)
SO_2	Acute	7.4	107.4	114.7
NO_2	Acute	74.7	95.2	169.9
	Chronic	22.0	2.0	24.0
$PM_{2.5}$	Acute	11.7	2.0	13.7
	Chronic	4.8	0.3	5.1
Notes: Amended permit (normal) scenario reflects a more realistic emissions scenario meant to more closely simulate the actual emissions from the refinery as proposed in the Permit Application submitted to MVRD on August 20, 2021. 1 - Concentrations reflect predicted air contaminant concentrations at the MPOI.				

4.4 UNCERTAINTY ANALYSIS

A summary of the major assumptions made in the Exposure Assessment stage of the HHRA and resulting uncertainties is provided below:

- For both Scenario 1 (Ambient Monitoring 2017-2019) and Scenarios 2-4 (Dispersion Modelling) WSP relied on air quality and meteorological monitoring data gathered by the MVRD. As all MVRD monitoring data undergoes a rigorous data validation process that is compliant with Environment and Climate Change Canada ("ECCC") NAPS requirements, WSP has not performed further data validation procedures, and the data have been used "as-is". This not likely a significant source of uncertainty or bias, because MVRD requires all permittees to utilize these data for regulatory air quality assessments.
- Conservative assumptions were applied when calculating the exposure estimates (i.e., conservative assumptions for exposure durations and frequencies).
- For the purposes of exposure modelling, it has been assumed that human receptors, whether in an indoor environment or outdoor environment, would be continuously exposed to COPC concentrations in ambient air throughout the duration of their time at the given receptor location. Ambient indoor air concentrations are dependant on a multitude of variables including building infiltration rates, indoor decay rates, ventilation system setups, and other factors. To maintain a conservative approach, the assumption that equilibrium is established between outdoor and indoor ambient air was applied for this assessment.
- It is important to note that typical operation of the refinery at fully permitted emission rates for all sources is not viable and would not occur. This "maximum" scenario acts as a conservative upper bounding case that is not representative of how the refinery operates. Additional information is provided in the AQA report (WSP, 2021).

5 HAZARD ASSESSMENT

The hazard assessment step provides the basis for evaluating what is an acceptable exposure and what level of exposure may be harmful to human health. This step involves identification of potentially harmful effects associated with each COPC and determines the dose that a receptor can be exposed to without experiencing unacceptable effects. This exposure limit is called the toxicity reference value (TRV).

5.1 REVIEW OF TOXICOLOGICAL BASIS OF AVAILABLE JURISDICTIONAL AMBIENT AIR QUALITY OBJECTIVES OF IDENTIFIED COPCS

Exposure limits describe health-protective exposures for humans and are derived based on the duration of exposure. For this HHRA, exposure limits selected to evaluate short-term (acute) and long-term (chronic) exposures were based on the following definitions:

- **Acute** – single or intermittent exposures lasting up to 24-hours; and,
- **Chronic** – repeated exposures over longer term periods that are conservatively assumed to take place over a lifetime.

A toxicological review was completed of available jurisdictional ambient air quality objectives ("AAQOs") for SO_2, NO_2, $PM_{2.5}$, benzene, and 1,3-butadiene. This review considered the following:

- For the available acute and chronic AAQOs, the technical (toxicological) basis of the objective was assessed;
- The health endpoints of these objectives were identified and the toxicological study (human or animal data) upon which the objectives are based were identified. Uncertainties inherent in the studies were also described;
- The scientific rigour in the derivation of the objectives was assessed;
- Key regulatory considerations in the derivation process for the objectives were described; and,
- The jurisdictional AAQO for acute and chronic exposure durations that is health-protective was identified and applied as the TRV for each COPC in the HHRA.

Exposure limits used in the HHRA were obtained from reputable regulatory agencies that regularly review and update the science supporting the exposure limits, provide supporting documentation, and/or engage a peer-review process in their standards development process. For the purposes of this HHRA, these sources included: Federal agencies (e.g., Health Canada, Canadian Council of Ministers of the Environment ["CCME"], United States Environmental Protection Agency ["US EPA"]), provincial or state agencies (e.g., British Columbia Ministry of Environment and Climate Change Strategy ["BC MoECCS"], Alberta Environment ["AENV"], Ontario Ministry of the Environment, Conservation and Parks ["ON MECP"], California Office of Environmental Health Hazard Assessment ["Cal OEHHA"]), and international organizations (e.g., World Health Organization ["WHO"]). Human health-based exposure limits from B.C., Health Canada, and CCME were prioritized.

Scientifically defensible exposure limits applied in the HHRA for each COPC and for each exposure duration were selected based on the following considerations:

- Established or derived by reputable and credible regulatory agencies;
- Protective of public health based on the current scientific understanding of the health effects known and/or suspected to be associated with exposures to the given COPC;
- Protective of sensitive individuals through the use of appropriate uncertainty factors; and
- Supported by adequate documentation.

In the case that the above criteria were supported by more than one standard, guideline or objective, the most scientifically defensible limit was selected based on professional judgement and the rationale for the decision is

provided in the toxicity profile (**Section 5.2**). The findings of the jurisdictional review of available AAQOs for acute and chronic exposures and their toxicological basis are described in the sections below for each COPC.

5.1.1 SULPHUR DIOXIDE

Jurisdictional acute and chronic exposure limits for SO_2 are provided in **Table 5-1** and **Table 5-2**, respectively. The toxicological studies supporting these exposure limits are described in detail below.

Table 5-1 Acute Inhalation Exposure Limits for SO_2

Regulatory Agency	Type	Value (ppb)	Value (µg/m^3)	Reference
Metro Vancouver	1-hour AAQO	70	183	Metro Vancouver 2020
BC MoECCS	1-hour	70	183	BC MoECCS 2020
CCME 2020 CAAQS (2025 CAAQS)	1-hour	70 (65)	-	CCME 2017
AENV	1-hour	172	450	AENV 2011
ON MECP	10-min	67	-	MECP 2020
	1-hour	40	-	
US EPA	1-hour	75	-	US EPA, 2019
Cal OEHHA	1-hour	0.25 (ppm)	660	Cal OEHHA 2008
WHO	10-min	-	500	WHO 2005
	24-hour	-	200	

Notes:
AAQO - Ambient Air Quality Objective; CAAQS – Canadian Ambient Air Quality Standard
BC MoECCS – British Columbia Ministry of Environment and Climate Change Strategy; AENV – Alberta Environment; CCME – Canadian Council of Ministers of Environment; ON MECP – Ontario Ministry of Environment, Conservation and Parks; US EPA – United States Environmental Protection Agency; Cal OEHHA - California Office of Environmental Health Hazard Assessment; WHO – World Health Organization

Metro Vancouver and British Columbia Ministry of Environment and Climate Change Strategy

In 2016, the BC MoECCS (BC MoECCS, 2020) extended the 2014 1-hour provincial interim ambient air quality objective ("IAAQO") for SO_2 of 75 parts per billion ("ppb") to the years 2017-2019 and revised it to 70 ppb to facilitate the transition to the 2020 Canadian Ambient Air Quality Standard ("CAAQS"). The intention was to have the IAAQO values superseded by the CAAQS in 2020. The 70 ppb value is based on an annual 99th percentile value, averaged over the preceding three years. MV has also adopted the 2020 CAAQS as a "not to exceed" value, which is a more stringent application of the numerical value than applied by CAAQS or BC MoECCS.

CCME

The CCME was consulted to obtain detailed rationale for the derivation of the CAAQSs for SO_2; however, there was no technical documentation available. Ms. Krohn confirmed that the information is not currently available from the CCME website and provided to WSP a report entitled: "*Guidance Document on Achievement Determination for Canadian Ambient Air Quality Standards for Sulphur Dioxide*" (CCME, 2020). This CCME (2020) document provides guidance on methodologies for determining whether the CAAQS for SO_2 are achieved or exceeded; however, it does not provide epidemiological studies that supports either the 2020 or 2025 CAAQS for SO_2.

As described in **Section 5.2.1**, Health Canada (2016) completed a comprehensive review of relevant health- and exposure- related data during the conduct of a "Human Health Risk Assessment for Sulphur Dioxide" to provide scientific guidance to decision makers in the review and/or development of air quality policies, including the National Ambient Air Quality Objectives ("NAAQOs") and CAAQSs. Health Canada (2016) concluded the following:

- Short term SO_2 exposures and respiratory morbidity showed the strongest evidence of causality; this was largely based on 5 to 10-minute controlled human exposure studies. The assessment identified a 10-minute human health reference concentration of 67 ppb;

- More recent literature showed evidence of a "suggestive of causal" relationship between non-accidental and cardiopulmonary mortality risks and short-term exposure to SO_2;
- Recent literature has also identified additional endpoints including reproductive and developmental endpoints. However, limited data exists for these endpoints, and they have been designated as having a weakly "suggestive of causal" relationship with SO_2 exposures;
- Intermittent spikes in SO_2 exposures are linked to respiratory morbidity and likely to other endpoints such as reproductive and developmental; and,
- There is "inadequate evidence to infer a causal relationship" between long term SO_2 exposures and adverse health effects.

Alberta Environment

Alberta Environment (AENV, 2011) issued a 1-hour AAQO for SO_2 of 450 micrograms per cubic metre ("µg/m^3") (172 ppb) based on pulmonary effects. In the Alberta Health & Wellness document entitled "Health Effects Associated with Short-term Exposure to Low Levels of Sulphur Dioxide (SO_2) – A Technical Review" (AHW, 2006), it was reported that healthy individuals exposed to SO_2 may exhibit increased airway resistance and bronchoconstriction, decreased maximum expiratory flow and decreased pulmonary function. While asthmatics experience similar effects, increases in asthma symptoms, wheezing, chest tightness, and difficulty breathing were also reported. It was concluded that transitory pulmonary effects might be expected for asthmatics at exposure concentrations between 0.5 and 1 parts per million ("ppm"), and for healthy humans between 0.75 ppm and 25 ppm. It is unclear what uncertainty factors or derivation methods were applied by AENV to generate the 1-hour AAQO.

Ontario Ministry of the Environment, Conservation and Parks

The ON MECP (MECP, 2020) provides a 1-hour Ambient Air Quality Criterion ("AAQC") for SO_2 of 40 ppb. This value was converted from the 10-min AAQC of 67 ppb [likely adopted from Health Canada (2016), although not specifically mentioned] to allow assessment of 1-hour air quality data. While the ON MECP identifies that this numerical value is based on health endpoints, there were no technical supporting documents that provided detailed rationale supporting the derivation of this AAQC.

United States Environmental Protection Agency

Although no inhalation reference concentration ("RfC") was available from US EPA, a 1-hour National Ambient Air Quality Standard ("NAAQS") of 75 ppb has been derived by the US EPA (2010). This value is based on the 3-year average of the 99th percentile of the annual distribution of 1-hour daily maximum SO_2 concentrations. This value was derived in 2010, in which the US EPA significantly strengthened the SO_2 1-hour NAAQS and revoked the 24-hour and annual standards. This revised value was established to protect against respiratory effects associated with exposure to SO_2 as short as a few minutes, based on human health studies documenting the respiratory effects elicited in asthmatics following a 5 to 10 minute exposure to SO_2 at concentrations as low as 200 ppb under elevated breathing rates. In 2019, the US EPA conducted a review of the health-based 1-hour NAAQS for SO_2 (75 ppb) and concluded that the value should be retained as the current standard to protect public health with an adequate margin of safety.

California Office of Environmental Health Hazard Assessment

The Cal OEHHA (Cal OEHHA, 2008) derived a 1-hour inhalation reference exposure limit ("REL") of 660 µg/m^3 based on impairment of airway function, especially in asthmatics. After reviewing several studies on controlled human data on acute exposures of healthy, asthmatic, and atopic individuals at concentrations as low as 0.25-2.0 ppm, it was determined that 0.25 ppm is a SO_2 concentration level that would not result in adverse respiratory health effects in sensitive individuals for a period of 1-hour. According to the literature review conducted by Cal OEHHA, this value coincides with the No Observed Adverse Effect Level ("NOAEL") identified in sensitive individuals and is deemed to be protective of asthmatics, as it was determined that adverse respiratory effects were consistently observed only at higher concentrations.

World Health Organization

The WHO (WHO, 2005) derived a 10-minute and 24-hour guideline of 500 and 20 µg/m^3 for SO_2, respectively. Effects from acute exposure were largely gathered from controlled chamber experiments on volunteers, which indicated that changes in pulmonary function and development of respiratory symptoms were observed when asthmatics were subject to exposure of SO_2 for periods as short as 10 minutes. Based on this observation, it was recommended that a value of 500 µg/m^3 should not be exceeded over an averaging period of 10 minutes. The 24-hour guideline acts as a precautionary value, which is based on several epidemiological studies associating exposure to SO_2 with mortality. It is unclear what uncertainty factors or derivation methods were applied by WHO to generate the 10-minute and 24-hour guidelines.

Table 5-2 Chronic Inhalation Exposure Limits for SO_2

Regulatory Agency	Type	Value (ppb)	Value (µg/m^3)	Reference
Metro Vancouver	Annual	5	13	Metro Vancouver 2020
BC MoECCS	Annual	5	13	BC MoECCS 2020
CCME 2020 CAAQS (2025 CAAQS)	Annual	5 (4)	-	CCME 2021
AENV	Annual	8	20	AENV 2011
ON MECP	Annual	4	11	MECP 2020
US EPA	-	-	-	US EPA, 2019
Cal OEHHA	-	-	-	Cal OEHHA 2008
WHO	-	-	-	WHO 2005

Notes:
BC MoECCS – British Columbia Ministry of Environment and Climate Change Strategy; CAAQS – Canadian Ambient Air Quality Standard; AENV – Alberta Environment; CCME – Canadian Council of Ministers of Environment; ON MECP – Ontario Ministry of Environment, Conservation and Parks; US EPA – United States Environmental Protection Agency; Cal OEHHA - California Office of Environmental Health Hazard Assessment; WHO – World Health Organization

Metro Vancouver and British Columbia Ministry of Environment and Climate Change Strategy

The British Columbia Ministry of Environment and Climate Change Strategy (BC MoECCS 2020) and Metro Vancouver (2020) adopted the CAAQS for 2020 promulgated by CCME. Achievement of this target is based on annual average of 1-hr concentrations over one year.

CCME

Technical supporting documents were not available to determine the basis for the annual CAAQS for SO_2. Additional information is provided below in **Section 5.2.1**

Alberta Environment

Alberta Environment (AENV, 2011) issued an annual AAQO for SO_2 of 20 µg/m^3 (8 ppb). This value was adopted from the European Union and was based on the protection of ecosystems, not human health. An annual AAQO designed to protect human health was not provided.

Ontario Ministry of the Environment, Conservation and Parks

The ON MECP (2020) provides an annual AAQC of 4 ppb for SO_2. The ON MECP identifies that the basis of this numerical value is vegetation (i.e., provides a level of protection against toxicity due to deposition on or uptake of the contaminant by plants). An annual AAQC designed to protect human health was not provided.

5.1.2 NITROGEN DIOXIDE

Jurisdictional acute and chronic exposure limits for NO_2 are provided in **Table 5-3** and **Table 5-4**, respectively. The toxicological studies supporting these exposure limits are described in detail below.

Table 5-3 Acute Inhalation Exposure Limits for NO_2

Regulatory Agency	Type	Value (ppb)	Value (µg/m^3)	Reference
Metro Vancouver	1-hour AAQO	60	113	Metro Vancouver 2020

BC MoECCS	1-hour AAQO	60	113	BC MoECCS 2020
CCME 2020 CAAQS (2025 CAAQS)	1-hour CAAQS	60 (42)	-	CCME 2017
AENV	1-hour AAQO	159	300	AENV 2011
ON MECP	1-hour AAQC	200	400	MECP 2020
	24-hour AAQC	100	200	
US EPA	1-hour Standard	100	-	US EPA 2018
Cal OEHHA	1-hour REL	-	470	California OEHHA 2008
WHO	1-hour AQG	-	200	WHO 2005

Notes:
AAQO - Ambient Air Quality Objective; AAQC – Ambient Air Quality Criteria; AQG - Air Quality Guideline; CAAQS – Canadian Ambient Air Quality Standard; REL – Reference Exposure Level
BC MoECCS – British Columbia Ministry of Environment and Climate Change Strategy; AENV – Alberta Environment; CCME – Canadian Council of Ministers of Environment; ON MECP – Ontario Ministry of Environment, Conservation and Parks; US EPA – United States Environmental Protection Agency; Cal OEHHA - California Office of Environmental Health Hazard Assessment; WHO – World Health Organization

Metro Vancouver and British Columbia Ministry of Environment and Climate Change Strategy

The British Columbia Ministry of Environment and Climate Change Strategy (BC MoECCS 2020) and MV (2020) revised their acute 1-hour AAQOs for NO_2 to further reduce NO_x emissions and minimize impacts to public health resulting from increasing population density. Both BC MoECCS and MV adopted the 2020 CAAQS for NO_2 endorsed by the CCME in 2017. The Provincial Framework (2020) lays out an approach for setting AAQO relative to the CAAQS. Whenever CAAQS are available, CAAQS and their supporting science assessments form the basis from which the provincial AAQO are developed. The process of adopting AAQO involves consideration of B.C.-specific factors that include vulnerable populations and other sensitive receptors, achievability, and clarifications of how AAQO will be implemented.

The proposed change in the CAAQS by the CCME is based on strong correlation between increasing NO_2 ambient air levels and respiratory effects, and contribution to early mortality at ambient concentrations commonly found in Canada particularly for sensitive individuals including the young, elderly and those with pre-existing respiratory conditions (Metro Vancouver 2020).

CCME

CCME was consulted to obtain detailed rationale for the derivation of the CAAQS for NO_2; however, there was no technical documentation available. WSP contacted Ms. Megan Krohn, Program Coordinator at CCME, to request technical scientific documentation that supports the CAAQS for NO_2. Ms. Krohn confirmed that the information is not currently available from the CCME website and provided to WSP a report entitled: "Guidance Document on Achievement Determination for Canadian Ambient Air Quality Standards for Nitrogen Dioxide" (CCME, 2020). This CCME (2020) document provides guidance on methodologies for determining whether the CAAQS for NO_2 are achieved or exceeded; however, it does not provide epidemiological studies that supports either the 2020 or 2025 CAAQS for NO_2.

As detailed in **Section 5.2.2**, Health Canada (2016) completed a comprehensive review of relevant health- and exposure-related data during the conduct of a "Human Health Risk Assessment for Ambient Nitrogen Dioxide" to support the development of the CAAQS for NO_2 to replace the previous NAAQOs. Health Canada (2016) concluded the following:

- There is strong evidence that ambient NO_2 causes both short-term and long-term respiratory effects, and short-term mortality, as well as suggestive evidence linking it to a wide range of other adverse health outcomes;
- These effects have been observed in epidemiological studies at NO_2 concentrations that commonly occur in Canada, well below the levels of the NAAQOs and other ambient standards, such as provincial/territorial guidelines and the US National Ambient Air Quality Standards;
- In studies examining the shape of the concentration-response curve, there is an approximately linear relationship between ambient NO_2 concentrations and health effects, with no clear evidence of a threshold; hence, based on the balance of the evidence it should be assumed that any increment in levels of ambient

NO_2 presents an increased risk for health effects, up to and including mortality (see detailed discussion provided in **Section 5.2.2** and **5.3**); and

- The health evidence supports the establishment of both short-term and long-term standards to protect against the full suite of health effects associated with ambient NO_2.

Alberta Environment

Alberta Environment (AENV 2011) has issued a 1-hour AAQO for NO_2 of 159 parts per billion (ppb; 300 µg/m^3) based on respiratory effects. The previous 24-hour AAQO of 200 µg/m^3 has been withdrawn by AENV. However, limited information is provided regarding the rationale for the derivation of 300 µg/m^3 as the 1-hour objective. The report titled: "*Assessment Report on Nitrogen Dioxide for Developing Ambient Air Quality Objectives"* (AENV 2007) provides a general overview of the potential health effects associated with NO_2; however, it did not detail the derivation of the 1-hour value. The report noted that healthy individuals may experience airway inflammation following acute exposures to NO_2 concentrations of 2000 ppb or lower. Individuals with pre-existing respiratory conditions including those with asthma, COPD or chronic bronchitis will experience greater sensitivity to acute NO_2 exposures compared to healthy individuals. Pre-exposure to NO_2 can also increase responsiveness to allergens by asthmatic individuals. It is unclear what effect thresholds or uncertainty factors were selected by AENV in the derivation of the 1-hour AAQO of 300 µg/m^3.

Ontario Ministry of Environment, Conservation and Parks

The ON MECP provides a 1-hour AAQC of 200 ppb (400 µg/m^3) and a 24-hour AAQC of 100 ppb (200 µg/m^3). While the ON MECP identifies that these numerical values are based on health, there was no technical supporting document that provides detailed rationale supporting the derivation of these AAQCs.

United States Environmental Protection Agency

Although no inhalation RfC was available from US EPA (2012), a 1-hour NAAQS has been derived by the US EPA (2010). This value is based on a 3-year average 98th percentile of the annual distribution of daily maximum 1-hour concentrations. Although it is derived from NO_2 exposure data, it is intended to apply to all NO_x compounds. Experimental evidence from human and animal studies indicates that respiratory effects attributable to NO_2 can occur after brief exposures (e.g., less than 1 hour up to 3 hours). The US EPA's 2008 Integrated Science Assessments concluded that 1-hour exposures of 100 ppb may result in small, yet significant increases in airway responsiveness. This is based in part on the observations from human clinical studies where airway inflammation and increased airway responsiveness were observed in asthmatics at concentrations less than 2 ppm. In contrast, airway inflammation has been observed at much higher concentrations (100 to 200 ppm/minute or 1 ppm for 2 to 3 hours) in healthy individuals. The 1-hour standard of 100 ppb (188 µg/m^3) is intended to be protective of sensitive individuals in the population, including asthmatics and individuals with pre-existing respiratory conditions. On April 6, 2018 based on a review of the full body of scientific evidence, US EPA issued a decision to retain the current NAAQS for oxides of nitrogen. US EPA concluded that the current NAAQS provide adequate protection of public health, including at-risk populations of older adults, children, and people with asthma, with an adequate margin of safety.

California Office of Environmental Health Hazard Assessment

The California Office of Environmental Health Hazard Assessment (OEHHA, 2008) derived a 1-hour REL of 470 µg/m^3 based upon respiratory effects. While OEHHA (2008) identified that the REL is based on a NOAEL of 250 ppb (470 µg/m^3) in sensitive asthmatics exposed for 1 hour with increase in airway reactivity as the critical effect, the key study upon which this is based is not well described. Also, the supporting document cited (CARB, 1992) is not readily available.

World Health Organization

The WHO (WHO, 2005) derived a 1-hour guideline of 200 µg/m^3 for NO_2. This value is based on short-term animal and human experimental toxicology studies which associate significant health effects (including adverse respiratory effects) with exposure to NO_2 levels greater than 200 µg/m^3. In a 1992 meta-analysis of 20 broncho-constrictor studies of asthmatics and 5 studies of normal subjects, researchers identified a statistically significant increase in airway responsiveness to a range of constrictor stimuli when asthmatic subjects were exposed to levels of NO_2 greater than 200 µg/m^3. WHO has specified that as this short-term guideline of 200 µg/m^3 has yet to be

challenged by more recent studies (at the time of writing), the guideline should therefore remain. WHO has not updated their guideline for NO_2 since 2005.

Table 5-4 Chronic Inhalation Exposure Limits for NO_2

Regulatory Agency	Type	Value (ppb)	Value (µg/m³)	Reference
Metro Vancouver	Annual AAQO	17	32	Metro Vancouver 2020
BC MoECCS	Annual AAQO	17	32	BC MoECCS 2020
CCME 2020 CAAQS (2025 CAAQS)	Annual CAAQS	17 (12)	-	CCME 2017
AENV	Annual AAQO	24	45	AENV AAQO 2019
ON MECP	Annual AAQC	-	-	Ontario MECP 2020
US EPA	Annual Standard	53	100	US EPA 2018
WHO	Annual AQG	-	40	WHO 2005

Notes:
AAQO - Ambient Air Quality Objective; AAQC – Ambient Air Quality Criteria; AQG - Air Quality Guideline; CAAQS – Canadian Ambient Air Quality Standard; REL – Reference Exposure Level
BC MoECCS – British Columbia Ministry of Environment and Climate Change Strategy; AENV – Alberta Environment; CCME – Canadian Council of Ministers of Environment; ON MECP – Ontario Ministry of Environment, Conservation and Parks; US EPA – United States Environmental Protection Agency; Cal OEHHA - California Office of Environmental Health Hazard Assessment; WHO – World Health Organization

Metro Vancouver and British Columbia Ministry of Environment and Climate Change Strategy

Similar to the 1-hour AAQOs, the BC MoECCS (2020) and MV (2020) revised their annual AAQOs for NO_2 by adopting the 2020 annual CAAQS for NO_2 endorsed by CCME in 2017. The Provincial Framework (2020) lays out an approach for setting AAQO relative to the CAAQS. Whenever CAAQS are available, CAAQS and their supporting science assessments form the basis from which the provincial AAQO are developed. The process of adopting AAQO involves consideration of B.C.-specific factors that include vulnerable populations and other sensitive receptors, achievability, and clarifications of how AAQO will be implemented.

This proposed change is based on strong correlation between increasing NO_2 ambient air levels and respiratory effects, and contribution to early mortality at ambient concentrations commonly found in Canada particularly for sensitive individuals including the young, elderly and those with pre-existing respiratory conditions (MV 2019).

CCME

As detailed in **Section 5.2.2**, technical supporting documents were not available to determine the basis for the annual CAAQS for NO_2.

Alberta Environment

Alberta Environment (2011) derived an annual AAQO of 24 ppb (45 µg/m³) based on effects to vegetation. The report titled: "Assessment Report on Nitrogen Dioxide for Developing Ambient Air Quality Objectives" (AENV 2007) provides a general overview of the potential chronic human health and plant health effects but does not provide detailed information regarding exposure concentrations above which adverse effects would be anticipated in humans.

Ontario Ministry of Environment, Conservation and Parks

The ON MECP has not determined an annual AAQC for NO_2.

United States Environmental Protection Agency

The US EPA (2012) has not derived a chronic inhalation RfC for NO_2. In 1971, US EPA derived a NAAQS of 53 ppb (100 µg/m³) which remains current to date based on a scientific and regulatory review that was completed (US EPA, 2018). Although the 1971 document is not readily available, the scientific reviews conducted in 1993 and 2018 by US EPA suggested that the annual standard is associated with the potential for human health effects. A scientific review of the annual air standard conducted in 1993 suggested that the standard of 53 ppb (100 µg/m³) should be upheld, based upon the results of a meta-analysis of epidemiological studies conducted in children ages 5

to 12. Within this review, an increase of 0.015 ppm or 28 µg/m^3 of NO_2 over an averaging period of 2 weeks was associated with a 20% increase in respiratory symptoms. The NO_2 sources included both indoor and outdoor sources, and average concentrations in the studies were noted to range from 0.008 to 0.065 ppm (US EPA 1993). In 1996, the annual standard was maintained by the US EPA on the basis that, in combination with the short-term standard, the annual standard was protective of both the potential short-term and long-term human health effects of NO_2 exposure (US EPA 1996). The most recent edition of the Final Rule (US EPA, 2018) indicates that the annual standard of 53 ppb (100 µg/m^3) should be retained due to the uncertainty associated with the potential long-term effects of NO_2.

World Health Organization

The WHO (2005) guideline value of 23 ppb (40 µg/m^3) represents an annual value recommended by the WHO International Program on Chemical Safety ("IPCS"). WHO IPCS (1997) indicates that the 23 ppb (40 µg/m^3) value is based on consideration of background concentrations and the observation that harmful health effects occur with an additional level of 15 ppb (or 28.2 µg/m^3) or more. It should be noted that some population studies have identified an association between adverse health effects and exposure to NO_2 levels below 40 µg/m^3. While the results of these studies may warrant a lowering of the current guideline, it is also important to consider that adverse effects may be a consequence of co-exposure since NO_2 is an important constituent of combustion generated air pollution and is highly correlated with other primary and secondary combustion products. As such, WHO has determined that it is unclear to what extent the health effects observed are attributable to NO_2 itself, therefore, the guideline value of 40 µg/m^3 has been retained until challenged by sufficient evidence.

5.1.3 FINE PARTICULATE MATTER (<2.5 µm)

Jurisdictional acute and chronic exposure limits for $PM_{2.5}$ are provided in **Table 5-5** and **Table 5-6**, respectively. The toxicological studies supporting these exposure limits are described in detail below.

Table 5-5 Acute Inhalation Exposure Limits for $PM_{2.5}$

Regulatory Agency	Type	Value (ppb)	Value (µg/m^3)	Reference
Metro Vancouver	24-hour		25	Metro Vancouver 2020
BC MoECCS	24-hour	-	25	BC MoECCS 2020
AENV	1-hour	-	80	AENV 2018
	24-hour	-	29	
CCME 2020 CAAQS	24-hour	-	27	CCME 2017
ON MECP	24-hour	-	27	MECP 2020
US EPA	24-hour	-	35	US EPA 2021
Cal OEHHA	24-hour	-	-	Cal OEHHA 2016
WHO	24-hour	-	25	WHO 2005

Notes:
BC MoECCS – British Columbia Ministry of Environment and Climate Change Strategy; AENV – Alberta Environment; CCME – Canadian Council of Ministers of Environment; CAAQS – Canadian Ambient Air Quality Standard; ON MECP – Ontario Ministry of Environment, Conservation and Parks; US EPA – United States Environmental Protection Agency; Cal OEHHA - California Office of Environmental Health Hazard Assessment; WHO – World Health Organization

Metro Vancouver and British Columbia Ministry of Environment and Climate Change Strategy

The 24-hour provincial air quality objective ("AQO") is 25 µg/m^3 and is based on annual 98th percentile of daily averages, over one year. No technical supporting documents detailing the derivation of the AQO were made available. MV (2020) has also adopted this value and determines compliance based on a rolling average.

CCME

The CCME provides a 24-hour 2020 CAAQS for $PM_{2.5}$ (27 µg/m^3); however, unlike other pollutants such as SO_2 and NO_2, a 2025 CAAQS is not provided for fine PM. CCME was consulted to obtain detailed rationale for the derivation of the CAAQS for fine PM; however, there was no technical documentation available. Ms. Krohn confirmed that the information is not currently available from the CCME website and provided to WSP a report entitled: "*Guidance Document on Achievement Determination Canadian Ambient Air Quality Standards for Fine*

Particulate Matter and Ozone" (CCME, 2020). This CCME (2020) document provides guidance on methodologies for determining whether the CAAQS for fine PM are achieved or exceeded; however, it does not provide epidemiological studies that support the 2020 CAAQS for $PM_{2.5}$.

Alberta Environment

Alberta Environment (AENV, 2019) issued a 1-hour and 24-hour AAQO of 80 µg/m^3 and 29 µg/m^3, respectively. The 1-hour value is intended for use in monitoring and reporting of the Ambient Air Quality Index. The 24-hour value is reported as being based on health effects (AENV, 2018). AENV (2018) outlines that exposure to fine PM may be associated with respiratory health effects including: reduced lung function, asthma, emphysema and bronchitis, or cardiovascular effects such as: angina, heart attacks and hypertension. Fine PM has also been linked with increased emergency room visits and hospitalizations. AENV (2018) also referenced a 2011 Health Canada report which identified a linear relationship between the concentration of $PM_{2.5}$ and the health response, with no clear evidence of a threshold for effects. Beyond this information, it is unclear how AENV came to derive the 1-hour and 24-hour AAQOs.

Ontario Ministry of the Environment, Conservation and Parks

The ON MECP (MECP, 2020) provides a 24-hour AAQC for $PM_{2.5}$ of 27 µg/m^3. This value reflects the 3-year average of the annual 98th percentile of the daily 24-hr average concentrations and is based on the 2020 CAAQS value. While the MECP (2020) identifies that this numerical value is based on health endpoints, there were no technical supporting documents that provide rationale supporting the derivation of this AAQC. For more details, the MECP references a 2012 CCME document entitled "*Guidance Document on Achievement Determination Canadian Ambient Air Quality Standards for Fine Particulate Matter and Ozone*". However, the document only focuses on methodologies, criteria, and procedures for reporting on achievement of the CAAQS and makes no mention of how the CAAQS value was derived.

United States Environmental Protection Agency

In 2006, the 24-hour NAAQS for $PM_{2.5}$ was lowered from 65 to 35 µg/m^3. This value (35 µg/m^3) is identified as a 98th percentile value, averaged over 3 years. US EPA (2006) concluded that a 24-hour standard of 35 µg/m^3 would protect public health with an adequate margin of safety from serious health effects including premature mortality and hospital admissions for cardiorespiratory causes that are likely associated with short-term exposure to fine PM. In 2012, US EPA re-evaluated the 24-hour value of 35 µg/m^3 for fine PM and retained it as the current standard.

World Health Organization

The WHO (2005) provided a 24-hour guideline for $PM_{2.5}$ of 25 µg/m^3. This value represents the 99th percentile of the distribution of daily values and is intended to protect against peaks of pollution that would lead to substantial excess morbidity or mortality. This value is largely based on published risk coefficients from multicentre studies and meta-analyses, which reported an average short-term mortality effect for PM_{10} of approximately 0.5% per 10 µg/m^3. This value is considered to provide a significant reduction in risks from acute exposure health effects such as short-term mortality.

Table 5-6 Chronic Inhalation Exposure Limits for $PM_{2.5}$

Regulatory Agency	Type	Value (ppb)	Value (µg/m^3)	Reference
Metro Vancouver	Annual		8	Metro Vancouver 2020
BC MoECCS	Annual	-	8	BC MoECCS 2020
CCME 2020 CAAQS	Annual	-	8.8	CCME 2017
AENV	-	-	-	AENV 2019
ON MECP	Annual	-	8.8	MECP 2020
US EPA	Annual	-	12	US EPA 2021
Cal OEHHA	Annual	-	12	Cal OEHHA 2016
WHO	Annual	-	10	WHO 2005
Notes:				

Regulatory Agency	Type	Value (ppb)	Value (µg/m^3)	Reference
BC MoECCS – British Columbia Ministry of Environment and Climate Change Strategy; AENV – Alberta Environment; CCME – Canadian Council of Ministers of Environment; CAAQS – Canadian Ambient Air Quality Standard; ON MECP – Ontario Ministry of Environment, Conservation and Parks; US EPA – United States Environmental Protection Agency; Cal OEHHA - California Office of Environmental Health Hazard Assessment; WHO – World Health Organization				

Metro Vancouver and British Columbia Ministry of Environment and Climate Change Strategy

In 2009, BC MoECCS (2020) provided an annual AQO of 8 µg/m^3 for $PM_{2.5}$. No technical supporting documents detailing the derivation of the AQO were made available. Metro Vancouver has adopted the same AQO and evaluates compliance based on annual average of 1-hour concentrations over one year.

CCME

The CCME provides an annual 2020 CAAQS for $PM_{2.5}$ (8.8 µg/m^3); however, unlike other pollutants such as SO_2 and NO_2, a 2025 CAAQS is not provided for fine PM. CCME was consulted to obtain detailed rationale for the derivation of the CAAQS for fine PM; however, there was no technical documentation available. Ms. Krohn confirmed that the information is not currently available from the CCME website and provided to WSP a report entitled: "*Guidance Document on Achievement Determination Canadian Ambient Air Quality Standards for Fine Particulate Matter and Ozone*" (CCME, 2020). This CCME (2020) document provides guidance on methodologies for determining whether the CAAQS for $PM_{2.5}$ are achieved or exceeded; however, it does not provide epidemiological studies that support the 2020 CAAQS for $PM_{2.5}$.

Ontario Ministry of the Environment, Conservation and Parks

The ON MECP (2020) provides an annual AAQC of 8.8 µg/m^3 for $PM_{2.5}$. The value reflects a 3-year average of the annual average concentrations. While the MECP identifies that this numerical value is based on health endpoints, there were no technical supporting documents that provide rationale supporting the derivation of this AAQC. For more details, the MECP references a 2012 CCME document entitled "*Guidance Document on Achievement Determination Canadian Ambient Air Quality Standards for Fine Particulate Matter and Ozone*". However, the document only focuses on methodologies, criteria, and procedures for reporting on achievement of the CAAQS and makes no mention of how the CAAQS was derived.

United States Environmental Protection Agency

In 2013, US EPA lowered the annual NAAQS for $PM_{2.5}$ from 15 to 12 µg/m^3, a value identified as an annual arithmetic mean, averaged over 3 years. Growing evidence since the last review showed that a lowering of the 15 µg/m^3 standard (originally set in 1997) was warranted given the multiple, multi-city studies over long periods of time demonstrating clear evidence of premature death, cardiovascular and respiratory harm as well as reproductive and developmental harm at concentrations below 15 µg/m^3. US EPA (2013) determined that an annual standard of 12 µg/m^3 is below the long-term mean $PM_{2.5}$ concentrations reported in each of the key multi-city, long- and short-term exposure studies that identified numerous serious health effects such as premature mortality and increased hospitalization for cardiovascular and respiratory effects. Additionally, a standard of 12 µg/m^3 takes into account the evidence of reproductive and developmental effects such as infant mortality and low birth weight which were identified in studies that provided evidence suggestive of a causal relationship with long-term $PM_{2.5}$ concentrations. A level of 12 µg/m^3 is approximately the same level as the lowest long-term mean concentration reported in these studies. US EPA (2013) concluded that an annual standard of 12 µg/m^3 provides the requisite degree of public health protection including the health of sensitive populations, with an adequate margin of safety.

California Office of Environmental Health Hazard Assessment

Cal OEHHA recommended an annual CAAQS of 12 µg/m^3 for $PM_{2.5}$, which places significant weight on the long-term exposure studies using the American Cancer Society ("ACS") and Harvard Six-Cities data. In both studies, robust associations were identified between long-term exposure to $PM_{2.5}$ and mortality; the mean $PM_{2.5}$ concentrations were 18 and 18.2 µg/m^3 in the Harvard and ACS studies, respectively. In addition, the annual CAAQS placed weight on the results of multiple studies investigating the relationship between $PM_{2.5}$ and adverse health outcomes. These studies had long-term (three- to four-year) means in the range of 13 to 18 µg/m^3. It was concluded by Cal OEHHA (2001) that an annual $PM_{2.5}$ standard of 12 µg/m^3 would provide adequate public health protection, including that of infants and children, against adverse effects of long-term exposure.

World Health Organization

An annual average guideline value of 10 µg/m^3 for $PM_{2.5}$ was set by WHO (2005) to represent the lower end of the range over which significant effects on survival have been observed in the previously mentioned ACS study. This value also places significant weight on the long-term exposure studies using the ACS and Harvard Six Cities data which demonstrated a robust association between long-term exposure to $PM_{2.5}$ and mortality (also discussed above). This annual standard is believed to be both achievable in large urban settings and is expected to effectively reduce health risks.

5.1.4 BENZENE

Jurisdictional acute and chronic exposure limits for benzene are provided in **Table 5-7** and **Table 5-8**, respectively. There are no available jurisdictional limits from BC MoECCS or CCME. The toxicological studies supporting the available exposure limits are described in detail below.

Table 5-7 Acute Inhalation Exposure Limits for Benzene

Regulatory Agency	Type	Value (ppb)	Value (µg/m^3)	Reference
BC MoECCS	1-hr AAQO	-	-	BC MoECCS 2020
	8-hr AAQO	-	-	
AENV	1-hr AAQO	9.0	30	AENV 2019
	8-hr AAQO	-	-	
ATSDR	Acute MRL	9	30	ATSDR 2007
	Intermediate MRL	6	19.44	
CCME	1-hr CAAQS	-	-	CCME 2017
Health Canada	REL	-	-	Health Canada 2021
	Inhalation Tolerable Concentration	-	-	
ON MECP	1-hr AAQC	-	-	MECP 2020
	8-hr AAQC	-	-	
US EPA	1-hr Standard	-	-	US EPA NAAQS Table 2021
	8-hr Standard	-	-	
Cal OEHHA	1-hr REL	8	26	California OEHHA 2014
	8-hr REL	0.1	3	
WHO	1-hr AQG	-	-	WHO 2000
	8-hr AQG	-	-	

Notes:
AAQO - Ambient Air Quality Objective; AAQC – Ambient Air Quality Criteria; AQG - Air Quality Guideline; CAAQS – Canadian Ambient Air Quality Standard; MRL – Minimum Risk Level; NAAQS – National Ambient Air Quality Standard; REL – Reference Exposure Level

AENV – Alberta Environment, BC MoECCS – British Columbia Ministry of Environment and Climate Change Strategy; ATSDR- Agency for Toxic Substances and Disease Registry, Cal OEHHA - California Office of Environmental Health Hazard Assessment; CCME – Canadian Council of Ministers of Environment; ON MECP – Ontario Ministry of Environment, Conservation and Parks; US EPA – United States Environmental Protection Agency; WHO – World Health Organization

Table 5-8 Chronic Inhalation Exposure Limits for Benzene

Regulatory Agency	Type	Value (ppb)	Value (µg/m^3)	Reference
BC MoECCS	Annual AAQO	-	-	BC MoECCS 2020
AENV	Annual AAQO	0.9	3	AENV AAQO 2019
CCME	Annual CAAQS	-	-	CCME 2017
Health Canada	Risk-Specific Concentration	0.19 to 1.4	0.6 to 4.5	Health Canada 2021; Risk-Specific Concentration that corresponds with derived Inhalation Unit Risks of 1.6 x 10^{-2} (mg/m^3)$^{-1}$

ON MECP	Annual AAQC	0.14	0.45	MECP 2020
	24-hour AAQC	0.72	2.3	
Cal OEHHA	Chronic REL	1	3	OEHHA 2014; based on health effects to hematologic system, nervous system, and developmental effects
ATSDR	Chronic MRL	3	9	ATSDR 2007
TCEQ	Annual Average	1.4	4.5	TCEQ 2015; based on long-term effect screening level used for permitting and an incremental lifetime cancer risk of 1-in-100,000 of developing leukemia
US EPA	Reference Concentration	9	30	US EPA 2003 based on decreased lymphocyte count based on human occupational inhalation study (Rothman *et al* 1996)
	Risk-Specific Concentrations	0.4 to 1.4	1.3 to 4.5	US EPA 2003 ; Risk-Specific Concentrations that correspond with derived Inhalation Unit Risks that range from 2.2×10^{-6} $(\mu g/m^3)^{-1}$ to 7.8×10^{-6} $(\mu g/m^3)^{-1}$
WHO	Risk-Specific Concentrations	0.53	1.7	WHO 2017; based on protection of leukaemia effects and an incremental lifetime cancer risk of 1-in-100,000

Notes:
AAQO - Ambient Air Quality Objective; AAQC – Ambient Air Quality Criteria; AQG - Air Quality Guideline; CAAQS – Canadian Ambient Air Quality Standard; MRL – Minimum Risk Level, REL – Reference Exposure Level

AENV – Alberta Environment, BC MoECCS – British Columbia Ministry of Environment and Climate Change Strategy; ATSDR-Agency for Toxic Substances and Disease Registry, Cal OEHHA - California Office of Environmental Health Hazard Assessment; CCME – Canadian Council of Ministers of Environment; ON MECP – Ontario Ministry of Environment, Conservation and Parks; US EPA – United States Environmental Protection Agency; WHO – World Health Organization

Alberta Environment

Alberta Environment (AENV, 2019) reports a 1-hour AAQO for benzene of 30 µg/m^3 (9 ppb) based on haematological effects. This value was adopted from Texas and the guideline was developed in 1999. According to the Texas Commission on Environmental Quality ("TCEQ"), the basis for the development of short-term and long-term effects screening level are unknown; however, these levels are based on data concerning health effects, odour nuisance potential, effects with respect to vegetation and corrosion effects and are not ambient air standards. If predicted or measured airborne levels of a chemical do not exceed the screening level, adverse health or welfare effects would not be expected to result. If ambient levels of constituents in air exceed the screening levels, it does not necessarily indicate a problem, rather, it triggers a more in-depth review.

The annual average AAQO for benzene is 3 µg/m^3 (0.9 ppb) based on carcinogenic effects.

United States Environmental Protection Agency

The US EPA (2002) derived a RfC for benzene of 30 µg/m^3, which represents a daily inhalation exposure of the human population (including sensitive subgroups) that is likely to be without appreciable risk of deleterious haematological (blood) effects during a lifetime of exposure. The RfC was derived based on benchmark dose ("BMD") modelling of the absolute lymphocyte count data from the occupational epidemiologic study of Rothman *et al.* (1996), in which workers were exposed to benzene by inhalation. A comparative analysis based on BMD modelling of haematological data from the Ward *et al*. (1985) subchronic experimental animal inhalation study was also conducted. In addition, comparative analyses using the Lowest-Observed Adverse Effect Level ("LOAEL") from the Rothman *et al.* (1996) study and the NOAEL from the Ward *et al.* (1985) study were performed.

The RfC was derived by dividing the adjusted benchmark concentration level of 8.2 mg/m^3 by the overall uncertainty factor ("UF") of 300 (i.e., RfC = $BMCL_{ADJ/UF}$ = 8.2 mg/m^3 ÷ 300 = 0.03 mg/m^3). The overall UF of 300 comprises a UF of 3 for effect-level extrapolation, 10 for intraspecies differences (human variability), 3 for subchronic-to-chronic extrapolation, and 3 for database deficiencies.

US EPA (2003) derived Inhalation Unit Risks ("IUR") of 2.2 x10^{-6} (µg/m^3)$^{-1}$ to 7.8 x 10^{-6} (µg/m^3)$^{-1}$ based on leukemia effects, mainly acute myelogenous leukemia, by extrapolation of low dose linearity utilizing maximum likelihood estimates. The corresponding Risk-Specific Concentrations from these IURs are 1.3 to 4.5 µg/m^3. For this HHRA, the risk-specific concentration of 4.5 µg/m^3 was applied based on Health Canada (2021), TCEQ (2015) and US EPA (2003).

Agency for Toxic Substances and Disease Registry

ATSDR has derived an acute-duration inhalation minimum risk level ("MRL") of 0.009 ppm (9 ppb) for benzene based on a LOAEL of 10.2 ppm for immunological effects in mice exposed for 6 hours/day for 6 consecutive days. The LOAEL of 10.2 ppm was adjusted from intermittent to continuous exposure ($LOAEL_{ADJ}$= 2.55 ppm) and converted to a human equivalent concentration ($LOAEL_{HEC}$= 2.55 ppm); an uncertainty factor of 300 (10 for use of a LOAEL, 3 for extrapolation from animals to humans using dosimetric conversion, and 10 to protect sensitive individuals) was applied.

ATSDR has derived an intermediate-duration inhalation MRL of 0.006 ppm (6 ppb) for benzene based on a LOAEL of 10 ppm for significantly delayed splenic lymphocyte reaction to foreign antigens evaluated in in-vitro mixed lymphocyte reaction following the exposure of male C57Bl/6 mice to benzene vapours for 6 hours/day, 5 days/week for 20 exposure days. The concentration was adjusted from intermittent to continuous exposure ($LOAEL_{ADJ}$= 1.8 ppm) and converted to a human equivalent concentration ($LOAEL_{HEC}$= 1.8 ppm); an uncertainty factor of 300 (10 for the use of LOAEL, 3 for extrapolation from animals to humans using dosimetric conversion, and 10 for human variability) was applied.

ATSDR has derived a chronic-duration inhalation MRL of 0.003 ppm (3 ppb) for benzene based on the results of BMD modelling of B cell counts in workers of shoe manufacturing industries in Tianjin, China. The resulting value was adjusted from intermittent to continuous exposure by applying an uncertainty factor of 10 (to protect sensitive individuals).

California Office of Environmental Health Hazard Assessment

The Cal OEHHA is required to develop guidelines for conducting health risk assessments under the Air Toxics Hot Spots Program. In 2014, Cal OEHHA derived a 1-hour inhalation Reference Exposure Level ("REL") of 27 µg/m^3 based on effects to the reproductive/development system and aplastic anemia and acute myelogenous leukemia. The critical effects were developmental hematotoxicity in fetal and neonatal mice.

The chronic REL is 3 µg/m^3 based on critical effects of decreased peripheral blood cells in Chinese workers affecting hematologic system. The target endpoint following chronic benzene exposure is the hematopoietic (blood) system. Neurological effects are also of concern at slightly higher concentrations. Impairment of immune function and/or various types of anemia may result from the hematotoxicity. Repeated benzene exposures can also lead to life-threatening aplastic anemia. These lesions may lead to the development of leukemia years later, after apparent recovery from the hematologic damage.

Health Canada

Health Canada has not established an inhalation RfC; however, they provide an IUR of 1.6E-02 (mg/m^3)$^{-1}$ which corresponds to an excess lifetime risk of 1-in-100,000 and 0.6 µg/m^3 concentration in air. The IUR to protect the general population against leukemia was derived based on chronic inhalation occupational exposures from two studies: Ohio Pliofilm Cohort (0.044 (ppm)$^{-1}$ or 0.014 (mg/m^3)$^{-1}$) and Chinese Cohorts (0.056 (ppm)$^{-1}$ or 0.018 (mg/m^3)$^{-1}$).

For the recommended IUR, Health Canada cites two references: Guidance for Benzene in Residential Indoor Air (Health Canada, 2013) and Public Health Goal for Benzene in Drinking Water (OEHHA, 2014). Based on these documents, the risk-specific concentrations associated with a 1 x 10^{-6} (or one-in-one million) risk of leukemia range from 0.06 µg/m^3 (OEHHA, 2014) to 0.45 µg/m^3. For 1 in 100,000 risk, which is the target for BC, the risk-specific concentrations range from 0.6 µg/m^3 to 4.5 µg/m^3.

Texas Commission on Environmental Quality

Epidemiological studies following short-term (i.e., acute, subacute) inhalation exposures to benzene demonstrated limited hematologic effects as per the review conducted by TCEQ. The Midzenski *et al.* (1992) study cited in the TCEQ benzene profile reported leukopenia, anemia, thrombocytopenia, and increased mean corpuscular volume in 15 male workers following subacute occupational exposure (mean of 5 days) at a LOAEL of 60 ppm. Dizziness and nausea were also reported in workers with more than 2 days of exposure. However, review of the study indicates that the reported sampling results (after exposure had ended) were "greater than 60 ppm" to 653 ppm (and could have been even higher due to sampling breakthrough), which does not allow for identification of a reliable LOAEL. Additionally, the study did not identify a NOAEL. The inability to identify a reliable LOAEL (or NOAEL) from the Midzenski *et al.* study (1992) precludes its use in the calculation of an acute Reference Value ("ReV") and acute Effects Screening Level ("ESL").

The chronic REL of 4.5 µg/m^3 (1.4 ppb) is based on a cancer endpoint of acute myelogenous and acute monocytic leukemia in occupationally exposed workers. Epidemiologic and case studies provide clear and consistent evidence of a causal association between benzene exposure and acute myelogenous (nonlymphocytic) leukemia, the dominant leukemia type observed among benzene-exposed workers in the studies reviewed. To a lesser extent, benzene exposure may be associated with chronic myelogenous (nonlymphocytic) leukemia and chronic lymphocytic leukemia, but studies have not yielded consistent results.

World Health Organization

The World Health Organization (WHO) current guidance relies on 1994 risk calculations that have not been updated (at the time of writing). The geometric mean of the range of estimates of the excess lifetime risk of leukaemia at an air concentration of 1 µg/m^3 is 6 x 10^{-6}. The concentrations of airborne benzene associated with an excess lifetime risk of 1-in-10 000, 1-in-100 000 and 1-in-1 000 000 are 17, 1.7 and 0.17 µg/m^3, respectively (WHO 2017).

5.1.5 1,3-BUTADIENE

Jurisdictional acute and chronic exposure limits for 1,3-butadiene are provided in **Table 5-9** and **Table 5-10**, respectively. There are no available jurisdictional limits from BC MoECCS, AENV, ATSDR, CCME, Health Canada or WHO. Jurisdictions with established values were reviewed and studies supporting these exposure limits are described in detail below.

Table 5-9 Acute Inhalation Exposure Limits for 1,3-Butadiene

Regulatory Agency	Type	Value (ppb)	Value (µg/m^3)	Reference
BC MoECCS	1-hr AAQO	-	-	BC MoECCS 2020
	8-hr AAQO	-	-	
AENV	1-hr AAQO	-	-	AENV AAQO 2019
	8-hr AAQO	-	-	
ATSDR	Acute MRL	-	-	ATSDR 2012
	Intermediate MRL	-	-	
CCME	1-hr CAAQS	-	-	CCME 2017
ON MECP	1-hr AAQC	-	-	MECP 2020; converted from the annual AAQC to allow assessment of 24-hour air quality data
	8-hr AAQC	-	-	
	24-hr AAQC	-	10	
US EPA	1-hr Standard	-	-	US EPA NAAQS 2021
	8-hr Standard	-	-	
Cal OEHHA	Acute REL	297	660	Cal OEHHA 2014
	8-hr REL	4	9	
WHO	1-hr AQG	-	-	WHO 2000
	8-hr AQG	-	-	
Environment/Health Canada	IARL-indoor air reference levels	-	1.7	Environment Canada/Health Canada 2000 Health Canada 2021

Regulatory Agency	Type	Value (ppb)	Value (µg/m^3)	Reference
	Inhalation Tolerable Concentration	-	-	

Notes:
AAQO - Ambient Air Quality Objective; AAQC – Ambient Air Quality Criteria; AQG - Air Quality Guideline; CAAQS – Canadian Ambient Air Quality Standard; MRL – Minimal Risk Level; REL – Reference Exposure Level

AENV – Alberta Environment, BC ENV – British Columbia Ministry of Environment and Climate Change Strategy; ATSDR-Agency for Toxic Substances and Disease Registry, Cal OEHHA - California Office of Environmental Health Hazard Assessment; CCME – Canadian Council of Ministers of Environment; ON MECP – Ontario Ministry of Environment, Conservation and Parks; US EPA – United States Environmental Protection Agency; WHO – World Health Organization

Table 5-10 Chronic Inhalation Exposure Limits for 1,3-Butadiene

Regulatory Agency	Type	Value (ppb)	Value (µg/m^3)	Reference
ATSDR	Chronic MRL	-	-	ATSDR 2012
BC MoECCS	Annual AAQO	-	-	BC MoECCS 2020
AENV	Annual AAQO	-	-	AENV 2019
CCME	Annual CAAQS	-	-	CCME 2017
Health Canada	TRV/Inhalation unit risk	-	-	Health Canada 2021
Environment/Health Canada	IARL-indoor air reference levels	0.8	1.7	Environment Canada/Health Canada 2000
ON MECP	Annual AAQC	1	2	MECP 2020
Cal OEHHA	Chronic REL	1	2	Cal OEHHA 2014; based on ovarian atrophy
US EPA	Reference Concentration	0.9	2	US EPA 2002; based on reproductive system – ovarian atrophy
	Risk Specific Concentrations	0.13	0.3	US EPA IRIS 2002; based on inhalation IUR of 3E-05 (µg/m^3)$^{-1}$
WHO	-	-	-	WHO 2005

Notes:
AAQO - Ambient Air Quality Objective; AAQC – Ambient Air Quality Criteria; AQG - Air Quality Guideline; CAAQS – Canadian Ambient Air Quality Standard; REL – Reference Exposure Level

AENV – Alberta Environment; ATSDR – Agency of Toxic Substances and Disease Registry; BC ENV – British Columbia Ministry of Environment and Climate Change Strategy; OEHHA - California Office of Environmental Health Hazard Assessment; CCME – Canadian Council of Ministers of Environment; ON MECP – Ontario Ministry of Environment, Conservation and Parks; US EPA – United States Environmental Protection Agency; WHO – World Health Organization

Environment Canada/Health Canada

Many VOCs are present in the indoor air of Canadian homes, some of which may pose a risk to human health at certain exposure concentrations. Health Canada has developed exposure limits for select VOCs which were prioritized for full assessment because they are commonly found in Canadian homes and have the potential to cause adverse health effects. To assist public health professionals, including those involved in standard development processes, who may need to assess the possible risk from exposure to other VOCs potentially found in indoor air, Health Canada has developed screening values called Indoor Air Reference Levels ("IARLs"). The IARLs are intended to supplement Health Canada's Residential Indoor Air Quality Guidelines. The IARLs represent concentrations that are associated with acceptable levels of risk after long-term exposure (over several months or years) for each specific VOC, as determined by the organization that performed the risk assessment.

The listed critical effects are the health endpoints used as the basis of the IARL, which is typically the most sensitive effect (i.e., occurs at the lowest exposure concentrations) that is considered relevant to humans. The IARL for 1,3-

butadiene is 1.7 µg/m^3 based on a publication by Environment Canada and Health Canada (2000) where the critical health endpoint is leukemia.

Ontario Ministry of Environment, Conservation and Parks

The ON MECP provides a 24-hour AAQC of 10 µg/m^3 and an annual AAQC of 2 µg/m^3. The MECP completed a jurisdictional review of available air standards for 1,3-butadiene from Health Canada and Environment Canada, the Province of Quebec, US EPA, California EPA, Louisiana, Massachusetts, and other agencies (MECP, 2011). MECP considered the IUR from TCEQ as the most appropriate unit risk factor given its derivation is based on an extensive risk analysis that included a detailed peer review process. Based on the IUR from TCEQ, a cancer risk-specific concentration of 2 µg/m^3, corresponding to an excess lifetime cancer risk level of 1-in-a million is used to derive the MECP AAQC. The risk-specific concentration of 2 µg/m^3 is adjusted by 10-fold to 20 µg/m^3 to correspond with a 1-in-100,000 target risk level.

The ON MECP derived the 24-hour AAQC by converting the annual AAQC to allow assessment of 24-hour air quality. A conversion factor of 5 was applied to derive the 24-hour AAQC. Based on this, the adjusted 24-hour AAQC is 100 µg/m^3 (i.e., 20 µg/m^3 * 5 = 100 µg/m^3).

United States Environmental Protection Agency

A NAAQS for 1,3-butadiene has not been derived by US EPA. An inhalation Reference Concentration of 2 µg/m^3 (0.9 ppb) has been developed by US EPA based on a 2-year mouse inhalation study with ovarian atrophy as a critical health effect (US EPA 2002). Exposure concentrations were adjusted to 24-hour continuous daily exposure; that is, exposure concentration × [6/24] × [5/7]). 1 ppm = 2.25 mg/m^3.

A variety of reproductive and developmental effects have been observed in mice exposed to 1,3-butadiene following inhalation. There are no human data on reproductive or developmental effects. Few adverse non-cancer health effects, other than reproductive and developmental effects, have been observed, except for hematological effects in mice exposed to higher concentrations.

The most sensitive short-term developmental endpoint is decreased fetal weight in the mouse. Decreases in fetal weight were observed at the lowest exposure concentration (40 ppm, 6 hours/day, gestation days 6-15); thus, a NOAEL was not established for this health endpoint. No developmental toxicity was observed in rats.

From chronic exposure studies (2-year bioassays), the most sensitive reproductive effects were ovarian atrophy in female mice and testicular atrophy in male mice. Testicular atrophy was observed following high exposures whereas, ovarian atrophy, was observed at the lowest exposure level (6.25 ppm, 6 hours/day, 5 days/week, for 2 years). Uterine atrophy was also observed in the highest exposure groups; however, this is likely to be a secondary effect of the ovarian atrophy. The mechanisms of ovarian atrophy are unknown, although there is strong evidence that the effect is mediated by the diepoxide metabolite.

There is "sufficient evidence" from epidemiologic studies of exposed workers to consider 1,3-butadiene carcinogenic to humans. Excesses of lymphohematopoietic cancers have been observed in 1,3-butadiene polymer production workers and monomer production workers in North America. A significant excess of leukemias was observed in polymer production workers, and significant excesses of non-Hodgkin's lymphomas (previously diagnosed as lymphosarcoma and reticular sarcoma, but now included in non-Hodgkin's lymphomas per the classification in the International Classification of Diseases of Oncology) have been observed in monomer workers. In summary, the findings of excess lymphohematopoietic cancers in polymer and monomer production workers are consistent with a causal association with exposure to 1,3-butadiene.

Risk-specific concentrations of 3 µg/m^3, 0.3 µg/m^3 and 0.03 µg/m^3 have been derived based on target risk levels of 1-in-10,000, 1-in-100,000 and 1-in-1,000,000, respectively.

California Office of Environmental Health Hazard Assessment

The Cal OEHHA (OEHHA, 2008) derived a 1-hour REL of 660 µg/m^3 (297 ppb) based upon developmental effects of lowered male fetal weight. An 8-hour inhalation REL of 9 µg/m^3 (4 ppb) was derived based on female reproductive effects of increased incidence of ovarian atrophy in mice. A chronic REL of 2 µg/m^3 (1 ppb) was also derived based on increased incidence of ovarian atrophy.

5.2 TOXICOLOGICAL REVIEW OF IDENTIFIED COPCS

A complete toxicology review of associated health effects following inhalation exposures to the identified COPCs was also performed. The health outcomes related to inhalation exposures to identified COPCs following short- and long-term exposures and the available human (or epidemiological) toxicological data were summarized in the sections below.

5.2.1 SULPHUR DIOXIDE

SO_2 belongs to a group of sulphur-containing gases called sulphur oxides ("SO_x"). It is emitted primarily during the burning of fossil fuels or sulphur-containing raw materials, or when these products are used in industrial processes such as metal ore smelting or electric power generation (CCME 2021).

SO_2contributes to the formation of $PM_{2.5}$ and smog, and when combined with water molecules in the atmosphere it can form sulfuric acid (CCME 2021).

In general, exposure to SO_2 can result in adverse effects on respiratory health, including reduced lung function, increased respiratory symptoms, and airway inflammation. Sensitive populations including individuals with asthma, children, and those with pre-existing respiratory diseases, are considered particularly vulnerable to exposure to SO_2 (CCME 2021).

5.2.1.1 *SHORT-TERM HEALTH EFFECTS*

Health Canada (2016) examined health-related literature on SO_2 from epidemiological, controlled exposure, toxicological and *in vitro* studies between the years 2007 and 2011. Health Canada (2016) also used the 2008 US EPA *Integrated Science Assessment of Oxides of Sulphur Oxides – Health Criteria* as a starting point for summarizing previous epidemiological data.

DATA GATHERED BY US EPA (HEALTH CANADA, 2016)

US EPA mentioned that several studies have consistently demonstrated that there is a causal relationship between respiratory morbidity and short-term exposure to SO_2. The strongest evidence for this relationship came from controlled human exposure studies involving asthmatics exercising at mild to moderate intensity, which identified respiratory symptoms and decreased lung function (e.g., wheeze and chest tightness) following exposure to 0.4-0.6 ppm of SO_2. It was also identified that for asthmatics who were otherwise healthy, both the magnitude of decrements in lung function and the percentage of individuals affected were directly correlated to increased SO_2 exposure at concentrations between 0.2 and 1.0 ppm.

Decrements in lung function [measured as increased specific airway resistance ("sR_{aw}")], decreased forced expiratory volume in 1 second ("FEV_1"), and peak expiratory flow rate ("PEFR") have been associated with increases in respiratory symptoms among asthmatics. In one key study, as discussed by US EPA (2008), asthmatic subjects were exposed to different dose groups of SO_2 for 10 minutes under exercising conditions. The authors reported that compared to clean air, the concentration of SO_2 required to produce a doubling of sR_{aw} was <0.5 ppm. Approximately 35% of the asthmatic subjects experienced a doubling of sR_{aw} at SO_2 concentrations ≤0.6 ppm.

In asthmatic children, the Childhood Asthma Management Program study identified an association between an increased risk of asthma symptoms and SO_2 concentrations ranging from 2.2 to 7.4 ppb. In a Harvard Six Cities study, exposure to SO_2 concentrations of 4.1 ppb were associated with cough incidence and lower respiratory tract symptoms.

Numerous single-city and multi-city epidemiological studies have demonstrated an association between short-term (≥1 h, generally 24-hour average) SO_2 exposure and adverse respiratory health effects in children. Moreover, the development of lung inflammation and airway hyperresponsiveness ("AHR") following exposure to SO_2 concentrations as low as 0.1 ppm have been identified in animal toxicological studies, which supports the positive associations in the epidemiological studies.

Several studies reported by the US EPA also provided evidence to support an association between emergency department ("ED") visits and hospitalizations for asthma with exposure to ambient SO_2 levels.

DATA GATHERED FROM 2007 TO 2011 STUDIES (HEALTH CANADA, 2016)

In one review examining controlled human exposure studies involving asthmatic volunteers, asthmatics were reported to experience bronchoconstriction and respiratory effects following short-term exposure to SO_2 at concentrations as low as 0.4 ppm, whereas healthy subjects showed respiratory effects at concentrations as low as 1 ppm.

Several more recent panel studies examined the relationship between short-term SO_2 exposure and respiratory endpoints of PEFR and FEV_1 for asthmatic individuals. In summary, inconsistent results were identified for SO_2-related changes in PEFR and asthma symptoms, and no association with changes in FEV_1 were reported.

Panel studies examining the effects of short-term exposure to SO_2 in children with respiratory diseases identified an association between SO_2 exposure and decreased PEFR and FEV_1. In one particular Canadian study involving asthmatic children, authors reported a significant association between decreased FEV_1 during the daytime and daytime SO_2 concentrations, with a mean 24-hour SO_2 concentration of 6 ppb. Although co-pollutants such as $PM_{2.5}$, ozone ("O_3"), and NO_2 were present, adverse effects associated with SO_2 exposures were weakly correlated with all co-pollutants examined.

Studies examining the relationship between hospital visits due to asthma or acute respiratory symptoms for adults and short-term exposure to SO_2 identified inconsistent results, including positive, negative, and no associations between SO_2 levels and hospital visits. For instance, a seven-city time-series study investigated the relationship between ED visits for asthma and air pollution; it was reported that percentages of ED visits for asthma were negatively related to increases to SO_2 concentration of 5.1 ppb. Moreover, another case study looking at ED visits between 1992 and 2002 in Edmonton, Alberta, did not identify an association between ED visits and ambient SO_2 levels; whereas positive associations were observed for other pollutants such as NO_2 and carbon monoxide ("CO"). Conversely, other studies have demonstrated a positive association between ED visits and SO_2 exposure, including a study in Italy which identified a positive relationship between ED visits for respiratory causes and SO_2 exposure in all seasons, particularly warm seasons, with a reported mean 24-hour average SO_2 concentration of 1.3 ppb.

Similarly, studies examining the relationship between hospital visits due to asthma for children and short-term exposure to SO_2 identified inconsistent results; both positive associations and no associations were reported. For instance, a case study in Sydney, Australia reported an increase in ED visits for asthmatic children for each interquartile range ("IQR") increase of 0.8 ppb of SO_2. Whereas an Italian study examining the relationship between air pollution and ED visits among asthmatic children did not identify significant associations between ED visits for respiratory disorders and daily levels of SO_2 (mean 24-hour average SO_2 concentration of 3.55 ppb).

5.2.1.2 *LONG-TERM HEALTH EFFECTS*

DATA GATHERED BY US EPA (HEALTH CANADA, 2016)

It was reported by the US EPA that the epidemiological studies examined did not provide sufficient evidence to infer a causal relationship between long-term exposure to SO_2 and respiratory effects. Although some studies reported positive associations in children, outcomes among the different studies were varied and inconsistent, making it difficult to conclusively determine the effects of long-term exposure of SO_2 on respiratory symptoms.

The few animal toxicology studies reviewed by the US EPA reported no effects on physiological lung function at SO_2 concentrations $\leq$ 5 ppm in rabbits and dogs. However, one study found decreased residual volume at 1 ppm of SO_2 in rats. Additionally, despite one animal study reporting mild bronchiolar epithelial hyperplasia in rats exposed for 4 months to 1 ppm of SO_2, the changes were no longer observed following 8 months of exposure.

DATA GATHERED FROM 2007 TO 2011 STUDIES (HEALTH CANADA, 2016)

Epidemiological studies evaluating effects of long-term SO_2 exposure in adults identified inconsistent results for changes to FEV_1, and no significant changes to forced vital capacity ("FVC"), pneumonia hospitalizations or gas transfer coefficient ("KCO"). For instance, in a British study examining respiratory effects to chronic SO_2 exposure, results indicated that a 3.82 ppb increase in SO_2 exposure was associated with lower FEV_1. Conversely, a study in Japan involving residents who were subject to SO_2 concentrations of 0.019, 0.026, 0.032, and 0.045 ppm, showed no significant differences among any of the subjects in terms of lung function tests including FEV_1 and FVC

measurements. Additionally, a Canadian case-control study involving pneumonia patients and controls aged 65 and older, determined that exposures to ambient SO_2 (annual mean concentration of 4.7 ppb or 5.8 ppb, depending on the method of calculation), were not associated with hospitalization for community-acquired pneumonia.

Similarly, epidemiological studies evaluating effects of long-term SO_2 exposure in children also identified inconsistent results, with positive or no associations identified. Although some studies were able to identify a correlation between SO_2 exposure (annual 24-hour averages between 3.90 and 6.3 ppb) and respiratory effects such as hay fever and symptoms of asthma, SO_2 exposure often showed very high correlation with other co-pollutants such as CO and NO_2; therefore, it was difficult to identify the independent respiratory effects of SO_2 from long-term exposure.

5.2.1.3 *CARCINOGENIC EFFECTS*

Numerous human studies examining the carcinogenic effects of SO_2 exposure identified inconsistent results. In one study involving a large cohort of Dutch people from 1986 to 1997 that were exposed to a mean SO_2 concentration in ambient air measured at 5.23 ppb, no increase of lung cancer was identified (Health Canada, 2016). Conversely, some Asian studies identified a significant association between SO_2 exposure and lung cancer mortality. For instance, in one multi-city study involving a large cohort of people aged 40 or older in China who were exposed to a reported average concentration of SO_2 of 27.86 ppb between 1991 and 2000, a 4.2% increase in lung cancer mortality was associated with a 10 $\mu g/m^3$ increase in SO_2 concentration (Health Canada, 2016). Even after considering the effects of co-pollutants such as NO_x, the association between SO_2 exposure and lung cancer mortality did not change.

The US EPA also reviewed numerous studies examining the genotoxic effects of SO_2 and reported that SO_2 was not found to be mutagenic or have potential to damage genetic material (i.e., DNA) *in vitro* (Health Canada, 2016). Additionally, studies in rats exposed to SO_2 concentrations between 0 and 30 ppm via inhalation for up to 20 months did not show evidence of carcinogenic potential (Health Canada, 2016).

According to the International Agency for Research on Cancer ("IARC"), SO_2 has not been classified as to its carcinogenicity (i.e., Group 3) as there is inadequate evidence to support the carcinogenicity of SO_2 in humans (IARC, 1992).

5.2.2 *NITROGEN DIOXIDE*

Oxides of nitrogen (NO_x) include NO and NO_2 and are produced from nitrogen and oxygen during fuel combustion; as such, ambient NO_2 comes primarily from the burning of fossil and biomass fuels. Of the NO_x species, NO_2 is the primary driver of health effects, and exposure to NO_2 can cause pulmonary irritation and contributes to respiratory health effects. Vulnerable individuals with heightened sensitivity to NO_2 include children, older adults, people with asthma and COPD, and those engaged in vigorous physical activity or who spend substantial amounts of time near major roadways (BC MoECCS 2021).

NO_2 in ambient air is chemically reactive and can react with volatile organic compounds to form ground level ozone. NO_2 also combines with water vapour to form nitric acid ("HNO_3"), that can subsequently react with ammonia and other organic chemicals to produce secondary particles such as ammonium nitrate. Ammonium nitrate can contribute to the harmful effects of particulate pollution and reduce visibility. NO_2 can also react with hydrocarbons in the atmosphere to produce ozone and other photochemical by-products.

5.2.2.1 *SHORT-TERM HEALTH EFFECTS*

In support of CAAQS development, Health Canada conducted a comprehensive HHRA based on most recent and relevant health studies to investigate the impacts of ambient NO_2 on the vulnerable population. Health Canada (2016) reviewed epidemiological studies of health effects associated with short-term exposure to ambient NO_2 with a focus on relevant studies from Canada and United States. Health Canada (2016) uses the 2008 US EPA Integrated Science Assessment of Oxides of Nitrogen – Health Criteria (US EPA ISA, 2008) as a starting point for summarizing previous epidemiological data.

Health Canada (2016) reports the effect of estimates for health outcomes as a percentage change in the outcome relative to a baseline mortality or morbidity rate, based on an incremental change in exposure. To enhance

comparability of the risk estimates between studies, these relative risks need to be presented by a uniform increment of exposure. Health Canada (2016) compared risks associated with short-term indices from many studies using a standard exposure increment of 30 ppb for 1-hour maximum NO_2 and 20 ppb for 24-hour average NO_2. However, different NO_2 exposure indices with different averaging times have been used in the existing epidemiological literature. Since concentrations are lower and less variable for longer averaging times, risks of health outcomes for a given concentration range are not directly comparable across exposure metrics, which complicates the determination of a standard increment.

In short-term epidemiological studies of asthmatics (including controlled, single-city and multi-city exposure studies), exposure to near-ambient levels of NO_2 elicited a range of adverse respiratory effects, including decreased lung function, increased AHR, and airway inflammation. Respiratory endpoints typically include asthma, bronchitis and emphysema (collectively referred to as COPD), upper and lower respiratory infections and other minor categories. Consistent associations were observed for children and older adults ≥65 years of age, with an IQR of 1 to 13% risk per 20 ppb increment in 24-hour average NO_2 or 30 ppb increase in 1-hour max NO_2. Risk estimates were often greater for those studies that considered combined exposures over several days, though the magnitude was also quite variable between studies.

Health Canada (2016) reported positive associations between ambient NO_2 and hospital admissions ("HAs") and emergency room visits ("ERVs") for above mentioned respiratory endpoints combined, for participants of all ages based on US EPA ISA (2008). Findings were generally very similar in studies of different designs, including time-series, case crossover, and multi-city studies. In two-pollutant models, the associations of HAs/ERVs with NO_2 were generally not very sensitive to adjustment for PM or other gaseous pollutants. With respect to HAs and ERVs, the 2008 US EPA ISA considered that there was suggestive evidence of an association between these outcomes and ambient NO_2 levels. Risk estimates were most often positive, and they were generally greater for children than for adults and older adults (≥65 years of age), with an IQR of 1–25% excess risk estimated per 20 ppb 24-hour average NO_2 or 30 ppb 1-hour max NO_2. Those for adults as a whole and for older adults (aged ≥65) were generally positive, but few were statistically significant. In analyses for subjects of all ages combined, associations were overwhelmingly positive, especially in relation to daily NO_2. The risk estimates with NO_2 were generally robust to adjustment for other gaseous and particulate pollutants in co-pollutant models.

As for the possible role of ambient NO_2 in HAs or ERVs for other respiratory outcomes, the 2008 US EPA ISA reported that a limited number of studies had investigated COPD, and still fewer had examined upper respiratory tract infections ("URTIs"), pneumonia, bronchitis, allergic rhinitis, and lower respiratory disease. While some of these studies reported positive and statistically significant associations, others reported null or negative associations, and based on the limited available data the US EPA concluded that it was difficult to draw conclusions with respect to the effects of NO_2 on these other respiratory conditions.

In more recent population-based studies, there continues to be evidence that ambient NO_2 is associated with increases in HAs for respiratory endpoints, primarily asthma hospitalizations and asthma ERVs. A large Canadian time-series study in 10 Canadian cities between 1993 and 2000 (Cakmak et al (2006) as cited in Health Canada, 2016) observed that all-age admissions were significantly related to ambient NO_2. The relationship between ambient NO_2 and ERVs for asthma was investigated in many studies, and findings indicated positive and significant associations were consistently observed for children's asthma ERVs and restricted to the warm season.

5.2.2.2 *LONG-TERM HEALTH EFFECTS*

While studies of the health effects of long-term exposure to air pollution are generally more complex to conduct than studies on daily variations in air pollutants, there is an increasing database that examines the consequences of long-term exposure to NO_2 and other air pollutants. Several authors used NO_2, NO_X and/or NO as markers of the traffic air pollution mixture, not specifically attributing the effects observed to NO_2 per se. The independent relation of NO_2 to mortality has not been widely characterized in these epidemiological studies, given the high collinearity among the various air pollutants, and uncertainty remains with respect to possible confounding by co-pollutants. Most studies utilized single-pollutant models. In studies that included co-pollutant analyses (with traffic indicators, PM indices) the results were somewhat inconsistent, though the effects of NO_2, which were mostly attenuated, often remained significant or at least presented some evidence of association with adverse outcomes.

The effects of long-term exposure to ambient NO_2 have been mostly examined with prospective cohort studies. There have been relatively few studies that examined the health effects of longer-term exposure to air pollutants.

Health Canada (2016) focused on studies that are particularly relevant to the risks associated with exposure to ambient NO_2 in Canada. Based on the quartiles of exposure, the effects appeared to increase at daily NO_2 levels above 21 ppb in the youngest men (aged 51–70); a linear dose–response relationship was observed for the oldest men (aged 71–90) for NO_2 daily levels between 10.6 and 32 ppb. The high correlation between NO_2 and the PM indices made the interpretation of the independent contribution of NO_2 difficult to determine. The US EPA concluded at that time that the health database was inadequate to infer the presence or absence of a causal relationship between total mortality and long-term exposure to NO_2.

Annual ambient concentrations of NO_2 (8.99–24.15 ppb) observed in the European studies reporting significant associations were relevant to those in Canada. Several cohort studies conducted in North America and in Europe showed positive associations between long-term NO_2 exposure and increased mortality due to cancer, but most of these associations were not significant. Deficits in lung function growth have been associated with long-term exposures to NO_2 in many epidemiologic studies 2008 US EPA ISA (US EPA, 2008). Overall, previous epidemiological studies indicated positive associations between long-term exposure to low NO_2 levels and both decrements in lung function measurements and partially irreversible deficits in lung function growth. It should, however, be noted that it has been difficult to distinguish the independent effects of NO_2, due to the high correlations with the other air pollutants for which similar risk estimates have been found.

Significant associations were observed between NO_2 exposure and decrements in markers at 33.9 ppb NO_2, in 48% of children. Among children with high parental stress, decrements in markers were measured at 21.8 ppb increase in residential and school NO_x, NO and NO_2. No significant associations were measured in low-stress households.

In Stockholm, Sweden, lifetime residential, day care, and school addresses were geocoded, and time-weighted average outdoor levels were calculated using emission inventories and air /m^3 dispersion models. A significant association between exposure to NO_x levels during the first year of life (23.40 ppb) and persistent wheeze was found using a small sub-cohort of the BAMSE[4] cohort study, which mainly focused on the genetic interactions between exposure to traffic-related air pollution for development of childhood allergic diseases.

Fewer studies have investigated the relationship between long-term exposure to air pollutants and asthma in adults. No significant cross-sectional associations were observed between hay fever and modelled NO_2 levels based on the highest (19.57 ppb) versus lowest quintile (<18.04 ppb) in adults aged 18–70 in the population-based study conducted in Nottingham, England. This study also found no evidence to suggest that living near traffic is a major determinant of allergic diseases in adults. No cross-sectional associations were found in adults aged 18–70 in a population-based study conducted in Nottingham between long-term exposure to NO_2 and total immunoglobulin E ("IgE"), based on the highest (>19.57 ppb) versus lowest quintile (<18.04 ppb).

NO_2 was the principal focus of a study involving 2,360 patients from a respiratory disease clinic in Toronto, Ontario. Non-significant associations were observed between long-term exposures to NO_2 and respiratory mortality, while results for lung cancer were inconclusive. Some positive associations were also reported with all cardiovascular mortality based on NO_x increases at 49.31 ppb.

A small number of studies, including a few conducted in Canada, investigated the relationship between long-term exposure to ambient NO_2 and a variety of cardiovascular outcomes. Most of these new publications studied the impact of traffic air pollutants on stroke incidence or hospitalization due to stroke. Studies in Canada, the US and Europe found positive associations of stroke with NO_2/NO_x, though these results are generally not statistically significant. Overall, the database is currently limited and provides inconsistent results on the relationship between long-term exposure to ambient NO_2 and cardiovascular morbidity. Moreover, most of these studies only reported single-pollutant models and several of these associations were more strongly related to PM air pollution.

In epidemiological studies, long-term exposure to ambient NO_2 was associated with adverse respiratory effects, especially in children, including reduced measures of lung function and reduced lung function growth. In children, several cohort studies also showed relationships between long-term exposure to NO_2 and the development of asthma and/or allergic responses. Long-term exposure to NO_2 levels appears to increase the incidence of asthma in adults as

[4] The BAMSE (Swedish abbreviation for Children, Allergy, Milieu, Stockholm, Epidemiology) study is an ongoing longitudinal, population-based prospective birth cohort including 4,089 children born between 1994 and 1996 in Stockholm, Sweden.

well. However, some uncertainty remains about the possible role of other co-occurring pollutants in the NO_2-related respiratory effects.

The epidemiological associations with respiratory health endpoints exhibit consistency, strength of association, and coherence across disciplines, as well as some indication of robustness and biological plausibility. However, considering the questions surrounding the possible role of co-pollutants, the overall evidence indicates that there is likely a causal relationship between long-term exposures to current levels of ambient NO_2/NO_x and respiratory effects related to the development of asthma or allergic-related disease.

5.2.2.3 *CARCINOGENIC EFFECTS*

The relationship between long-term exposures to NO_x/NO_2 and lung cancer has been assessed in Europe using data from major cohorts. In the Dutch cohort, in which 2,183 lung cancer cases were identified among participants, no evidence of an association was found between NO_2 and lung cancer incidence at 15.96 ppb in NO_2 concentration. Positive but non-significant associations were also observed for several other types, including buccal cavity and pharynx, oesophagus, liver, uterus, kidney, bladder, and breast cancer and non-Hodgkin's lymphoma.

A Canadian study suggested a possible association between long-term exposure to NO_2 levels and post-menopausal breast cancer incidence, while in France acute leukemia was found to be associated with traffic-NO_2 levels and other indicators of traffic. Additional studies are required, however, to confirm these observations on cancer incidence given the difficulty in disentangling any effect associated with NO_2 from those of other co-occurring pollutants.

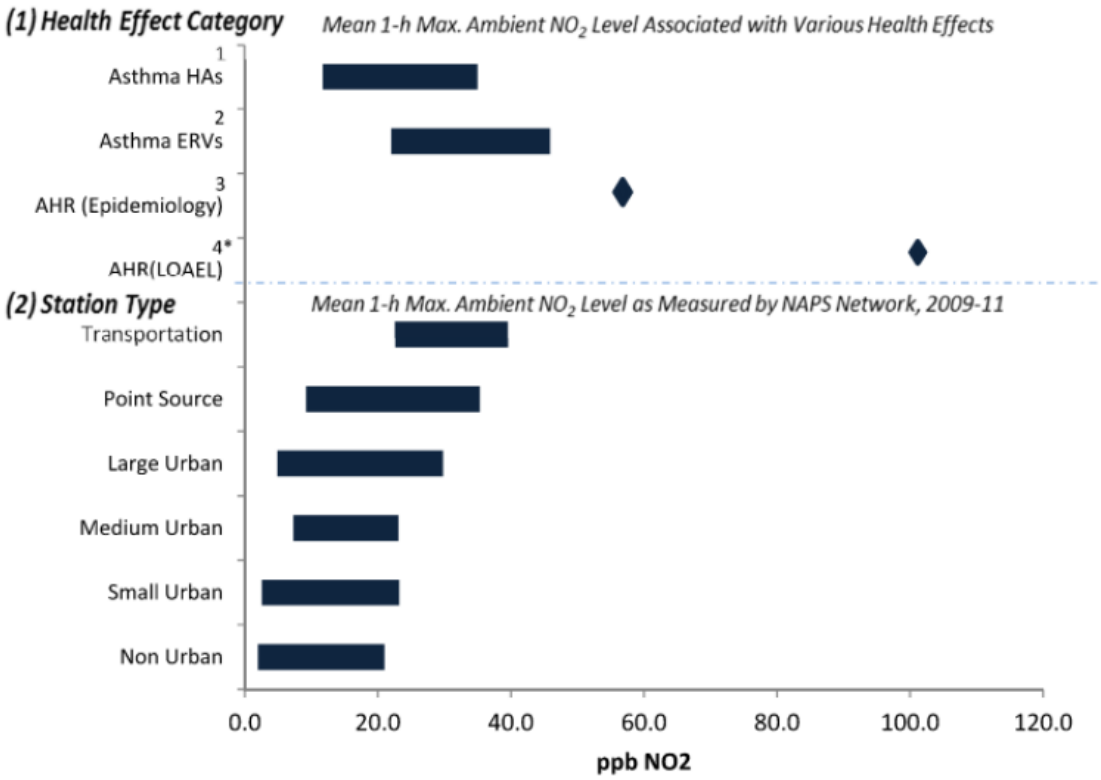
Effects of NO_2 on reproduction in humans are not known. IARC and US EPA have not classified nitrogen oxides for potential carcinogenicity. Nitrogen oxides have caused changes in the genetic material of animal cells, but it is not known if these can cause developmental effects in humans.

5.2.2.4 *COMPARISON OF AMBIENT CONCENTRATIONS IN CANADA AND KEY EPIDEMIOLOGICAL STUDIES*

Health Canada (2016) characterized health risks associated with exposure to ambient NO_2 in Canada by comparing the concentrations at which health effects are observed in key epidemiological studies with the levels measured at monitoring stations in the NAPS network across Canada. Health Canada (2016) carried out the comparison as follows:

- Focused on health endpoints for which the weight of evidence concluded "causal" or "likely to be causal" including mortality associated with short-term exposure to ambient NO_2 and respiratory disease associated with each of short-term and long-term exposure;
- Reviewed key health effect studies conducted in Canada and United States that involved primarily human epidemiological studies of ambient NO_2-related effects;
- Studies were further limited to those that reported significant association between ambient NO_2 and key health endpoint categories which provided effect estimates for NO_2 for the same metrics as are commonly used for ambient standards; that is, daily 1-hour max, 24-hour average and long-term average; and
- For those studies that reported associations for short-term exposures, studies were only included if the findings for NO_2 were robust to adjustment for other pollutants, or if exclusively single-pollutant models were run and health outcomes were significantly related to NO_2 and not to other pollutants. These latter criteria were not applied in selecting long-term studies because almost none of the long-term exposure studies adjusted for co-pollutants, given the high collinearity among the various air pollutants.

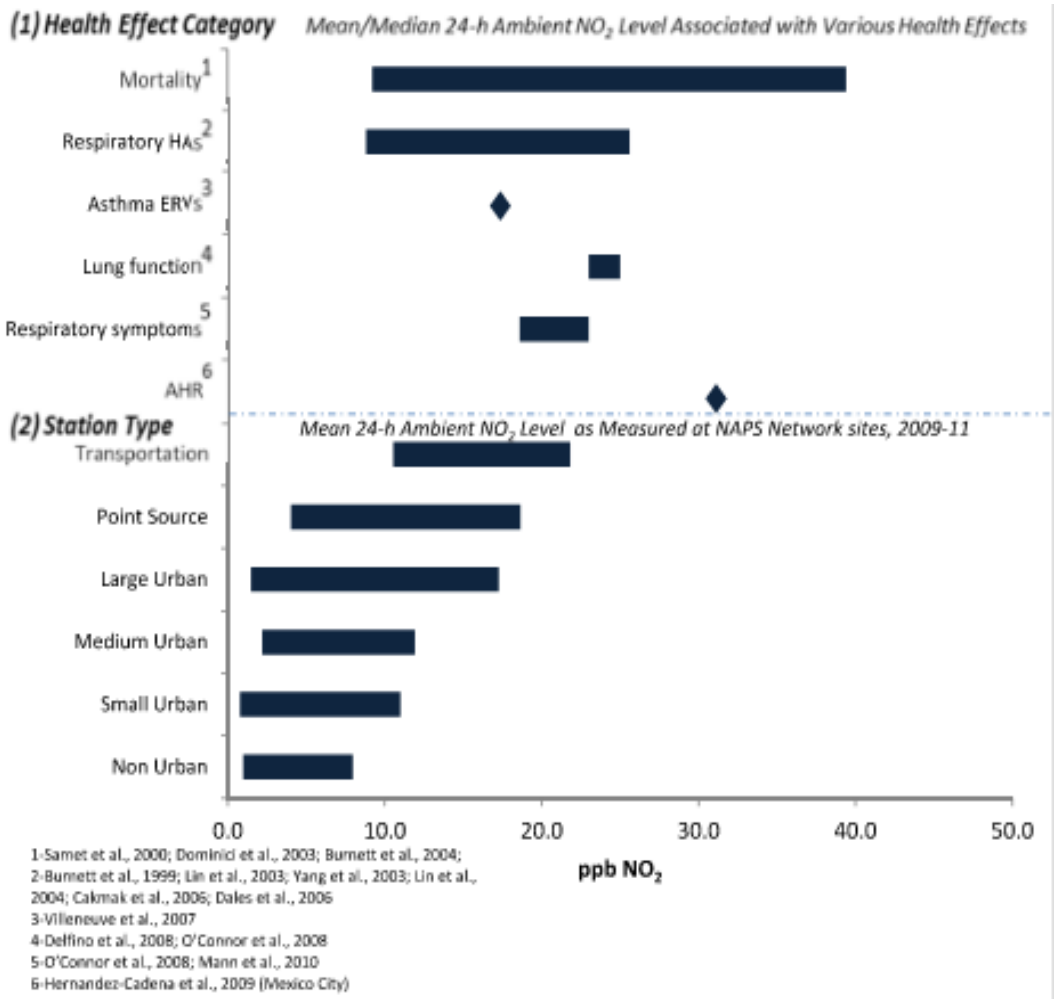
Health Canada (2016) presented the analyses in **Figure 5-1** for the daily 1-hour max NO_2, in **Figure 5-2** for the 24-hour average NO_2, and in **Figure 5-3** for NO_2 as the long-term (annual/multi-year) average. For each figure, the top panel presents the mean or median NO_2 levels associated with various categories of health effects; while the lower panel presents the mean concentrations of NO_2 measured at the NAPS stations, grouped by station type. In cases where there is more than one data point, they are presented as a bar that represents the range of mean/median concentrations, whereas if there is only a single data point, it is presented as a diamond.



1-Magas et al., 2007; Grineski et al., 2011
2-Peel et al., 2005; Strickland et al., 2010
3-Hernandez-Cadena et al., 2009 (Mexico City)
4-1-hour LOAEL in Controlled Human Exposure studies in US EPA Meta-Analysis (US EPA, 2008)

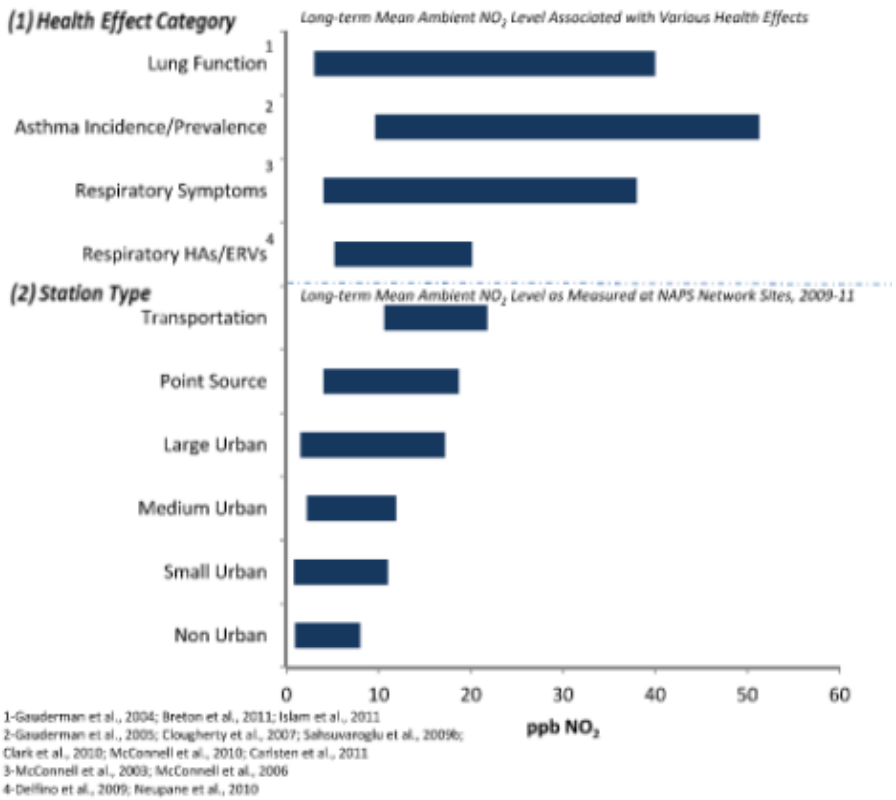
Notes:
HA – hospital admissions
ERV – emergency room visits
AHR – airway hyper-responsiveness
LOAEL – lowest observed adverse effect level

Figure 5-1 Comparison between Daily 1-h Maximum Ambient NO2 Levels (1) Associated with Various Health Effects in the Selected Canadian/US Epidemiology Studies and (2) Measured at Canadian NAPS Monitoring Stations (Figure 12.1 from Health Canada (2016))



Notes:
HA – hospital admissions
ERV – emergency room visits
AHR – airway hyper-responsiveness

Figure 5-2 Comparison between Mean 24-h Average Ambient NO_2 Levels (1) Associated with Various Health Effects in the Selected Canadian/US Epidemiology Studies and (2) Measured at Canadian NAPS Monitoring Stations (Figure 12.2 from Health Canada (2016))



Notes:
HA – hospital admissions
ERV – emergency room visits

Figure 5-3 Comparison between Mean Long Term Ambient NO_2 Levels (1) Associated with Various Health Effects in the Selected Canadian/US Epidemiology Studies and (2) Measured at Canadian NAPS Monitoring Stations (Figure 12.3 from Health Canada (2016))

5.2.3 FINE PARTICULATE MATTER (<2.5 µm)

Particulate matter is identified as all solid and liquid airborne particles (except water) that are microscopic in size. $PM_{2.5}$, also known as fine PM, is identified as those particles that are 2.5 µm or less in aerodynamic diameter. Sources of $PM_{2.5}$ primarily include fossil fuel combustion processes, industrial processes, and biomass burning. In general, exposure to $PM_{2.5}$ can lead to adverse health effects to the heart and lungs and may also lead to other health issues including asthma attacks, chronic bronchitis, and heart attacks (CCME 2021). In addition, exposure to $PM_{2.5}$ has been linked to increased ERVs and hospitalization due to respiratory and cardiovascular problems, as well as increased risk of premature mortality (CCME 2021).

Unlike SO_2 and NO_2, Health Canada has not prepared a comprehensive risk assessment report for $PM_{2.5}$. The most comprehensive assessment for $PM_{2.5}$ health science currently available is the US EPA Integrated Science Assessment (ISA) for Particulate Matter (US EPA, 2019), which builds upon a previous Integrated Science Assessment for Particulate Matter published in 2009 (US EPA, 2009). The US EPA (2019) reviewed hundreds of studies investigating a wide of potential health effects and, as shown in Table 5-X below, determined that the weight of scientific evidence supported causal links between $PM_{2.5}$ exposure and cardiovascular effects, as well as total mortality. Links between $PM_{2.5}$ exposure and respiratory effects, nervous system effects and cancer were determined “likely to be causal”.

Table 5-11 Summary of US EPA Integrated Science Assessment for Particulate Matter Causality Determinations

	Short-Term Exposure	Long-Term Exposure
Respiratory Effects	Likely to be causal	Likely to be causal
Cardiovascular Effects	Causal	Causal
Metabolic Effects	Suggestive of, but not sufficient to infer	Suggestive of, but not sufficient to infer
Nervous System Effects	Suggestive of, but not sufficient to infer	Likely to be causal
Reproductive and Developmental Effects	N/A	Suggestive of, but not sufficient to infer
Cancer	N/A	Likely to be causal
Mortality	Causal	Causal

The following sections provide further detailed discussion for each of the health effects identified in Table 5-X.

5.2.3.1 *SHORT-TERM HEALTH EFFECTS*

RESPIRATORY EFFECTS

US EPA (2019) examined possible short-term respiratory effects of $PM_{2.5}$ including exacerbation of asthma and allergy symptoms, development of COPD, and increasing incidences of respiratory-related HA and ERV visits, respiratory infection, respiratory health effects in healthy populations, respiratory effects in population with cardiovascular disease and respiratory mortality. The US EPA ISA (2019) concluded that there was a "likely to be causal relationship" between short-term $PM_{2.5}$ exposure and respiratory effects.

The collective data of animal and epidemiologic studies were evaluated for strength of causality. Overall evidence links COPD HA and ERV visits to short-term $PM_{2.5}$ exposures; however, uncertainty exists related to lack of assessment of co-pollutants and potential for confounding and comparison to previous findings showing attenuation of the $PM_{2.5}$ associations with adjustment for NO_2 (US EPA, 2019). The causal link between COPD HA and ERV visits to short-term $PM_{2.5}$ exposures is further supported by the findings of controlled human exposure and animal toxicologic studies that demonstrate increases in COPD symptoms, medication use, pulmonary inflammation, lung injury and decreases in lung function following short-term exposures to $PM_{2.5}$ (US EPA, 2019).

Regarding HA and ERV for combined respiratory-related diseases and infections, associations are seen in children, people of all ages, and older adults from single-city studies and in people of all ages in multicity studies (US EPA, 2019). Studies of respiratory mortality also report associations in single-and multicity studies, although confidence intervals are sometimes wide.

Regarding respiratory infections and short-term $PM_{2.5}$ exposures, the previous 2009 ISA reported consistent findings between $PM_{2.5}$ concentrations and HA or ERV visits for respiratory infections; however, recent studies are not consistent with the results of older studies because the respiratory infection-related outcomes examined were heterogeneous (US EPA, 2019). Many studies of respiratory infection did not examine any co-pollutants, making it unclear whether $PM_{2.5}$ associations are independent of co-pollutants (USEP 2019). Animal data demonstrate biological plausibility based on altered host defense and greater susceptibility to bacterial infection as a result of short-term $PM_{2.5}$ exposure (US EPA, 2019).

Regarding respiratory effects in healthy populations and short-term $PM_{2.5}$ exposures, epidemiologic studies reported changes in lung function and pulmonary inflammation. However, changes tend to be transient and co-pollutant confounding is inadequately examined (US EPA, 2019). Controlled human exposure and animal toxicologic studies provide evidence for lung function decrements and pulmonary effects including inflammation, injury, oxidative stress, morphologic changes, and allergic sensitization; but these effects were not observed in every study (US EPA, 2019).

CARDIOVASCULAR EFFECTS

US EPA (2019) examined possible short-term cardiovascular effects of $PM_{2.5}$ including ischemic heart disease and myocardial infarction, heart failure and impaired heart function, ventricular depolarization, repolarization and arrhythmia, cerebrovascular disease and stroke, blood pressure and hypertension, venous thromboembolism disease and pulmonary embolism, HA and ERV, cardiovascular mortality, heart rate and heart rate variability, systemic inflammation, oxidative stress, coagulation, endothelial dysfunction and arterial stiffness. The US EPA ISA (2019) concluded that there was a "causal relationship" between short-term $PM_{2.5}$ exposure and cardiovascular effects.

The collective data of animal controlled human exposure and epidemiologic panel studies were evaluated for strength of causality. Overall evidence links HA and ERV for cardiovascular-related effects, particularly, for ischemic heard disease and heart failure. These results are supported by experimental evidence from animal studies and controlled human exposure of endothelial dysfunction, impaired cardiac function, increased risk of arrhythmia, changes in heart rate variability, increases in blood pressure, systemic inflammation, oxidative stress, and coagulation (US EPA, 2019).

Evidence demonstrates a continuum of cardiovascular-related health effects following short-term exposure to $PM_{2.5}$ (US EPA, 2019). These cardiovascular-related health effects range from relatively modest increases in biomarkers related to inflammation and coagulation, to subclinical cardiovascular endpoints such as endothelial dysfunction, to HAs and ERVs for outcomes such as ischemic heart disease and heart failure (US EPA, 2019). In coherence with this continuum of effects is a body of epidemiologic studies reporting a relatively consistent relationship between short-term $PM_{2.5}$ exposure and cardiovascular-related mortality (US EPA, 2019). The current body of evidence also reduces uncertainties from the previous review related to potential co-pollutant confounding and limited biological plausibility for cardiovascular effects following short-term $PM_{2.5}$ exposure (US EPA, 2019).

METABOLIC EFFECTS

US EPA (2019) examined possible short-term metabolic effects of $PM_{2.5}$ including glucose and insulin homeostasis, inflammation, and liver function. The collective data of animal and epidemiologic studies were evaluated for strength of causality. Overall, the collective evidence is "suggestive of, but not sufficient to infer, a causal relationship between short-term $PM_{2.5}$ exposure and metabolic effects" (US EPA, 2019).

Recent studies provide some evidence supporting the effects of exposure on glucose and insulin homeostasis and other indicators of metabolic function. However, causal evidence is based on a small number of epidemiologic and toxicologic studies reporting effects on glucose and insulin homeostasis and other indicators of metabolic function such as inflammation in the visceral adipose tissue and liver (US EPA, 2019).

NERVOUS SYSTEM EFFECTS

US EPA (2019) examined possible short-term nervous system effects of $PM_{2.5}$ including effects on the autonomic nervous system, and changes in hypothalamic neurotransmitters. The collective data of animal and epidemiologic studies were evaluated for strength of causality. Overall, the collective evidence is "suggestive of, but not sufficient to infer, a causal relationship between short-term exposure to $PM_{2.5}$ and nervous system effects" (US EPA, 2019).

Animal data provides the strongest evidence that indicate an effect of short-term $PM_{2.5}$ exposure on the autonomic nervous system and changes in hypothalamic neurotransmitters. US EPA (2019) states that these studies provide evidence that $PM_{2.5}$ exposure leads to changes in norepinephrine which in turn, indicates that the hypothalamus plays an important role in mediating effects. However, human studies related to short-term $PM_{2.5}$ exposures and diseases of the nervous system remain limited (US EPA, 2019).

Regarding short-term exposure to $PM_{2.5}$ and diseases of the nervous system or depression, evidence is limited to a small number of analyses. Positive associations were not observed in studies of HAs for depression, dementia, or Alzheimer's disease (US EPA, 2019). A small increase in HAs for Parkinson's disease was reported in a large US study of Medicare recipients (age 65+) indicating that short-term exposure to $PM_{2.5}$ may exacerbate a range of symptoms experienced by Parkinson's disease patients (US EPA, 2019). A study of school children reported associations of $PM_{2.5}$ with some tests of neuropsychological function (US EPA, 2019). None of the epidemiologic studies considered confounding by co-pollutant exposures (US EPA, 2019).

MORTALITY

US EPA (2019) concluded that there was a "causal relationship" between short-term $PM_{2.5}$ exposure and non-accidental total mortality. This conclusion was supported by a large number of single and multi-city times series studies that indicate a consistent association between short term $PM_{2.5}$ exposures and total mortality. The strongest evidence is based primarily from the assessment of $PM_{2.5}$-related cardiovascular morbidity, with more limited evidence from respiratory morbidity, which collectively provides biological plausibility for mortality from short-term $PM_{2.5}$ exposures. This association has been shown to hold for a range of exposure assessment approaches, as well across both rural and urban study locations. Studies assessing the impacts of co-pollutant confounding and other sources of confounding (i.e. weather) generally indicated that association between short-term $PM_{2.5}$ exposure and short term mortality are robust and independent of confounding effects.

5.2.3.2 *LONG-TERM HEALTH EFFECTS*

RESPIRATORY EFFECTS

US EPA (2019) examined possible long-term respiratory effects of $PM_{2.5}$ including lung function and development; development of asthma, allergy, COPD and respiratory infection; severity of respiratory disease; subclinical respiratory effects in healthy population; subclinical effects in populations with cardiovascular disease; and respiratory mortality. The collective data of animal and epidemiologic studies were evaluated for strength of causality. The US EPA ISA (2019) concluded that sufficient evidence supports a "likely to be causal relationship" between long-term $PM_{2.5}$ exposure and respiratory effects.

This conclusion was based mainly on epidemiologic evidence demonstrating associations between long-term $PM_{2.5}$ exposure and changes in lung function or lung function growth rate in children with more limited evidence for asthma development and prevalence in children, childhood wheeze, and pulmonary inflammation. These associations were observed across numerous cohort studies that differed in location, exposure assessment methodology and study period. Recent studies of long term $PM_{2.5}$ exposure show pulmonary oxidative stress, inflammation, and morphologic changes in the upper (nasal) and lower airways. Other results show changes consistent with the development of allergy and asthma and impaired lung development. Biological plausibility for these observed effects was provided by long-term toxicologic studies that demonstrated impaired lung development and increased airway responsiveness in animal models. Epidemiologic studies indicated that long-term $PM_{2.5}$ exposure accelerated lung function decline, but also indicated that declining $PM_{2.5}$ concentrations over time have resulted in measurable improvements in pulmonary function growth and bronchitic symptoms in children and improvements in lung function in adults.

As with short-term respiratory effects, there was the potential for a confounding impact of co-pollutant exposure, but the US EPA ISA (2019) concluded that there was likely sufficient toxicologic evidence of $PM_{2.5}$-induced effects to support the independent effect of $PM_{2.5}$ exposure on long-term respiratory health outcomes.

CARDIOVASCULAR EFFECTS

US EPA (2019) examined possible long-term cardiovascular effects of $PM_{2.5}$ including ischemic heart disease and myocardial infarction, cerebrovascular disease and stroke, atherosclerosis, heart failure and impaired heart function, ventricular depolarization, repolarization and arrhythmia, blood pressure and hypertension, venous thromboembolism disease and pulmonary embolism, cardiovascular mortality, heart rate and heart rate variability, systemic inflammation, oxidative stress and blood lipids, coagulation, impaired vascular function and arterial stiffness.

The collective data of animal and epidemiologic studies were evaluated for strength of causality. The US EPA ISA (2019) concluded that there was a "causal relationship" between long-term $PM_{2.5}$ exposure and cardiovascular effects. This conclusion was based primarily on numerous mortality studies of U.S. and Canadian cohorts that have shown consistent strong associations between long-term $PM_{2.5}$ exposure and cardiovascular mortality, even in areas with relatively low annual mean $PM_{2.5}$ levels (4.08−17.9µg/m^3). The causal link between cardiovascular mortality and long-term $PM_{2.5}$ exposures were consistently reported in studies that differed in location, exposure assessment and statistical methodology and study period. The study findings remained relatively unchanged or increased in co-pollutant models adjusted for ozone, NO_2, $PM_{10–2.5}$, or SO_2 (US EPA, 2019). Analyses of the concentration response function relating cardiovascular mortality to long-term $PM_{2.5}$ exposure generally supported a linear, no-threshold relationship, particularly at low $PM_{2.5}$ concentrations,

Associations with coronary heart disease, stroke, and atherosclerosis progression were also observed in several additional epidemiologic studies, providing coherence with the mortality findings. Recent studies have also shown associations between long-term $PM_{2.5}$ exposure and cardiovascular morbidity, including heart failure, high blood pressure and hypertension. Biological plausibility for these observed effects was provided by long-term animal toxicologic studies that demonstrated increased atherosclerosis and coronary artery wall thickness, decreased cardiac contractility and output, and changes in blood pressure in response to long term $PM_{2.5}$ exposure (US EPA, 2019).

METABOLIC EFFECTS

US EPA (2019) examined possible long-term metabolic effects of $PM_{2.5}$ including metabolic syndrome, glucose and insulin homeostasis, Type 2 diabetes mellitus, inflammation, liver function, endocrine hormones, adiposity and

weight gain, and gestational diabetes. The collective data of animal and epidemiologic studies were evaluated for strength of causality. The US EPA ISA (2019) concluded that the collective evidence is "suggestive of, but not sufficient to infer, a causal relationship between long-term $PM_{2.5}$ exposure and metabolic effects" (US EPA, 2019).

This conclusion is based on epidemiologic studies that report positive associations between long-term $PM_{2.5}$ exposure and diabetes-related mortality in well-established cohorts in the U.S. and Canada. Although results were not consistent across cohorts, some epidemiologic studies report positive associations with incident diabetes, metabolic syndrome, and glucose and insulin homeostasis. Consideration of co-pollutant confounding was limited. Some support was provided by experimental studies demonstrating increased blood glucose, insulin resistance, and inflammation and visceral adiposity but the experimental evidence was not entirely consistent.

NERVOUS SYSTEM EFFECTS

US EPA ISA (2019) concluded that there was a "likely to be causal relationship" between long-term $PM_{2.5}$ exposure and nervous system effects. This conclusion is primarily based on toxicologic studies from multiple research groups that show inflammation, oxidative stress, morphologic changes, and neurodegeneration in multiple brain regions following long-term exposure of adult animals to $PM_{2.5}$ concentrated ambient particles (US EPA, 2019). Both experimental and epidemiologic evidence are well substantiated and coherent, supporting a pathway involving neuroinflammation in specific regions of the brain (i.e., the hippocampus, cerebral cortex and hypothalamus) and morphologic changes in the brain indicative of neurodegeneration (US EPA, 2019). In addition to the nervous system effects primarily observed in adults, there is preliminary but limited epidemiologic evidence of neurodevelopmental effects, specifically autism spectrum disorder. Evidence for this outcome is supported by an animal toxicologic study demonstrating $PM_{2.5}$-induced inflammatory and morphologic changes in regions of the brain consistent with autism spectrum disorder (US EPA, 2019). Evidence for a relationship between long-term $PM_{2.5}$ exposure and Alzheimer's disease and dementia is provided by both animal toxicologic and epidemiologic studies (US EPA, 2019). There has been limited assessment of the impact of co-pollutant exposure, but the above-noted toxicologic studies provided evidence of an independent effect of long term $PM_{2.5}$ exposure on nervous system effects (US EPA, 2019).

REPRODUCTIVE AND DEVELOPMENTAL EFFECTS

US EPA (2019) examined possible long-term reproductive and developmental effects of $PM_{2.5}$ including male and female fertility and reproduction, pregnancy and birth outcomes and developmental outcomes. The body of animal and epidemiologic studies were evaluated for strength of causality. Overall, the collective evidence is "suggestive of, but not sufficient to infer, a causal relationship between long-term $PM_{2.5}$ exposure and reproductive and developmental effects" (US EPA, 2019).

Regarding male fertility and reproduction, strongest evidence with $PM_{2.5}$ exposure come from studies on sperm motility (from human data) and spermiation (from animal data) (US EPA, 2019). However, uncertainties exist from lack of evaluation of co-pollutant confounding or multiple potential sensitive windows of exposure. Other studies on sperm including the epidemiologic literature on sperm morphology have inconsistent results. Studies of female reproduction in association with $PM_{2.5}$ exposure also have mixed results (US EPA, 2019). In rodents, ovulation and estrus are affected by $PM_{2.5}$ exposure. In the epidemiologic literature, results on human fertility and fecundity in association with $PM_{2.5}$ exposure is limited, with evidence from *in vitro* fertilization showing a modest association of $PM_{2.5}$ concentrations with decreased odds of becoming pregnant. Animal toxicologic studies show inconsistent results from $PM_{2.5}$ exposure and its effects on reproduction. Biological plausibility for outcomes on male and female fertility and reproduction come from laboratory animal studies that show genetic and epigenetic changes to germ cells with $PM_{2.5}$ exposure (US EPA, 2019)."

Regarding pregnancy and birth outcomes, several studies indicated an association between $PM_{2.5}$ and low birth weight and preterm birth in animal studies. The epidemiologic and toxicologic literature generally show positive associations of $PM_{2.5}$ exposure with reduced fetal growth and reduced birth weight. Most of the epidemiologic studies do not control for co-pollutant confounding and do not have a specific sensitive window of exposure, but there is biological plausibility from the animal toxicologic literature in support of these outcomes as well as support for multiple sensitive windows for $PM_{2.5}$ exposure associated outcomes. Various pregnancy-related pathologies, including gestational hypertension, pre-eclampsia, and gestational diabetes, show inconsistent results in association with $PM_{2.5}$ exposure (US EPA, 2019).

MORTALITY

US EPA (2019) examined possible long-term effects of $PM_{2.5}$ and total mortality. Available epidemiologic studies were evaluated for strength of causality. The US EPA ISA (2019) concluded that there was a "causal relationship" between long-term $PM_{2.5}$ exposure and non-accidental total mortality. This conclusion was supported by numerous epidemiologic studies mainly in North America and Europe that show association between long-term $PM_{2.5}$ exposures and total mortality, even in study areas with relatively low $PM_{2.5}$ levels (≤12 µg/m^3) (US EPA, 2019). The strongest evidence is based on the Harvard Six Cities Study and the American Cancer Study, adding mortality data due to cardiovascular disease (including ischemic heart disease) and respiratory disease (including COPD), and extending the follow-up period of the American Cancer Study to 22 years (1982−2004). U.S. and Canadian cohort studies demonstrate consistent, positive associations between long-term $PM_{2.5}$ exposure and mortality across various locations, exposure assessment and statistical methods, where mean annual average concentrations are ≤12 µg/m^3.

The association for total mortality was also supported by the associations for cause-specific mortality (i.e., cardiovascular mortality) reported above. In same way that early cohort studies indicated that increased levels of long-term $PM_{2.5}$ exposure decreased life expectancy, more recent studies have indicated the converse: over time, decreasing $PM_{2.5}$ exposure levels led to increases in life expectancy. As with short-term exposures, the association between long-term $PM_{2.5}$ exposure and mortality was robust across different exposure assessment approaches, co-pollutant models, and other confounders such as smoking and socioeconomic status, indicating an independent effect of long term $PM_{2.5}$ exposure on total mortality.

5.2.3.3 *CARCINOGENIC EFFECTS*

US EPA, 2019 concluded that there was a "likely to be causal relationship" between long-term $PM_{2.5}$ exposure and cancer. A number of epidemiologic studies indicated associations between long-term $PM_{2.5}$ exposure and lung cancer. However, studies of cancer development have often focused on exposure to whole particulate matter, rather than the $PM_{2.5}$ size fraction, or exposure to individual components of particulate such as metals. Despite this, biological plausibility for an association between long-term $PM_{2.5}$ exposure and cancer was provided by a wide range of toxicologic studies that indicated that components of $PM_{2.5}$ are mutagenic, cytogenic and can cause DNA damage and differential expression of genes potentially relevant to genotoxicity, as well as exhibiting carcinogenic potential. Assessment of pollutant confounding was limited but did indicate that multipollutant models including ozone did not change the association between long-term $PM_{2.5}$ exposure and lung cancer incidence.

Notwithstanding the conclusions of the US EPA, 2019, it is important to note that IARC have not classified the carcinogenicity of $PM_{2.5}$. The IARC determination of carcinogenicity for "outdoor air pollution" (IARC 2013) considers a range of individual gaseous and particulate pollutants including $PM_{2.5}$ but stops short of assigning carcinogenicity to individual components of the "outdoor air pollution" mixture.

5.2.4 BENZENE

Benzene is a clear, colourless, volatile, highly flammable liquid with a characteristic sweet aromatic odour. It is formed from both natural processes and human activities. Natural sources include emissions from volcanoes and forest fires. Industrial processes are the main source of benzene in the environment. Benzene is found in crude oil and is also formed in oil refineries and other petrochemical operations for use in the manufacturing of other chemical products. It is a component of gasoline (regulated in Canada to below 1% by volume on an annual basis, with an absolute ceiling of 1.5%). Small amounts of benzene are created whenever an organic (i.e., carbon-based) material is burned, e.g., gasoline or cigarettes, or during a forest fire.

Benzene is degraded rapidly in the upper atmosphere. Because of its solubility in water, a minor amount may be removed by rain to contaminate surface waters and soil. However, it is not persistent in surface water or soil, either volatilizing back to air or being degraded by bacteria. Airborne benzene exists almost exclusively in the vapour phase and is transformed primarily by reaction with hydroxyl radicals, resulting in a residence time ranging from 2 hours (at higher hydroxyl radical concentrations) to 8 days (at lower hydroxyl radical concentrations). The most significant route of exposure to human is through inhalation.

5.2.4.1 *SHORT-TERM HEALTH EFFECTS*

Brief exposure (5–10 minutes) to very high levels of benzene in air (10,000–20,000 ppm) can result in death. Lower levels (700–3,000 ppm) can cause drowsiness, dizziness, rapid heart rate, headaches, tremors, confusion, and unconsciousness. In most cases, people will stop feeling these effects when they are no longer exposed and begin to breathe fresh air.

The cause of death from acute overexposure to benzene has been reported to result from asphyxiation, respiratory arrest, Central Nervous System depression or cardiac collapse (ATSDR, 2007). Brief exposure (30 minutes) to 300 ppm (978 mg/m^3) benzene produced drowsiness, dizziness and headaches in exposed workers (ATSDR, 2007).

Occupational exposure of males to benzene air concentrations >60 ppm (196 mg/m^3) for up to 3 weeks (2.5 to 8 hours/day) during the removal of residual fuel from shipyard tanks produced respiratory effects (mucus membrane irritation and dyspnea), reduced blood cell counts (leukocytes, erythrocytes, and thrombocytes), and neurological effects (dizziness, nausea, headache, fatigue) (ATSDR, 2007).

Uncertainty in exposure levels and duration, the potential for confounding exposures to other chemicals, and lack of corresponding control groups, limit the use of data collected from an occupational setting; however, the ATSDR (2007) has identified well conducted occupational studies with effects linked to specific benzene exposure concentrations. Adverse health effects reported in human studies following the acute inhalation of benzene and the air concentration at which they are predicted to occur are summarized in the table below.

Table 5-12 Acute Effects Following Human Exposure to Benzene

Acute Effects	Exposure Period	Air Concentration ppm (mg/m^3)	Reference
Death	5 to 10 minutes	20,000 (65,200)	Flury *et al*. 1928
Neurological: drowsiness, dizziness, headaches	30 min	300 (978)	Flury *et al*. 1928
Neurological: dizziness, headaches, nausea, fatigue (males)	1-21 days, 2.5-8 hr/day	60 (196)	Midzenski *et al*. 1992
Respiratory: mucus membrane irritation and dyspnea (males). Hematological: leucopenia, anemia, and thrombocytopenia (males).	1-21 days, 2.5-8 hr/day	60 (196)	Midzenski *et al*. 1992

5.2.4.2 *LONG-TERM HEALTH EFFECTS*

The major effect of benzene from long-term exposure is on the blood. Benzene causes harmful effects on the bone marrow and can cause a decrease in red blood cells leading to anemia. It can also cause excessive bleeding and can affect the immune system, increasing the chance for infection. Reduction in other components in the blood can cause excessive bleeding. Blood production may return to normal after exposure to benzene stops. Some women who breathed high levels of benzene for many months had irregular menstrual periods and a decrease in the size of their ovaries, but it is not known for certain that benzene caused the effects. It is not known whether benzene will affect fertility in men (ATSDR, 2007).

Long-term exposure to benzene can cause cancer of the blood-forming organs. This condition is called leukemia. Exposure to benzene has been associated with development of a particular type of leukemia called acute myeloid leukemia (“AML”). Most information on effects of long-term exposure to benzene are from studies of workers employed in industries that make or use benzene. These workers were exposed to levels of benzene in air far greater than the levels normally encountered by the general population. Current levels of benzene in workplace air are much lower than in the past. Because of this reduction and the availability of protective equipment such as respirators, fewer workers have symptoms of benzene poisoning (ATSDR, 2007).

Similar to the effects reported following acute exposures, subchronic and chronic exposure to relatively low levels of benzene produced measurable depression of one or more circulating blood cells, resulting in haematotoxic and immunotoxic effects. Subchronic and chronic studies in humans and animals have reported pancytopenia or the

reduction in number of all major blood cells, including leukocytes (white blood cells), erythrocytes (red blood cells), and thrombocytes (platelets). Blood cells are produced by the bone marrow and therefore pancytopenia is a condition that results from the inability of the bone marrow to adequately produce mature blood cells. A more severe effect of benzene exposure is aplastic anaemia in which the bone marrow is unable to function and stem cells do not mature. The progression of aplastic anaemia can result in AML, or cancer of the myeloid line of white blood cells (ATSDR, 2007).

Pancytopenia was reported in workers occupationally exposed to benzene concentrations ranging from 3 to 210 ppm (10 to 685 mg/m^3) over periods of 4 months to 3 years (ATSDR, 2007). Decreased production of white blood cells (leucocytes and lymphocytes) occurred in workers occupationally exposed for 1 to 21 years to benzene concentrations ranging from 0.57 to 75 ppm (1.86 to 245 mg/m^3) (ATSDR, 2007). Decreased red blood cell counts and anaemia were reported following subchronic and chronic occupational exposure to benzene concentrations ranging from 2.26 to 29 ppm (7.37 to 95 mg/m^3) (ATSDR, 2007).

There was a lack of observed adverse effects on blood cells in male refinery workers exposed to 0.53 ppm (1.73 mg/m^3) benzene for 1 to 21 years (ATSDR, 2007). This exposure level was selected by the Cal OEHHA and adjusted for continuous exposure and variation in human sensitivity to develop a chronic REL of 0.02 ppm or 60 µg/m^3 (OEHHA, 2014).

The study reporting the lowest air concentration at which white blood cell (lymphocyte) levels were reduced was selected by the ATSDR for the development of the MRL for chronic inhalation exposure (>365 days) to benzene. Significant decreases in B-lymphocyte counts were reported for male shoe manufacturing workers in Tianjin, exposed to 0.57 ppm (1.86 mg/m^3) benzene for an average of 6.1 years (ATSDR, 2007). A chronic MRL of 0.003 ppm (0.01 mg/m^3) was determined using BMD modelling and adjusting from occupational to continuous exposure. A 10-fold uncertainty factor was also applied to account for variations in human sensitivity (ATSDR, 2007).

The US EPA developed a RfC also based on a study reporting decreased lymphocyte counts following occupational exposure to 7.6 ppm (24 mg/m^3) benzene (US EPA, 2002). The US EPA used benchmark dose modelling and adjusted for human variability, subchronic-to-chronic exposures, and database deficiencies to arrive at a RfC of 30 µg/m^3 for lifetime chronic human exposure to benzene (US EPA, 2002).

The Cal OEHHA, the ATSDR, and the US EPA have all developed chronic exposure guidelines for benzene based on effects (or lack thereof) on blood cell counts following occupational exposures.

Exposure to benzene may be harmful to the reproductive organs. Some women workers who breathed high levels of benzene for many months had irregular menstrual periods. When examined, these women showed a decrease in the size of their ovaries. However, exact exposure levels were unknown, and the studies of these women did not prove that benzene caused these effects. It is not known what effects exposure to benzene might have on the developing fetus in pregnant women or on fertility in men. Studies with pregnant animals show that breathing benzene has harmful effects on the developing fetus. These effects include low birth weight, delayed bone formation, and bone marrow damage.

Several studies linked the occupational exposure of women to benzene with reproductive effects, including menstrual disorders, reduced fertility, and increased frequency of spontaneous abortions (ATSDR, 2007). One case study reported severe pancytopenia and increased chromosomal aberrations in a woman exposed to benzene throughout her pregnancy but not in her child (ATSDR). In contrast, another study reported chromosomal effects in the lymphocytes of children born of women exposed to benzene (and other solvents) during pregnancy (ATSDR, 2007).

Several case-control studies reported significant associations between childhood leukemia and parental exposure to benzene (US EPA, 2002). Maternal exposure to benzene during pregnancy was associated with acute nonlymphocytic leukemia (“ANL”) in second or later-born (versus firstborn) children (US EPA, 2002). Maternal exposure to pesticides, petroleum products, and solvents (including benzene) during pregnancy was associated with an increased occurrence of ANL in offspring (ATSDR, 2007). Paternal exposure to benzene prior to conception was also associated with childhood leukemia (US EPA, 1998).

5.2.4.3 CARCINOGENIC EFFECTS

Both the IARC and the US EPA have determined that benzene is carcinogenic to humans. The IARC has classified benzene as a Group I human carcinogen. Based on "several studies of increased incidence of nonlymphocytic leukemia from occupational exposure, increased incidence of neoplasia in rats and mice exposed by inhalation and gavage, and some supporting data", benzene has been placed in the US EPA weight-of-evidence classification A, human carcinogen (US EPA).

Long-term exposure to high levels of benzene in the air can cause leukemia, particularly AML. This is a cancer of the blood-forming organs. Studies of controlled animal exposure to benzene have also reported leukemia as well as non-Hodgkin's lymphoma, and tumours in the lung, liver, mammary gland, and Zymbal gland (US EPA 2002).

Occupational exposure to benzene, and solvents containing benzene, has been associated with ANL as well as non-Hodgkin's lymphoma and multiple myelomas (ATSDR). Although limited by confounding exposures to other chemicals and lack of precise exposure monitoring, the available occupational studies demonstrate a consistent increase in the risk of leukemia with exposure to benzene (ATSDR).

A cohort of rubber hydrochloride manufacturing workers at three facilities in Ohio (Pliofilm workers cohort) is considered to be the most thoroughly studied occupational group with respect to the risk of developing leukemia following exposure to benzene (ATSDR). Data from this cohort have been used for the development of AAQGs for benzene by Health Canada, the US EPA, as well as the WHO, European Union, and Health Council of the Netherlands.

An IUR of 2.2 x 10-6 per µg/m^3 has been derived by US EPA based on hematologic effects of leukemia.

5.2.5 1,3-BUTADIENE

1,3-Butadiene is a product of incomplete combustion resulting from natural processes and human activity. It is a colourless gas with a mild gasoline-like odour. It is also an industrial chemical used primarily in the production of polymers, including polybutadiene, styrene-butadiene rubbers and latexes, and nitrile-butadiene rubbers. 1,3-Butadiene enters the Canadian environment from exhaust emissions from gasoline- and diesel-powered vehicles, from non-transportation fuel combustion, from biomass combustion and from industrial on-site uses.

While 1,3-butadiene is not persistent, it is ubiquitous in the urban environment because of its widespread combustion sources. Highest atmospheric concentrations have been measured in air in cities and close to an industrial source. Inhalation is the predominant route of exposure for the general and occupational populations. The general population is exposed to 1,3-butadiene primarily through ambient and indoor air. In comparison, other media, including food and drinking-water, contribute negligibly to exposure to 1,3-butadiene. Tobacco smoke may contribute significant amounts of 1,3-butadiene. Workers in the production of rubber, plastics and resins are more likely to be exposed than the general population.

1,3-Butadiene is absorbed from the lungs into the bloodstream following inhalation exposure. 1,3-Butadiene is broken down to its metabolites in the liver. About half of inhaled 1,3-butadiene is broken down and exhaled. The remaining chemical is broken down and excreted in the urine. 1,3-Butadiene metabolites in urine can be used as biomarkers of exposure. 1,3-Butadiene-derived hemoglobin adducts, which are surrogate biomarkers for 1,3-butadiene metabolites, have been shown to correlate. Average concentration in cities and suburban air is 0.04 to 1 ppb.

5.2.5.1 SHORT-TERM HEALTH EFFECTS

Occupational exposure at 2,000, 4,000 or 8,000 ppm concentrations of 1,3-butadiene is reported to cause irritation of the skin, eyes, nose, and throat. Coughing, drowsiness, and fatigue have also been reported at higher, but not specified, exposure concentrations (ATSDR, 2012). These physiological responses dissipated upon removal of the workers from the area where 1,3-butadiene had accumulated based on studies cited in a Centers for Disease Control ("CDC") 1984 publication and in ATSDR 2012. High gas concentrations may cause mild skin irritation.

In a survey of 1,3-butadiene monomer, polymer and end-user industries in the United States, the geometric mean concentration for full-shift exposure for all job categories was 0.098 ppm and the arithmetic mean was 2.12 ppm. Although data for ambient air levels in Europe are limited, reported concentrations in urban air generally ranged from less than 2 $\mu g/m^3$ to 20 $\mu g/m^3$. Mean levels in indoor air in a small number of Canadian homes and offices were 0.3 $\mu g/m^3$. Sidestream cigarette smoke contains 1,3-butadiene at approximately 0.4 mg/cigarette, and levels of butadiene in smoky indoor environments are typically 10–20 $\mu g/m^3$ (WHO, 2001).

In available surveys in Canada, 1,3-butadiene was detected up to 6 times more frequently in indoor air in homes than in corresponding samples of outdoor air, with concentrations being up to 10-fold higher indoors than outdoors (studies cited in WHO, 2001). Air concentrations in indoor environments are highly variable and depend largely on individual activities and circumstances, including the use of consumer products (e.g., cigarettes), the infiltration of vehicle exhaust from nearby traffic and possibly from attached garages, and cooking activities involving heated fats and oils. While data are inadequate to determine the relative contributions of each of these potential indoor sources, the highest concentrations of 1,3-butadiene in indoor air in Canada have generally been detected in indoor environments contaminated with tobacco smoke.

5.2.5.2 *LONG-TERM HEALTH EFFECTS*

Information on the lethality of 1,3-butadiene via inhalation in humans is limited (ATSDR, 2012). Based on studies reviewed by ATSDR (2012), it is unclear if cardiovascular disease is likely to be caused by 1,3-butadiene exposure. No studies were located regarding noncancer gastrointestinal effects, musculoskeletal effects, hepatic effects, renal effects, immunological effects, dermal effects, reproductive effects, or developmental effects in humans after inhalation exposure to 1,3-butadiene (ATSDR, 2012). Psychomotor responses of two men inhaling 2,000, 4,000, or 8,000 ppm 1,3-butadiene for 6–8 hours/day on different days were evaluated. Results after 1,3-butadiene exposure were identical to those obtained before exposure.

Based on epidemiology studies by CDC, a retrospective cohort study was conducted at two styrene-butadiene rubber (“SBR”) production facilities in the US. The combined cohorts consisted of 2,756 white males who had an average length of employment of approximately 10 years. No historical exposure data were available. Environmental sampling conducted at the time of the study characterized the most likely chemical exposures to be 1,3-butadiene, styrene, and benzene. Average exposure concentrations of 1,3-butadiene in the two facilities were 1.24 ppm (range, 0.11-4.17 ppm) and 13.5 ppm (range, 0.34-174 ppm). No statistically significant excesses in total or cause-specific mortality were observed for the total worker populations of either facility. However, a subgroup of workers from one cohort had a non-statistically significant excess mortality for cause-specific categories of the lymphatic and hematopoietic tissues.

Eight facilities that produced SBR in the US and Canada provided data for another retrospective study. The study covered a period of 36 years and included a total worker population of 13,920 black and white males. No significant excesses in cause-specific mortality were observed; however, some cancers (digestive system, kidney, lymph nodes, and larynx) occurred at a higher rate in white males compared with the general population, and the black male population had a non-statistically significant elevated risk of arteriosclerotic disease. The small number of workers in the cohorts from the 8 facilities studied and the relatively short latency periods of workers exposed inhibited the capability to identify statistically significant increases in risk of mortality or cause-specific disease. Also, environmental data were insufficient to characterize and quantify the workers’ chemical exposures.

Studies reviewed by WHO in 2000 suggested that there is no evidence for a measurable effect of 1,3-butadiene on hematological parameters at recent exposure levels in United States industry when they studied epidemiological data of a cohort mortality study of butadiene monomer workers, two cohort mortality studies of SBR workers and a lympho-haematopoietic cancer case-control study.

5.2.5.3 *CARCINOGENIC EFFECTS*

An association between exposure to 1,3-butadiene in the occupational environment and leukemia fulfils several of the traditional criteria for causality; there is also some limited evidence that 1,3-butadiene is genotoxic in exposed workers. Therefore, in view of the weight of evidence of available epidemiological and toxicological data, 1,3-butadiene is considered highly likely to be carcinogenic in humans; it is also considered likely to be genotoxic in humans (WHO, 2001).

The carcinogenicity of 1,3-butadiene has been investigated in several populations of workers occupationally exposed during its manufacture or use. Although most of these studies are limited by the paucity of historical monitoring data, there is evidence that occupational exposure to 1,3-butadiene in the SBR industry is associated with excess mortality due to leukemia and weaker evidence of an association with lymphosarcoma in 1,3-butadiene monomer production workers (Environment/Health Canada, 2000; WHO, 2001).

IARC has classified 1,3-butadiene as Group 1, carcinogenic to human, based on sufficient evidence in human for causing cancer of the haematolymphatic organs. There is strong evidence that the carcinogenicity of 1,3-butadiene in humans operates by a genotoxic mechanism that involves formation of reactive epoxides, interaction of these direct acting mutagenic epoxides with DNA, and resultant mutagenicity. The metabolic pathways for 1,3-butadiene in experimental animals have also been demonstrated in humans (IARC, 2008)

Under US EPA's 1999 Guidelines for Carcinogen Risk Assessment,1,3-butadiene is characterized as carcinogenic to humans by inhalation. This characterization is supported by the total weight of evidence provided by the following:

- Sufficient evidence from epidemiologic studies of the majority of US workers occupationally exposed to 1,3-butadiene, either to the monomer or to the polymer by inhalation, showing increased lymphohematopoietic cancers and a dose-response relationship for leukemias in polymer workers;
- Sufficient evidence in laboratory animal studies showing that 1,3-butadiene causes tumors at multiple sites in mice and rats by inhalation; and
- Numerous studies consistently demonstrating that 1,3-butadiene is metabolized into genotoxic metabolites by experimental animals and humans. The specific mechanisms of 1,3-butadiene-induced carcinogenesis are unknown, however, the scientific evidence strongly suggests that the carcinogenic effects are mediated by genotoxic metabolites of 1,3-butadiene (i.e., the monoepoxide, the diepoxide, and the epoxydiol).

5.3 SELECTED TOXICOLOGICAL REFERENCE VALUES FOR APPLICATION IN THE HHRA

Based on the review of available jurisdictional health-based standards for selected COPCs, as well as the health and exposure related data reviewed and discussed in the toxicological summary write-up, this HHRA adopted the health-based TRVs shown in **Table 5-13** below.

Table 5-13 Selected TRVs for the HHRA

COPC	Type	TRV ($\mu g/m^3$)	Source	Basis
Acute Exposure Duration				
SO_2	1-hr	106	Health Canada (2016)	**For protection against lung function decrements** The 1-hour TRV (106 $\mu g/m^3$) is based on the lowest observed adverse effect concentration ("LOAEC") of 0.4 ppm resulting in lung function decrements from controlled human exposure studies of asthmatics exposed to SO_2 for 5-10 minutes, at increased ventilation (Health Canada, 2016). Although the studies focused on asthmatics, an uncertainty factor of 6 was applied to account for further susceptibility that may exist in the general population due to genetic factors, age, or disease status, which resulted in a 10-minute reference concentration of 67 ppb. Health Canada (2016) did not derive 1-hour, 24-hour, or annual reference concentrations as the studies reviewed did not provide enough evidence to suggest causality. The 10-minute reference concentration of 67 ppb was converted to 40 ppb by the Ontario MECP (2020) to allow for the assessment of 1-hour air quality data.

COPC	Type	TRV ($\mu g/m^3$)	Source	Basis
NO_2	1-hr	113	Health Canada (2016)	**For protection of airway hyper-responsiveness (AHR)** This 1-hour TRV (113 $\mu g/m^3$) is primarily based on an exposure study involving 85 asthmatic children (aged 7-12) from Mexico City (Hernandez-Cadena et al, 2009 cited in Health Canada, 2016). In this study, exposure to ambient NO_2 was associated with reduced broncho-dilating response to inhaled corticosteroids in asthmatic children, indicating increased AHR. The study findings indicated elevated NO_2 levels were associated with a 15% decrease in lung function response to inhaled corticosteroids (as indicated by FEV_1 or forced expiratory volume in 1 second response to short-acting β agonists) per 10 ppb daily 1-hour maximum NO_2, with similar decreases in response 0 to 3 days following exposure inhaled corticosteroids.
	1-hr	79	Health Canada (2016)	**To reduce frequency of asthma emergency room visits (ERVs)** Asthma ERV is also considered as a health endpoint in this HHRA as ERVs associated with increased incidences of asthma in children or adults have been consistently associated with short-term ambient NO_2 in the studies reviewed by Health Canada (2016). However, ERVs were also related to exposures to other pollutants as few co-pollutant analyses were conducted (Health Canada, 2016).
$PM_{2.5}$	24-hr	25	WHO (2005)	**For protection against excess morbidity or mortality** This 24-hour TRV (25 $\mu g/m^3$) represents a 99th percentile of the distribution of daily values and is intended to protect against peaks of pollution that would lead to substantial excess morbidity or mortality. This value is largely based on published risk coefficients from multicentre studies and meta-analyses, which reported an average short-term mortality effect for PM_{10} of approximately 0.5% per 10 $\mu g/m^3$. This value is considered to provide a significant reduction in risks from acute exposure health effects such as short-term mortality.
Benzene	24-hr	30	US EPA (2003)	**Protection against hematopoietic effects** This TRV (30 $\mu g/m^3$) is based on benchmark dose modelling of the absolute lymphocyte count data from the occupational epidemiologic study of Rothman et al. (1996) cited in US EPA (2003), in which workers were exposed to benzene by inhalation.
1,3-Butadiene	24-hr	100	ON MECP (2011)	**For protection against irritation** The 24-hr AAQC was derived from the annual AAQC and is intended to protect against acute health effects including irritation, dryness of the eyes, nasal passages, throat and lungs as well as neurological effects including systemic effects (fatigue, lethargy). The Ontario MECP derived the 24-hour AAQC by converting the annual AAQC to allow assessment of 24-hour air quality. A conversion factor of 5 was applied to derive the 24-hour AAQC. Based on this, the adjusted 24-hour AAQC is 100 $\mu g/m^3$ (i.e., 20 $\mu g/m^3$ * 5 = 100 $\mu g/m^3$).
Chronic Exposure Duration				
SO_2	Annual	*No health-based value available*		A chronic (annual) TRV was not identified as part of this assessment, given that few jurisdictions were able to derive a value, and for the ones that did, the value was based on protection of ecological systems and vegetation, and not for human health. Therefore, SO_2 was not assessed on a chronic-health basis in the HHRA.
NO_2	Annual	23	Health Canada (2016)	**Protection of respiratory morbidity** This TRV (23 $\mu g/m^3$) is based on long-term exposure to ambient NO_2 and respiratory morbidity. Uncertainty remains with respect to possible confounding effects by co-pollutants.

COPC	Type	TRV (µg/m^3)	Source	Basis
$PM_{2.5}$	Annual	10	WHO (2005)	**Protection against excess mortality** This TRV (10 µg/m^3) represents the lower end of the range over which significant effects on survival have been observed in the ACS study.
Benzene	Annual	4.5	Health Canada (2021), TCEQ (2015) and US EPA (2003)	**Protection against leukemia, mainly acute myelogenous leukemia** This TRV (4.5 µg/m^3) was derived based on a risk specific concentration relating to a 1 in 100,000 risk of developing leukemia observed in workers exposed via inhalation.
1,3-Butadiene	Annual	20	ON MECP (2011)	**Protection against carcinogenicity** The AAQC of 2 µg/m^3 was derived by ON MECP based on carcinogenicity (1 in 1,000,000) associated with annual average exposure to this compound. The TRV (20 µg/m^3) is based on 1 in 100,000 risk.

5.4 UNCERTAINTY ANALYSIS

The major sources of uncertainty associated with the Hazard Assessment stage of the HHRA are briefly described below for each COPC:

5.4.1 SO_2

- The potential confounding health effects by co-pollutants such as $PM_{2.5}$ in epidemiology studies remains a major uncertainty in the health assessment for SO_2; for this reason, the LOAEC for lung function decrements was solely based on controlled human exposure studies.
- While Health Canada (2016) examined long-term epidemiological studies, a chronic (annual) TRV was not adopted largely due to the inconsistency across studies and inability to distinguish potential confounding by co-pollutants, as well as uncertainties regarding geographic scale of analysis.
- Health based 1-hour AAQOs are available from other jurisdictions that are higher than value adopted as part of this assessment (40 ppb); however, these exposure limits are either dated and/or documentation describing the technical basis of, or derivation of the standards, are lacking. As such, it is not possible to confirm whether exposure limits from other jurisdictions are adequately protective of human health.

5.4.2 NO_2

- While Health Canada (2016) details the health- and exposure-studies supporting the CCME 2020 and 2025 CAAQS, CCME does not provide any documentation that describes how the proposed numerical values for 2020 or 2025 CAAQS for NO_2 were derived.
- Exposure to co-pollutants remains the major uncertainty in the overall health database for air pollutants including NO_2:
  - Adjustments through statistical control can be completed to control for potential co-pollutant confounding in air pollution health effects studies. Co-pollutant regression models are the most widely used technique whereby, the NO_2 effect estimate represents the risk associated with NO_2 while keeping the level of the other co-pollutant(s) or other covariate(s) constant. There are limitations to multivariable models; in particular, high correlations between NO_2 levels and potential confounders can affect the magnitude or precision of the effect estimate for NO_2 or the covariate(s) and are a concern for models that include a traffic-related co-pollutant or that include three or more pollutants in the same model.
  - With respect to asthma and respiratory incidence in children, Health Canada (2016) states that overall findings were generally not highly sensitive to study design, but uncertainty remains about whether the effects related to NO_2 are independent of other pollutants. In a limited number of studies examining effects of NO_2 in co-pollutant models, robust associations were generally observed following adjustment for various air pollutants including particulate matter and/or ozone or sulphur dioxide. Results from these studies are coherent with associations found in children for asthma incidence and respiratory symptoms.
  - Human epidemiology studies are observational rather than experimental, and hence there can be uncertainty as to whether the effects reported in the epidemiology studies are in fact due to ambient NO_2 alone. The NO_2 may be a marker (in whole or in part) for other air pollutants, or the observed association may even be the result of some other factor (Health Canada, 2016).
  - Uncertainty associated with exposure to co-pollutants applies to HAs and ERVs as a health endpoint because it is challenging to separate the effect of each air pollutant when multiple pollutants are present.
  - This same uncertainty also applies to long-term exposure to NO_2 levels from traffic-related exposures as co-pollutant models adjusting for other key traffic-related air pollutants such as carbon monoxide or ultrafine particulates have not been performed.
- Health-based 1-hour and annual AAQOs are available from other jurisdictions that are higher than values adopted by MV, BC MoECCS and CCME; however, these exposure limits are either dated and/or

documentation describing the technical basis of or derivation of the standards are lacking. As such, it is not possible to confirm whether exposure limits from other jurisdictions are adequately protective of human health.

5.4.3 $PM_{2.5}$

- Considerable uncertainty remains as to which of the PM fractions (coarse or fine) are responsible for eliciting certain health effects. For instance, the extent to which fine PM may also contribute to the health effects observed as a result of exposure to coarse PM is an important source of uncertainty affecting the HHRA.
- Some acute- and chronic- health based standards from other jurisdictions are higher than the values adopted as part of this assessment; however, these exposure limits are either dated and/or documentation describing the technical basis or derivation of the standards are lacking. As such, it is not possible to confirm whether exposure limits from other jurisdictions are adequately protective of human health.

5.4.4 BENZENE

- It is noted that no jurisdictional limits were identified from BC MoECCS or CCME for benzene.
- Uncertainty in exposure levels and duration, as well as potential for confounding exposures to other chemicals, presents some uncertainty in the interpretation of health effects from occupational studies with benzene.

5.4.5 1,3-BUTADIENE

- No jurisdictional limits were identified from BC MoECCS, AENV, ATSDR, CCME, Health Canada, or WHO for 1,3-butadiene.
- Exposure to 1,3-butadiene via indoor sources and/or cigarette smoke remains a significant source of exposure to the chemical. Concentrations of 1,3-butadiene within indoor environments are highly variable and largely depend on individual activities and circumstances, including the use of consumer products (e.g., cigarettes), the infiltration of vehicle exhaust from nearby traffic and possibly from attached garages, and cooking activities involving heated fats and oils.
- No long-term studies were located regarding noncancer effects in humans after inhalation exposure to 1,3-butadiene.

6 RISK CHARACTERIZATION

Risk characterization is the final step in the HHRA process, during which the exposure and hazard (toxicity) assessments are integrated. The process of risk characterization conducted in this HHRA reflects the conservative approach used to generate risk estimates. The process and interpretation of these steps are discussed in the following sections. Key uncertainties that influence results, including data gaps, are also described.

6.1 QUANTIFYING HAZARDS FOR CARCINOGENIC CHEMICALS

Some chemicals are reported to have cancer-causing health effects, and generally these substances (also known as carcinogens) behave based on a non-threshold mechanism. To maintain a health-protective approach, regulatory agencies typically assume that there is no dose below which a harmful effect will not occur and any exposure to a carcinogen is associated with some level of risk. For carcinogenic chemicals, the potential for exposures to result in harmful effects is based on the Incremental Lifetime Cancer Risk ("ILCR"). The ILCR is calculated as the product of estimated exposure and Inhalation Unit Risk ("IUR").

$$ILCR = \text{Adj}EE \times IUR$$

Where:

ILCR = Incremental Lifetime Cancer Risk (Unitless)

Adj EE = Adjusted Exposure Estimate (µg/m^3)

IUR = Inhalation Unit Risk (µg/m^3)$^{-1}$

As described in **Section 5**, both benzene and 1,3-butadiene are classified as being carcinogenic to humans because there is sufficient animal and/or human evidence that demonstrates cancer causing activity.

Predicted cancer risks are based on the lifetime probability of developing cancer as a result of environmental exposure to a carcinogenic substance. An ILCR represents the increased probability of an individual developing cancer over an 80-year lifespan as a result of exposure to a carcinogenic COPC (i.e., incremental risk above the typical background risk that exists). Both Health Canada (2012) and B.C. Ministry of Health (2021) consider the acceptable ILCR to be one-in-one hundred thousand (1 x 10^{-5}). An ILCR greater than 1 x 10^{-5} is indicative of a potential health concern that should be more closely examined. An ILCR of less than 1 x 10^{-5} is considered essentially negligible (Health Canada, 2012).

6.2 QUANTIFYING HAZARDS FOR NON-CARCINOGENIC CHEMICALS

Most chemicals are reported to have associated health endpoints other than cancer and as such, these substances are often referred to as non-carcinogenic. Regulatory agencies assume that for non-carcinogens, there is a dose or level below which no harmful health effects will occur. As such for non-carcinogens, the potential for exposures to result in harmful human health effects is based on the ratio between the estimated exposure and health based TRV. This ratio is called the Exposure Ratio ("ER") or Hazard Quotient ("HQ") and is calculated as shown below:

$$HQ = \frac{EE}{TRV}$$

Where:

HQ = Hazard Quotient (unitless)

EE = Exposure Estimate (µg/m^3)

TRV = Chemical-Specific Toxicological Reference Value (µg/m^3)

The HQ provides an indication of whether estimated exposures are large enough to be of concern for human health. Typically, a HQ of less than 1 indicates that exposures would not be expected to result in adverse human health effects. Given that conservative assumptions are used by regulatory agencies in the development of TRVs, HQ values greater than 1.0 do not mean that adverse human health effects will occur, but the likelihood that an adverse effect will occur increases as the HQ value rises above 1.0

It should be noted that EE is derived differently for acute (1-hour or 24-hour) versus chronic (24-hour or annual) exposures. For acute exposures, EE is defined as shown in **Table 6-1** for each COPC:

Table 6-1 Averaging Period and Statistical Form for Acute Exposure Estimates

COPC	Statistical Form
SO_2	Maximum 1-hr concentration
NO_2	Annual 98th percentile of daily 1-hr maximum concentrations
$PM_{2.5}$	Maximum annual 99th percentile 24-hr average concentration
Benzene	Maximum daily 24-hr average concentration (2017-2019)
1,3-Butadiene	Maximum daily 24-hr average concentration (2017-2019)

For acute exposures, the exposure concentrations determined according to the averaging periods and statistical forms detailed in **Table 6-1** are compared directly to the acute TRV to calculate a HQ. A HQ benchmark (or "Target HQ") of 1.0 was applied to acute exposures (1-hour or 24-hour) for all COPCs and for all human receptors.

For chronic exposures, EE is defined as the annual mean air concentration (with adjustment for hours of exposure and averaging time for each receptor group, "Adj EE") because the timeframe of interest is related to longer term annual exposures. The adjusted concentration is then compared to the chronic TRV to calculate a HQ.

The equation used to derive the adjusted chronic (annual) EE is presented below:

$$AdjEE = C_{air} \times ET \times EF \times ED/AT$$

Where:

C_{air} = Measured or modelled concentration of contaminant in air (µg/m^3);

ET = Exposure time (hours/day);

EF = Exposure frequency (days/year);

ED = Exposure duration (years); and,

AT = Averaging time (days)

Details of the exposure parameters used for each receptor group are provided in **Section 4.2**. Despite the fact that all chronic exposures are determined based on annual air concentration averages, the statistical forms for these averages vary as shown in **Table 6-2** below.

Table 6-2 Averaging Period and Statistical Form for Chronic Exposure Estimates

COPC	Statistical Form
NO_2	Maximum annual average of all 1-hr concentrations
$PM_{2.5}$	Maximum annual average of 1-hr concentrations
Benzene	Maximum annual average of daily 24-hr average concentrations (2017-2019)
1,3-Butadiene	Maximum annual average of daily 24-hr average concentrations (2017-2019)

A target HQ of 1.0 was applied to chronic exposures for all COPC for residents and seniors living in retirement facilities. In accordance with BC guidance (2021), the HQ benchmark of 1.0 is applicable when baseline exposure is considered in the exposure assessment and all sources of exposure are evaluated. This assumption is considered to be met for these receptors/exposure scenarios.

A HQ benchmark of 0.2 was applied to chronic exposures for all COPCs for the following receptors and exposure scenarios: young children in daycare, students attending elementary school or high-school, patients in a hospital facility, workers, visitors, and TWN members participating in outdoor cultural activities on TWN Reserve Lands. The HQ benchmark of 0.2 is applicable in these cases because these receptors may receive only a portion of their theoretical exposure within the HHRA study area. The lower HQ benchmark allows for exposures outside of those considered in this assessment.

6.3 RESULTS OF THE QUANTITATIVE ASSESSMENT

In this section, the contribution to overall risk from each source-receptor-pathway is discussed, with emphasis in the figures placed on Refinery-only contributions. The predicted exposure estimates, ILCR, and HQs for acute and chronic exposures for each of the identified receptors and COPCs are provided in **Figure 6-1** through **Figure 6-58** and **Table 6-3** through **Table 6-14** below.

Supporting information for the HHRA is provided in **Appendix C**. Scenario 4 results are provided in **Appendix D**.

6.3.1 SULPHUR DIOXIDE (SO_2) – ACUTE EXPOSURES

As detailed in **Section 5.3** one TRV (106 µg/m^3) for acute SO_2 exposures has been applied in the risk characterization step of the HHRA. **Table 6-3** and **Figure 6-1** through **Figure 6-9** present the predicted exposure estimates and HQs for 1-hour maximum SO_2 exposures for each of the identified receptors associated with lung function decrements.

Figure 6-1 presents results for Scenario 1 – Ambient Monitoring 2017-2019, based on air quality measurements at monitoring stations near the refinery. **Figure 6-2** through **Figure 6-9** present results for Scenario 2 - Dispersion Modelling Current Permit Maximum and Scenario 3 - Dispersion Modelling Amended Permit Maximum. Exposure estimates for these scenarios were developed using a dispersion model that predicts ambient air concentrations of COPCs based on emissions from the Parkland refinery.

The coloured shading within **Table 6-3** corresponds to the colour of the applicable concentration / risk isopleths in **Figure 6-2** through **Figure 6-9. Table 6-3** also contains risk estimates for the maximally impacted receptors of each type for Scenarios 2 (S2) and 3 (S3) (see "Receptor Maxima" column).

Table 6-3 Predicted Health Risks Associated with a Decrease in Lung Function Following 1-hour Maximum Exposure to SO_2 for Identified Receptors

1-Hr Acute TRV (µg/m^3)	Baseline Conc. (µg/m^3)	HQ (Baseline)	Receptor Maxima	Predicted Conc. From Refinery (µg/m^3)	HQ (Refinery-Only)	Cumulative Conc. (µg/m^3)	HQ (Cumulative)	% HQ Attributable to Baseline
106	7.4	0.07	▲ Hospital - S3	5.2	0.05	12.6	0.12	59%
			▲ Hospital - S2	15.5	0.15	22.9	0.22	32%
			Isopleth 4	21.2	0.20	28.6	0.27	26%
			■ Seniors – S3	31.6	0.30	39	0.37	19%
			■ School – S3	40.3	0.38	47.7	0.45	16%
			◆ Daycare – S3	51.7	0.49	59.1	0.56	13%
			■ Workplace – S3	57	0.54	64.4	0.61	11%
			Isopleth 3	67.6	0.64	75	0.71	10%
			■ Seniors – S2	82.6	0.78	90	0.85	8%
			⬟ TWN – S3	97.1	0.92	104.5	0.99	7%
			▲ Residents – S3	97.4	0.92	104.8	0.99	7%

1-Hr Acute TRV (µg/m^3)	Baseline Conc. (µg/m^3)	HQ (Baseline)	Receptor Maxima	Predicted Conc. From Refinery (µg/m^3)	HQ (Refinery-Only)	Cumulative Conc. (µg/m^3)	HQ (Cumulative)	% HQ Attributable to Baseline
			TRV	106	1.00	113.4	**1.07**	7%
			School – S2	121.2	**1.14**	128.6	**1.21**	6%
			Daycare – S2	123.4	**1.16**	130.8	**1.23**	6%
			Recreation – S3	144.2	**1.36**	151.6	**1.43**	5%
			Isopleth 2	162.6	**1.53**	170	**1.60**	4%
			Workplace – S2	175.6	**1.66**	183	**1.73**	4%
			Residents – S2	217.7	**2.05**	225.1	**2.12**	3%
			TWN – S2	227.1	**2.14**	234.5	**2.21**	3%
			Isopleth 1	292.6	**2.76**	300	**2.83**	2%
			Recreation – S2	304.5	**2.87**	311.9	**2.94**	2%

Notes:
Cumulative Concentration/HQ = Baseline + Refinery Contribution
Refinery-only and cumulative HQs presented in **bold** and shaded if >1.0

The results presented above for the acute health endpoint associated with lung function decrements (TRV = 106 µg/m^3) are interpreted as follows:

- A Target HQ of 1.0 was selected for sensitive receptors as the HHRA assumed that all receptors could potentially receive their theoretical 1-hr SO_2 exposure within the HHRA study area. These receptors include: residents of all ages, seniors in LTC facilities, toddlers and young children in daycare, children and teens in school, adult patients in a hospital, workers, visitors, and TWN members participating in outdoor cultural activities within the HHRA study area.
- Air quality monitoring data from 2017 – 2019 (Scenario 1) shown in **Figure 6-1** indicates that only the Burnaby Capitol Hill (T23) monitoring station near the refinery shows a HQ greater than 1.0, with a maximum measured HQ of 3.05. Analysis of the underlying hourly data indicates that over the 3-year monitoring period, the HQ exceeded 1.0 for a total of 25 hours, with only 2 hours exceeding a HQ of 2. There were 18 hours exceeding during 2017, 6 hours exceeding during 2018, and only a single hour exceeding during 2019. This decreasing number of exceedance hours is consistent with Parkland's increased usage of SO_2 reduction additive starting in 2018.
- Baseline (ambient) SO_2 concentrations contribute relatively little to the cumulative risk within the HHRA study area. The refinery is the largest SO_2 contributor in the airshed, and as such its contributions make up the largest proportion of total risk, particularly for Scenario 2 (Current Permit Maximum) ranging from 41% of the risk for the Scenario 3 maximum hospital receptor to more than 98% for the Scenario 2 maximum recreation receptor. For the amended permit Scenarios 3 (maximum) and 4 (normal), SO_2 concentrations within the HHRA study area are greatly reduced. **Table 6-15** shows the percent change in maximum predicted 1-hour SO_2 concentrations relative to current permit maximum (Scenario 2) for the amended permit scenarios (3 and 4); the percent change from the current permit maximum results in a net reduction of 51% (Scenario 2 versus Scenario 3) and 78% (Scenario 2 versus Scenario 4) for refinery-only contributions.
- Cumulative HQs based on the modelled SO_2 concentrations presented in **Table 6-3** range from 0.12 for the Scenario 3 maximum hospital receptor to 2.94 for the Scenario 2 maximum recreation receptor. **Figure 6-2** through **Figure 6-9** present a graphical representation of risk results for Scenarios 2 and 3. Scenario 4 results are presented in **Appendix D**, **Figure D-1** to **Figure D-4**. The highest predicted 1-hr SO_2 concentrations for Scenarios 3 and 4 are spatially limited and do not overlap with the presence of sensitive receptors, with the exception of the Scenario 3 maximum recreational receptor located immediately beside the refinery.
- Given that the highest predicted refinery-only concentrations result in a HQ greater than 1.0 in certain locations within the HHRA study area, a statistical evaluation was performed to further refine the probability of risk to sensitive receptors.

  - Frequency of exceedance ("FOE") statistics[5] were generated to determine the number of hours over the full 1-year modelling period (i.e., 8760 hours) that would be predicted to exceed the Target HQ of 1.0 at each of the maximally exposed receptor locations. This analysis counts the number of hours when the refinery-only SO_2 concentrations exceed this level. These results are provided in **Appendix C** for all modelled scenarios.
  - For Scenario 2, the total number of hours predicted to exceed the Target HQ of 1.0 at each of the maximally exposed receptors ranged from 0 hours (maximum hospital receptor and maximum senior care receptor) to 9 hours (maximum recreation receptor). Note that 9 hours represents a very small proportion of the year (0.1%).
  - For Scenario 3 the total number of hours predicted to exceed the Target HQ of 1.0 at each of the maximally exposed receptors was predicted to be 0 hours for all sensitive receptors, except the maximum recreation receptor located beside the refinery (2 hours predicted to exceed the TRV for the 1-year modelling period). The FOE statistics for Scenario 4 show that the maximum number of hours exceeding the Target HQ of 1.0 at the Trans Canada Trail is further reduced from 2 hours to nil (0 hours).
- The predicted (cumulative) 1-hour SO_2 concentrations greater than the TRV of 106 µg/m^3 (corresponding to a predicted refinery contribution of 98.6 µg/m^3) may result in a decrease of lung function for sensitive receptors present within these areas. Based on the modelling, a significant reduction in SO_2 concentrations (i.e., up to a 78% decrease) is achieved in Scenarios 3 and 4 (compared to Scenario 2), virtually eliminating the spatial extent of cumulative concentrations greater than 106 µg/m^3 under the amended permit scenarios.
- Conservative assumptions made in the HHRA that may lead to an overestimation of risks are further discussed in **Section 6.5**.

[5] FOE calculations were based on refinery-only 1-hour SO_2 concentrations for all modelled hours (i.e., the 100th percentile dataset = 8760 hours).

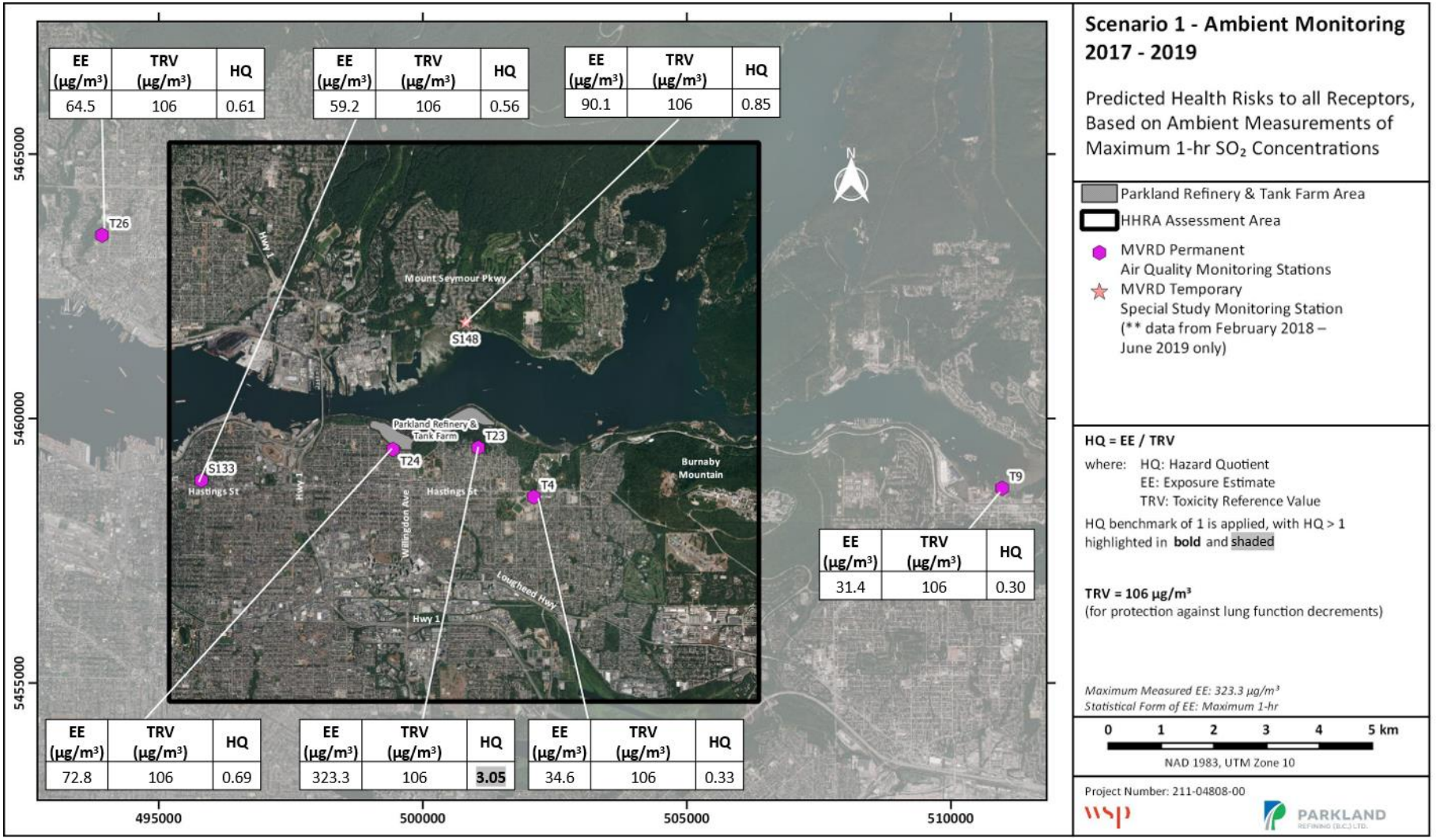


Figure 6-1: Scenario 1 – Predicted Health Risks to All Receptors Based on Ambient Measurements of Maximum 1-hr SO_2 Concentrations

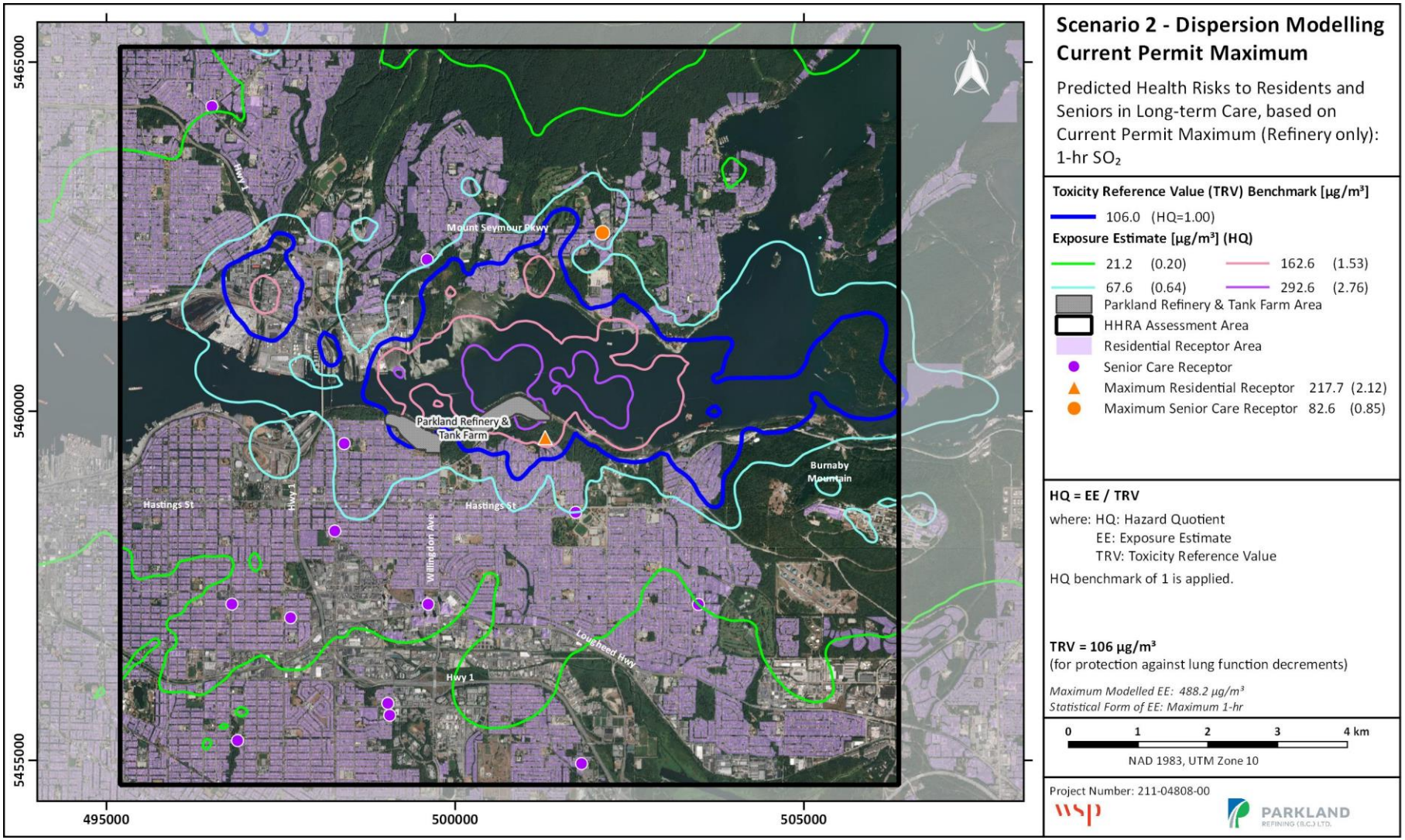


Figure 6-2: Scenario 2– Predicted Health Risks to Residents and Seniors in Long-term Care Based on Current Permit Maximum (Refinery-Only) 1-hr SO_2

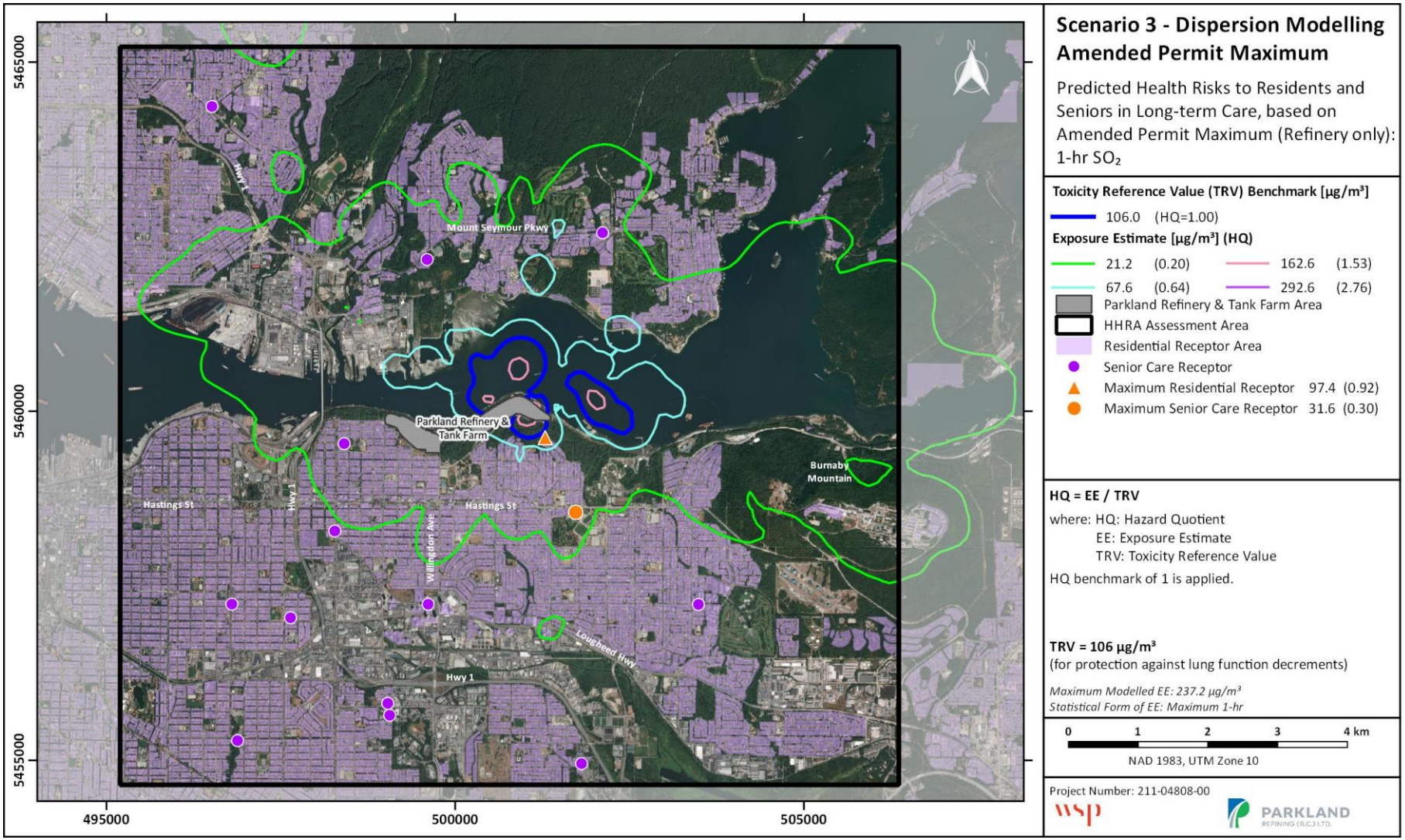


Figure 6-3: Scenario 3 – Predicted Health Risks to Residents and Seniors in Long-term Care Based on Amended Permit Maximum (Refinery-Only) 1-hr SO_2

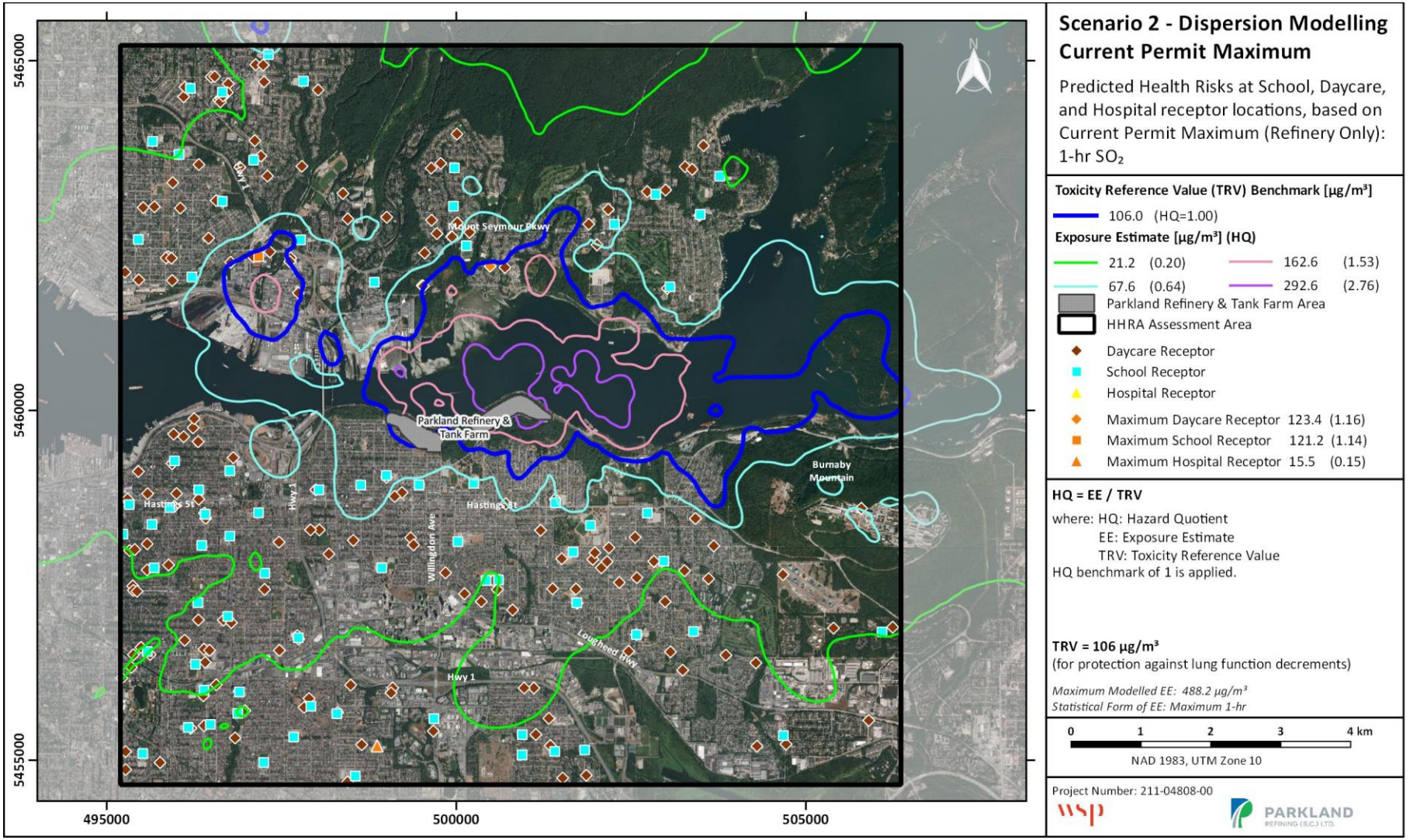


Figure 6-4: Scenario 2 – Predicted Health Risks at School, Daycare, and Hospital Receptor Locations Based on Current Permit Maximum (Refinery-Only) 1-hr SO_2

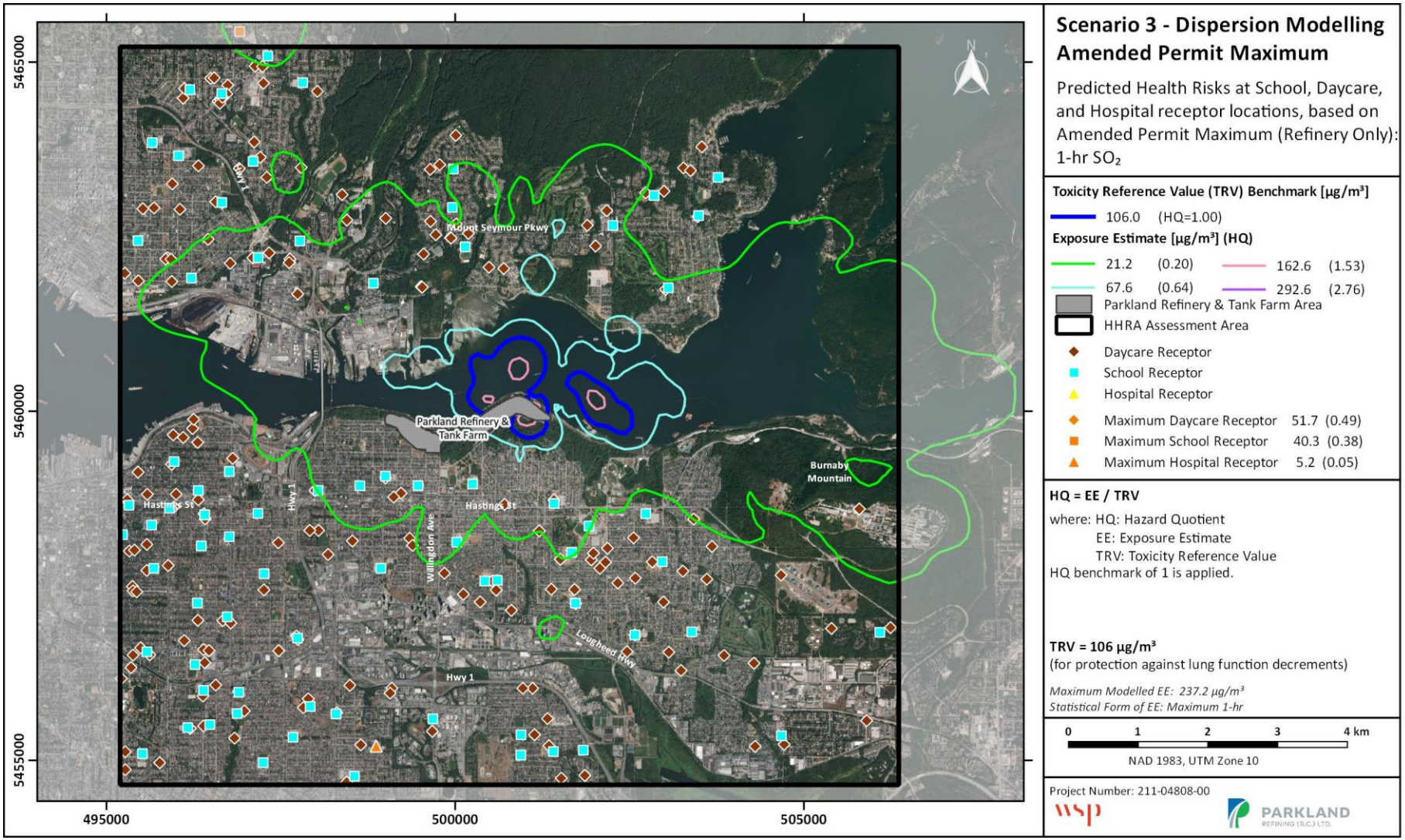


Figure 6-5: Scenario 3 – Predicted Health Risks at School, Daycare, and Hospital Receptor Locations Based on Amended Permit Maximum (Refinery-Only) 1-hr SO_2

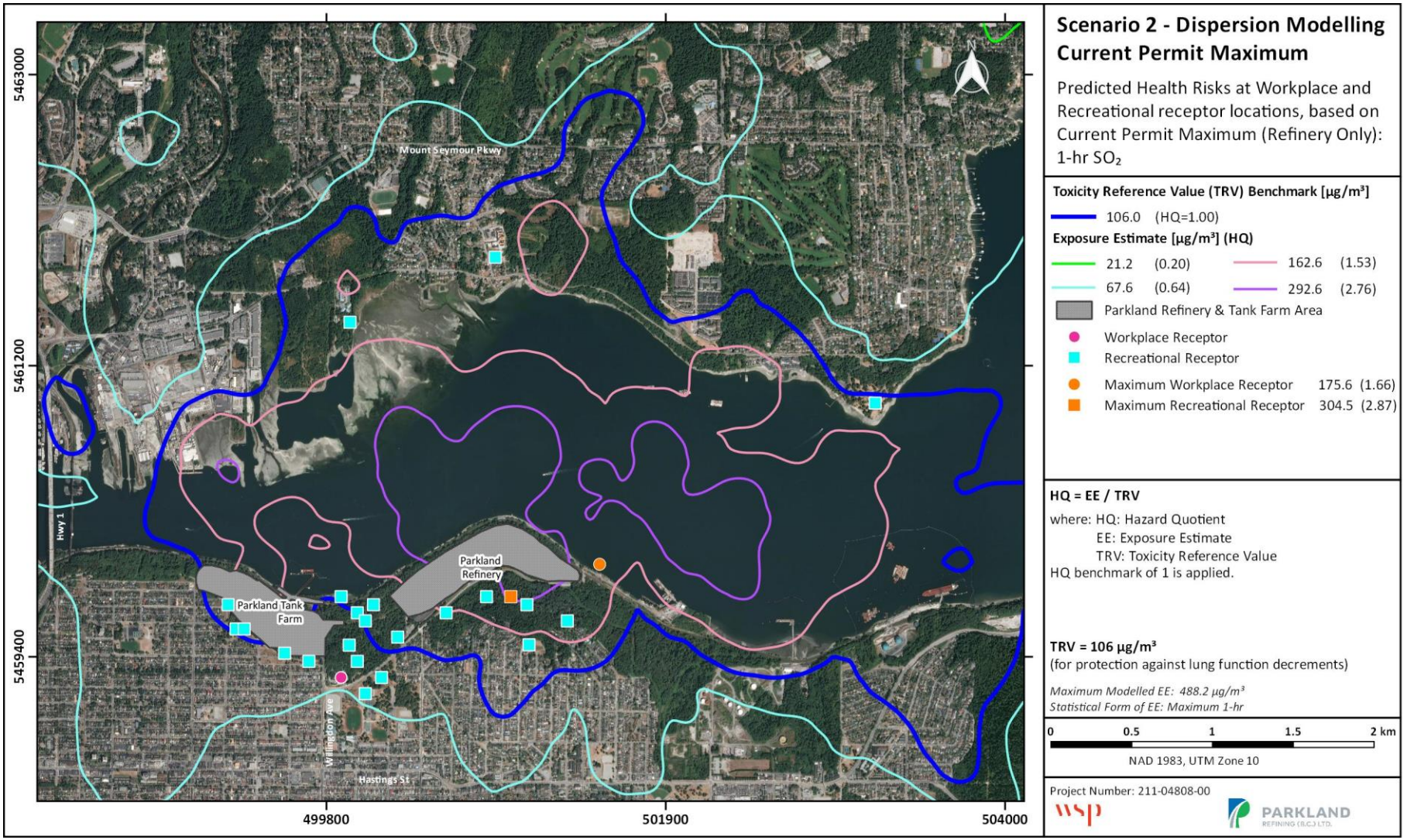


Figure 6-6: Scenario 2 – Predicted Health Risks at Workplace and Recreational Receptor Locations Based on Current Permit Maximum (Refinery-Only) 1-hr SO_2

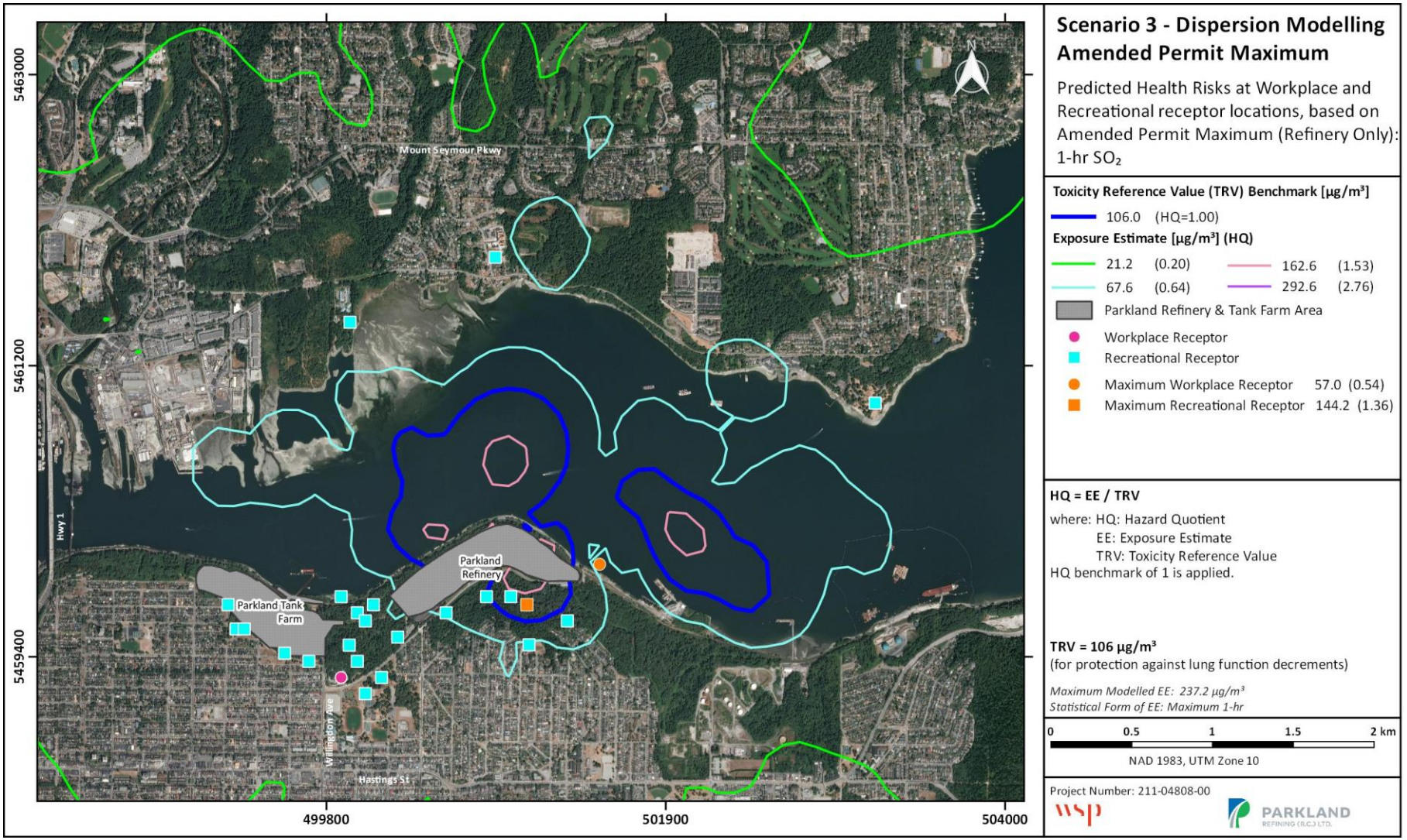


Figure 6-7: Scenario 3 – Predicted Health Risks at Workplace and Recreational Receptor Locations Based on Amended Permit Maximum (Refinery-Only) 1-hr SO_2

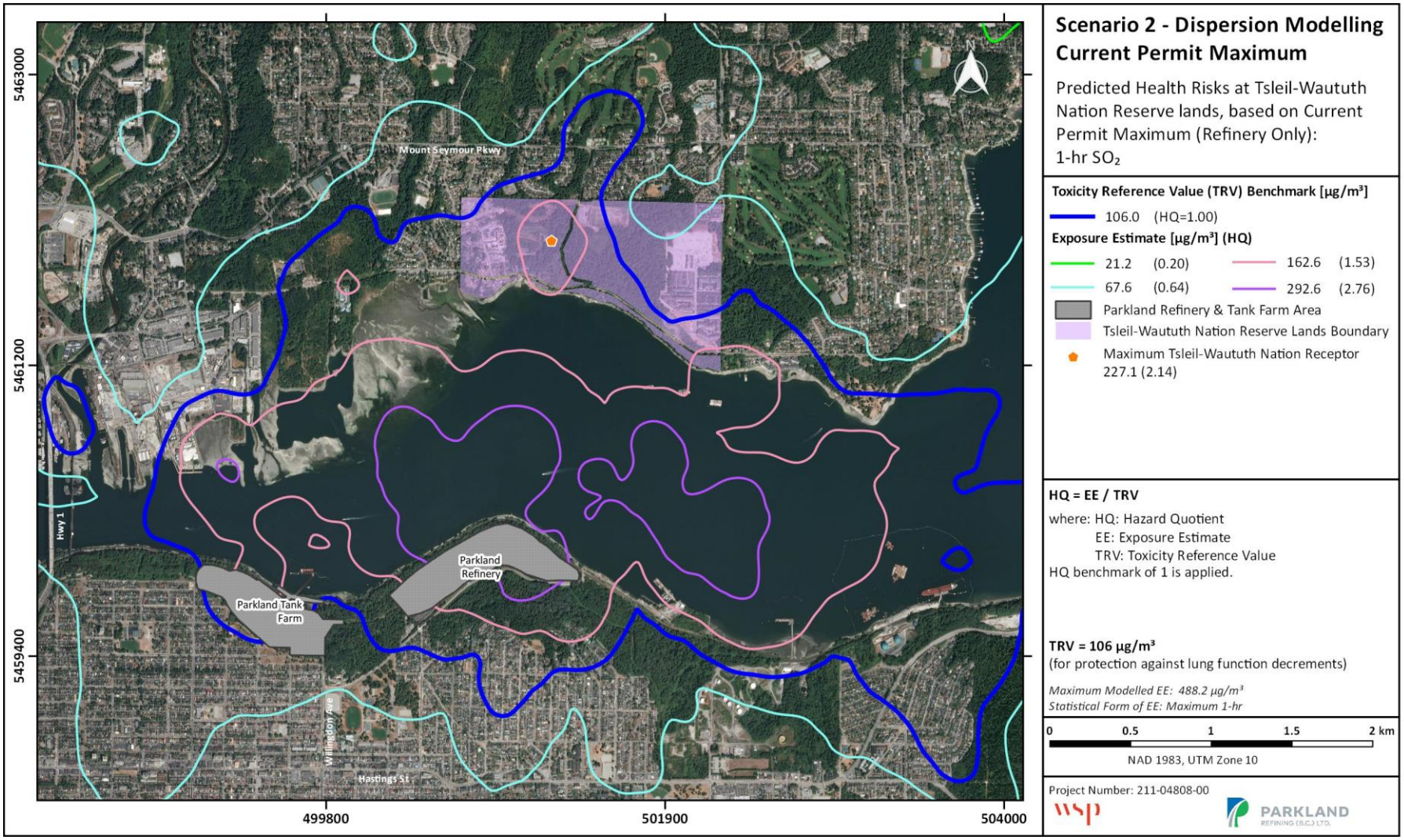


Figure 6-8: Scenario 2 – Predicted Health Risks at TWN Reserve Lands Based on Current Permit Maximum (Refinery-Only) 1-hr SO_2

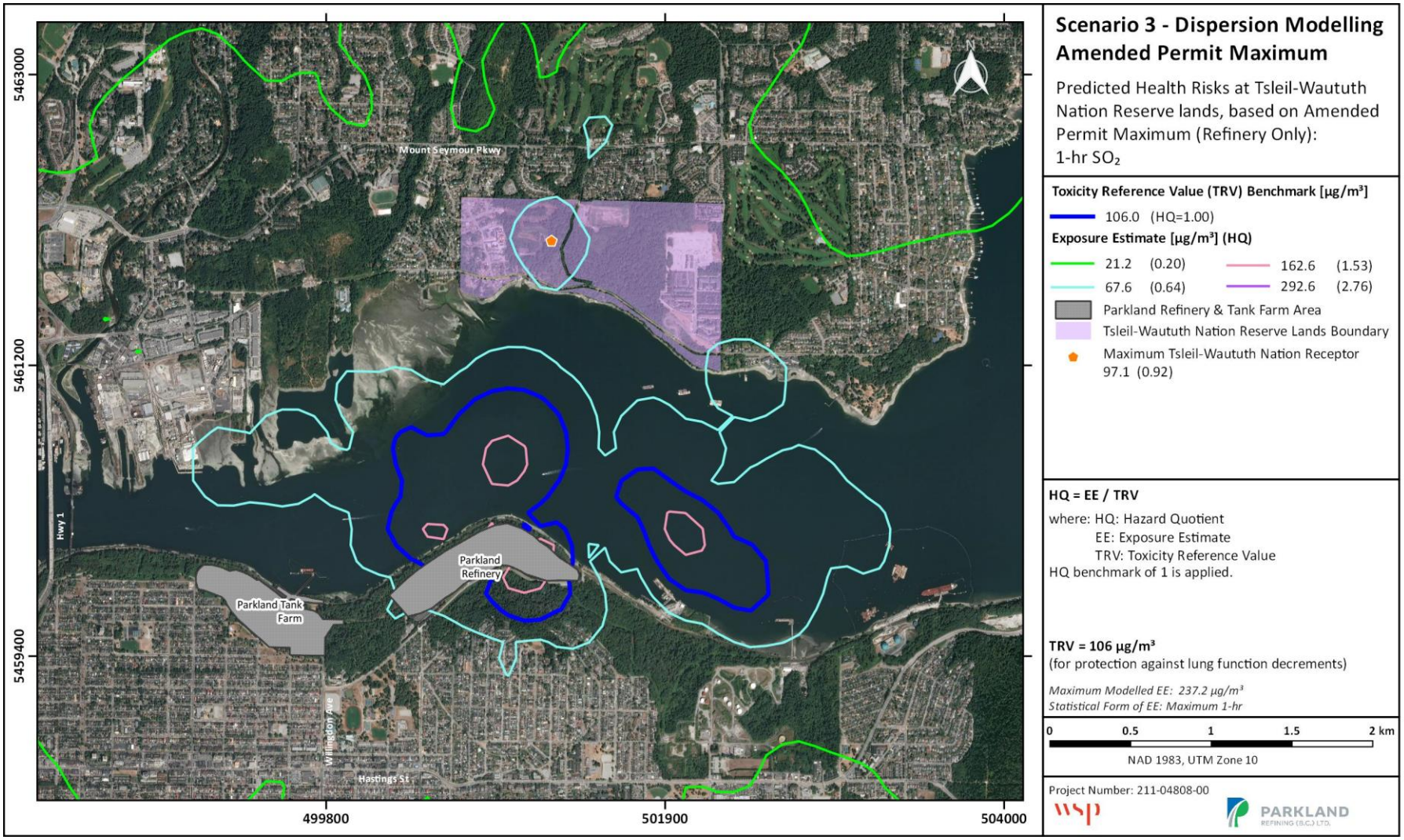


Figure 6-9: Scenario 3 – Predicted Health Risks at TWN Reserve Lands Based on Amended Permit Maximum (Refinery-Only) 1-hr SO_2

6.3.2 NITROGEN DIOXIDE (NO_2) – ACUTE EXPOSURES (TRV=113 µg/m³)

As detailed in **Section 5.3** two TRVs for acute NO_2 exposures have been applied in the risk characterization step of the HHRA. This section presents the results for the TRV of 113 µg/m^3, which was derived based on risks associated with airway hyper-responsiveness. **Table 6-4** and **Figure 6-10** through **Figure 6-18** below present the predicted exposure estimates and HQs associated with airway hyper-responsiveness for predicted 98th percentile daily 1-hour maximum NO_2 exposures for each of the identified receptors.

Figure 6-10 presents results for Scenario 1 – Ambient Monitoring 2017-2019, based on air quality measurements at monitoring stations near the refinery. **Figure 6-11** through **Figure 6-18** present results for Scenario 2 - Dispersion Modelling Current Permit Maximum and Scenario 3 - Dispersion Modelling Amended Permit Maximum. Exposure estimates for these scenarios were developed using a dispersion model that predicts ambient air concentrations of COPCs based on emissions from the Parkland refinery.

The coloured shading within **Table 6-4** corresponds to the colour of the applicable concentration / risk isopleths in **Figure 6-11** through **Figure 6-18**. **Table 6-4** also contains risk estimates for the maximally impacted receptors of each type for Scenarios 2 (S2) and 3 (S3) (see "Receptor Maxima" column).

Table 6-4 Predicted Health Risks Associated with Airway Hyper-Responsiveness Following Daily 1-hour Maximum Exposure to NO_2 for Identified Receptors

1-Hr Acute TRV (µg/m^3)	Baseline Conc. (µg/m^3)	HQ (Baseline)	Receptor Maxima	Predicted Conc. From Refinery (µg/m^3)	HQ (Refinery -Only)	Cumulative Conc. (µg/m^3)	HQ (Cumulative)	% Cumulative HQ Attributable to Baseline
113	74.7	0.66	▲ Hospital - S3	3.4	0.03	78.1	0.69	96%
			▲ Hospital - S2	3.9	0.03	78.6	0.70	95%
			Isopleth 4	4.3	0.04	79	0.70	95%
			Isopleth 3	22.6	0.20	97.3	0.86	77%
			■ School – S3	22.9	0.20	97.6	0.86	77%
			◆ Daycare – S3	27.2	0.24	101.9	0.90	73%
			■ Workplace – S3	27.9	0.25	102.6	0.91	73%
			■ Seniors – S3	29.1	0.26	103.8	0.92	72%
			■ School – S2	30.0	0.27	104.7	0.93	71%
			◆ Daycare – S2	33.4	0.30	108.1	0.96	69%
			■ Workplace – S2	33.5	0.30	108.2	0.96	69%
			■ Seniors – S2	36.8	0.33	111.5	0.99	67%
			⬟ TWN – S3	40.6	0.36	115.3	**1.02**	65%
			⬟ TWN – S2	46.7	0.41	121.4	**1.07**	62%
			▲ Residents – S3	47.9	0.42	122.6	**1.08**	61%
			Isopleth 2	50.3	0.45	125	**1.11**	60%
			■ Recreation – S3	59.7	0.53	134.4	**1.19**	56%
			▲ Residents – S2	62.7	0.55	137.4	**1.22**	54%
			■ Recreation – S2	75	0.66	149.7	**1.32**	50%
			Isopleth 1	79.0	0.70	153.7	**1.36**	49%

Notes:
Cumulative Concentration/HQ = Baseline + Refinery Contribution.

1-Hr Acute TRV (µg/m^3)	Baseline Conc. (µg/m^3)	HQ (Baseline)	Receptor Maxima	Predicted Conc. From Refinery (µg/m^3)	HQ (Refinery -Only)	Cumulative Conc. (µg/m^3)	HQ (Cumulative)	% Cumulative HQ Attributable to Baseline
Refinery-only and cumulative HQs presented in **bold** and shaded if >1.0								

The results presented above for the acute health endpoint of airway hyper-responsiveness (TRV = 113 µg/m^3) are interpreted as follows:

- A Target HQ of 1.0 was selected for sensitive receptors as the HHRA assumed that all receptors could potentially receive their theoretical 1-hr NO_2 exposure within the HHRA study area. These receptors include: residents of all ages, seniors in LTC facilities, toddlers and young children in daycare, children and teens in school, adult patients in a hospital, workers, visitors, and TWN members participating in outdoor cultural activities within the HHRA study area.
- Air quality monitoring data from 2017 – 2019 (Scenario 1) shown in **Figure 6-10** indicates that none of the monitoring stations included in the study show HQs greater than 1.0, based on the TRV of 113 µg/m^3. It is important to note that the HQ are very similar for all of the monitoring stations, indicating very consistent maximum 1-hr NO_2 concentrations throughout the study area, regardless of their respective distance from the refinery. This suggests that baseline / non-refinery NO_2 sources are the driver of concentrations and associated NO_2 health risks throughout the study domain.
- Air quality modelling results (Scenarios 2, 3 and 4) corroborate the point above and indicate that baseline (ambient) NO_2 concentrations account for the majority of the cumulative NO_2 health risk within the HHRA study area, contributing50 to 96% of the HQ for the maximum sensitive receptors. The baseline 1-hour NO_2 concentrations result in a HQ of 0.66.
- HQs for refinery-only contributions ranged from 0.03 for the Scenario 3 maximum hospital receptor to 0.66 for the Scenario 2 maximum recreational receptor (corresponding to modelled refinery contributions of 3.4 to 75 µg/m^3). As shown on **Figure 6-11** through **Figure 6-18**, the zones of highest predicted refinery NO_2 concentrations for Scenarios 2 and 3 are spatially limited and generally do not overlap with the presence of sensitive receptors. Refinery-only HQs for the maximally exposed sensitive receptors are all less than 1.0 (see **Figure 6-11** through **Figure 6-18**). Scenario 4, which represents the most likely future exposure to human receptors, shows a further reduction in the spatial extent of highest modelled refinery NO_2 concentrations (see **Appendix D**, **Figure D-5** through **Figure D-8**).
- The predicted cumulative 1-hour NO_2 concentrations greater than the TRV of 113 µg/m^3 (corresponding to a predicted refinery contribution of 38.3 µg/m^3) result in an HQ greater than 1.0, and thus may result in the potential for increased airway hyper-responsiveness for select sensitive receptors (see **Table 6-4)**. However, as stated above, the majority of the predicted health risk is associated with baseline / non-refinery NO_2 concentrations that are beyond the control of the refinery.
- Conservative assumptions made in the HHRA that may lead to overestimation of risks are further discussed in Section 6.5. Additional discussion of the potential for health risks due to exposure to daily 1-hr maximum NO_2 concentrations is provided below in the context of the more stringent TRV (79 µg/m^3).

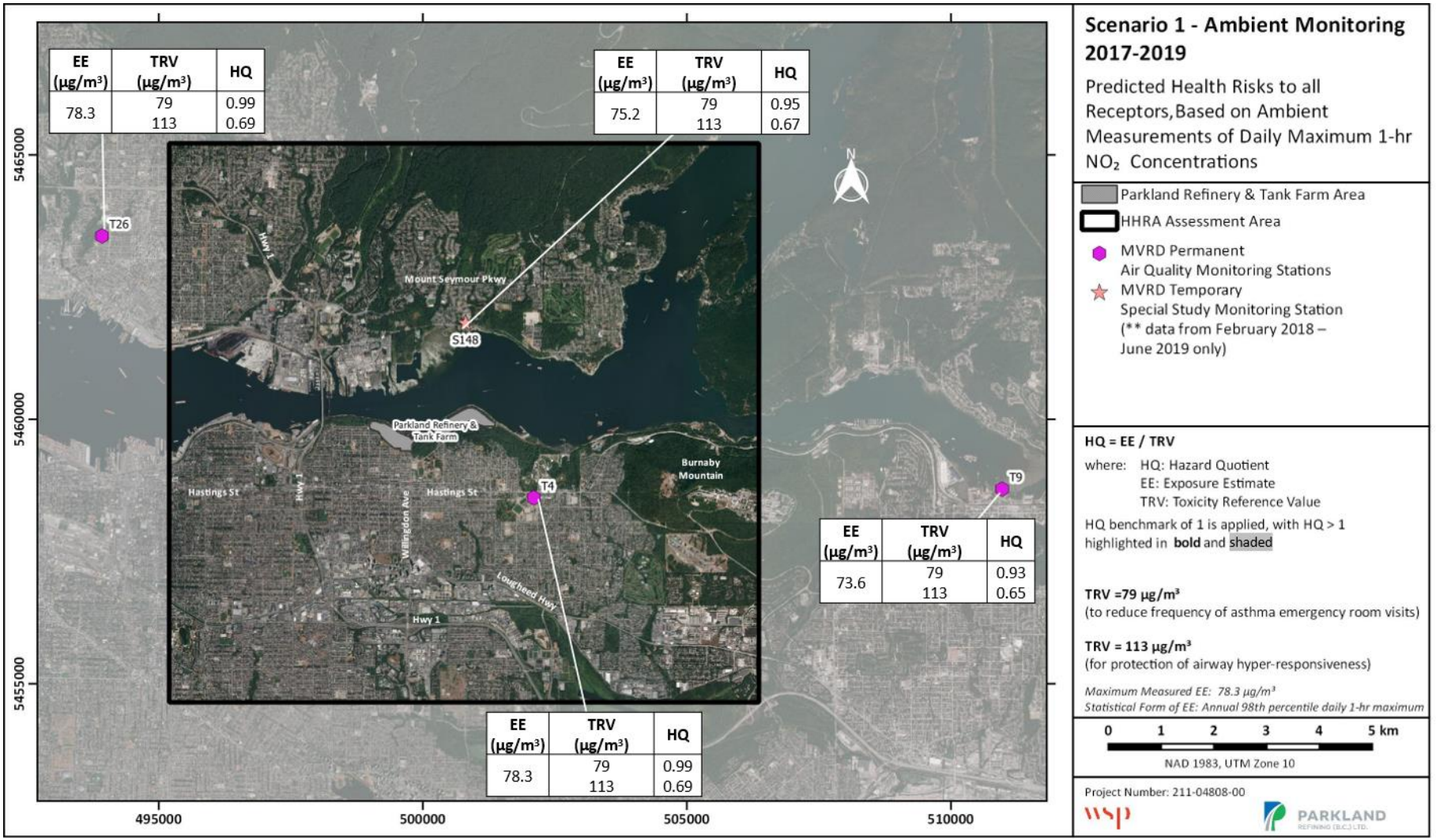


Figure 6-10: Scenario 1 – Predicted Health Risks to All Receptors Based on Ambient Measurements of Daily Maximum 1-hr NO_2 Concentrations

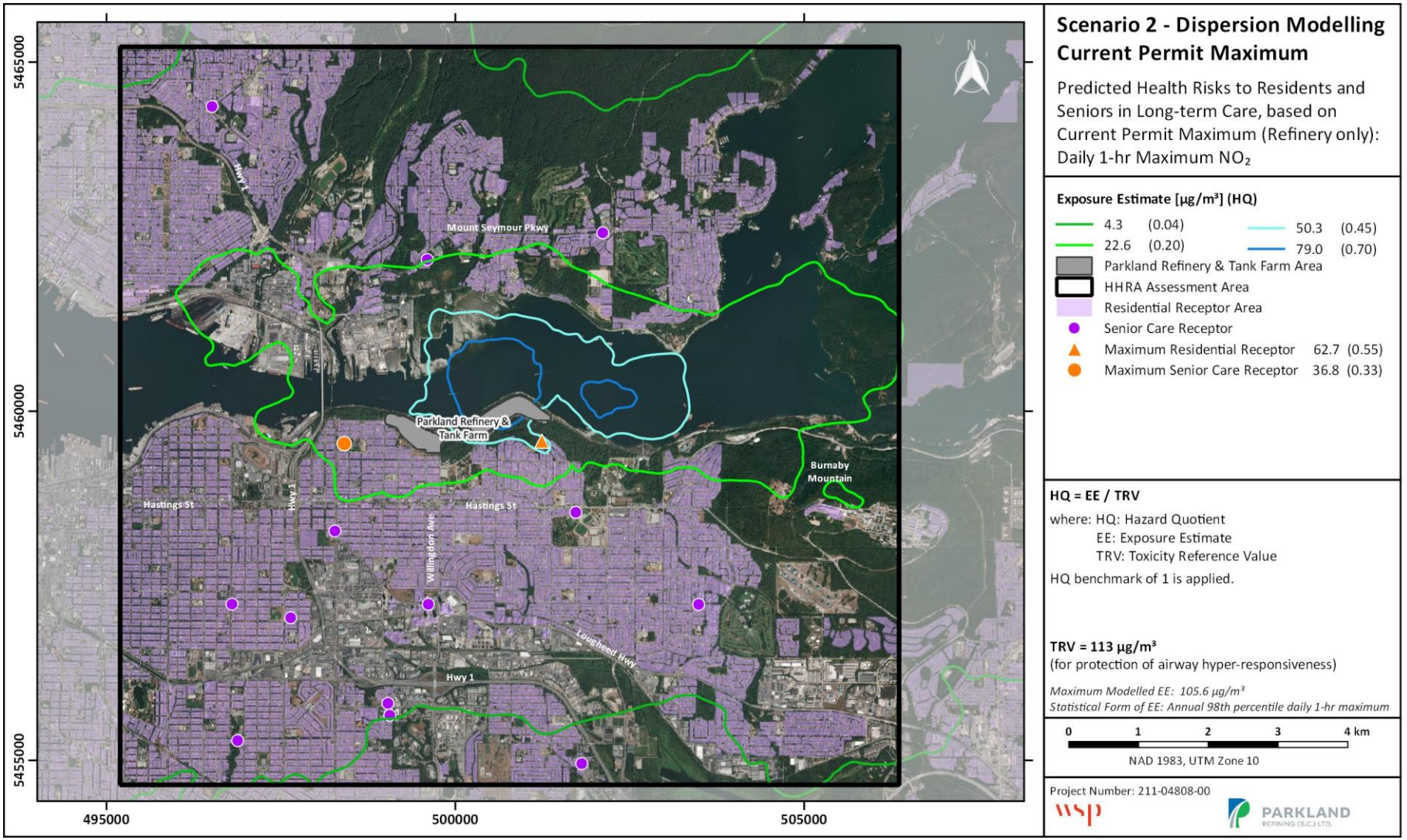


Figure 6-11: Scenario 2 – Predicted Health Risks to Residents and Seniors in Long-term Care Based on Current Permit Maximum (Refinery-Only) Daily 1-hr Maximum NO_2 (TRV=113 ug/m^3)

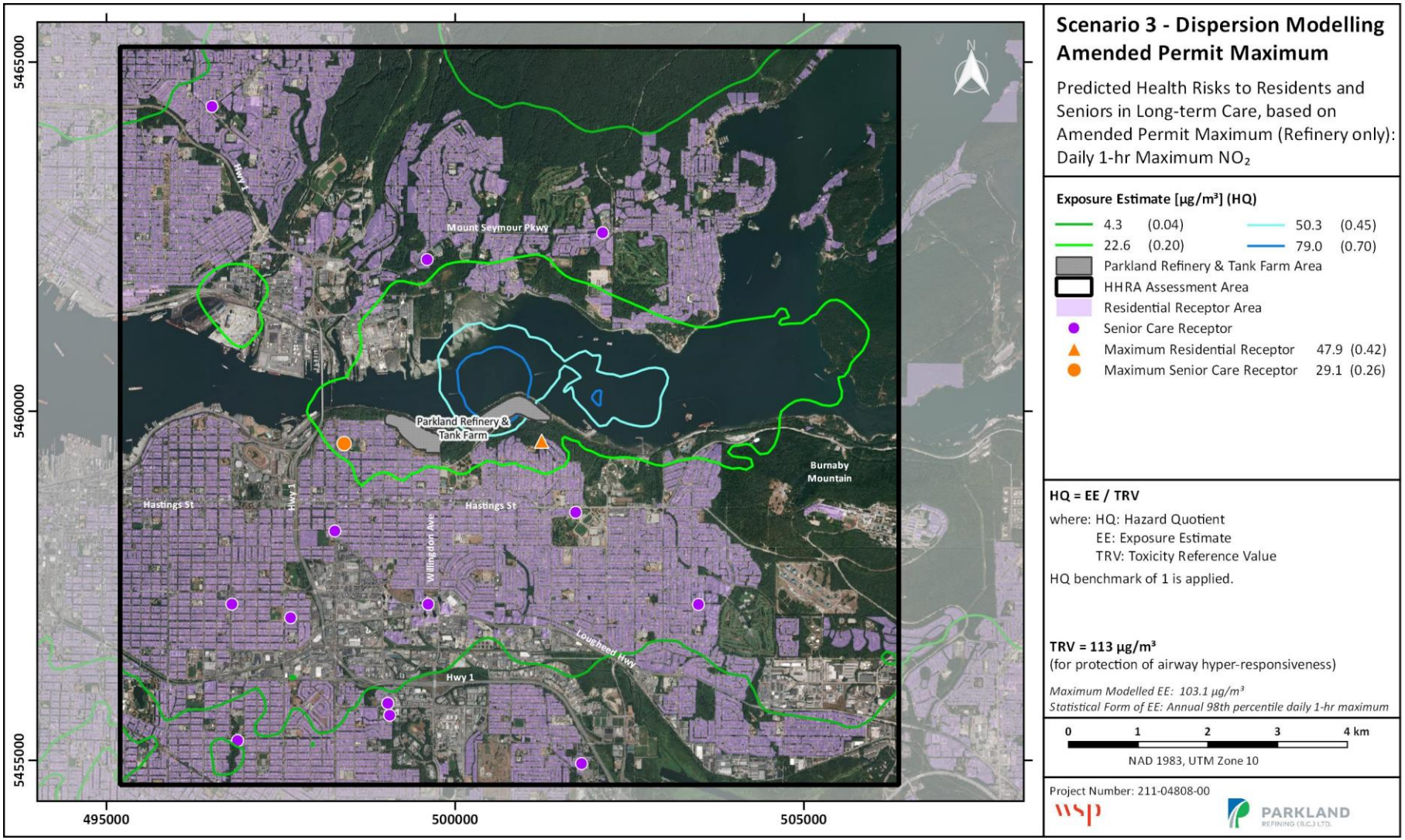


Figure 6-12: Scenario 3 – Predicted Health Risks to Residents and Seniors in Long-term Care Based on Amended Permit Maximum (Refinery-Only) Daily 1-hr Maximum NO_2 (TRV=113 ug/m^3)

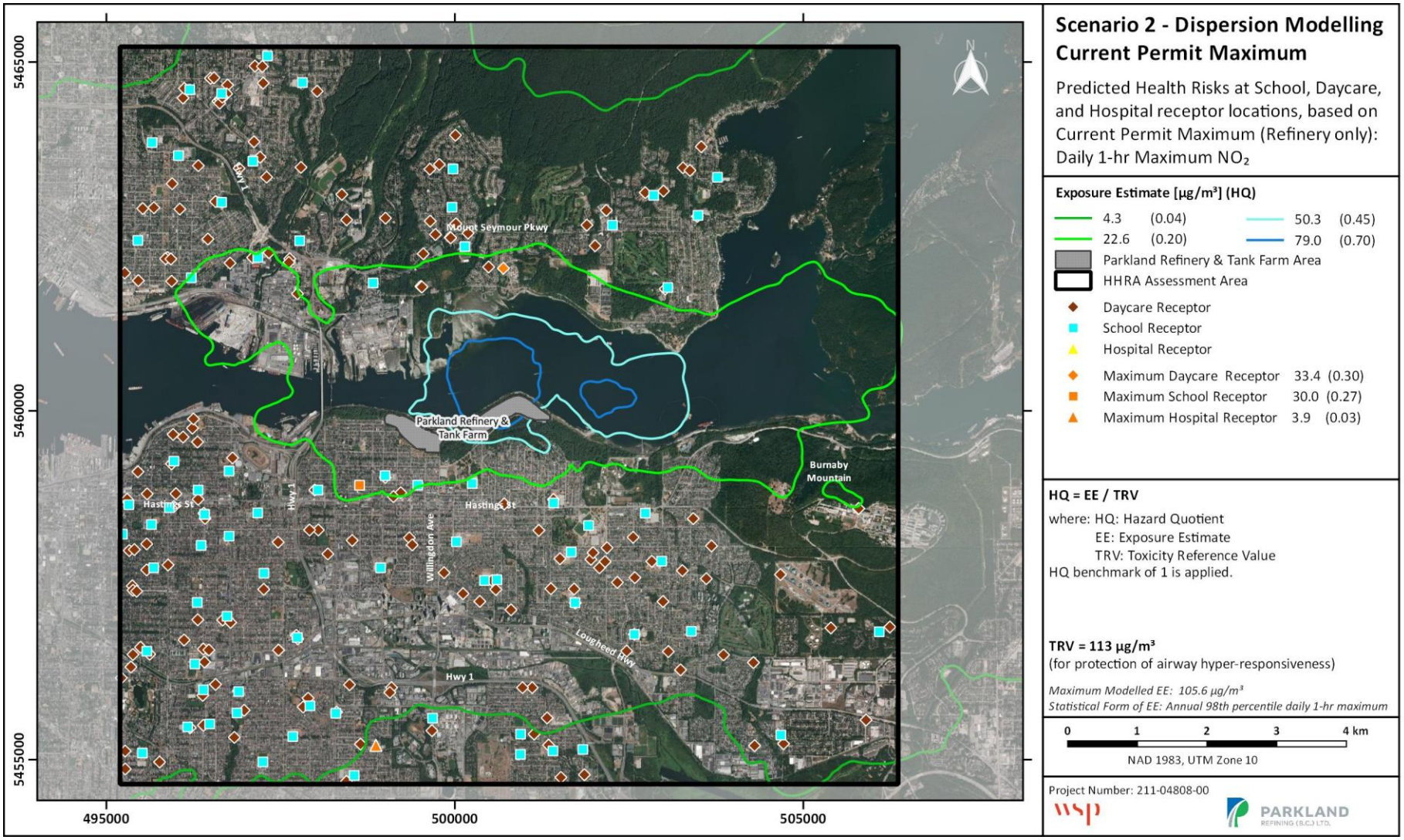


Figure 6-13: **Scenario 2 – Predicted Health Risks at School, Daycare, and Hospital Receptor Locations Based on Current Permit Maximum Scenario (Refinery-Only) Predicted Daily 1-hr Maximum NO_2 Concentrations (TRV=113 ug/m^3)**

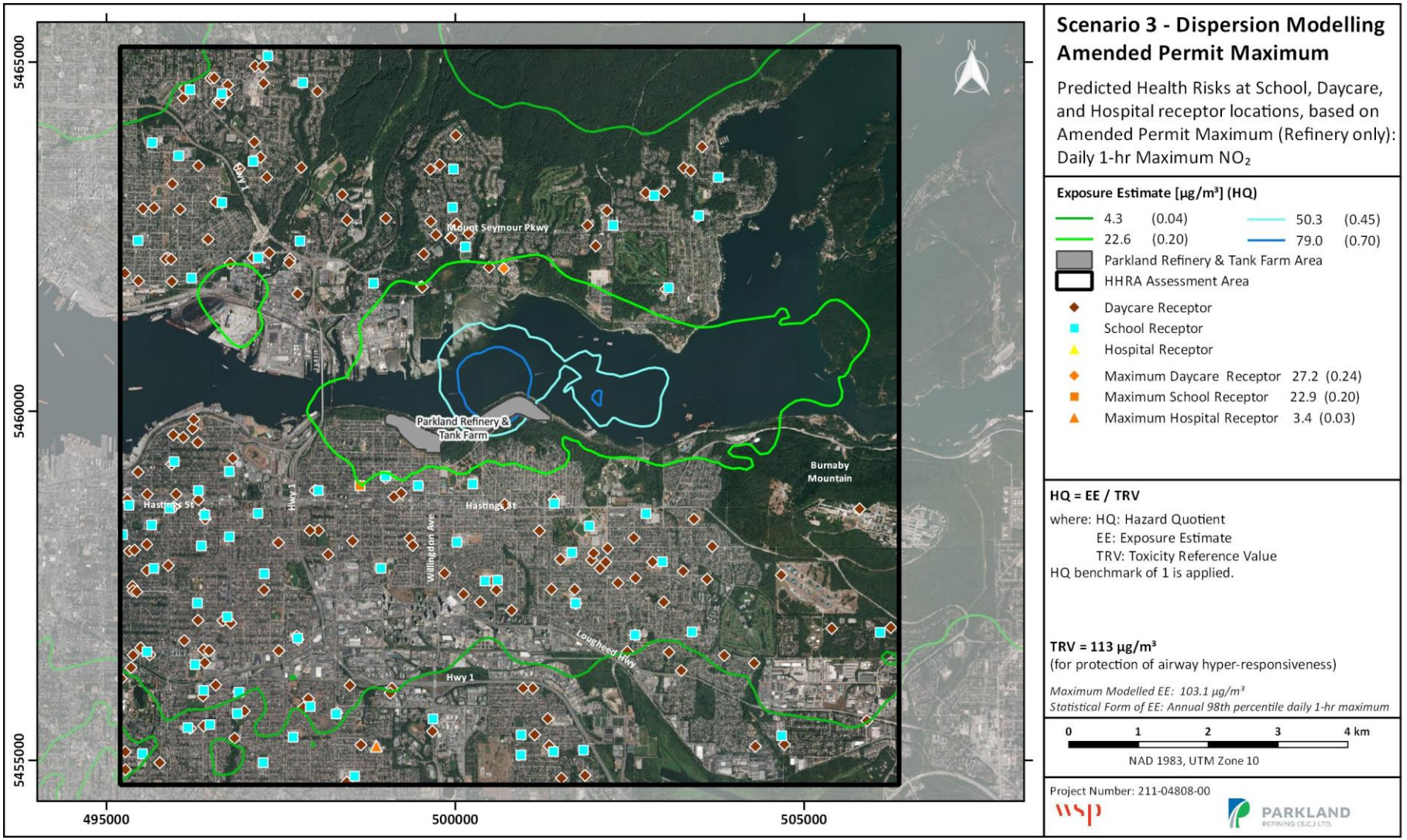


Figure 6-14: Scenario 3 – Predicted Health Risks at School, Daycare, and Hospital Receptor Locations Based on Amended Permit Maximum (Refinery-Only) Daily 1-hr Maximum NO_2 (TRV=113 ug/m^3)

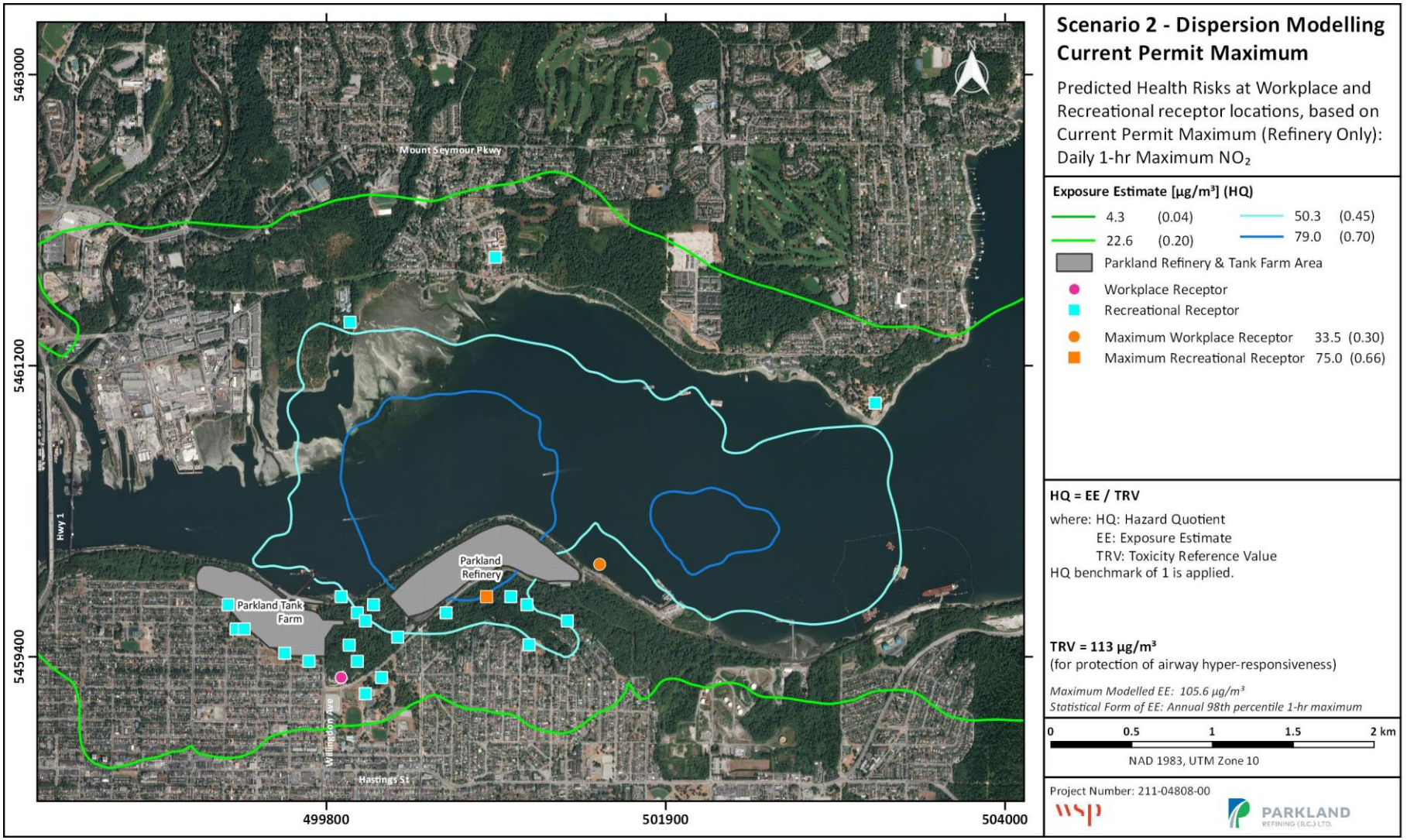


Figure 6-15: Scenario 2 – Predicted Health Risks at Workplace and Recreational Receptor Locations Based on Current Permit Maximum (Refinery-Only) Daily 1-hr Maximum NO_2 (TRV=113 ug/m^3)

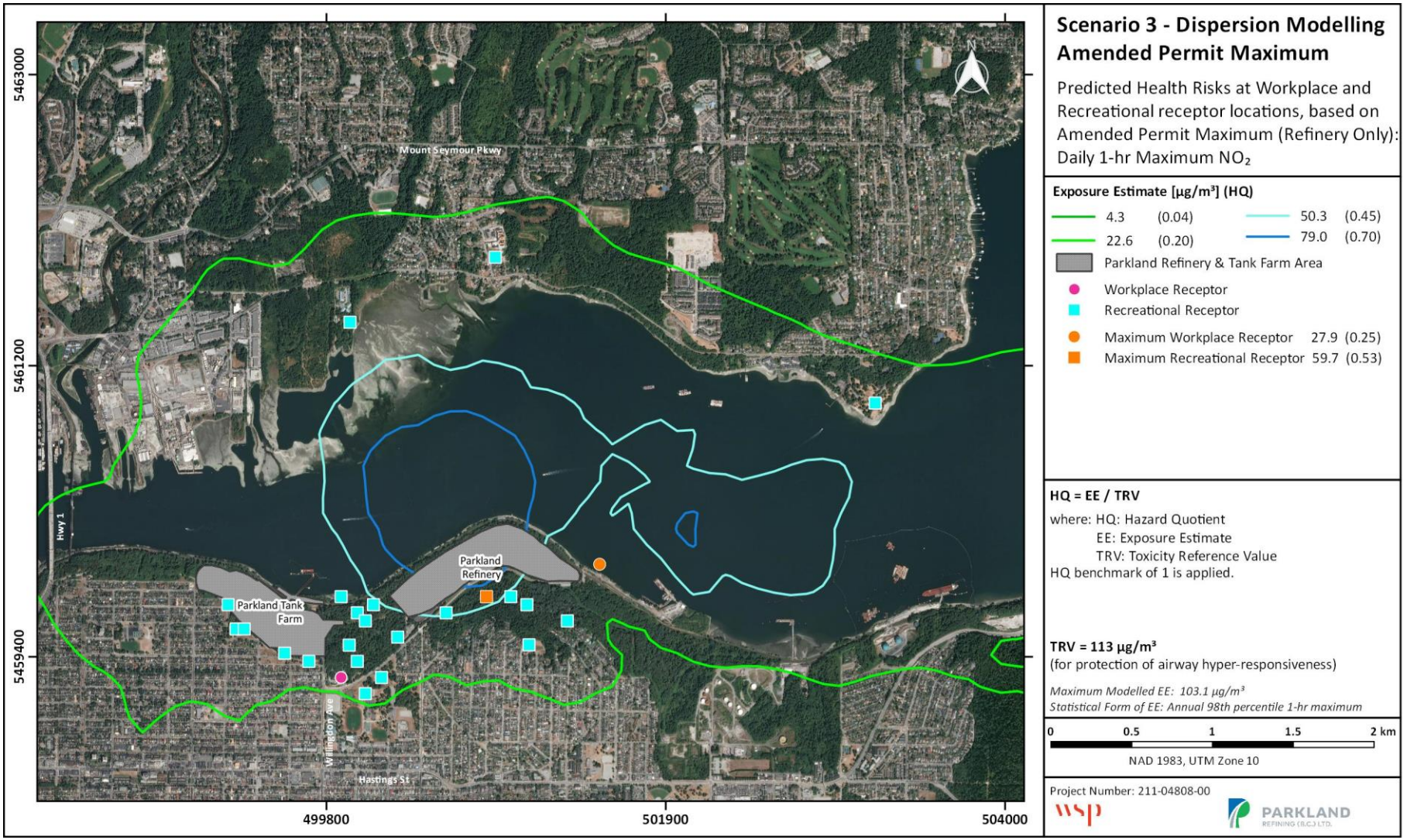


Figure 6-16: Scenario 3 – Predicted Health Risks at Workplace and Recreational Receptor Locations Based on Amended Permit Maximum (Refinery-Only) Daily 1-hr Maximum NO_2 (TRV=113 ug/m^3)

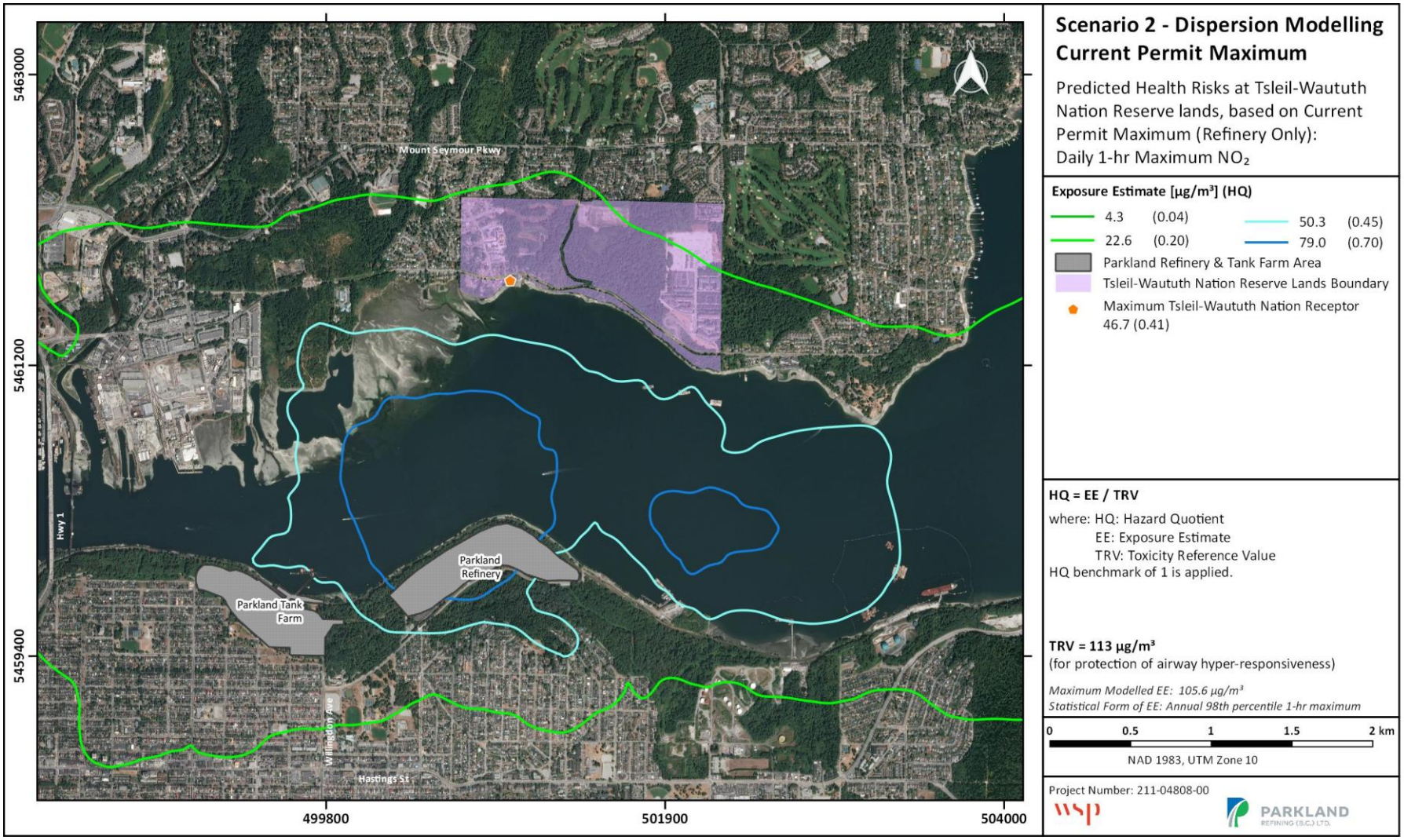


Figure 6-17: Scenario 2 – Predicted Health Risks at TWN Reserve Lands Based on Current Permit Maximum (Refinery-Only) Daily 1-hr Maximum NO_2 (TRV=113 ug/m^3)

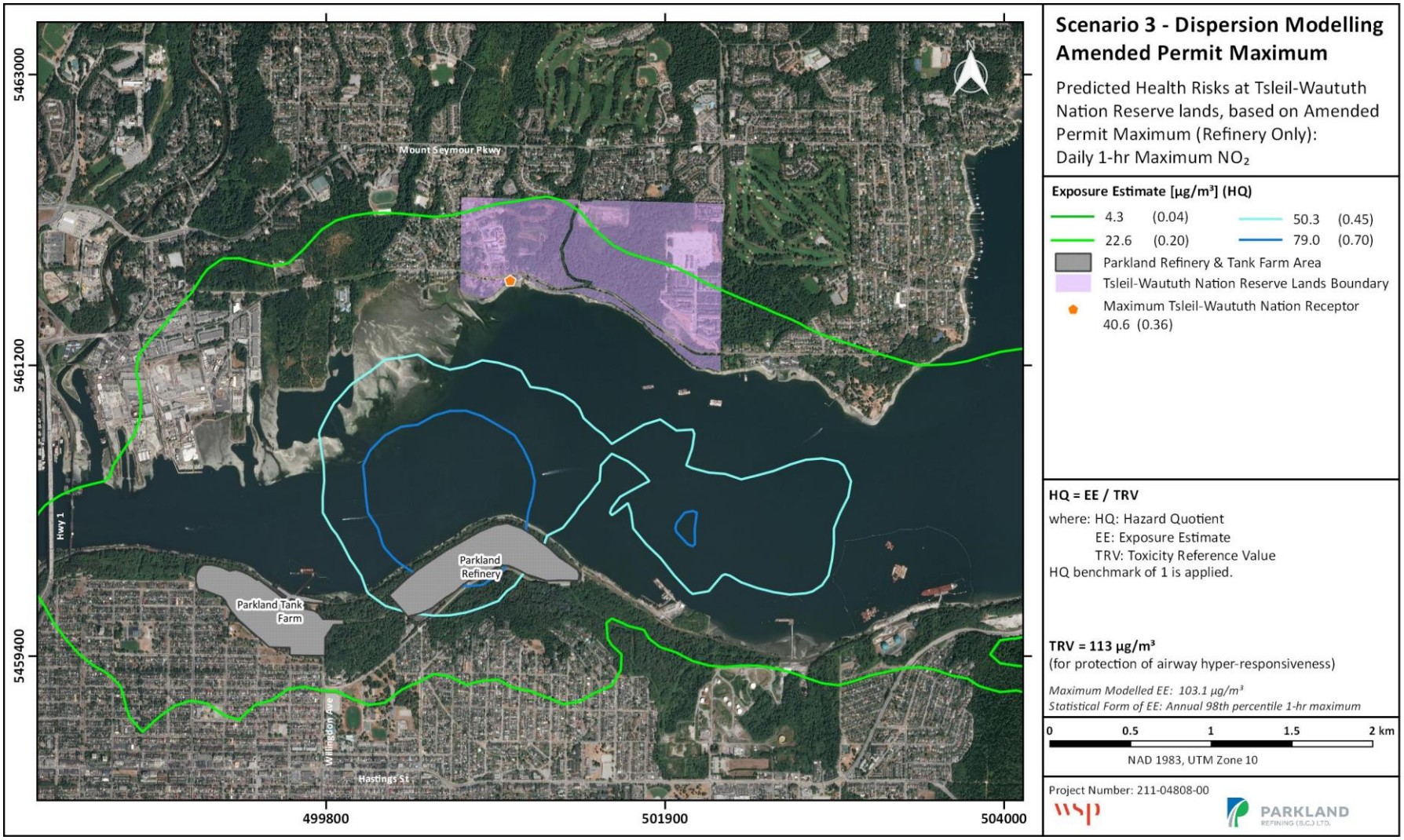


Figure 6-18: Scenario 3 – Predicted Health Risks at TWN Reserve Lands Based on Amended Permit Maximum (Refinery-Only) Daily 1-hr Maximum NO_2 (TRV=113 ug/m^3)

6.3.3 NITROGEN DIOXIDE (NO_2) – ACUTE EXPOSURES (TRV=79 µg/m^3)

As detailed in **Section 5.3** two TRVs for acute NO_2 exposures have been applied in the risk characterization step of the HHRA. This section presents the results for the TRV of 79 µg/m^3, which was derived based on risks associated with asthma emergency room visits. **Table 6-5** and **Figure 6-19** through **Figure 6-27** present the predicted exposure estimates and HQs associated with asthma emergency room visits for predicted 98th percentile daily 1-hour maximum NO_2 exposures for each of the identified receptors.

Figure 6-19 presents results for Scenario 1 –Ambient Monitoring 2017-2019, based on air quality measurements at monitoring stations near the refinery. **Figure 6-20** through **Figure 6-27** present results for Scenario 2 - Dispersion Modelling Current Permit Maximum and Scenario 3 - Dispersion Modelling Amended Permit Maximum. Exposure estimates for these scenarios were developed using a dispersion model that predicts ambient air concentrations of COPCs based on emissions from the Parkland refinery.

The coloured shading within **Table 6-5** corresponds to the colour of the applicable concentration / risk isopleths in **Figure 6-20** through **Figure 6-27 Table 6-5** also contains risk estimates for the maximally impacted receptors of each type for Scenarios 2 (S2) and 3 (S3) (see "Receptor Maxima" column).

Table 6-5 Predicted Health Risks Associated with Asthma Emergency Room Visits Following Daily 1-hour Maximum Exposure to NO_2 for Identified Receptors

1-Hr Acute TRV (µg/m^3)	Baseline Conc. (µg/m^3)	HQ (Baseline)	Receptor Maxima	Predicted Conc. From Refinery (µg/m^3)	HQ (Refinery-Only)	Cumulative Conc. (µg/m^3)	HQ (Cumulative)	% HQ Attributable to Baseline
79	74.7	0.95	▲ Hospital - S3	3.4	0.04	78.1	0.99	96%
			▲ Hospital - S2	3.9	0.05	78.6	0.99	95%
			Isopleth 5	4.3	0.05	79	1.00	95%
			Isopleth 4	15.8	0.20	91	**1.15**	83%
			■ School – S3	22.9	0.29	97.6	**1.24**	77%
			Isopleth 3	25.3	0.32	100	**1.27**	75%
			◆ Daycare – S3	27.2	0.34	101.9	**1.29**	74%
			■ Workplace – S3	27.9	0.35	102.6	**1.30**	73%
			■ Seniors – S3	29.1	0.37	103.8	**1.31**	72%
			■ School – S2	30	0.38	104.7	**1.33**	72%
			◆ Daycare – S2	33.4	0.42	108.1	**1.37**	69%
			■ Workplace – S2	33.5	0.42	108.2	**1.37**	69%
			■ Seniors – S2	36.8	0.47	111.5	**1.41**	67%
			⬟ TWN – S3	40.6	0.51	115.3	**1.46**	65%
			⬟ TWN – S2	46.7	0.59	121.4	**1.54**	62%
			▲ Residents – S3	47.9	0.61	122.6	**1.55**	61%
			Isopleth 2	50.3	0.64	125	**1.58**	60%
			■ Recreation – S3	59.7	0.76	134.4	**1.70**	56%
			▲ Residents – S2	62.7	0.79	137.4	**1.74**	55%
			■ Recreation – S2	75	0.95	149.7	**1.89**	50%
			TRV	79	1.00	153.7	**1.95**	49%
			Isopleth 1	100.3	**1.27**	175	**2.22**	43%

1-Hr Acute TRV (µg/m^3)	Baseline Conc. (µg/m^3)	HQ (Baseline)	Receptor Maxima	Predicted Conc. From Refinery (µg/m^3)	HQ (Refinery-Only)	Cumulative Conc. (µg/m^3)	HQ (Cumulative)	% HQ Attributable to Baseline
Notes: Cumulative Concentration/HQ = Baseline + Refinery Contribution Refinery-only and cumulative HQs presented in **bold** and shaded if >1.0								

The results presented above for the acute health endpoint associated with asthma emergency room visits (TRV = 79 µg/m^3) are interpreted as follows:

- A Target HQ of 1.0 was selected for sensitive receptors as the HHRA assumed that all receptors could potentially receive their theoretical 1-hr NO_2 exposure within the HHRA study area. These receptors include: residents of all ages, seniors in LTC facilities, toddlers and young children in daycare, children and teens in school, adult patients in a hospital, workers, visitors, and TWN members participating in outdoor cultural activities within the HHRA study area.
- Air quality monitoring data from 2017 - 2019 (Scenario 1) shown in **Figure 6-19** indicates that none of the monitoring stations included in the study show HQs greater than 1.0, based on the TRV of 79 µg/m^3. It is important to note that the HQ are similarly high (HQs = 0.93-0.99) for all of the monitoring stations, indicating very consistent maximum 1-hr NO_2 concentrations throughout the study area, regardless of their respective distance from the refinery. This suggests that baseline / non-refinery NO_2 sources are the driver of concentrations and associated NO_2 health risks throughout the study domain.
- Air quality modelling results (Scenarios 2, 3 and 4) corroborate the point above and indicate that baseline (ambient) NO_2 concentrations account for the majority of the cumulative NO_2 health risk within the HHRA study area, contributing 50 to 96% of the HQ for the maximum sensitive receptors. The baseline 1-hour NO_2 concentrations result in an HQ of 0.95.
- HQs for refinery-only contributions ranged from 0.04 for the Scenario 3 maximum hospital receptor to 0.95 for the Scenario 2 maximum recreational receptor (corresponding to modelled refinery contributions of 3.4 to 75 µg/m^3). As shown on **Figure 6-20** through **Figure 6-27**, the zones of highest refinery NO_2 concentrations for Scenarios 2 and 3 are spatially limited and generally do not overlap with the presence of sensitive receptors. Refinery-only HQs for the maximally exposed sensitive receptors are all less than 1.0 (see **Figure 6-20** through **Figure 6-27).** Scenario 4, which represents the most likely future exposure to human receptors, shows a further reduction in the spatial extent of highest modelled refinery NO_2 concentrations (see **Appendix D**, **Figure D-9** to **Figure D-12)**.
- Predicted cumulative 1-hour NO_2 concentrations greater than the TRV of 79 µg/m^3 (corresponding to a predicted refinery contribution of 4.3 µg/m^3) result in a HQ greater than 1.0, and thus may result in the potential for increased asthma emergency room visits for sensitive receptors (see **Table 6-5**). However, as stated above, the majority of the predicted health risk is associated with baseline / non-refinery NO_2 concentrations, which represent a HQ of 0.95 on their own. This significant baseline risk is beyond the control of the refinery and means that the 18% refinery NO_x emissions reduction from Scenario 2 to Scenario 3 does not sufficiently reduce the cumulative HQs for any of the maximally exposed sensitive receptors exceeding under Scenario 2 to below a HQ of 1.0 under Scenario 3.
- Given that predicted cumulative concentrations may result in a HQ greater than 1.0 for many receptors, a statistical evaluation was performed to further refine the probability of risk to sensitive receptors.
  - FOE statistics were generated to determine the number of hours over the full 1-year modelling period (i.e., 8760 hours) that the predicted cumulative NO_2 concentration exceeded the numerical value of the TRV (79 µg/m^3) at the maximally exposed residential receptor location. These results are shown in **Appendix C** for all modelled scenarios.
  - For Scenario 2, the total number of hours that that the predicted cumulative NO_2 concentration exceeded the numerical value of the TRV (79 µg/m^3) at the maximally exposed residential receptor location was 1,288 hours, or 14.7% of the year. The refinery-only predicted cumulative NO_2 concentration exceeded the numerical value of the TRV (79 µg/m^3) for a total of 4 hours, or 0.05% of the year.

  - For Scenario 3, the total number of hours that that the predicted cumulative NO_2 concentration exceeded the numerical value of the TRV (79 µg/m^3) at the maximally exposed residential receptor location was 1,165 hours, or 13.3% of the year. The refinery-only predicted cumulative NO_2 concentration exceeded the numerical value of the TRV (79 µg/m^3) for a total of 2 hours, or 0.02% of the year, and did not exceed the TRV at any sensitive receptor location.
- Conservative assumptions made in the HHRA that may lead to overestimation of risks are further discussed in **Section 6.5**.

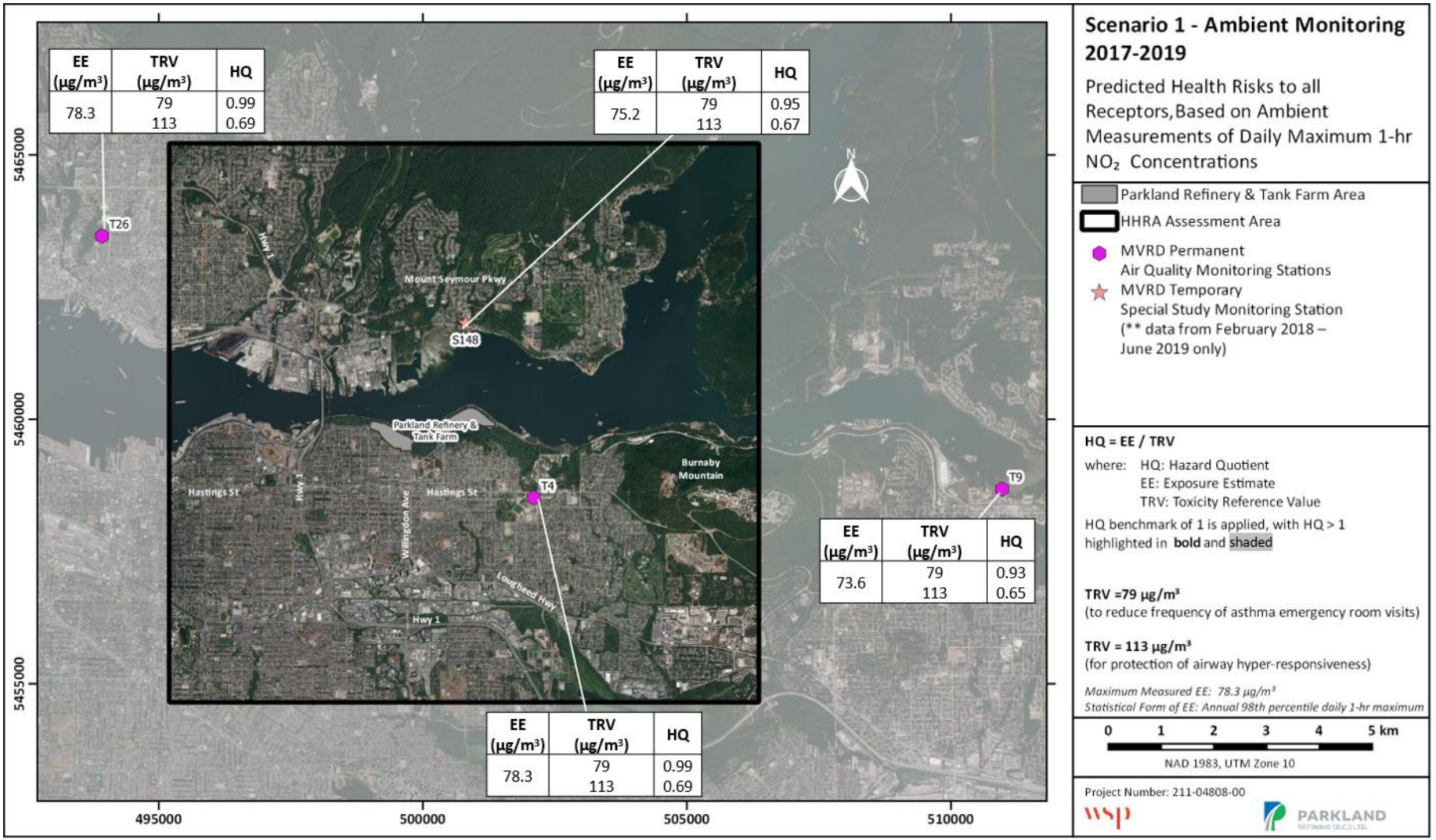


Figure 6-19: Scenario 1 – Predicted Health Risks to All Receptors Based on Ambient Measurements of Daily Maximum 1-hr NO_2 Concentrations

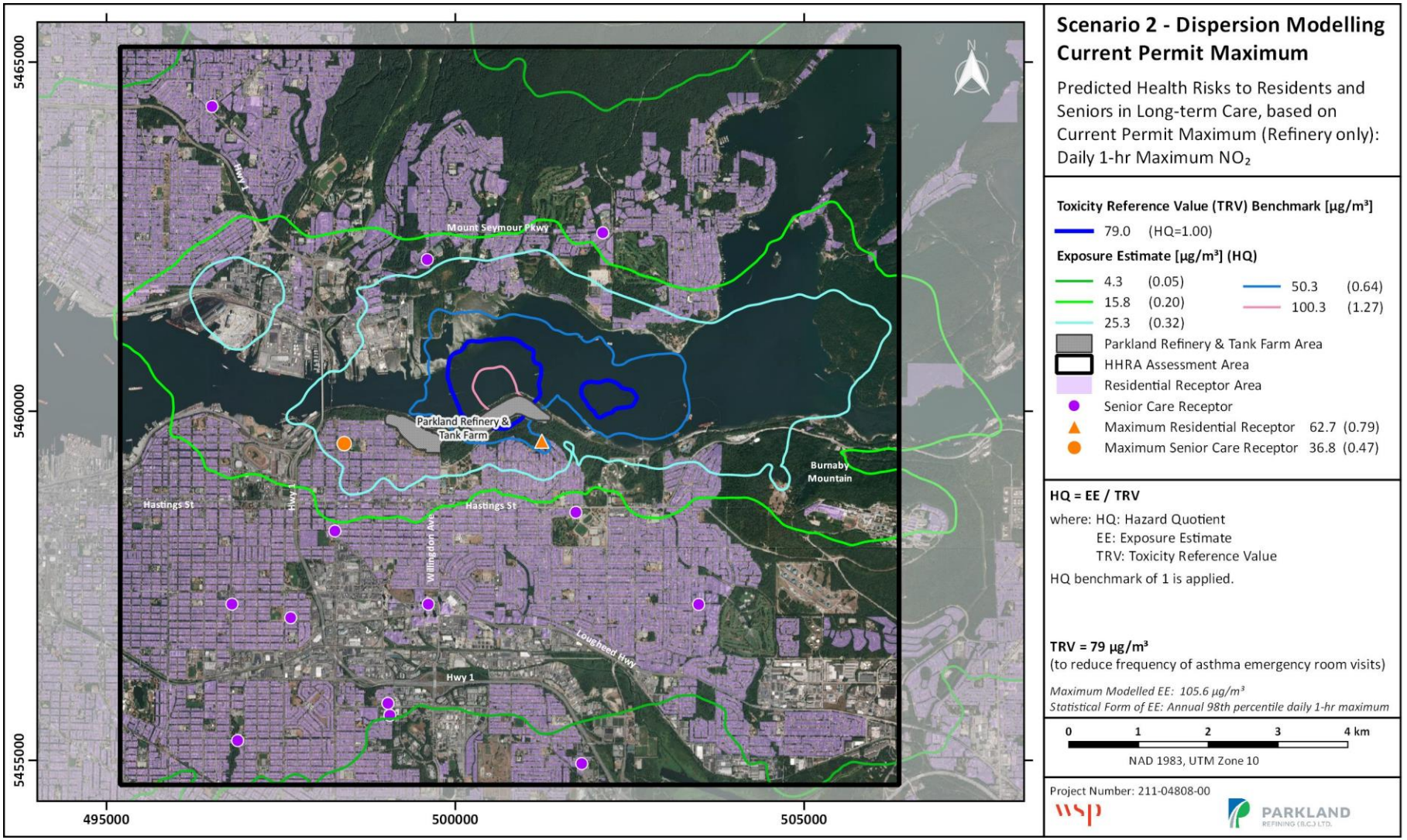


Figure 6-20: Scenario 2 – Predicted Health Risks to Residents and Seniors in Long-term Care Based on Current Permit Maximum (Refinery-Only) Daily 1-hr Maximum NO_2 (TRV=79 ug/m^3)

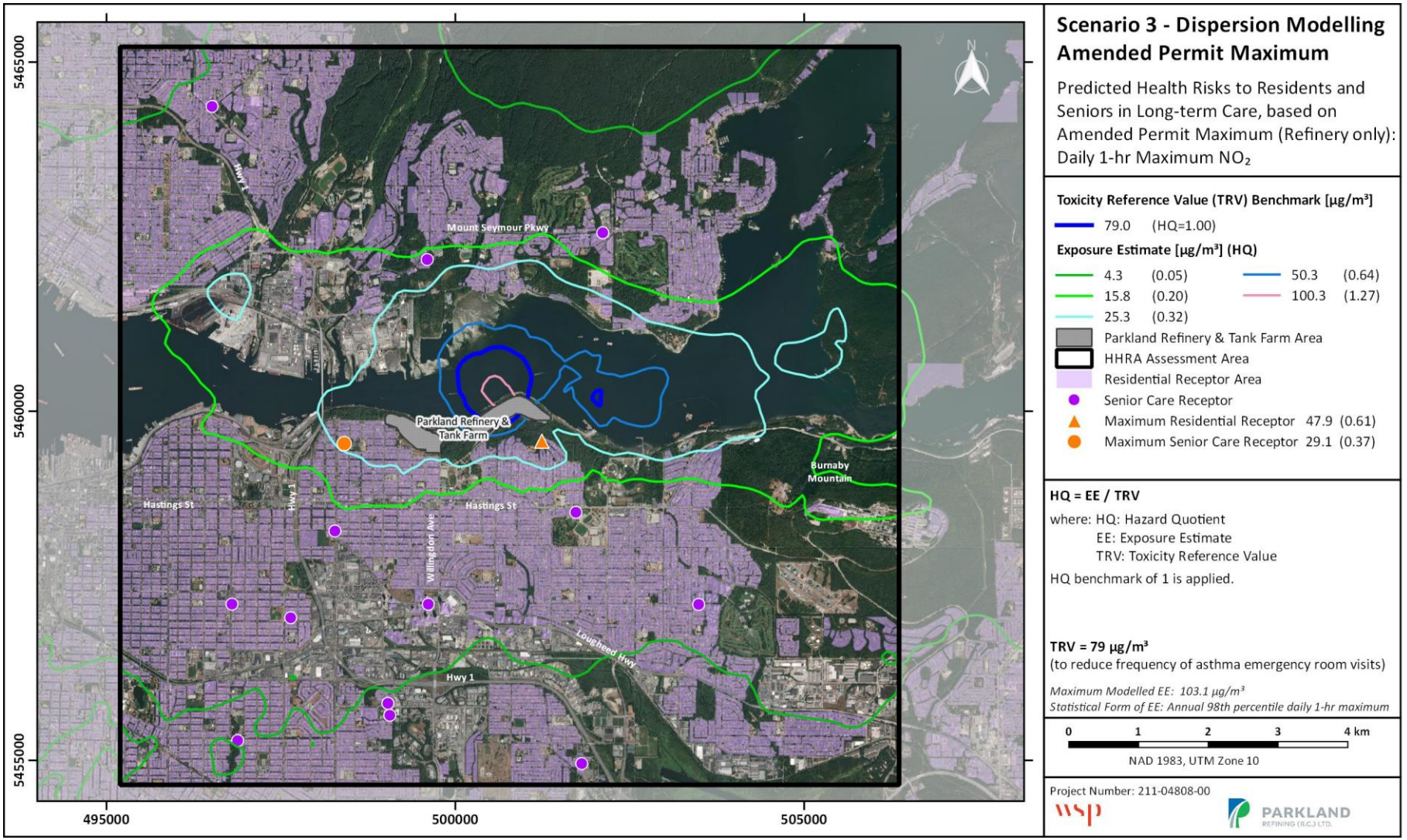


Figure 6-21: Scenario 3 – Predicted Health Risks to Residents and Seniors in Long-term Care Based on Amended Permit Maximum (Refinery-Only) Daily 1-hr Maximum NO_2 (TRV=79 ug/m^3)

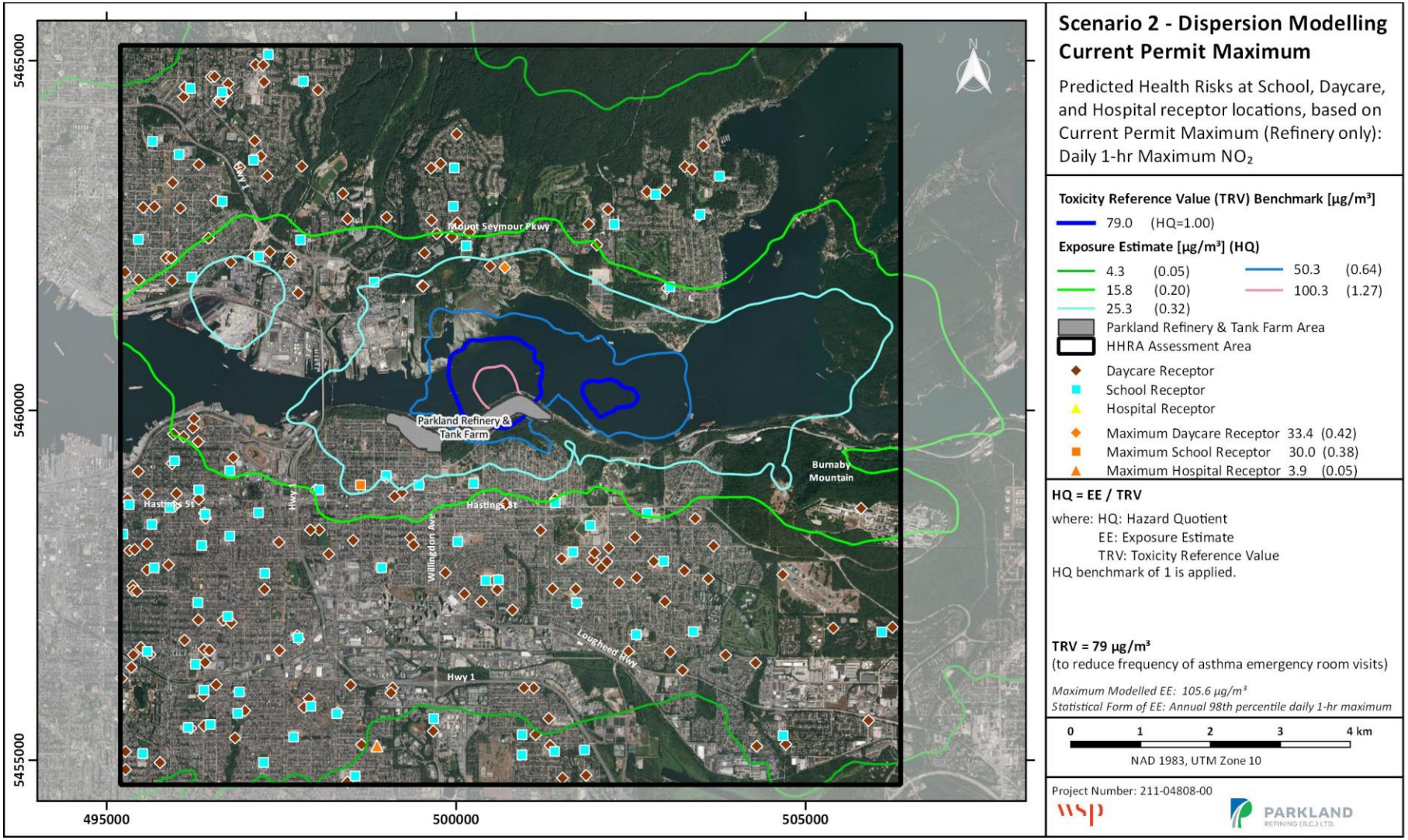


Figure 6-22: Scenario 2 – Predicted Health Risks at School, Daycare, and Hospital Receptor Locations Based on Current Permit Maximum (Refinery-Only) Daily 1-hr Maximum NO_2 (TRV=79 ug/m³)

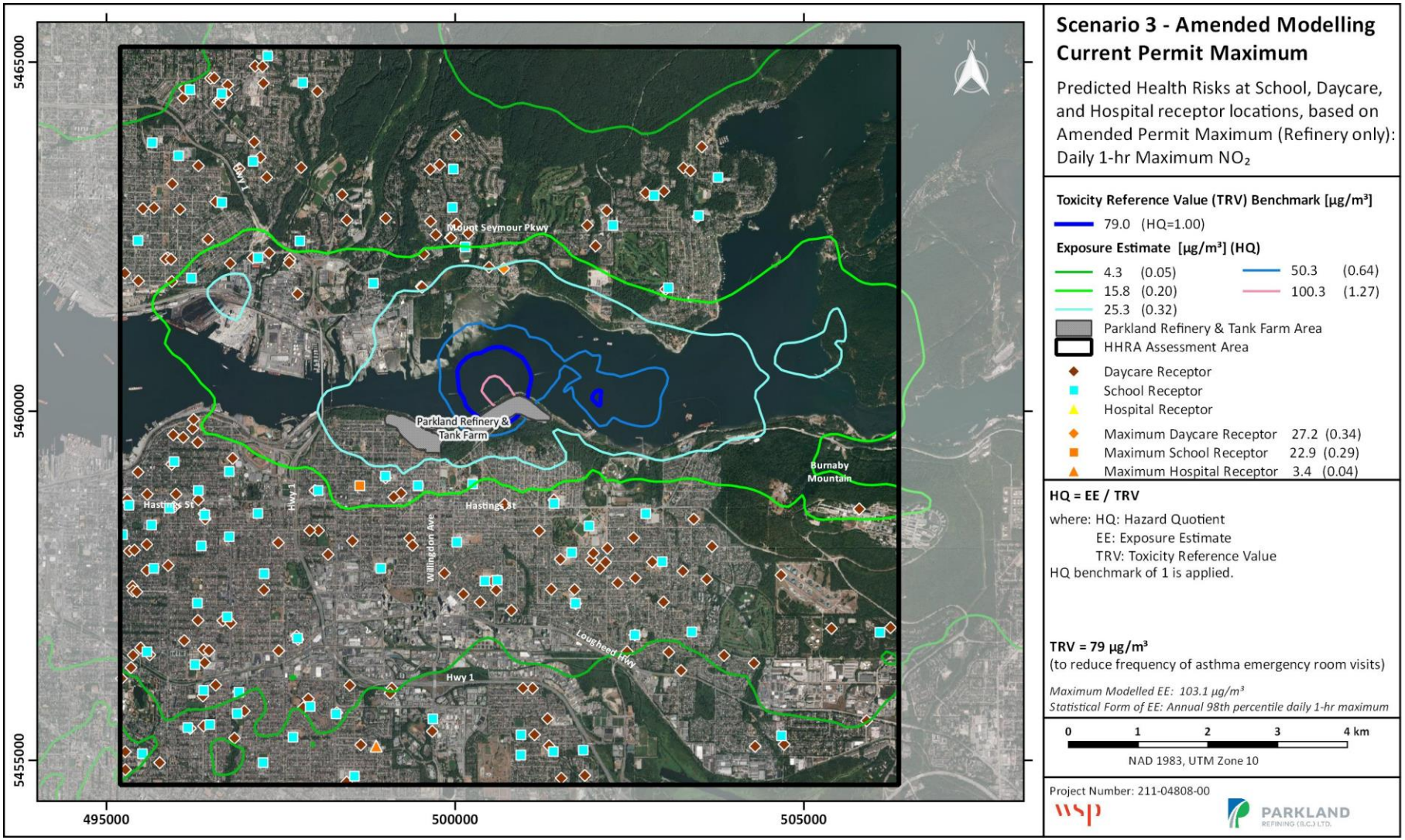


Figure 6-23: Scenario 3 – Predicted Health Risks at School, Daycare, and Hospital Receptor Locations Based on Amended Permit Maximum (Refinery-Only) Daily 1-hr Maximum NO_2 (TRV=79 ug/m³)

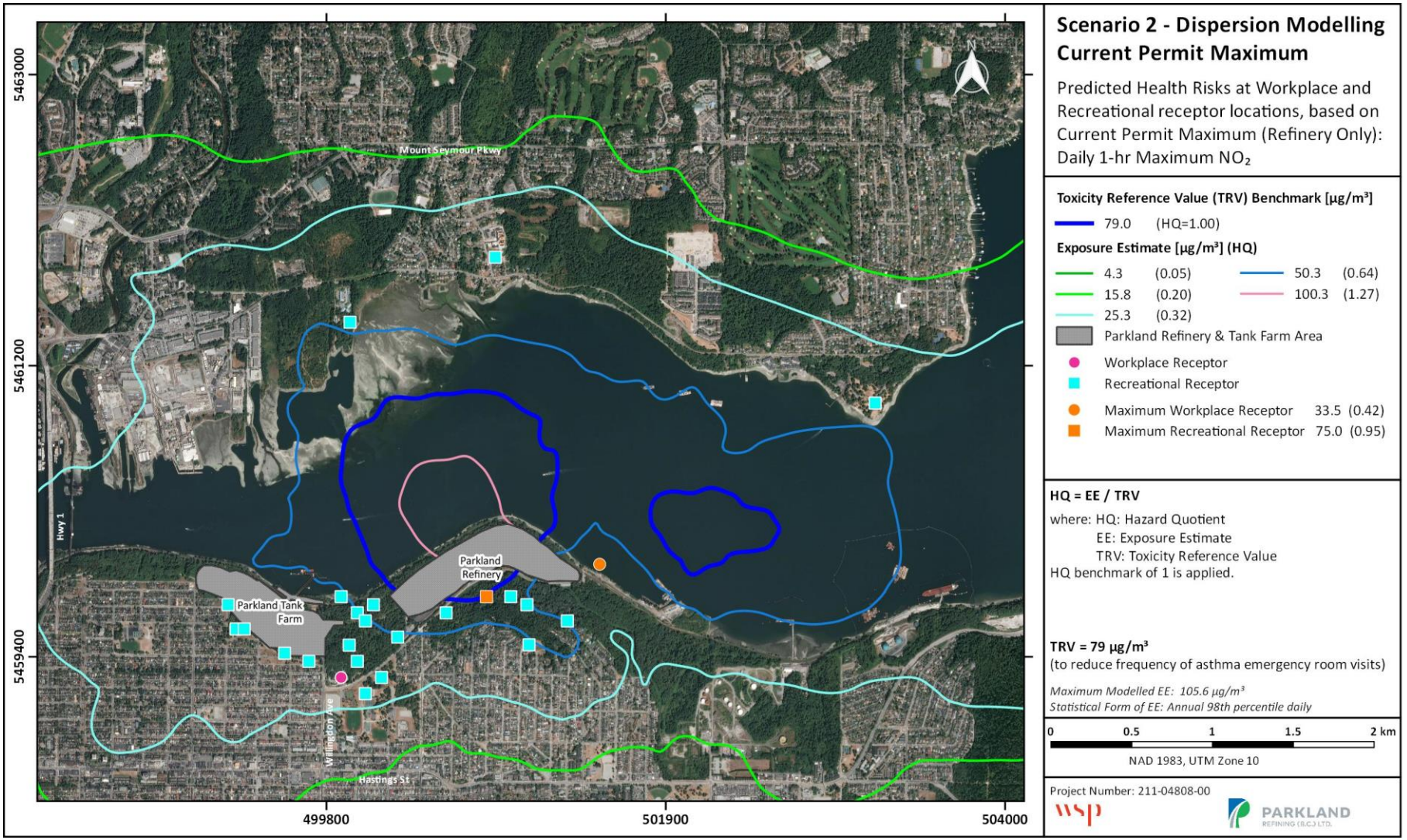


Figure 6-24: Scenario 2 – Predicted Health Risks at Workplace and Recreational Receptor Locations Based on Current Permit Maximum (Refinery-Only) Daily 1-hr Maximum NO_2 (TRV=79 ug/m^3)

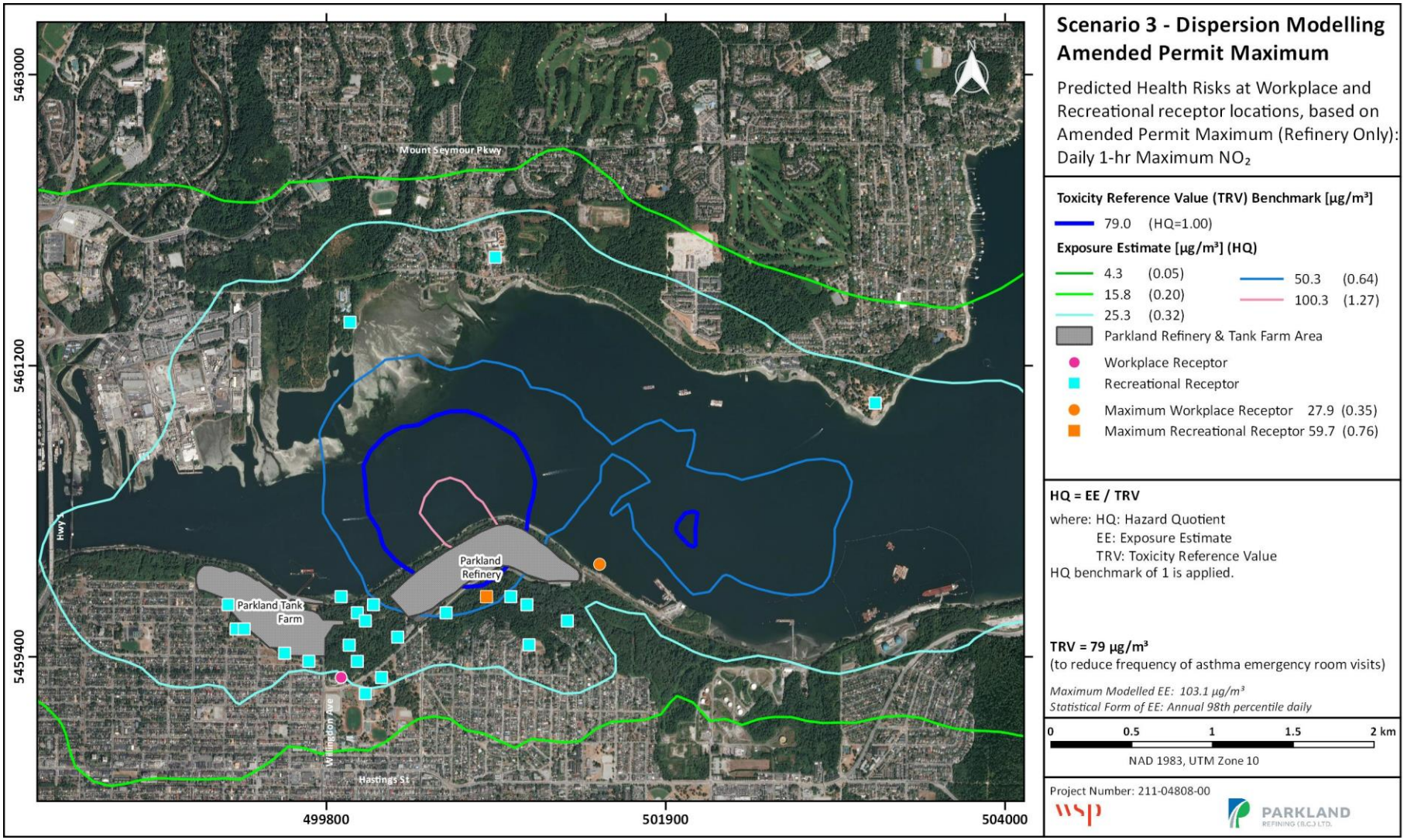


Figure 6-25: Scenario 3 – Predicted Health Risks at Workplace and Recreational Receptor Locations Based on Amended Permit Maximum (Refinery-Only) Daily 1-hr Maximum NO_2 (TRV=79 ug/m^3)

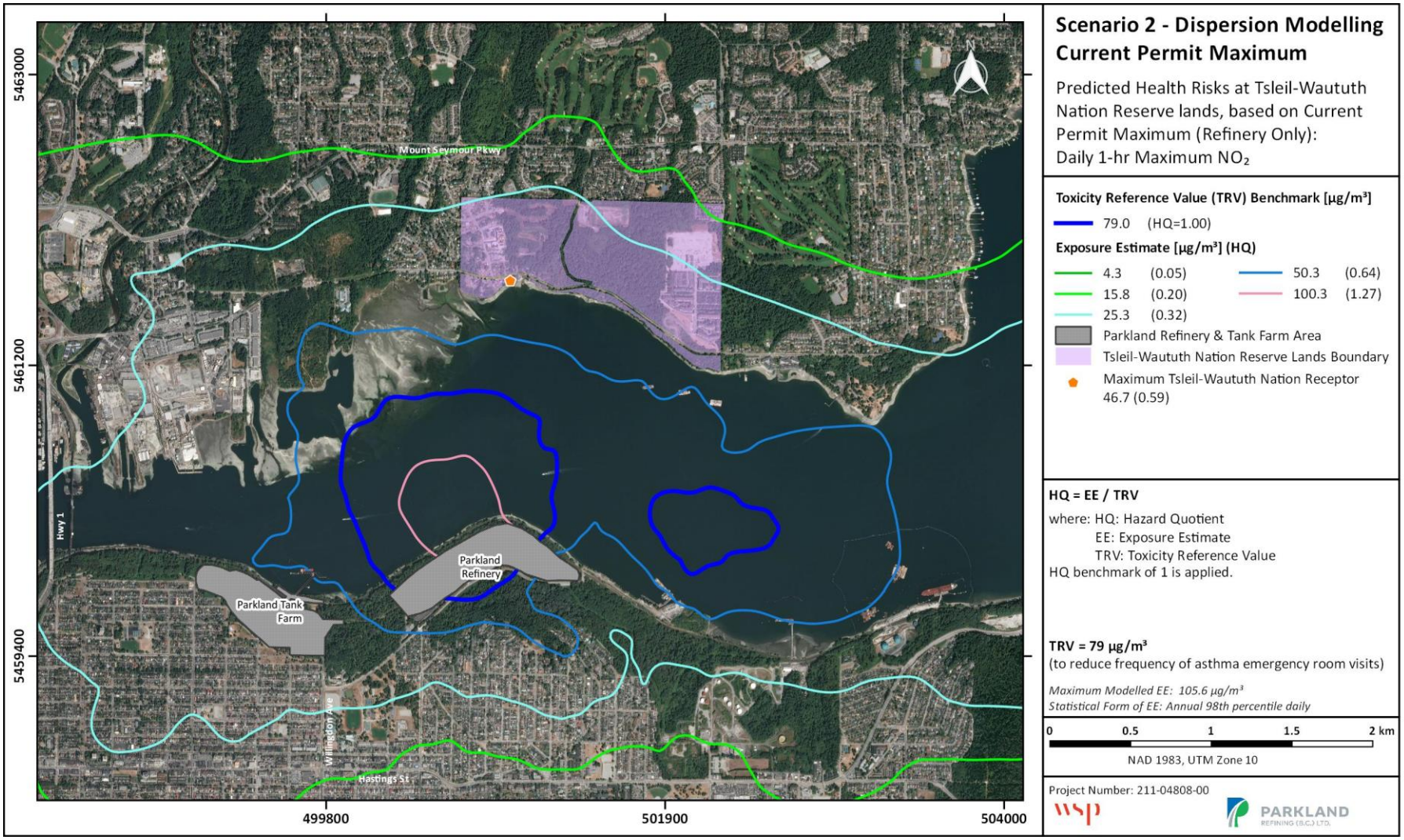


Figure 6-26: Scenario 2 – Predicted Health Risks at TWN Reserve Lands Based on Current Permit Maximum (Refinery-Only) Daily 1-hr Maximum NO_2 (TRV=79 ug/m³)

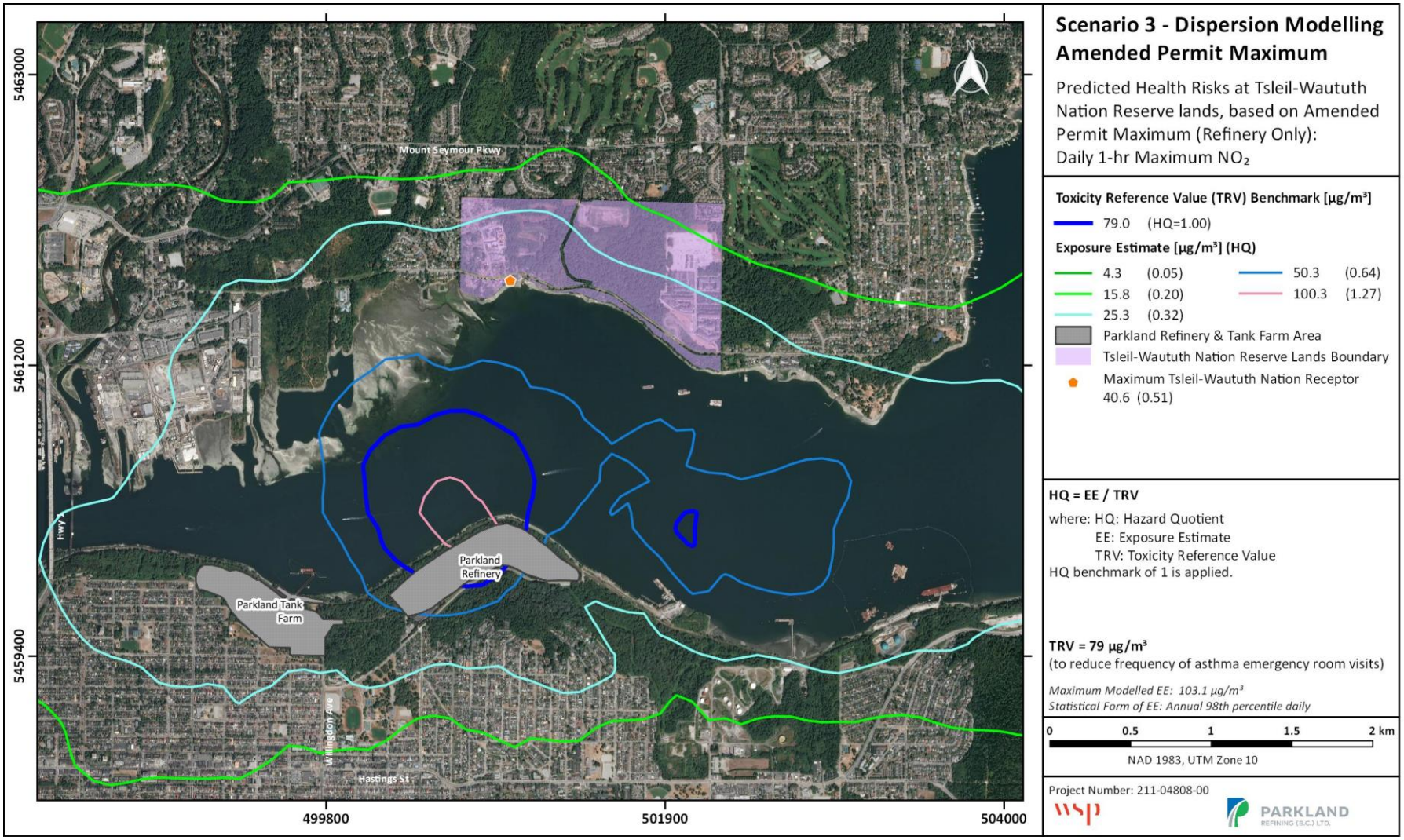


Figure 6-27: Scenario 3 – Predicted Health Risks at TWN Reserve Lands Based on Amended Permit Maximum (Refinery-Only) Daily 1-hr Maximum NO_2 (TRV=79 ug/m³)

6.3.4 NITROGEN DIOXIDE (NO_2) – CHRONIC EXPOSURES

As detailed in **Section 5.3** one TRV for chronic NO_2 exposures has been applied in the risk characterization step of the HHRA. This section presents the results for the chronic TRV of 23 µg/m^3, which was derived to protect against respiratory morbidity. **Table 6-6** through **Table 6-9**, and **Figure 6-28** through **Figure 6-36** present the predicted exposure estimates and HQs associated with respiratory morbidity for annual average NO_2 exposures for each of the identified receptors.

Figure 6-28 represents results for Scenario 1 –Ambient Monitoring 2017-2019, based on air quality measurements at monitoring stations near the refinery. **Figure 6-29** through **Figure 6-36** present results for Scenario 2 – Dispersion Modelling Current Permit Maximum and Scenario 3 - Dispersion Modelling Amended Permit Maximum. Exposure estimates for these scenarios were developed using a dispersion model that predicts ambient air concentrations of COPCs based on emissions from the Parkland refinery.

The coloured shading within **Table 6-6** through **Table 6-9** corresponds to the colour of the applicable concentration / risk isopleths in **Figure 6-29** through **Figure 6-36**. **Table 6-6** through **Table 6-9** also contains risk estimates for the maximally impacted receptors of each type for Scenarios 2 (S2) and 3 (S3) (see "Receptor Maxima" column).

6.3.4.1 LTC FACILITIES AND RESIDENTIAL RECEPTOR LOCATIONS

The results presented in **Table 6-6**, **Figure 6-29**, and **Figure 6-30** below for the predicted long-term health risks associated with maximum annual exposure to NO_2 for residents and seniors in LTC facilities are interpreted as follows:

- A Target HQ of 1.0 was selected for residents and seniors in LTC facilities as the HHRA assumed that these receptors could potentially receive their theoretical annual exposure within the HHRA study area.
- Air quality monitoring data from 2017 – 2019 (Scenario 1) shown in **Figure 6-28** indicates that only the Port Moody (T9) monitoring station, which is located outside of the HHRA study area, presents a HQ greater than 1.0, with a maximum HQ of 1.1. It is important to note that the HQs are similarly high (0.95-0.996) for the other two permanent MVRD stations (T26 and T4), indicating consistent maximum annual average NO_2 concentrations throughout the study area regardless of their respective distance from the refinery. This suggests that baseline / non-refinery NO_2 sources are the driver of concentrations and associated NO_2 health risks throughout the study area. It is interesting to note the significantly lower HQ (0.66) for the special study monitoring location (S148) located immediately across the Burrard Inlet from the refinery.
- Air quality modelling results (Scenarios 2, 3 and 4) corroborate the point above and indicate that cumulative HQs for residents and seniors in LTC are driven by baseline (ambient) NO_2 concentrations. Baseline accounts for more than 88% of the cumulative risk for these receptors long-term exposure to NO_2. See **Figure 6-29** and **Figure 6-30** for a graphical presentation of predicted concentrations applicable to these receptors for Scenarios 2 and 3, respectively.
- HQs for refinery-only contributions ranged from 0.05 for the maximum Scenario 3 senior receptor to 0.08 for the maximum Scenario 2 residential receptor. Spatially, the area predicted to contain modelled refinery concentrations resulting in a cumulative HQ greater than 1.0 is very small and does not overlap with locations of any of the residential receptors for either of Scenario 2 or 3 (**Figure 6-29**, **Figure 6-30**). This is also the case for seniors in LTC facilities. Thus, no risk to these receptors is expected as a result of the predicted maximum annual exposure to NO_2.

6.3.4.2 DAYCARE, SCHOOL, AND HOSPITAL RECEPTOR LOCATIONS

The results presented in **Table 6-7**, **Figure 6-31**, and **Figure 6-32** below for the predicted long-term health risks associated with maximum annual exposure to NO_2 for toddlers and young children in daycare, children and teenagers attending school, and adult hospital patients are interpreted as follows:

- A Target HQ of 0.2 was selected for school, daycare, and hospital receptor locations because the HHRA assumed that receptors may only receive a portion of their theoretical annual exposure within the HHRA study area.

- Air quality modelling results (Scenarios 2, 3 and 4) indicate that baseline (ambient) annual NO_2 concentrations account for more than 88% of the cumulative risk for the toddler and young child's long-term exposure to NO_2 in this exposure scenario, and already exceed a HQ of 0.2 even with no refinery contribution taken into account. The cumulative HQs for the maximum Scenario 2 and 3 daycare receptors was estimated to be 0.26 and 0.25 respectively, indicating a slight increase in the potential for health risks due to long-term exposure to NO_2. Refinery-only HQs for all sensitive receptors were well below the 0.2 threshold. Conservative assumptions made in the HHRA that may overestimate calculated risks are further discussed in **Section 6.5.**
- Air quality modelling results (Scenarios 2, 3 and 4) indicate that baseline (ambient) annual NO_2 concentrations account for more than 88% of the cumulative risk for the school-aged receptor's exposure to NO_2. Cumulative HQs for the maximum Scenario 2 and 3 school receptors were estimated to be 0.20 and 0.19 respectively. Refinery-only HQs were well below the 0.2 threshold.
- Air quality modelling results (Scenarios 2, 3 and 4) indicate that health risks due to annual NO_2 concentrations are not expected for the adult hospital patient given that cumulative HQs were estimated to be well below the Target HQ of 0.2.

6.3.4.3 *WORKPLACE & RECREATIONAL RECEPTOR LOCATIONS*

The results presented in **Table 6-8**, **Figure 6-33**, and **Figure 6-34** below for the predicted long-term health risks associated with maximum annual exposure to NO_2 for adult workers and recreational receptors (all life stages) are interpreted as follows:

- A Target HQ of 0.2 was selected for adult workers and recreational receptors because the HHRA assumed that receptors may only receive a portion of their theoretical annual exposure within the HHRA study area.
- Air quality modelling results (Scenarios 2, 3 and 4) indicate that baseline (ambient) annual NO_2 concentrations account for more than 88% of the cumulative risk for these receptors' long-term exposure to NO_2 in this exposure scenario, and already exceed a HQ of 0.2 even with no refinery contribution taken into account. Refinery-only HQs were all well below the 0.2 threshold. The cumulative HQ for the maximum adult worker receptor was estimated to be 0.23 for both Scenarios 2 and 3, indicating a slight increase in the potential for health risks due to exposure to NO_2 at workplaces close to the refinery.
- Air quality modelling results (Scenarios 2, 3 and 4) indicate that the cumulative HQs for the recreational receptor/visitor (all life stages) were all predicted to be less than the Target HQ of 0.2.

6.3.4.4 *TSLEIL-WAUTUTH RESERVE LANDS*

The results presented in **Table 6-9**, **Figure 6-35**, and **Figure 6-36** below for the predicted long-term health risks associated with maximum annual exposure to NO_2 for persons of all ages participating in outdoor cultural activities at TWN Reserve Lands are interpreted as follows:

- A Target HQ of 0.2 was selected for persons of all ages at TWN Reserve Lands because the HHRA assumed that receptors may only receive a portion of their theoretical annual exposures within the HHRA study area. Chronic residential, daycare, school, and recreational exposures occurring on TWN Reserve Lands are quantified in the previous sections.
- Air quality modelling results (Scenarios 2, 3 and 4) indicate that baseline (ambient) annual NO_2 concentrations account for more than 88% of the cumulative risk for these receptors' long-term exposure to NO_2 in this exposure scenario. Refinery-only HQs were all well below the 0.2 threshold. Cumulative HQs for all life stages were also well below the Target HQ of 0.2, indicating the potential for health risks due to long-term exposure to NO_2 during outdoor cultural activities is negligible at these receptor locations.

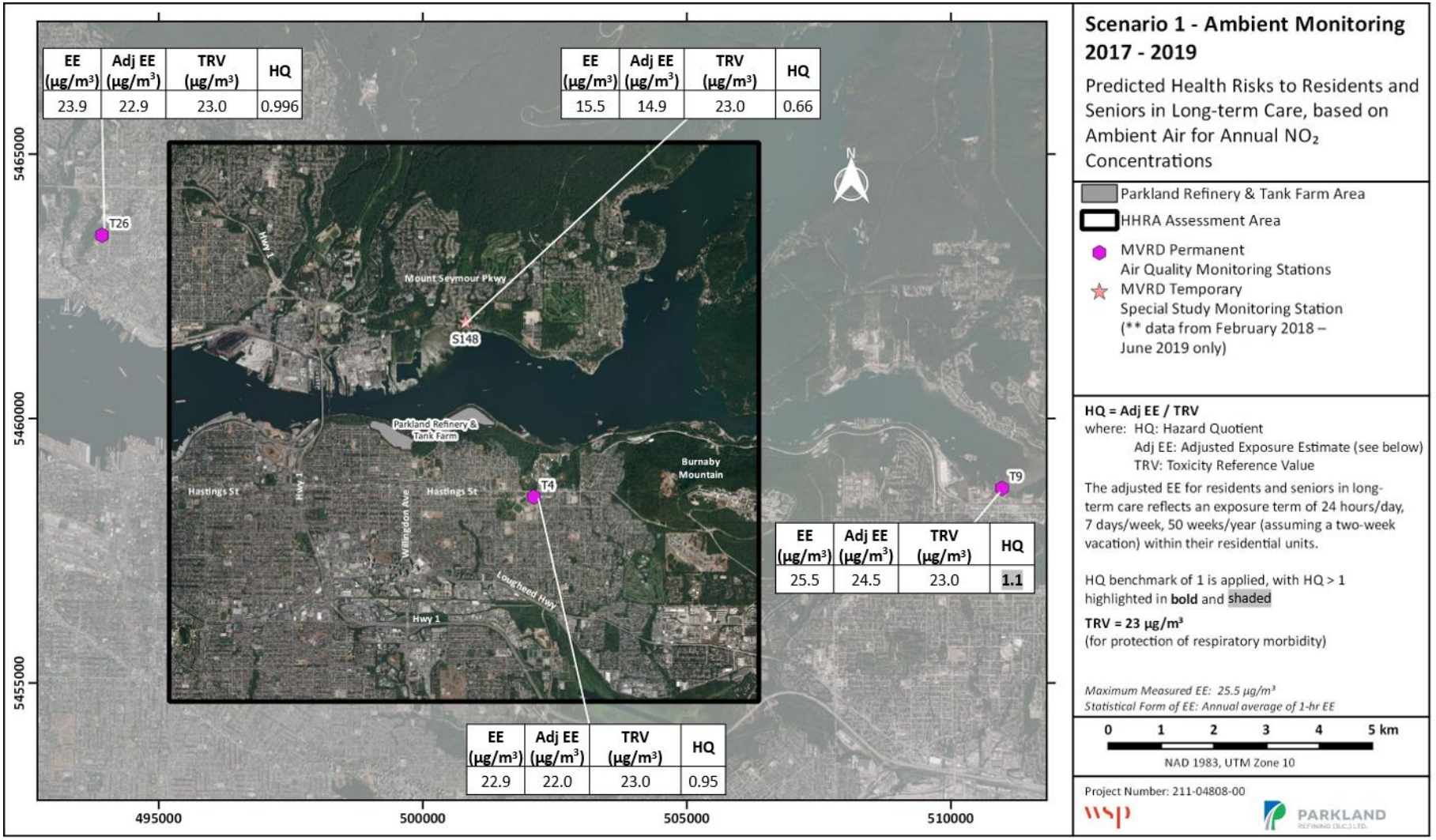


Figure 6-28: Scenario 1 – Predicted Health Risks to Residents and Seniors in Long-Term Care Based on Ambient Air Measurements for Annual NO_2 Concentrations

Table 6-6 Exposure Estimates and Predicted HQs Resulting from Maximum Annual Exposure to NO_2 for Identified Receptors at LTC Facilities and Residential Receptor Locations

Receptor	Annual TRV (µg/m³)	Baseline Conc. (µg/m³)	Adjusted Baseline Conc. (µg/m³)	HQ (Baseline)	Receptor Maxima	Predicted Conc. From Refinery (µg/m³)	Adjusted Refinery Conc. (µg/m³)	HQ (Refinery-Only)	Cumulative Conc. (µg/m³)	Adjusted Cumulative Conc. (µg/m³)	HQ (Cumulative)	% HQ Attributable to Baseline
LTC Home *Adult*	23	22	21	0.91	■ S3	1.2	1.2	0.05	23.2	22.2	0.97	95%
					■ S2 Isopleth 2	1.5	1.4	0.06	23.5	22.5	0.98	94%
					Isopleth 1	3.0	2.9	0.13	25.0	24.0	**1.04**	88%
Resident *Infant* *Toddler* *Child* *Teen* *Adult*	23	22	21	0.91	Isopleth 2	1.5	1.4	0.06	23.5	22.5	0.98	94%
					▲ S3	1.7	1.6	0.07	23.7	22.7	0.99	93%
					▲ S2	1.9	1.8	0.08	23.9	22.9	1.00	92%
					Isopleth 1	3.0	2.9	0.13	25.0	24.0	**1.04**	88%

Notes:
Cumulative Concentration/HQ = Baseline + Refinery Contribution
Target HQ=1.0
HQs presented in **bold** and shaded if Target HQ is exceeded.

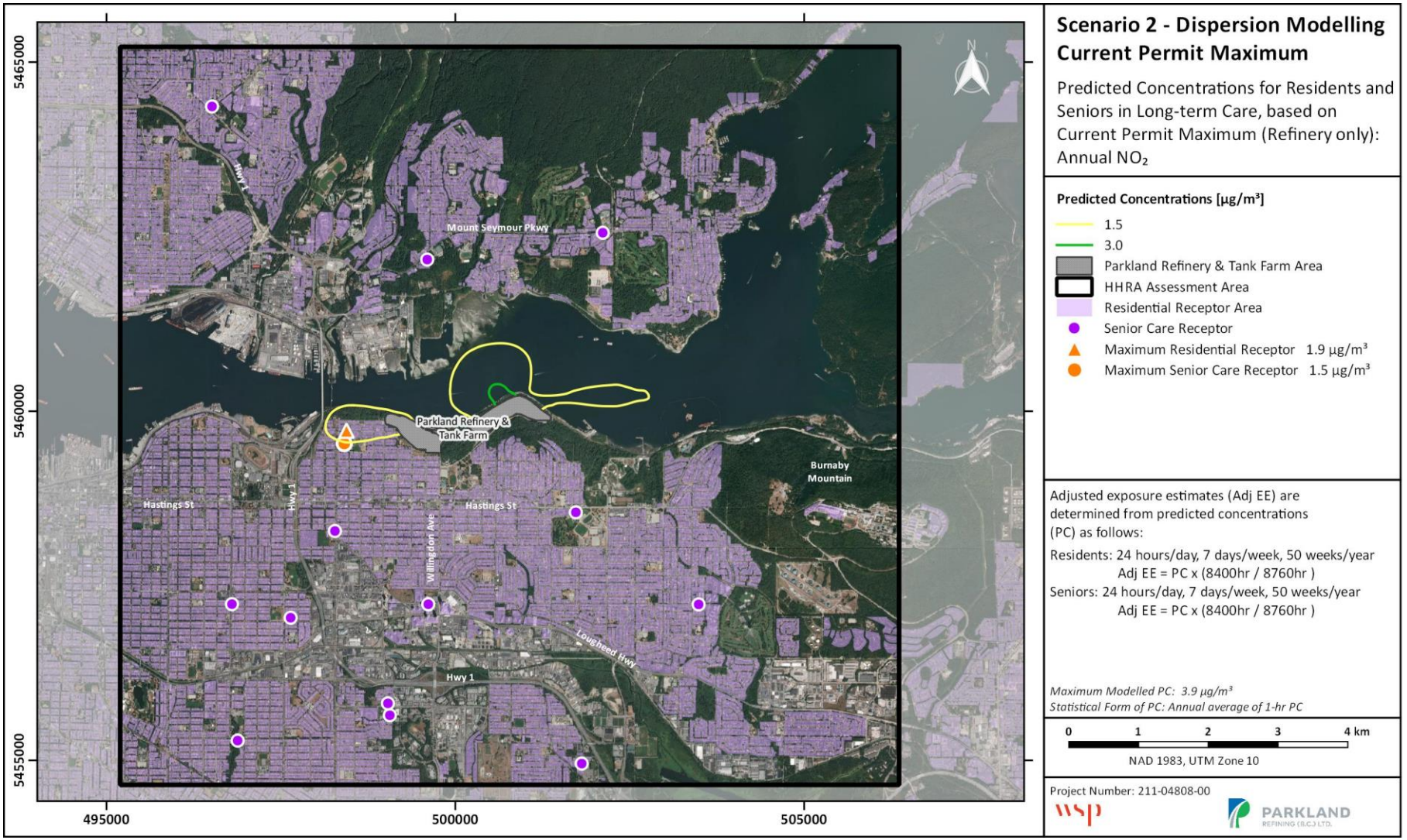


Figure 6-29: Scenario 2 – Predicted Concentrations for Residents and Seniors in Long-term Care Based on Current Permit Maximum (Refinery-Only) Annual NO_2

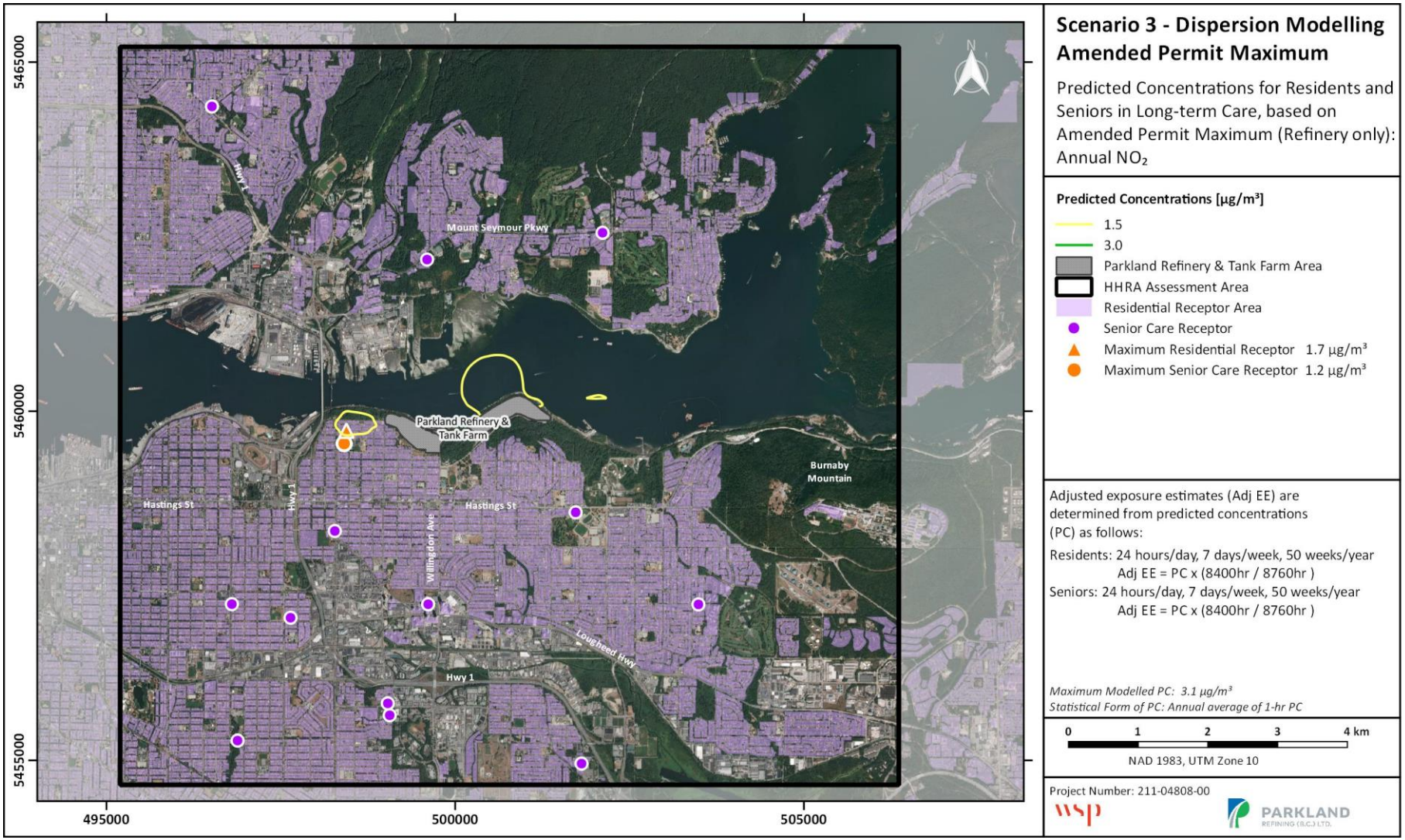


Figure 6-30: Scenario 3 – Predicted Concentrations for Residents and Seniors in Long-term Care Based on Amended Permit Maximum (Refinery-Only) Annual NO_2

Table 6-7 Exposure Estimates and Predicted HQs Resulting from Maximum Annual Exposure to NO_2 for Identified Receptors at Daycare, School, and Hospital Receptor Locations

Receptor	Annual TRV (µg/m³)	Baseline Conc. (µg/m³)	Adjusted Baseline Conc. (µg/m³)	HQ (Baseline)	Receptor Maxima	Predicted Conc. From Refinery (µg/m³)	Adjusted Refinery Conc. (µg/m³)	HQ (Refinery-Only)	Cumulative Conc. (µg/m³)	Adjusted Cumulative Conc. (µg/m³)	HQ (Cumulative)	% HQ Attributable to Baseline
Daycare *Toddler Child*	23	22	5.7	**0.25**	◆ S3	0.8	0.2	0.01	22.8	5.9	**0.25**	96%
					◆ S2	1.0	0.3	0.01	23.0	5.9	**0.26**	96%
					Isopleth 2	1.5	0.4	0.02	23.5	6.0	**0.26**	94%
					Isopleth 1	3.0	0.8	0.03	25.0	6.4	**0.28**	88%
School *Child Teen*	23	22	4.3	0.19	■ S3	0.8	0.2	0.01	22.8	4.5	0.19	97%
					■ S2	1.0	0.2	0.01	23.0	4.5	0.20	96%
					Isopleth 2	1.5	0.3	0.01	23.5	4.6	0.20	94%
					Isopleth 1	3.0	0.6	0.03	25.0	4.9	**0.21**	88%
Hospital *Adult*	23	22	2.1	0.09	▲ S3	0.1	0.01	0.0004	22.1	2.1	0.09	99%
					▲ S2	0.2	0.02	0.001	22.2	2.1	0.09	99%
					Isopleth 2	1.5	0.1	0.006	23.5	2.3	0.10	93%
					Isopleth 1	3.0	0.3	0.01	25.0	2.4	0.10	88%

Notes:
Cumulative Concentration/HQ = Baseline + Refinery Contribution
Target HQ=0.2
HQs presented in **bold** and shaded if Target HQ is exceeded.

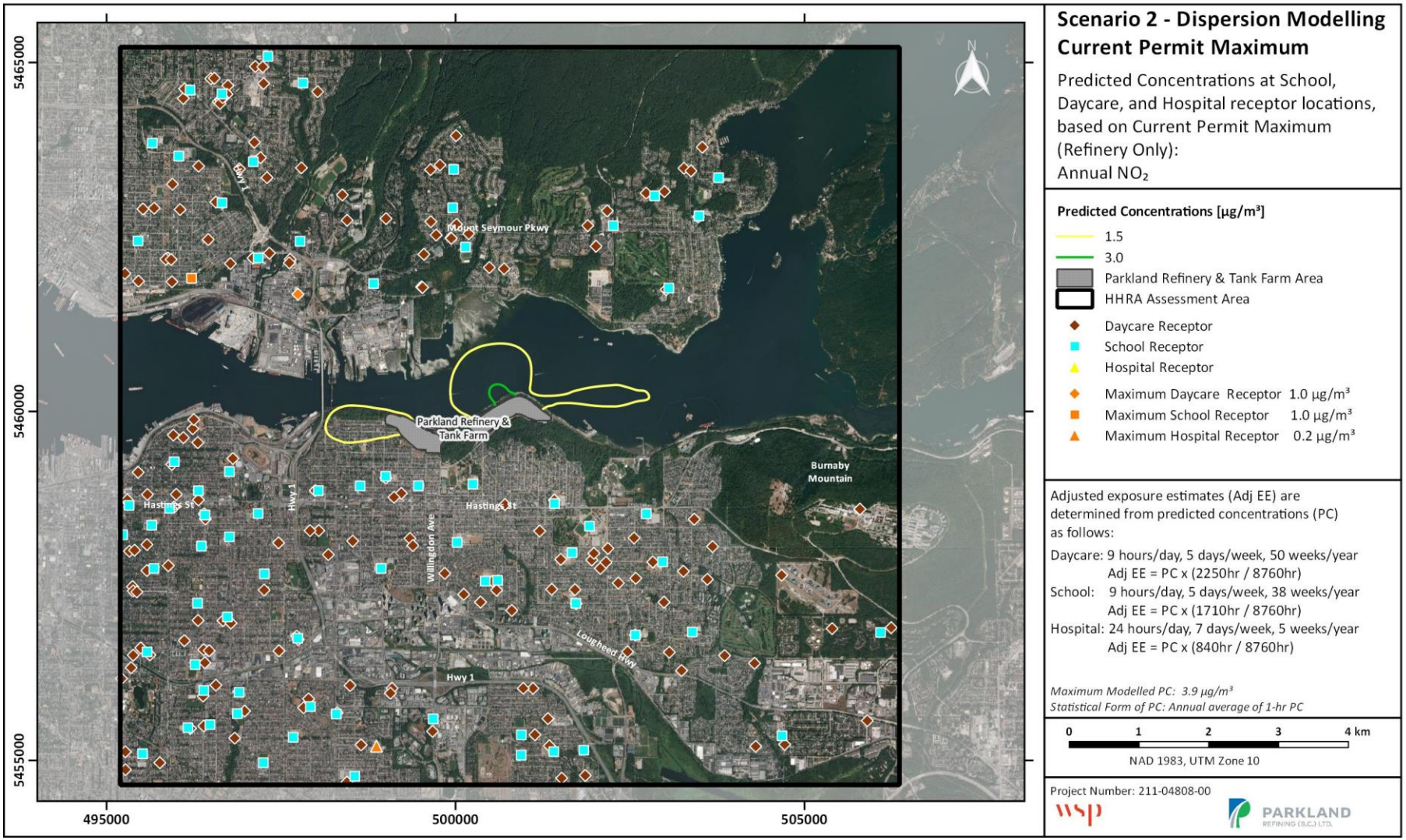


Figure 6-31: Scenario 2 – Predicted Concentrations at School, Daycare, and Hospital Receptor Locations Based on Current Permit Maximum (Refinery-Only) Annual NO_2

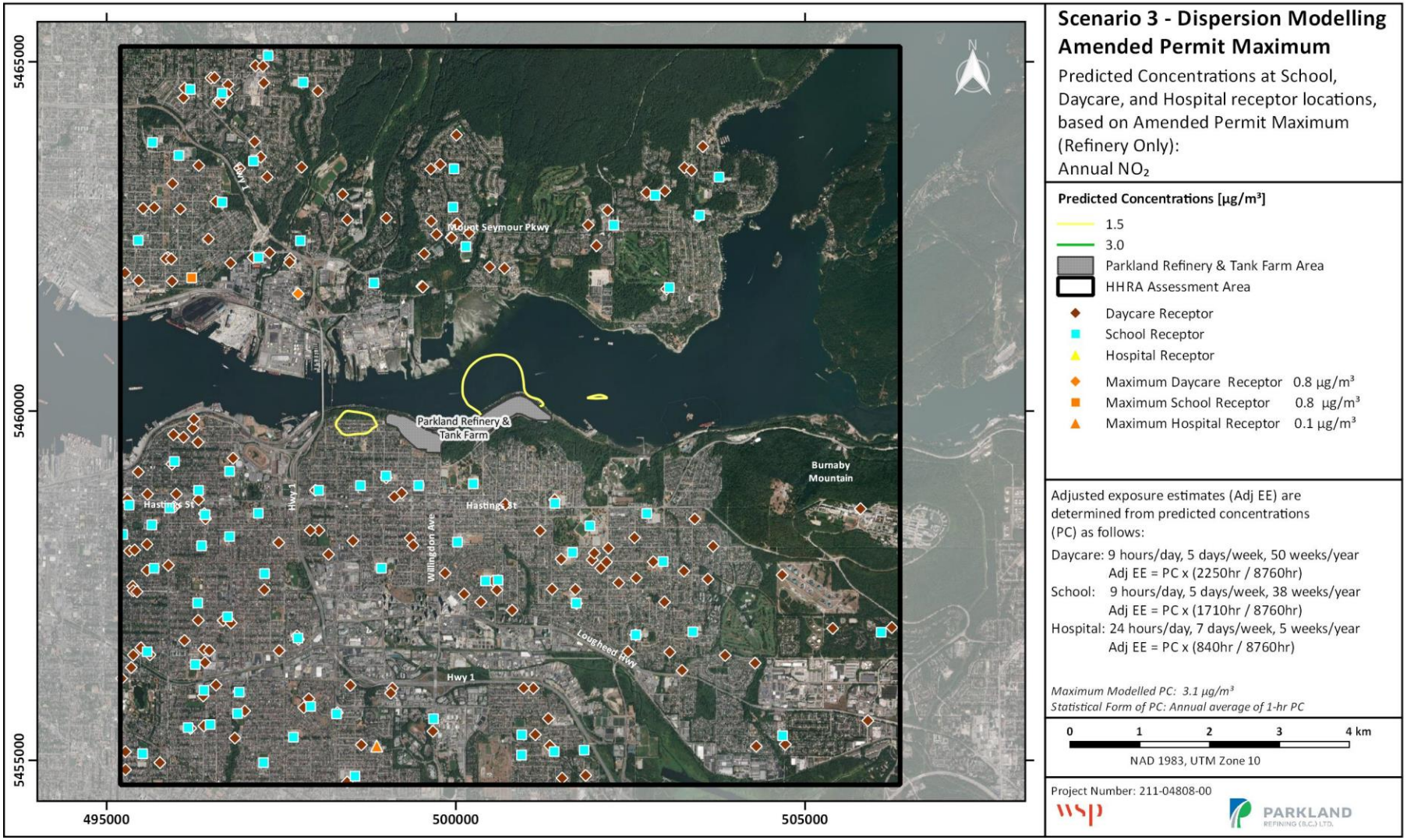


Figure 6-32: Scenario 3 – Predicted Concentrations at School, Daycare, and Hospital Receptor Locations Based on Amended Permit Maximum (Refinery-Only) Annual NO_2

Table 6-8 Exposure Estimates and Predicted HQs Resulting from Maximum Annual Exposure to NO_2 for Workplace & Recreational Receptor Locations

Receptor	Annual TRV (µg/m³)	Baseline Conc. (µg/m³)	Adjusted Baseline Conc. (µg/m³)	HQ (Baseline)	Receptor Maxima	Predicted Conc. From Refinery (µg/m³)	Adjusted Refinery Conc. (µg/m³)	HQ (Refinery-Only)	Cumulative Conc. (µg/m³)	Adjusted Cumulative Conc. (µg/m³)	HQ (Cumulative)	% HQ Attributable to Baseline
Workplace *Adult*	23	22	5.0	**0.22**	S3	0.7	0.2	0.007	22.7	5.2	**0.23**	97%
					S2	0.9	0.2	0.009	22.9	5.2	**0.23**	96%
					Isopleth 2	1.5	0.3	0.015	23.5	5.4	**0.23**	94%
					Isopleth 1	3.0	0.7	0.030	25.0	5.7	**0.25**	88%
Recreational Visitor *Infant* *Toddler* *Child* *Teen* *Adult*	23	22	1.8	0.08	S3	1.4	0.1	0.005	23.4	1.9	0.08	94%
					Isopleth 2	1.5	0.1	0.005	23.5	1.9	0.08	94%
					S2	1.6	0.1	0.006	23.6	1.9	0.08	93%
					Isopleth 1	3.0	0.2	0.010	25.0	2.0	0.09	88%

Notes:
Cumulative Concentration/HQ = Baseline + Refinery-Contribution
Target HQ=0.2
HQs presented in **bold** and shaded if Target HQ is exceeded.

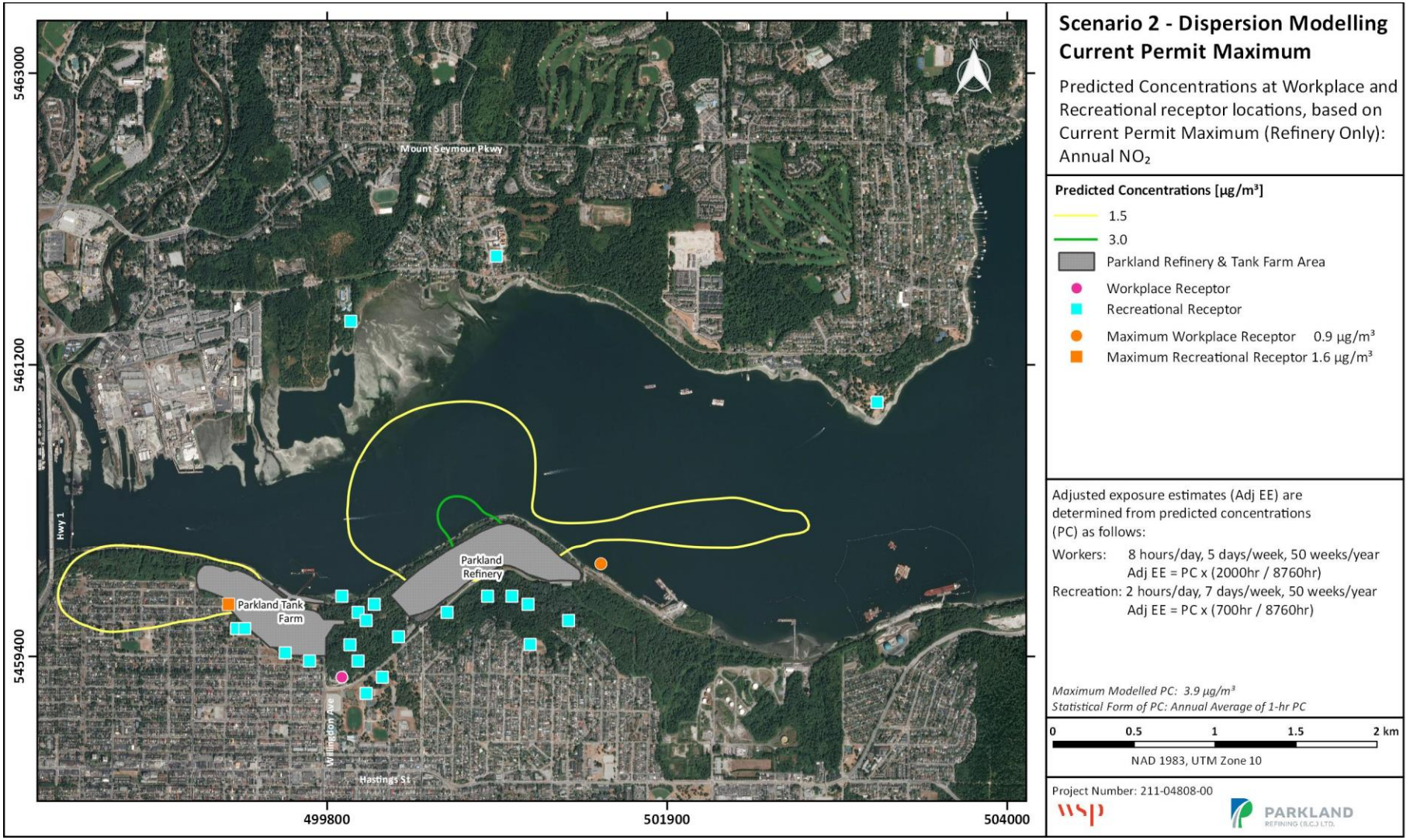


Figure 6-33: Scenario 2 – Predicted Concentrations at Workplace and Recreational Receptor Locations Based on Current Permit Maximum (Refinery-Only) Annual NO_2

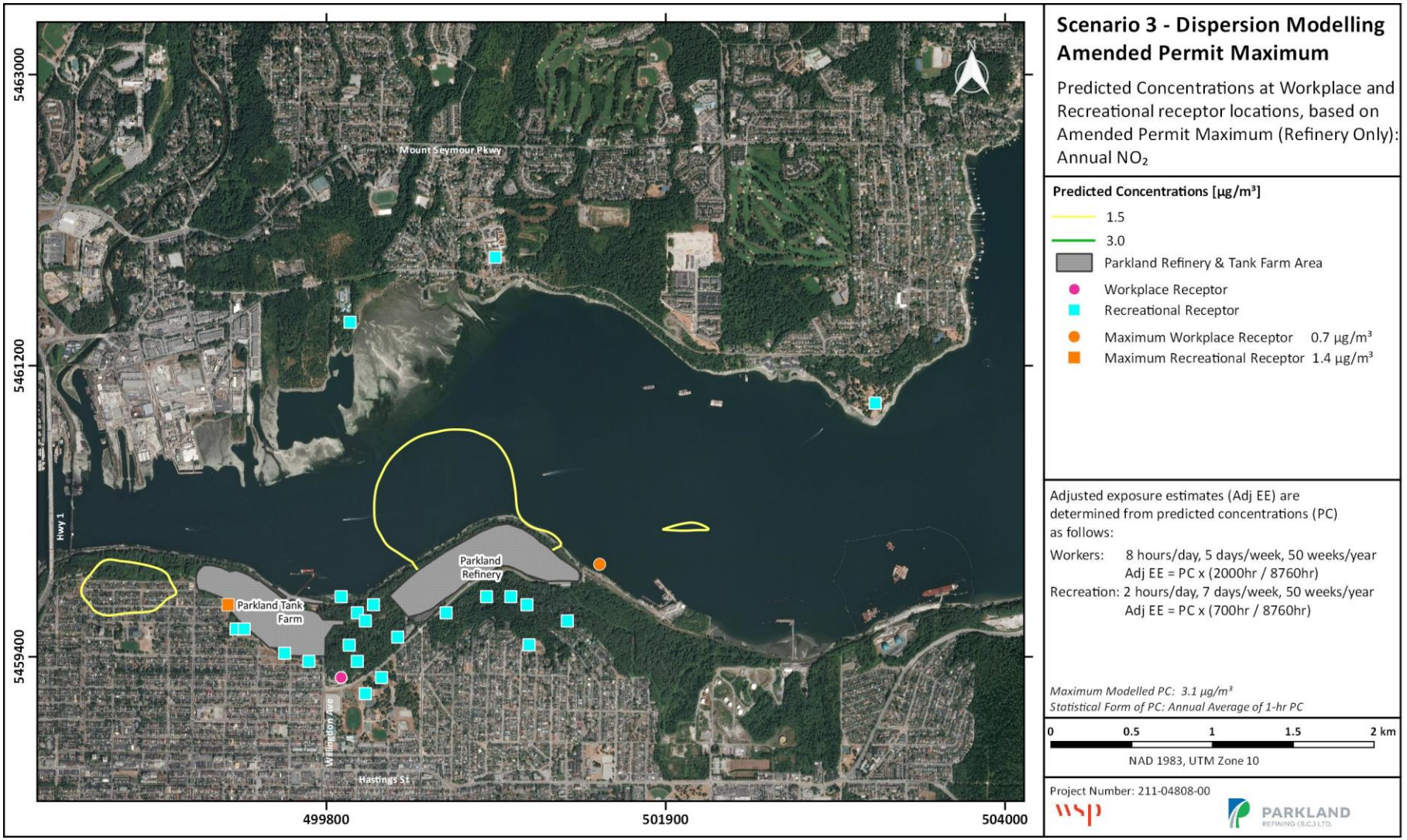


Figure 6-34: Scenario 3 – Predicted Concentrations at Workplace and Recreational Receptor Locations Based on Amended Permit Maximum (Refinery-Only) Annual NO_2

Table 6-9 Exposure Estimates and Predicted HQs Resulting from Maximum Annual Exposure to NO_2 for Identified Receptors at TWN Reserve Lands

Receptor	Annual TRV (µg/m³)	Baseline Conc. (µg/m³)	Adjusted Baseline Conc. (µg/m³)	HQ (Baseline)	Receptor Maxima	Predicted Conc. From Refinery (µg/m³)	Adjusted Refinery Conc. (µg/m³)	HQ (Refinery)	Cumulative Conc. (µg/m³)	Adjusted Cumulative Conc. (µg/m³)	HQ (Cumulative)	% HQ Attributable to Baseline
Reserve Lands *Infant* *Toddler* *Child* *Teen* *Adult*	23	22	1.3	0.06	⬟ S3	0.8	0.05	0.002	22.8	1.35	0.06	96%
					⬟ S2	1.0	0.06	0.003	23	1.37	0.06	96%
					Isopleth 2	1.5	0.09	0.004	23.5	1.4	0.06	94%
					Isopleth 1	3.0	0.18	0.008	25.0	1.5	0.06	88%

Notes:
Cumulative Concentration/HQ = Baseline + Refinery-Contribution
Target HQ=0.2
HQs presented in **bold** and shaded if Target HQ is exceeded.

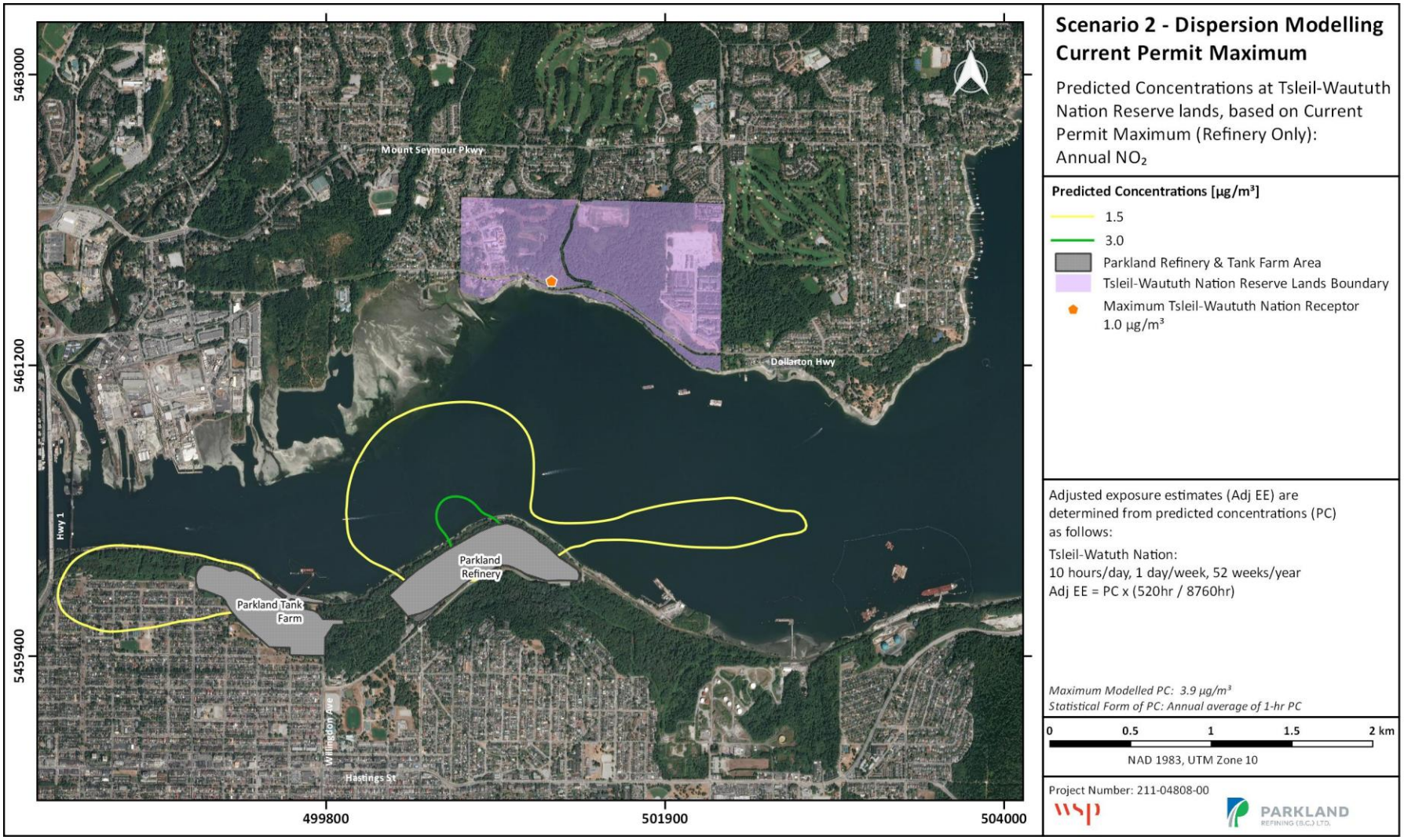


Figure 6-35: Scenario 2 – Predicted Concentrations at TWN Reserve Lands Based on Current Permit Maximum (Refinery-Only) Annual NO_2

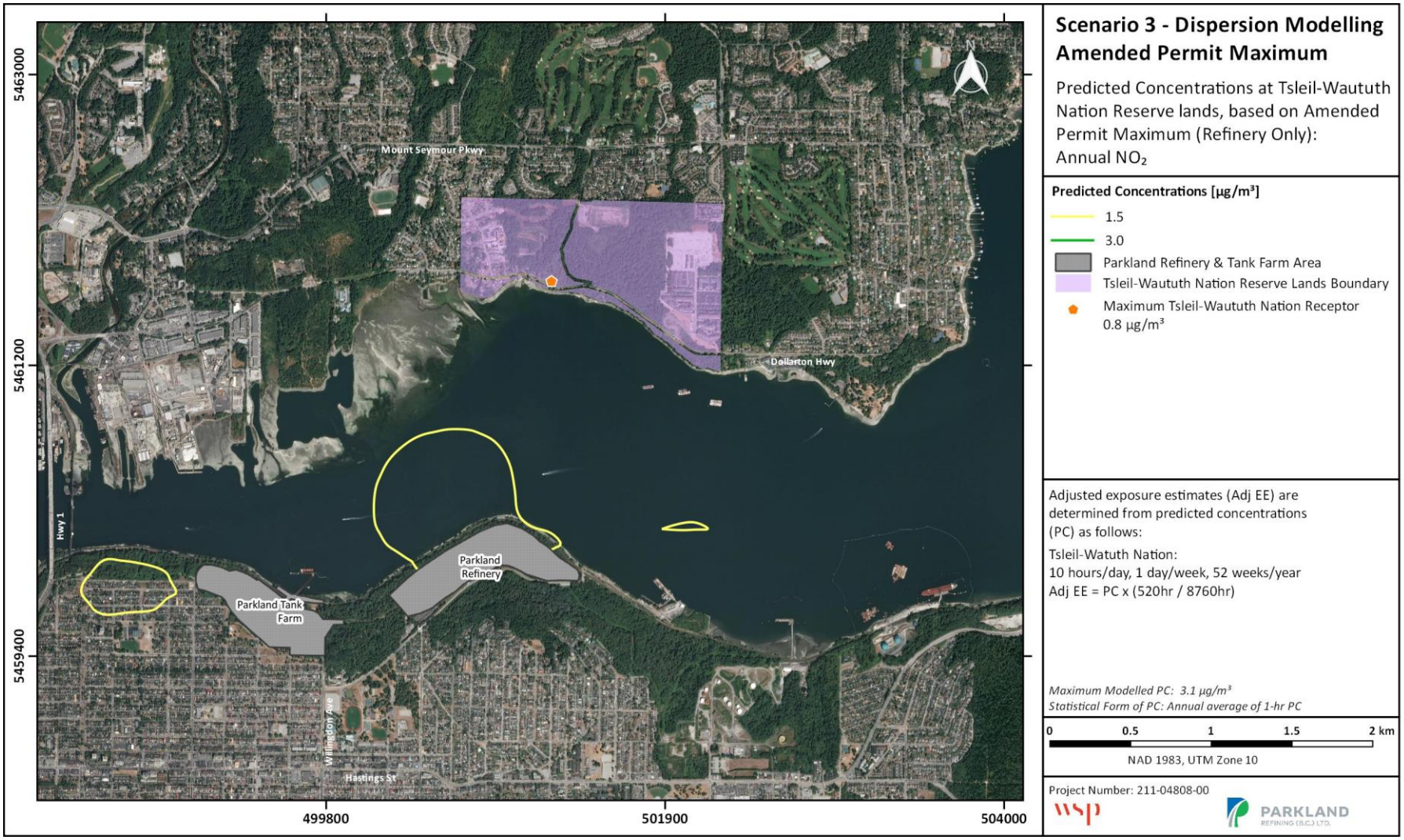


Figure 6-36: Scenario 3 – Predicted Concentrations at TWN Reserve Lands Based on Amended Permit Maximum (Refinery-Only) Annual NO_2

6.3.5 FINE PARTICULATE MATTER ($PM_{2.5}$) – ACUTE EXPOSURES

As detailed in **Section 5.3** one TRV (25 µg/m^3) for acute $PM_{2.5}$ exposures has been applied in the risk characterization step of the HHRA. **Table 6-10** and **Figure 6-37** through **Figure 6-45** present the predicted exposure estimates and HQs for 24-hour $PM_{2.5}$ exposures for each of the identified receptors associated with excess morbidity or mortality.

Figure 6-37 presents results for Scenario 1 – Ambient Monitoring 2017-2019, based on air quality measurements at monitoring stations near the refinery. **Figure 6-38** through **Figure 6-45** present results for Scenario 2 - Dispersion Modelling Current Permit Maximum and Scenario 3 - Dispersion Modelling Amended Permit Maximum. Exposure estimates for these scenarios were developed using a dispersion model that predicts ambient air concentrations of COPCs based on emissions from the Parkland refinery.

The coloured shading within **Table 6-10** corresponds to the colour of the applicable concentration / risk isopleths in **Figure 6-38** through **Figure 6-45**. **Table 6-10** also contains risk estimates for the maximally impacted receptors of each type for Scenarios 2 (S2) and 3 (S3) (see "Receptor Maxima" column).

Table 6-10 Predicted Health Risks Associated with Excess Morbidity or Mortality Following 24-hour Exposure to $PM_{2.5}$ for Identified Receptors

24-Hr Acute TRV (µg/m^3)	Baseline Conc. (µg/m^3)	HQ (Baseline)	Receptor Maxima	Predicted Conc. From Refinery (µg/m^3)	HQ (Refinery-Only)	Cumulative Conc. (µg/m^3)	HQ (Cumulative)	% HQ Attributable to Baseline
25	11.7	0.47	Hospital - S3	0.4	0.02	12.1	0.48	97%
			Hospital - S2	0.6	0.02	12.3	0.49	95%
			Seniors - S3	1.7	0.07	13.4	0.54	87%
			School - S3	2.1	0.08	13.8	0.55	85%
			Seniors - S2	2.2	0.09	13.9	0.56	84%
			Daycare - S3	2.2	0.09	13.9	0.56	84%
			Isopleth 2	2.3	0.09	14	0.56	84%
			School - S2	2.7	0.11	14.4	0.58	81%
			Daycare - S2	2.9	0.12	14.6	0.58	80%
			TWN - S3	3.2	0.13	14.9	0.60	79%
			Residents - S3	3.8	0.15	15.5	0.62	75%
			TWN - S2	4.1	0.16	15.8	0.63	74%
			Workplace - S3	4.1	0.16	15.8	0.63	74%
			Recreation - S3	4.5	0.18	16.2	0.66	72%
			Residents - S2	4.9	0.20	16.6	0.66	70%
			Isopleth 1	5	0.20	16.7	0.67	70%
			Workplace - S2	5.2	0.21	16.9	0.68	69%
			Recreation - S2	5.7	0.23	17.4	0.70	67%

Notes:
Cumulative Concentration/HQ = Baseline + Refinery Contribution
Refinery-only and cumulative HQs presented in **bold** and shaded if >1.0

The results presented above for the acute health endpoint associated with excess morbidity or mortality (TRV = 25 µg/m^3) are interpreted as follows:

- A Target HQ of 1.0 was selected for sensitive receptors as the HHRA assumed that all receptors could potentially receive their 24-hour $PM_{2.5}$ exposure within the HHRA study area. These receptors include: residents of all ages, seniors in LTC facilities, toddlers and young children in daycare, children and teens in school, adult patients in a hospital facility, workers, visitors, and TWN members participating in outdoor cultural activities within the HHRA study area.
- Air quality monitoring data from 2017 – 2019 (Scenario 1) shown in **Figure 6-37** indicates that none of the monitoring stations included in the study area shows a HQ greater than 1.0, based on the TRV of 25 µg/m^3. It is important to note that the HQ are in a similar range for all of the monitoring stations, indicating very consistent maximum 24-hr $PM_{2.5}$ concentrations throughout the study area, regardless of their respective distance from the refinery. This suggests that baseline / non-refinery $PM_{2.5}$ sources are the driver of concentrations and associated $PM_{2.5}$ health risks throughout the study area.
- Air quality modelling results (Scenarios 2, 3 and 4) corroborate the point above and indicate that baseline (ambient) $PM_{2.5}$ concentrations account for the majority of the cumulative $PM_{2.5}$ health risk within the HHRA study, ranging from 67% for the Scenario 2 maximum residential receptor to 97% for the Scenario 3 maximum hospital receptor. The baseline 24-hour $PM_{2.5}$ concentrations result in a HQ of 0.47.
- HQs for refinery-only contributions ranged from 0.02 for the Scenario 3 maximum hospital receptor to 0.23 for the Scenario 2 maximum residential receptor (corresponding to modelled concentrations of 0.4 to 5.7 µg/m^3). As shown on **Figure 6-38** through **Figure 6-45**, the highest predicted 24-hr $PM_{2.5}$ concentrations are spatially limited and generally do not overlap with the presence of sensitive receptors. Cumulative HQs presented in **Table 6-10** range from 0.48 to 0.7. Given that none of the cumulative HQs exceeded 1.0, no elevated risks to sensitive receptors are anticipated as a result of exposure to maximum predicted 24-hour $PM_{2.5}$ concentrations.

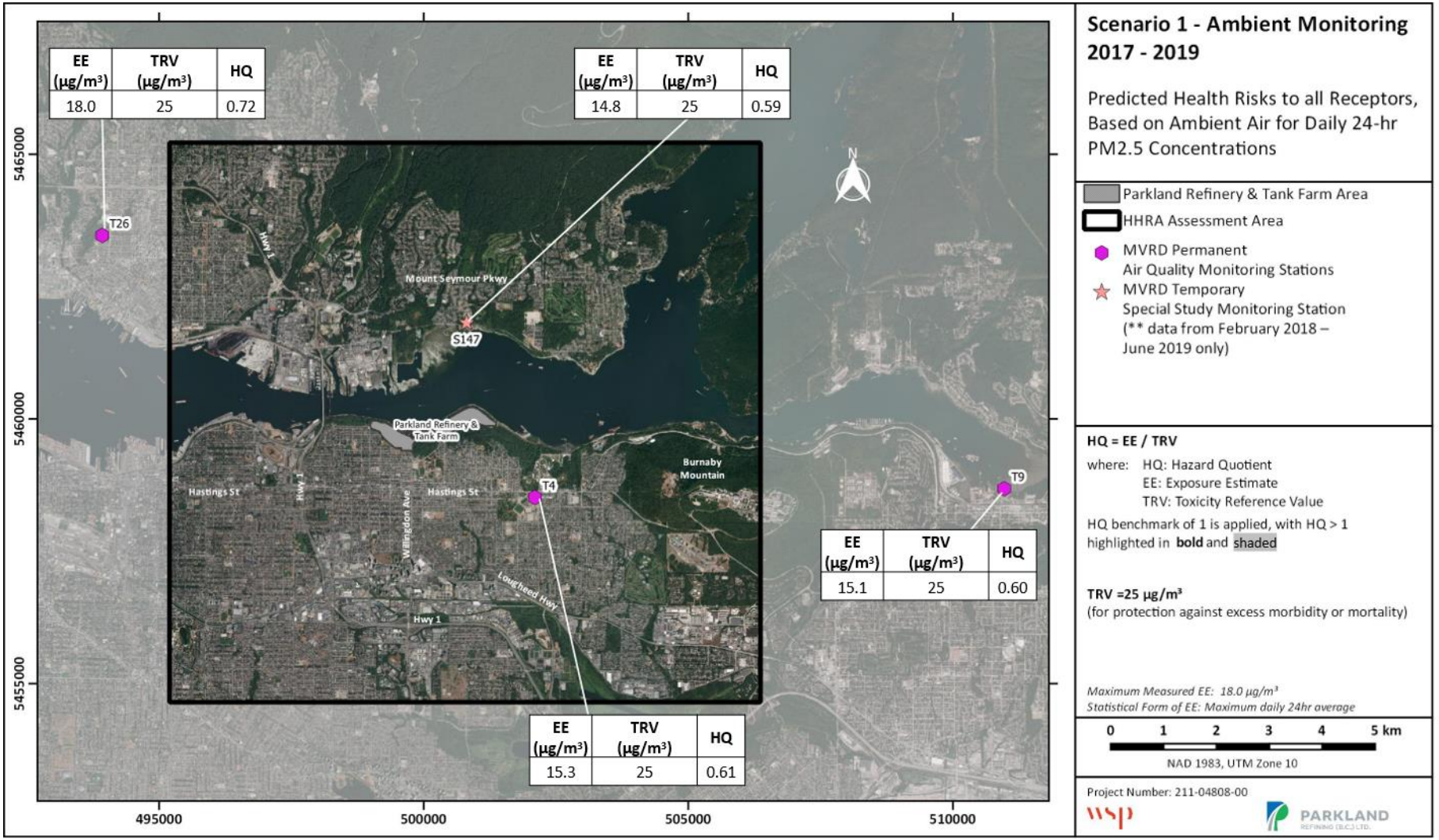


Figure 6-37 Scenario 1 – Predicted Health Risks to All Receptors Based on Ambient Air Measurements of 24-hr $PM_{2.5}$ Concentrations

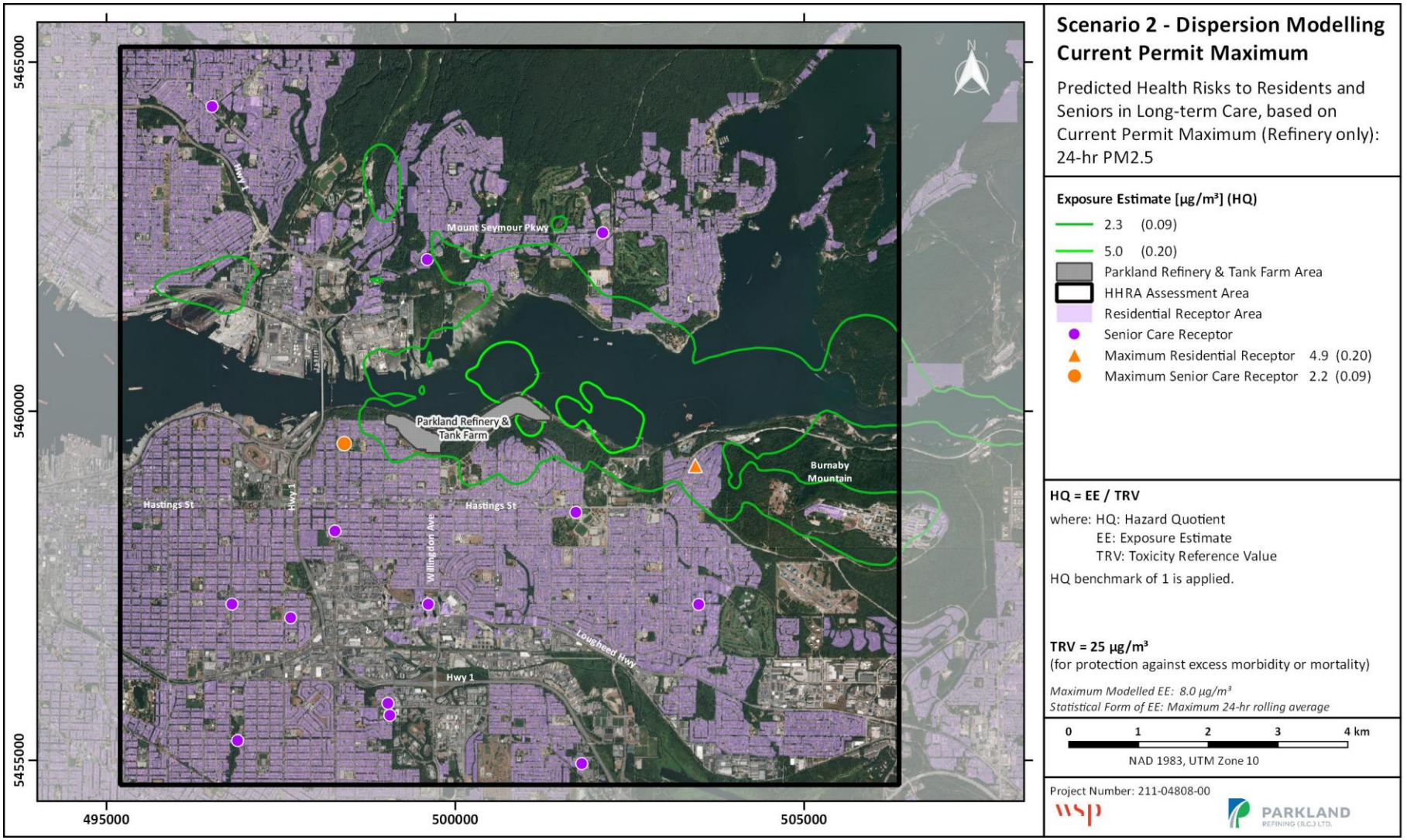


Figure 6-38: Scenario 2 – Predicted Health Risks to Residents and Seniors in Long-term Care Based on Current Permit Maximum (Refinery-Only) 24-hr $PM_{2.5}$

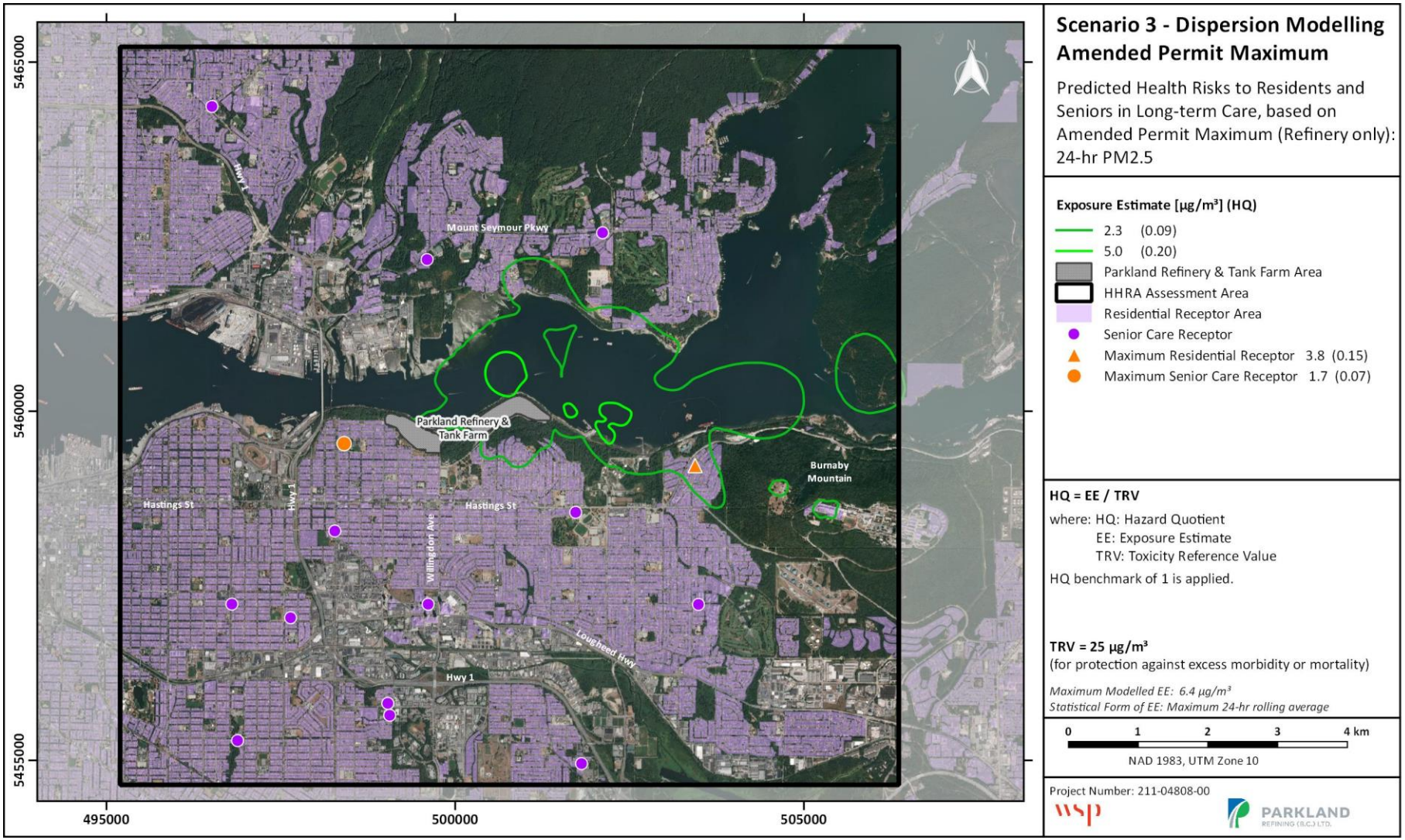


Figure 6-39: Scenario 3 – Predicted Health Risks to Residents and Seniors in Long-term Care Based on Amended Permit Maximum (Refinery-Only) 24-hr $PM_{2.5}$

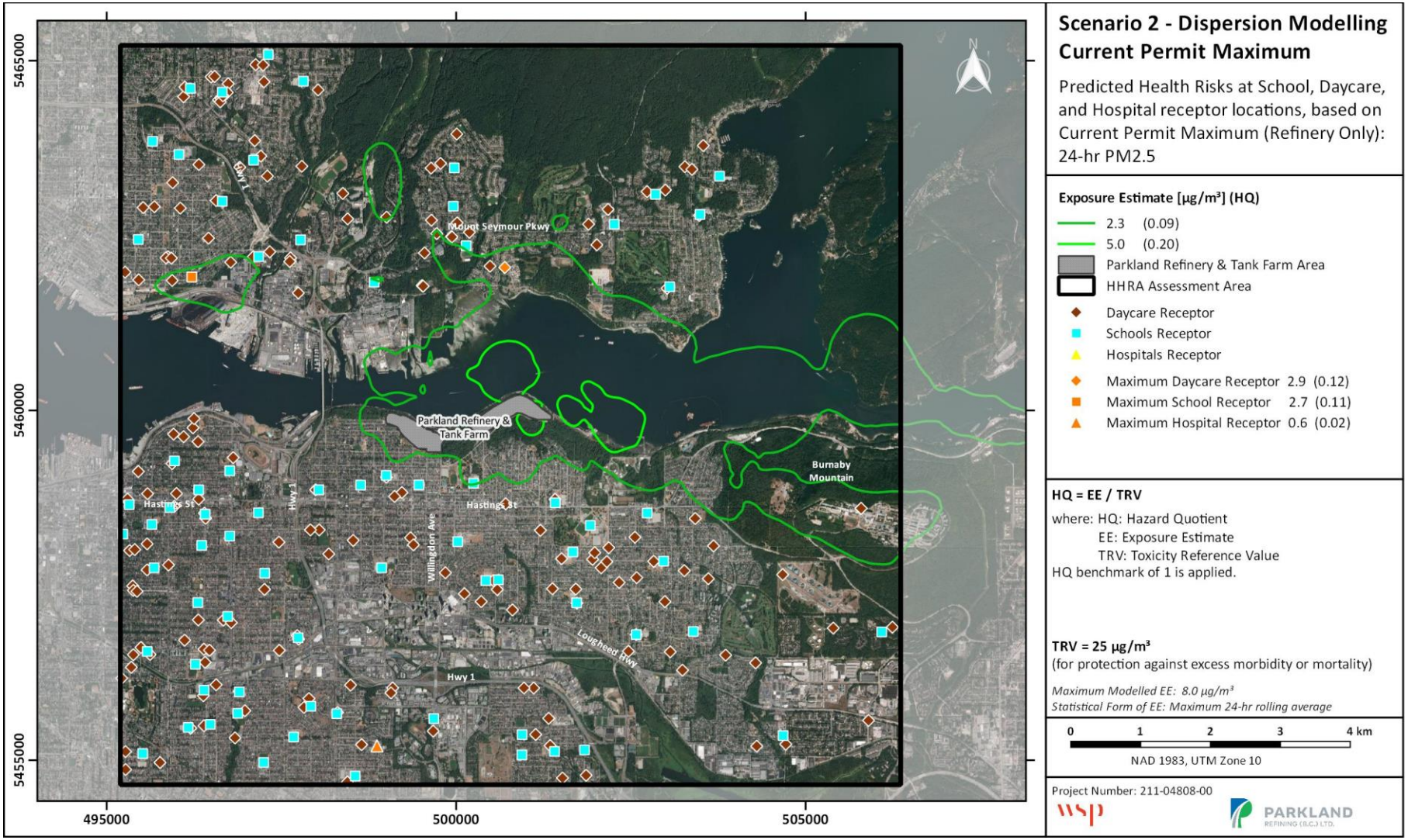


Figure 6-40: Scenario 2 – Predicted Health Risks at School, Daycare, and Hospital Receptor Locations Based on Current Permit Maximum (Refinery-Only) 24-hr $PM_{2.5}$

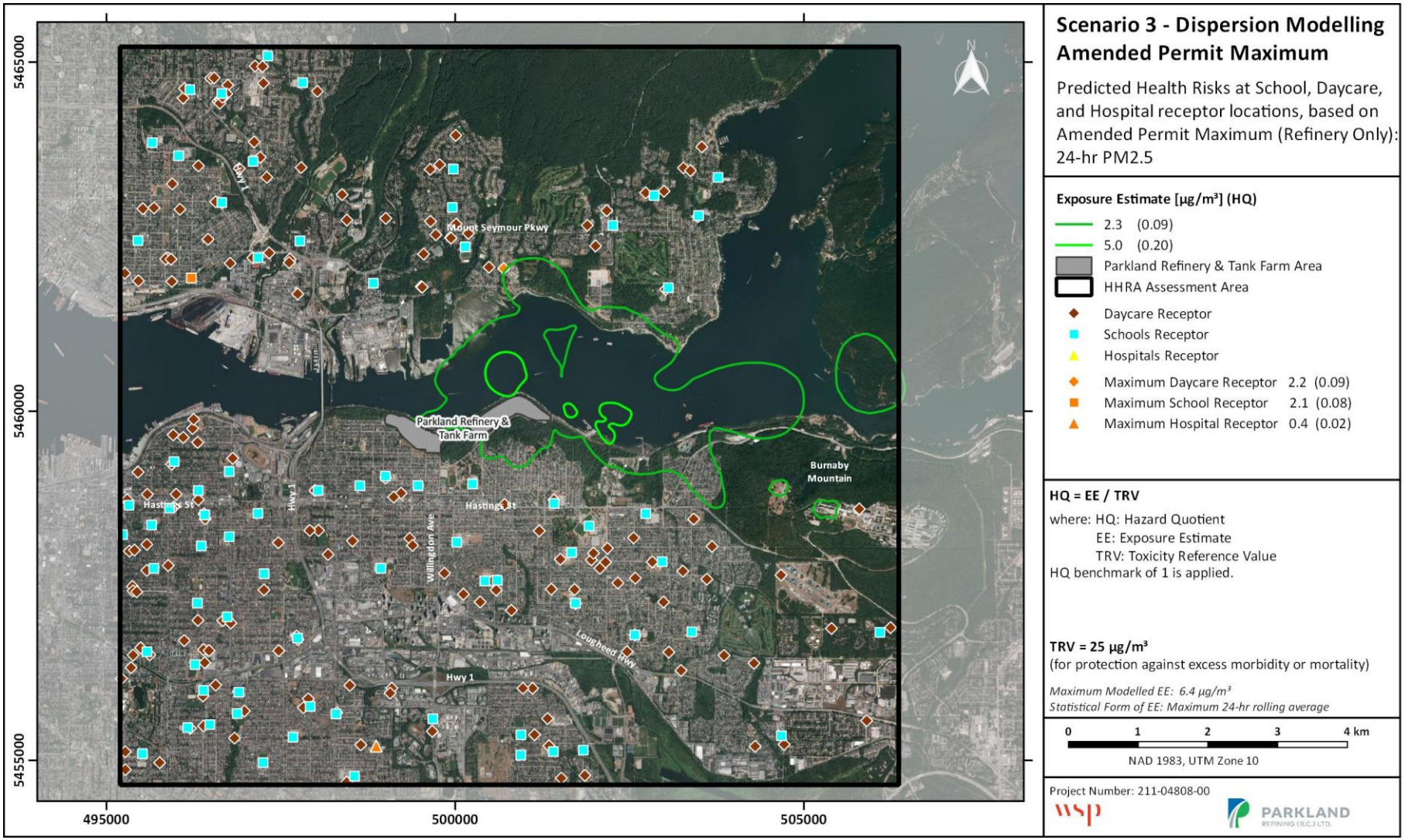


Figure 6-41: Scenario 3 – Predicted Health Risks at School, Daycare, and Hospital Receptor Locations Based on Amended Permit Maximum (Refinery-Only) 24-hr $PM_{2.5}$

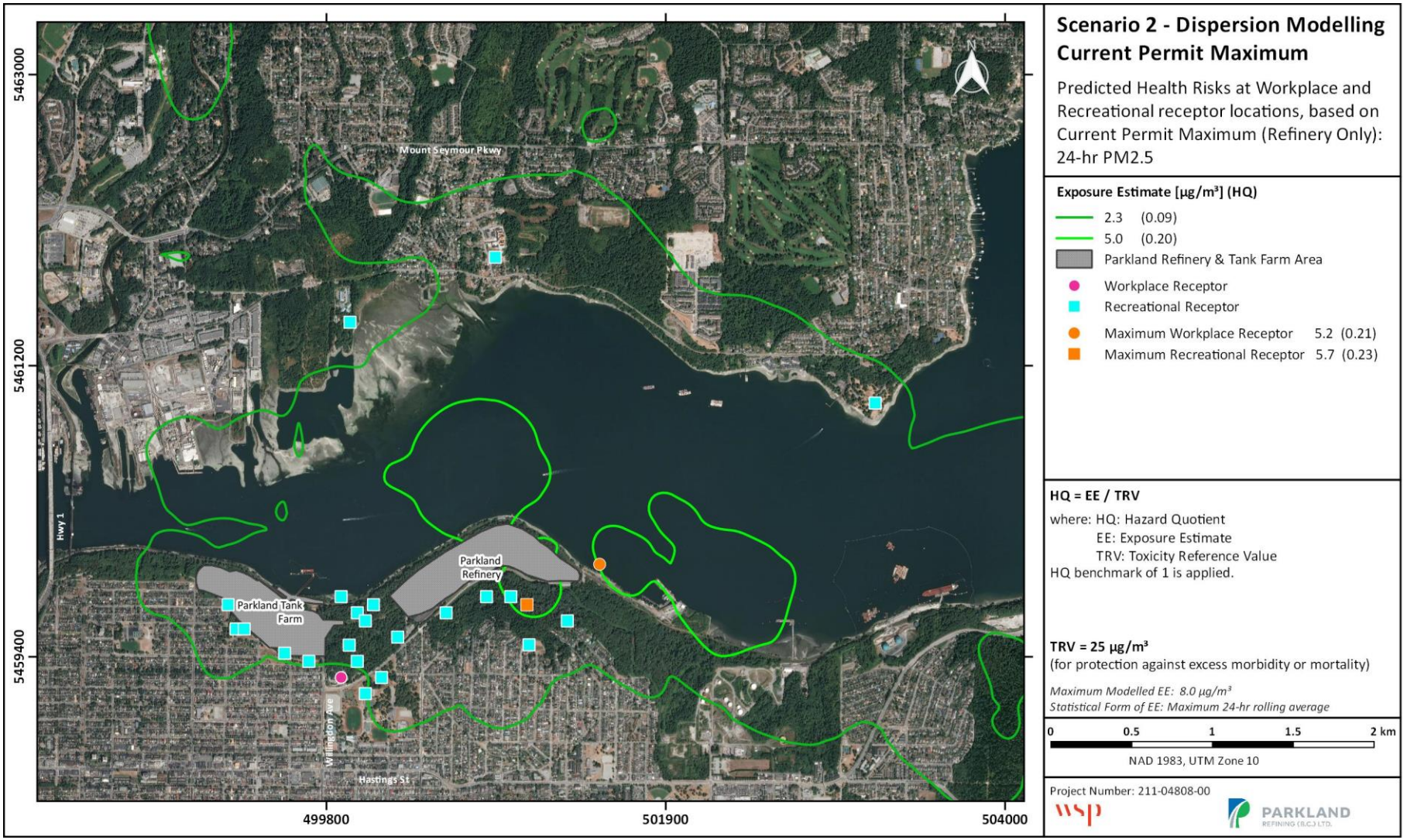


Figure 6-42: Scenario 2 – Predicted Health Risks at Workplace and Recreational Receptor Locations Based on Current Permit Maximum (Refinery-Only) 24-hr $PM_{2.5}$

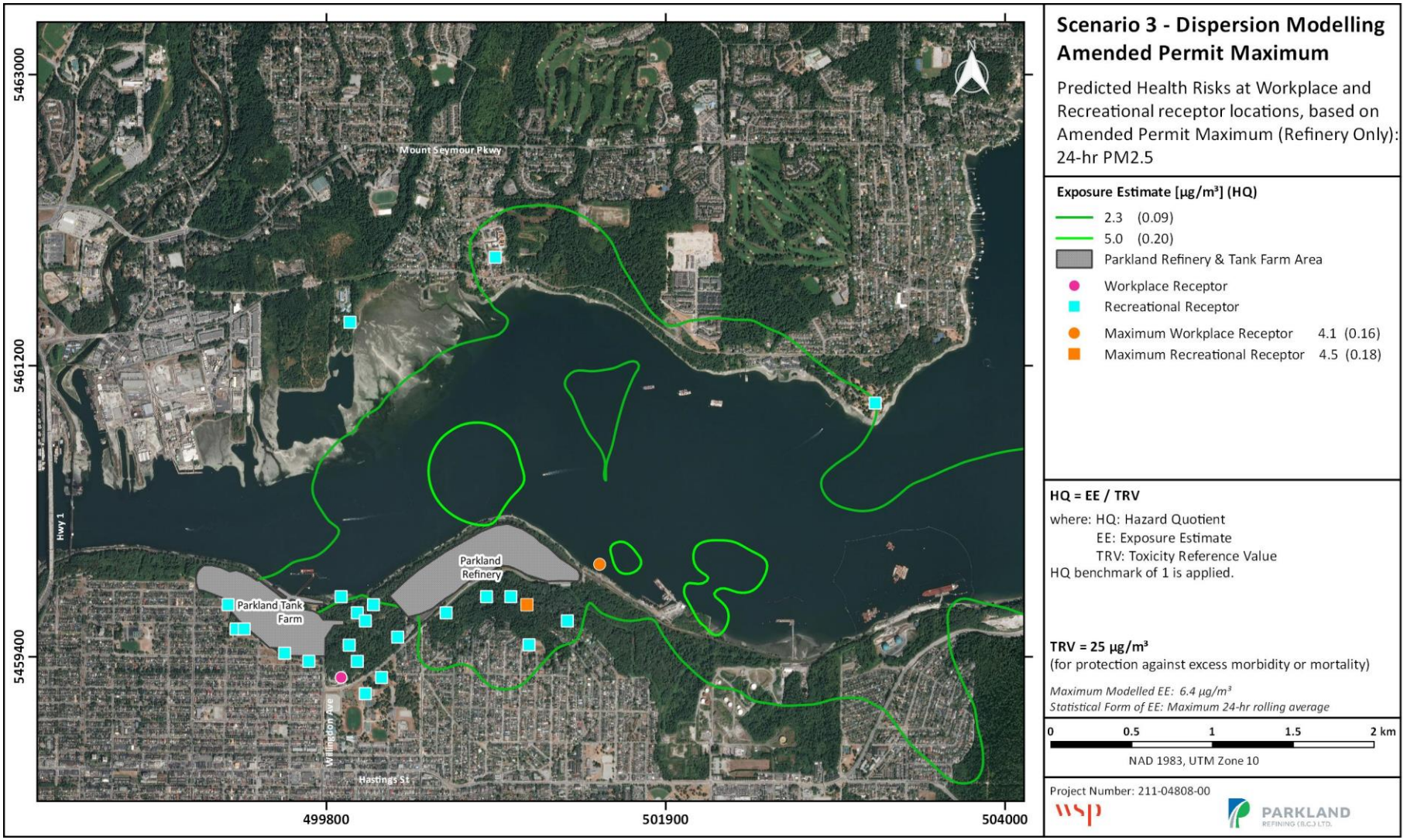


Figure 6-43: Scenario 3 – Predicted Health Risks at Workplace and Recreational Receptor Locations Based on Amended Permit Maximum (Refinery-Only) 24-hr $PM_{2.5}$

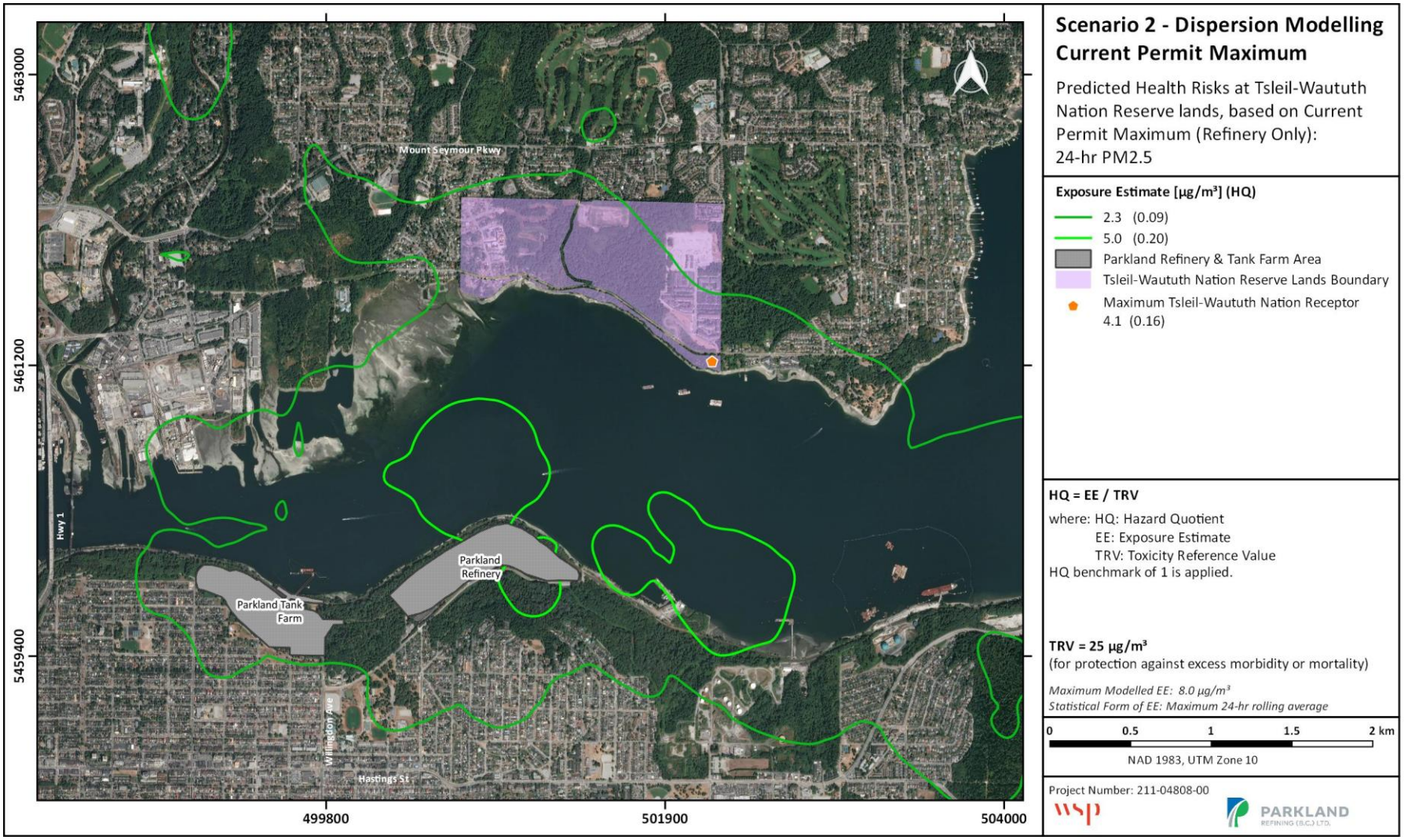


Figure 6-44: Scenario 2 – Predicted Health Risks at TWN Reserve Lands Based on Current Permit Maximum (Refinery-Only) 24-hr $PM_{2.5}$

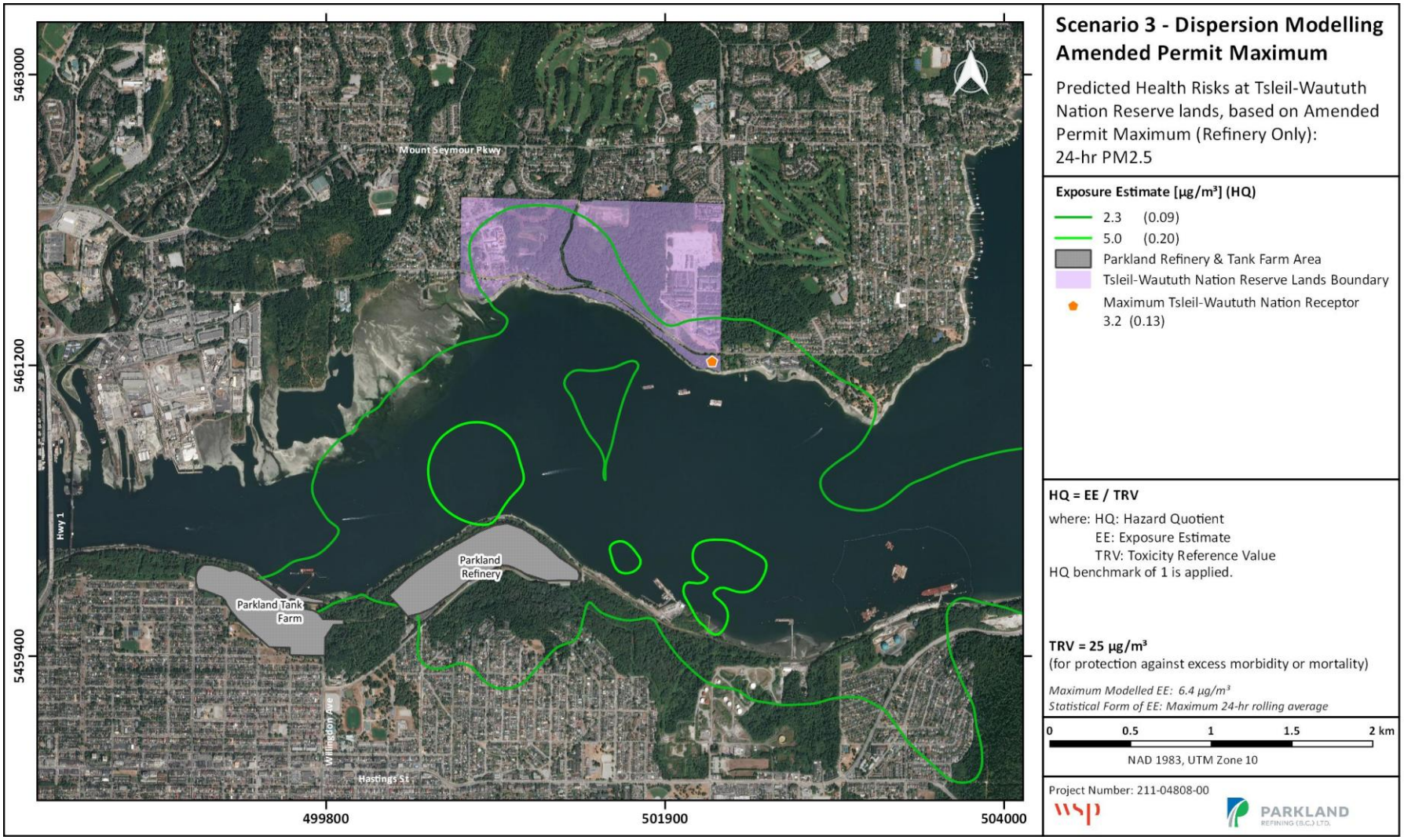


Figure 6-45: Scenario 3 – Predicted Health Risks at TWN Reserve Lands Based on Amended Permit Maximum (Refinery-Only) 24-hr $PM_{2.5}$

6.3.6 FINE PARTICULATE MATTER ($PM_{2.5}$) – CHRONIC EXPOSURES

As detailed in **Section 5.3** one TRV for chronic $PM_{2.5}$ exposures has been applied in the risk characterization step of the HHRA. This section presents the results for the TRV of 10 µg/m^3, which was derived to protect against excess mortality. **Table 6-11** through **Table 6-14,** and **Figure 6-46** through **Figure 6-54** present the predicted exposure estimates and HQs associated with excess mortality risks for annual average $PM_{2.5}$ exposures for each of the identified receptors.

Figure 6-46 represents results for Scenario 1 – Ambient Monitoring 2017 - 2019, based on air quality measurements at monitoring stations near the refinery. **Figure 6-47** through **Figure 6-54** present results for Scenario 2 – Dispersion Modelling Current Permit Maximum and Scenario 3- Dispersion Modelling Amended Permit Maximum. Exposure estimates for these scenarios were developed using a dispersion model that predicts ambient air concentrations of COPCs based on emissions from the Parkland refinery.

The coloured shading within **Table 6-11** through **Table 6-14** corresponds to the colour of the applicable concentration / risk isopleths in **Figure 6-47** through **Figure 6-54**. **Table 6-11** through **Table 6-14** also contains risk estimates for the maximally impacted receptors of each type for Scenarios 2 (S2) and 3 (S3) (see "Receptor Maxima" column).

6.3.6.1 LTC FACILITIES AND RESIDENTIAL RECEPTOR LOCATIONS

The results presented in **Table 6-11, Figure 6-46, Figure 6-47** and **Figure 6-48** below for the predicted long-term health risks associated with maximum annual exposure to $PM_{2.5}$ for seniors in LTC facilities and residents are interpreted as follows:

- A Target HQ of 1.0 was selected for residents and seniors in LTC facilities as the HHRA assumed that these receptors could potentially receive their theoretical annual exposure within the HHRA study area.
- Air quality monitoring data from 2017 - 2019 (Scenario 1) shown in **Figure 6-46** indicates that that none of the monitoring stations included in the study results in HQs greater than 1.0, based on the TRV of 10 µg/m^3. It is important to note that the HQs are similar (0.46-0.55) for all of the monitoring stations, indicating very consistent annual $PM_{2.5}$ concentrations throughout the study area, regardless of their respective distance from the refinery. This suggests that baseline / non-refinery $PM_{2.5}$ sources are the driver of concentrations and associated $PM_{2.5}$ health risks throughout the study area.
- Air quality modelling results (Scenarios 2, 3 and 4) indicate that cumulative HQs for the resident and seniors in LTC based on dispersion modelling results (Scenarios 2-4) are driven by baseline (ambient) $PM_{2.5}$ concentrations, which account for 94 – 96% of the cumulative HQs for the maximum resident and senior receptors. Potential health risks to the resident or seniors in LTC facilities are not expected as a result of the predicted maximum annual exposure to $PM_{2.5}$ given that all cumulative HQs were less than the Target HQ of 1.0.

6.3.6.2 DAYCARE, SCHOOL, AND HOSPITAL RECEPTOR LOCATIONS

The results presented in **Table 6-12**, **Figure 6-49** and **Figure 6-50** below for the predicted long-term health risks associated with maximum annual exposure to $PM_{2.5}$ for toddlers and young children in daycare, children and teenagers attending school, and hospital patients are interpreted as follows:

- A Target HQ of 0.2 was selected for school, daycare, and hospital receptor locations because the HHRA assumed that receptors may only receive a portion of their theoretical annual exposure within the HHRA study area.
- Air quality modelling results (Scenarios 2, 3 and 4) indicate that potential health risks to toddlers and young children in daycare facilities are not expected as a result of predicted maximum annual exposure to $PM_{2.5}$ given that all cumulative HQs were less than the benchmark HQ of 0.2.
- Air quality modelling results (Scenarios 2, 3 and 4) indicate that potential health risks to children and teens in elementary school and teenagers in high school are also not expected as a result of predicted maximum annual exposure to $PM_{2.5}$ given that all cumulative HQs were less than the benchmark HQ of 0.2.

- Air quality modelling results (Scenarios 2, 3 and 4) indicate that potential health risks to adult hospital patients are also not expected as a result of predicted maximum annual exposure to $PM_{2.5}$ given that all cumulative HQs were less than the benchmark HQ of 0.2.

6.3.6.3 *WORKPLACE & RECREATIONAL RECEPTOR LOCATIONS*

The results presented in **Table 6-13**, **Figure 6-51** and **Figure 6-52** below for the predicted long-term health risks associated with maximum annual exposure to $PM_{2.5}$ for adult workers and recreational receptors (all life stages) are interpreted as follows:

- A Target HQ of 0.2 was selected for these receptor locations because the HHRA assumed that receptors may only receive a portion of their theoretical annual exposure within the HHRA study area.
- Air quality modelling results (Scenarios 2, 3 and 4) indicate that cumulative HQs are driven by baseline (ambient) $PM_{2.5}$ concentrations. Baseline accounts for more than 92% of the cumulative risk for these receptors long-term exposure to $PM_{2.5}$. Potential health risks to the adult worker and recreational receptor (all life stages) are not expected as a result of the predicted maximum annual exposure to $PM_{2.5}$ given that all cumulative HQs were less than the benchmark HQ of 0.2.

6.3.6.4 *TSLEIL-WAUTUTH RESERVE LANDS*

The results presented in **Table 6-14**, **Figure 6-53** and **Figure 6-54** below for the predicted long-term health risks associated with maximum annual exposure to $PM_{2.5}$ for persons of all ages participating in outdoor cultural activities at TWN Reserve Lands are interpreted as follows:

- A Target HQ of 0.2 was selected for persons of all ages at TWN Reserve Lands because the HHRA assumed that receptors may only receive a portion of their theoretical annual exposure within the HHRA study area. Chronic residential, daycare, school, and recreational exposures occurring on TWN Reserve Lands are quantified in the previous sections.
- Air quality modelling results (Scenarios 2, 3 and 4) indicate that baseline accounts for more than 92% of the cumulative risk for these receptors' long-term exposure to $PM_{2.5}$. Cumulative HQs for all life stages were less than the Target HQ of 0.2, indicating the potential for health risks due to long-term exposure to $PM_{2.5}$ is negligible.

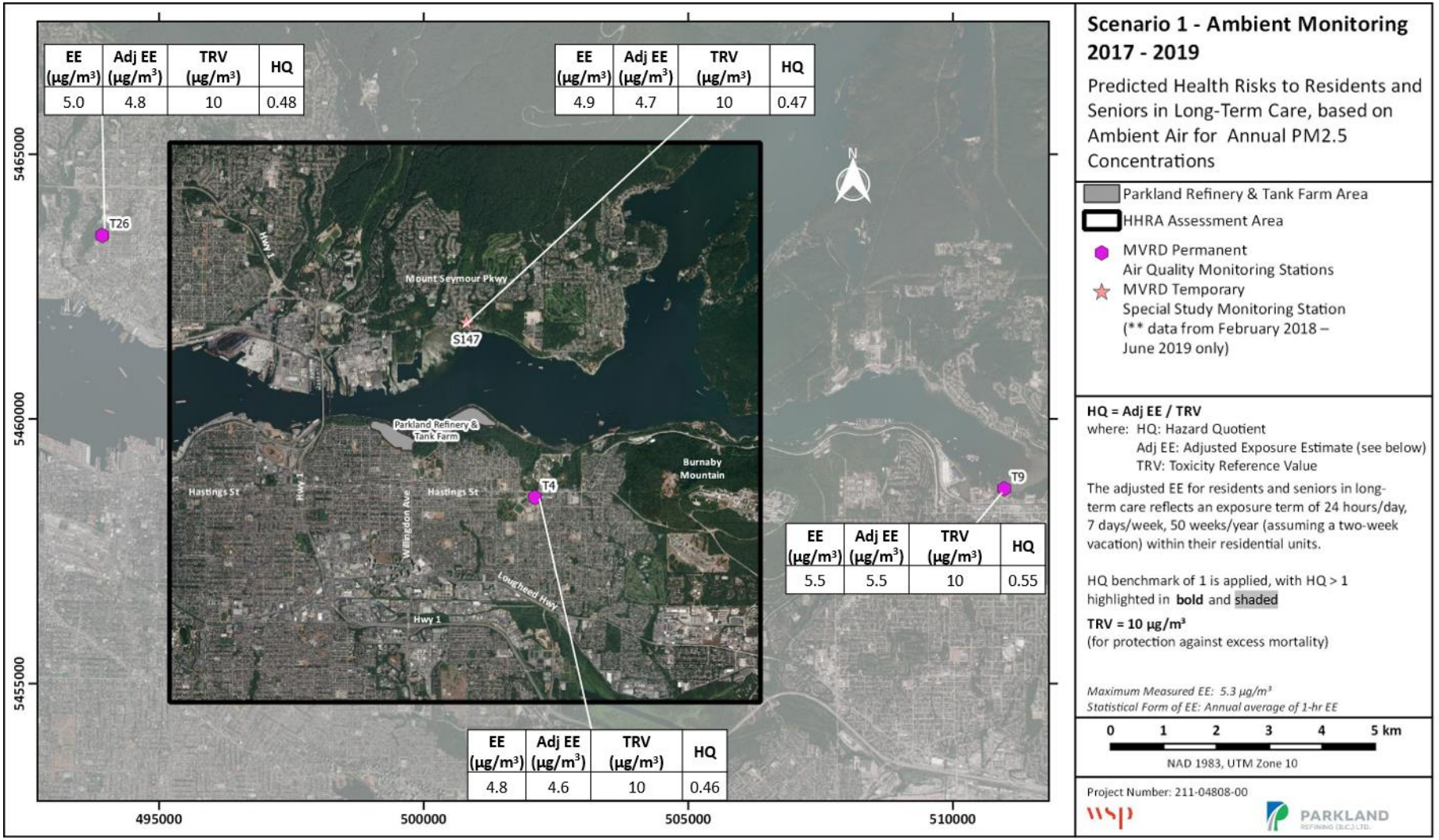


Figure 6-46 Scenario 1 – Predicted Health Risks to Residents and Seniors in Long-Term Care Based on Ambient Air Measurements for Annual $PM_{2.5}$ Concentrations

Table 6-11 Exposure Estimates and Predicted HQs Resulting from Maximum Annual Exposure to $PM_{2.5}$ for Identified Receptors – Seniors in LTC Facilities and Residents

Receptor	Annual TRV (µg/m³)	Baseline Conc. (µg/m³)	Adjusted Baseline Conc. (µg/m³)	HQ (Baseline)	Receptor Maxima	Predicted Conc. From Refinery (µg/m³)	Adjusted Refinery Conc. (µg/m³)	HQ (Refinery-Only)	Cumulative Conc. (µg/m³)	Adjusted Cumulative Conc. (µg/m³)	HQ (Cumulative)	% HQ Attributable to Baseline
LTC Home *Adult*	10	4.8	4.6	0.46	■ S3 Isopleth 2	0.2	0.19	0.02	5.0	4.8	0.48	96%
					■ S2	0.3	0.29	0.03	5.1	4.9	0.49	94%
					Isopleth 1	0.4	0.38	0.04	5.2	5.0	0.50	92%
Resident *Infant Toddler Child Teen Adult*	10	4.8	4.6	0.46	Isopleth 2	0.2	0.2	0.02	5.0	4.8	0.48	96%
					▲ S3	0.3	0.3	0.03	5.1	4.9	0.49	94%
					▲ S2 Isopleth 1	0.4	0.4	0.04	5.2	5.0	0.50	92%

Notes:
Cumulative Concentration/HQ = Baseline + Refinery Contribution
Target HQ=1.0
HQs presented in **bold** and shaded if Target HQ is exceeded.

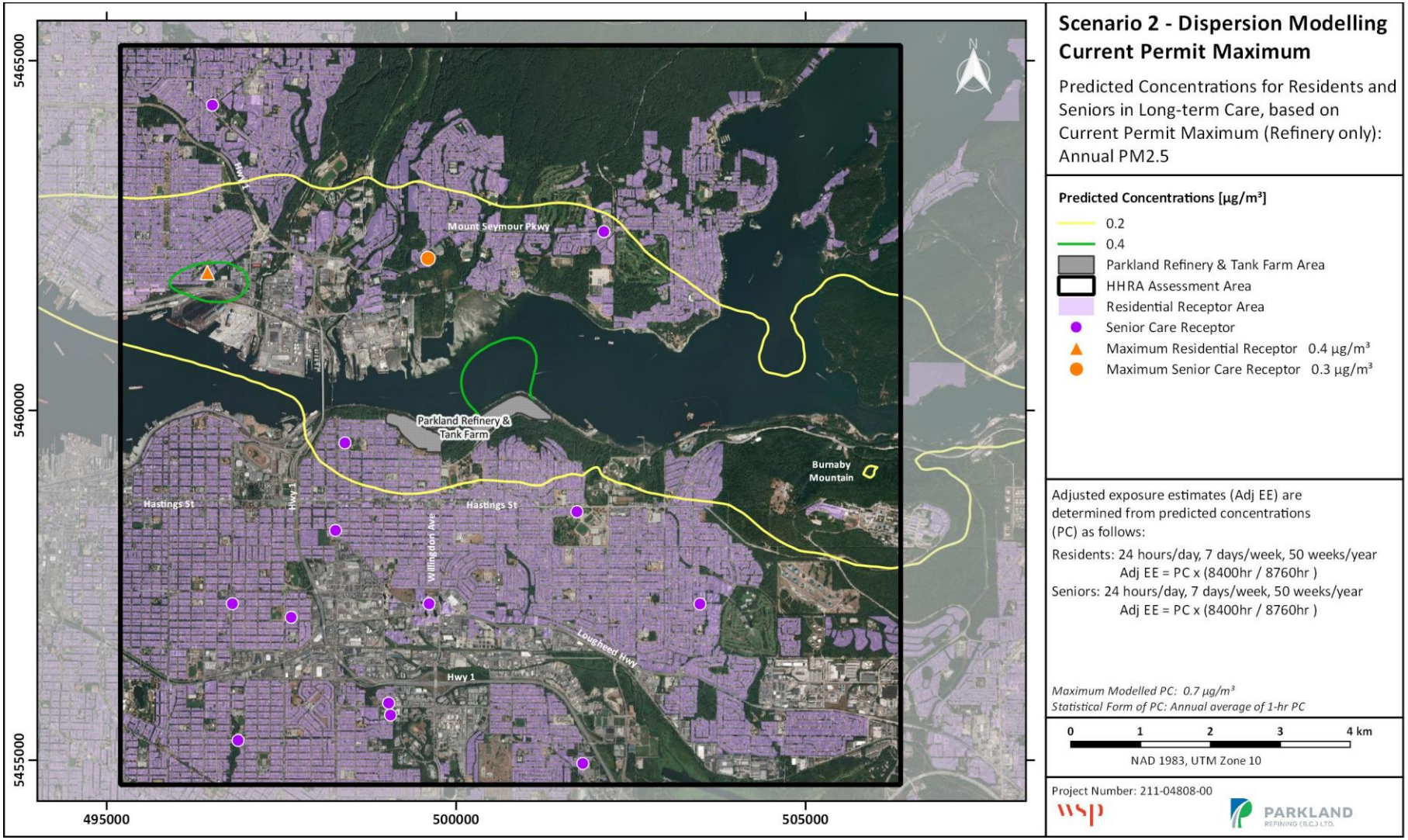


Figure 6-47: Scenario 2 – Predicted Health Risks to Residents and Seniors in Long-term Care Based on Current Permit Maximum (Refinery-Only) Annual $PM_{2.5}$ Concentrations

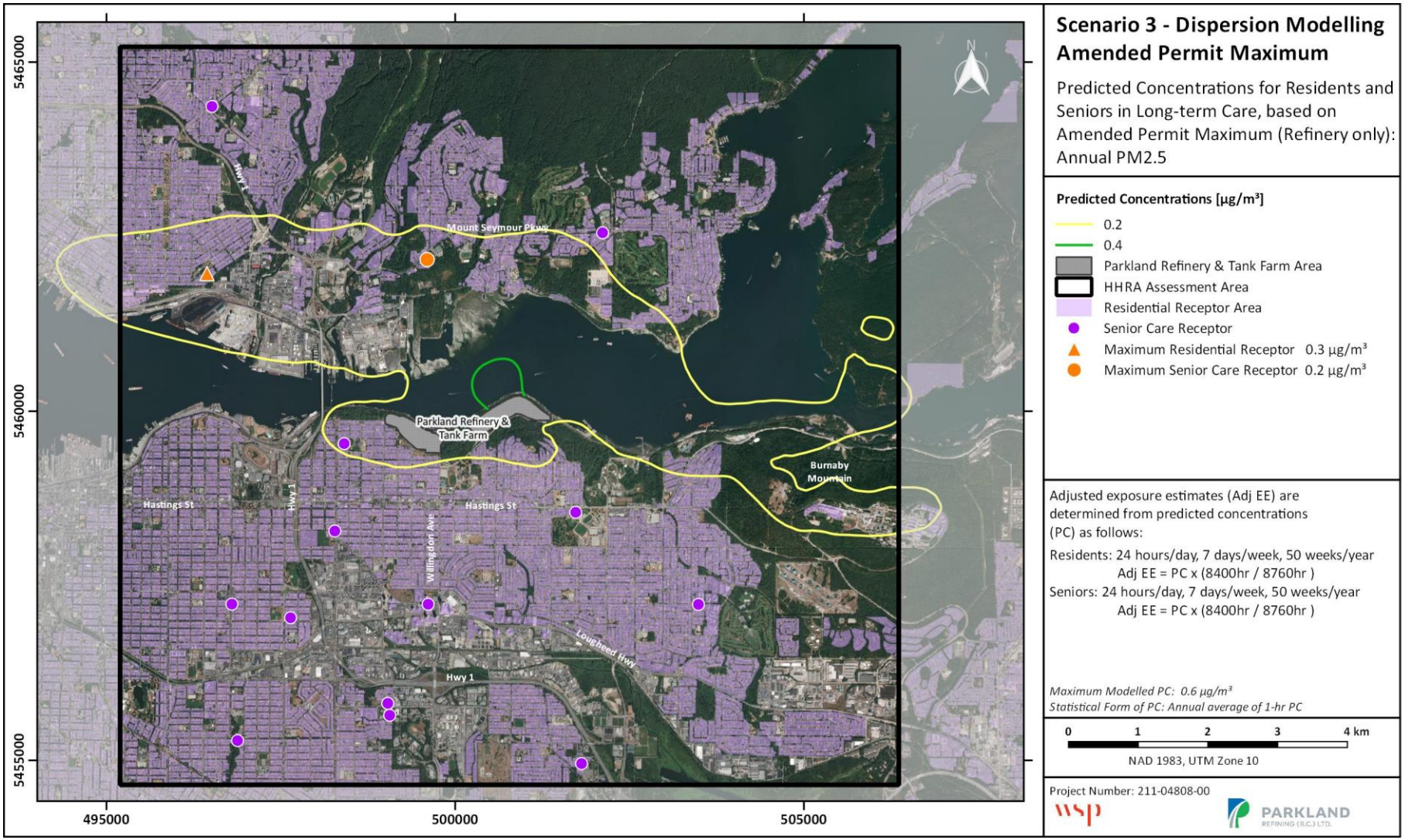


Figure 6-48: Scenario 3 – Predicted Health Risks to Residents and Seniors in Long-term Care Based on Amended Permit Maximum (Refinery-Only) Annual $PM_{2.5}$ Concentrations

Table 6-12 Exposure Estimates and Predicted HQs Resulting from Maximum Annual Exposure to $PM_{2.5}$ for Identified Receptors at Daycares, Schools, and Hospital

Receptor	Annual TRV (µg/m³)	Baseline Conc. (µg/m³)	Adjusted Baseline Conc. (µg/m³)	HQ (Baseline)	Receptor Maxima	Predicted Conc. From Refinery (µg/m³)	Adjusted Refinery Conc. (µg/m³)	HQ (Refinery-Only)	Cumulative Conc. (µg/m³)	Adjusted Cumulative Conc. (µg/m³)	HQ (Cumulative)	% HQ Attributable to Baseline
Daycare *Toddler Child*	10	4.8	1.2	0.1	Isopleth 2	0.2	0.05	0.005	5.0	1.28	0.128	96%
					◆ S3	0.3	0.08	0.008	5.1	1.31	0.131	94%
					◆ S2 Isopleth 1	0.4	0.10	0.010	5.2	1.34	0.134	92%
School *Child Teen*	10	4.8	0.94	0.09	Isopleth 2	0.2	0.04	0.004	5.0	0.98	0.098	96%
					■ S3	0.3	0.06	0.006	5.1	1.00	0.100	94%
					■ S2 Isopleth 1	0.4	0.08	0.008	5.2	1.02	0.102	92%
Hospital *Adult*	10	4.8	0.5	0.05	▲ S2, S3	0.1	0.01	0.001	4.9	0.47	0.047	98%
					Isopleth 2	0.2	0.02	0.002	5.0	0.48	0.048	96%
					Isopleth 1	0.4	0.04	0.004	5.2	0.50	0.050	92%

Notes:
Cumulative Concentration/HQ = Baseline + Refinery Contribution
Target HQ=0.2
HQs presented in **bold** and shaded if Target HQ is exceeded.

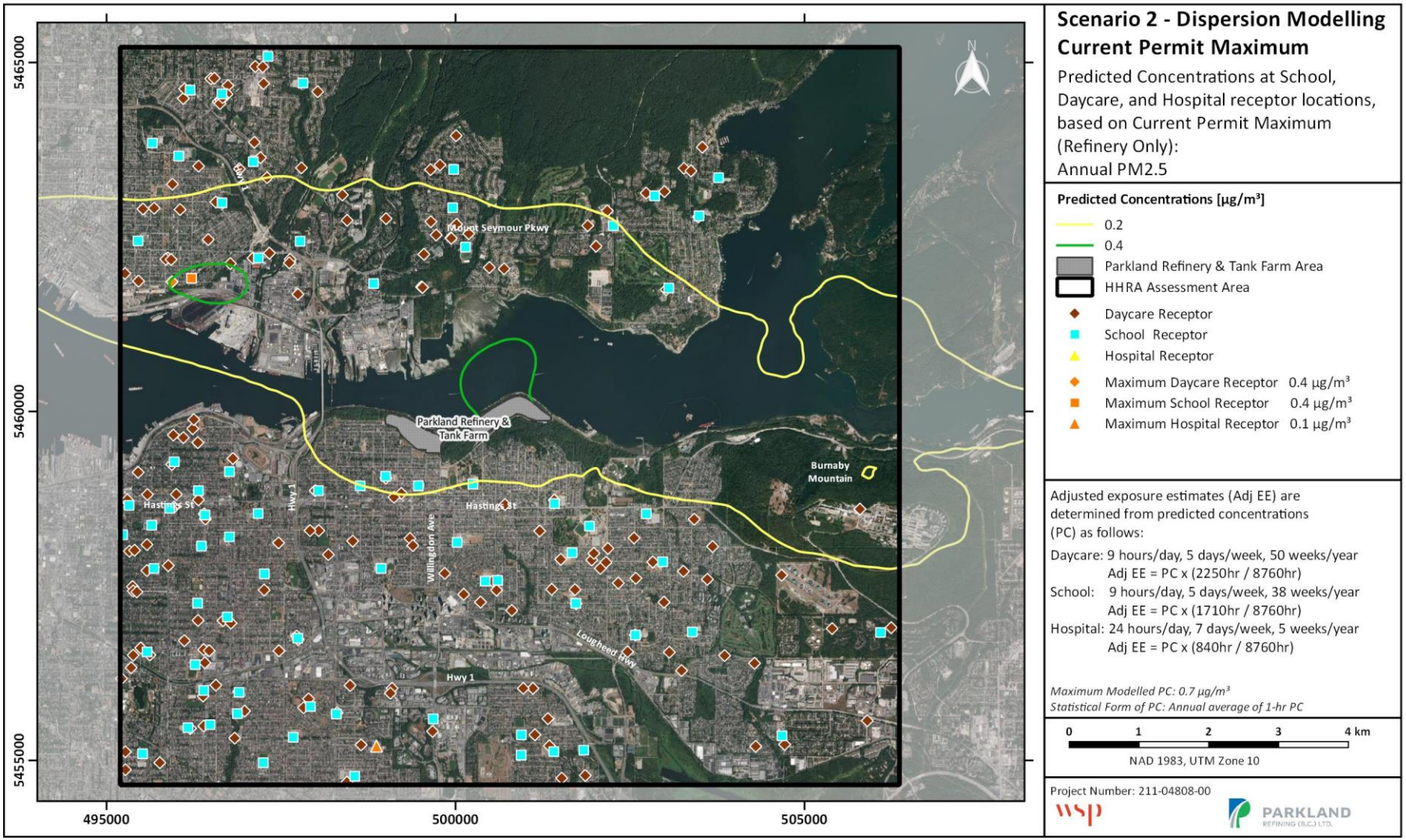


Figure 6-49: Scenario 2 – Predicted Health Risks at School, Daycare, and Hospital Receptor Locations Based on Current Permit Maximum (Refinery-Only) Annual $PM_{2.5}$ Concentrations

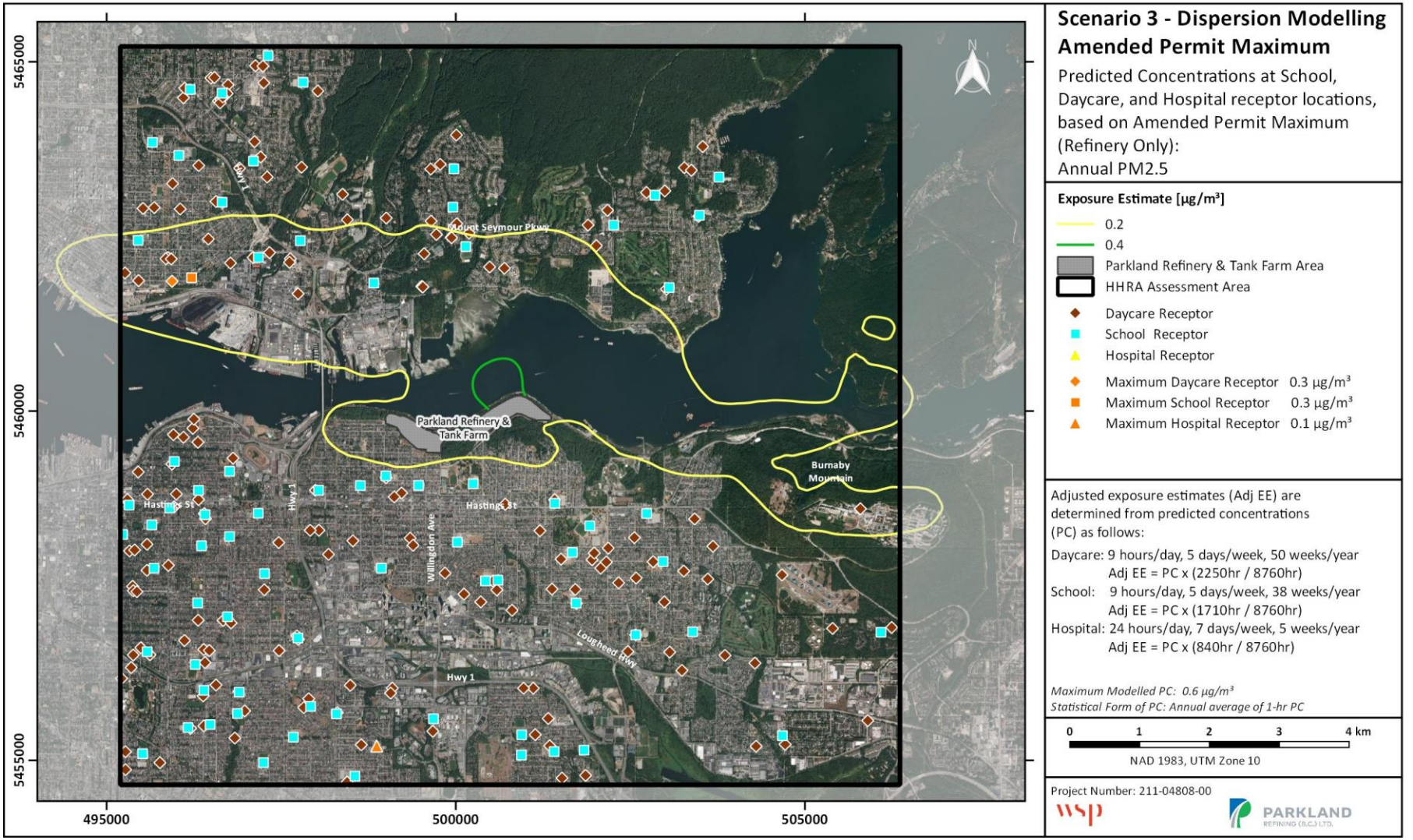


Figure 6-50: Scenario 3 – Predicted Health Risks at School, Daycare, and Hospital Receptor Locations Based on Amended Permit Maximum (Refinery-Only) Annual $PM_{2.5}$ Concentrations

Table 6-13 Exposure Estimates and Predicted HQs Resulting from Maximum Annual Exposure to $PM_{2.5}$ for Identified Receptors – Worker & Recreational Receptors

Receptor	Annual TRV (µg/m³)	Baseline Conc. (µg/m³)	Adjusted Baseline Conc. (µg/m³)	HQ (Baseline)	Receptor Maxima	Predicted Conc. From Refinery (µg/m³)	Adjusted Refinery Conc. (µg/m³)	HQ (Refinery-Only)	Cumulative Conc. (µg/m³)	Adjusted Cumulative Conc. (µg/m³)	HQ (Cumulative)	% HQ Attributable to Baseline
Workplace *Adult*	10	4.8	1.1	0.1	S3 Isopleth 2	0.2	0.046	0.0046	5	1.14	0.114	96%
					S2	0.3	0.068	0.0068	5.1	1.16	0.116	94%
					Isopleth 1	0.4	0.091	0.0091	5.2	1.19	0.119	92%
Visitor *Infant* *Toddler* *Child* *Teen* *Adult*	10	4.8	0.4	0.04	Isopleth 2	0.2	0.016	0.0016	5	0.40	0.040	96%
					S3	0.3	0.024	0.0024	5.1	0.41	0.041	94%
					S2 Isopleth 1	0.4	0.032	0.0032	5.2	0.42	0.042	92%

Notes:
Cumulative Concentration/HQ = Baseline + Refinery Contribution
Target HQ=0.2
HQs presented in **bold** and shaded if Target HQ is exceeded.

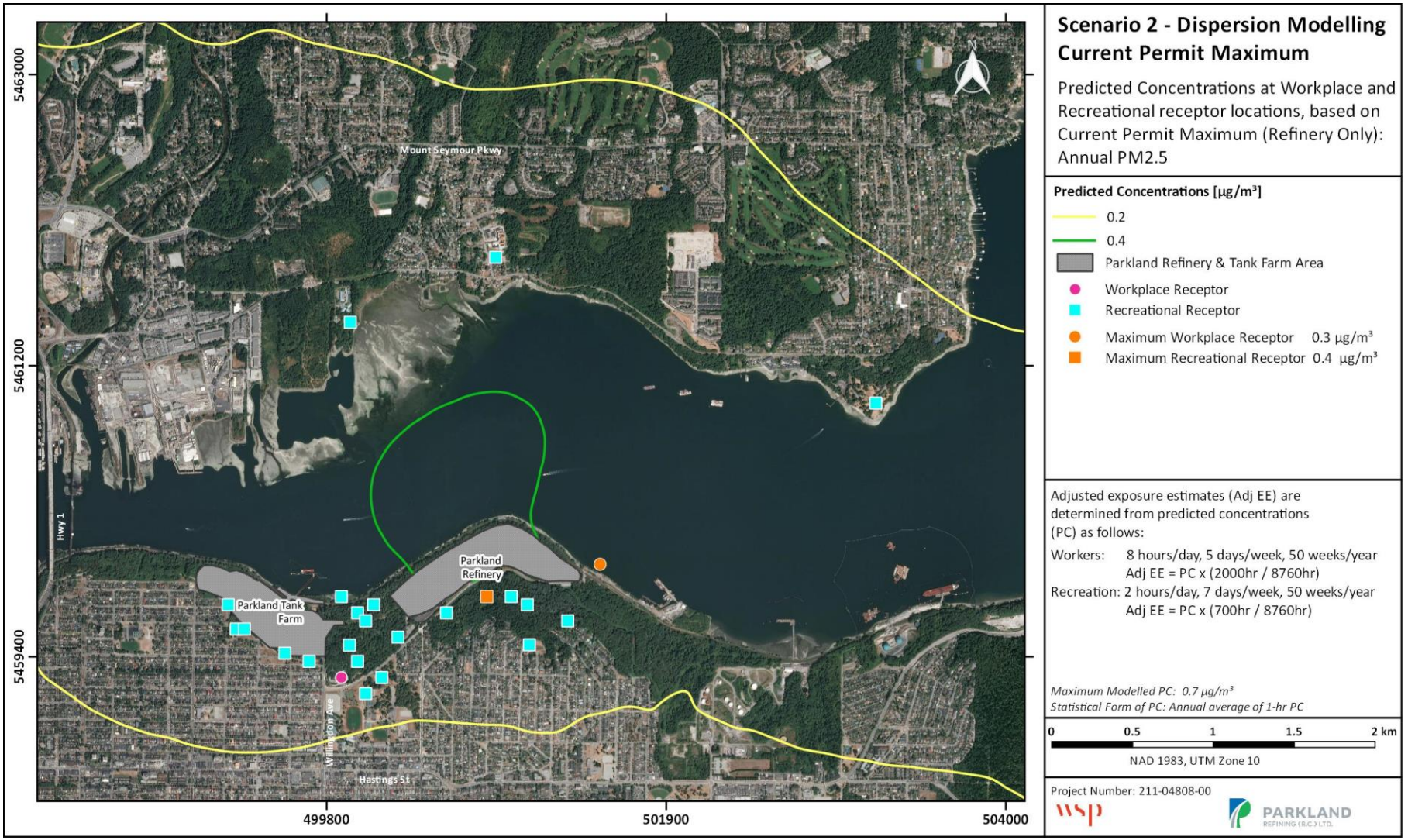


Figure 6-51: Scenario 2 – Predicted Health Risks at Workplace and Recreational Receptor Locations Based on Current Permit Maximum (Refinery-Only) Annual $PM_{2.5}$ Concentrations

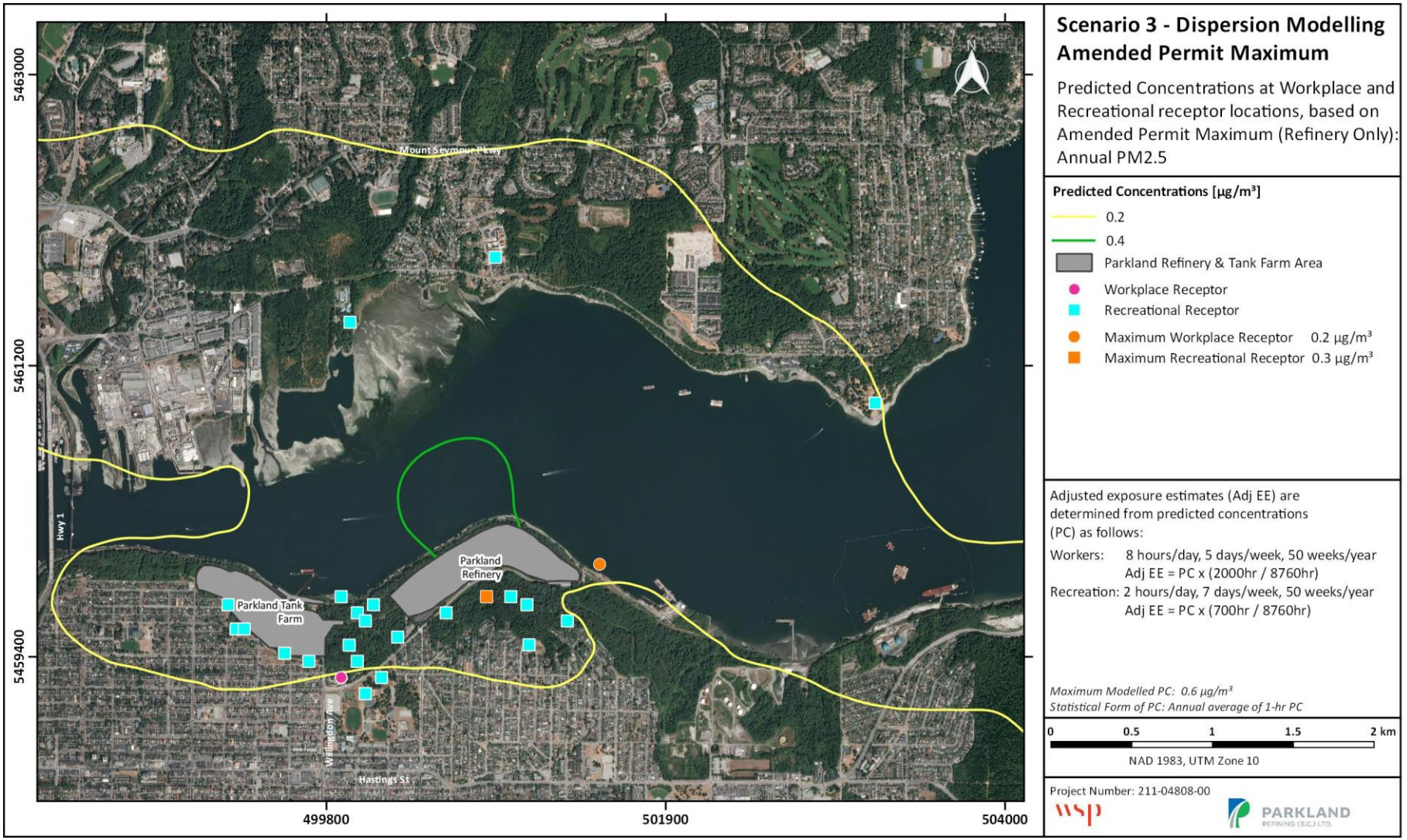


Figure 6-52: Scenario 3 – Predicted Health Risks at Workplace and Recreational Receptor Locations Based on Amended Permit Maximum (Refinery-Only) Annual $PM_{2.5}$ Concentrations

Table 6-14 Exposure Estimates and Predicted HQs Resulting from Maximum Annual Exposure to $PM_{2.5}$ for Identified Receptors – TWN Reserve Lands

Receptor	Annual TRV (µg/m³)	Baseline Conc. (µg/m³)	Adjusted Baseline Conc. (µg/m³)	HQ (Baseline)	Receptor Maxima	Predicted Conc. From Refinery (µg/m³)	Adjusted Refinery Conc. (µg/m³)	HQ (Refinery-Only)	Cumulative Conc. (µg/m³)	Adjusted Cumulative Conc. (µg/m³)	HQ (Cumulative)	% HQ Attributable to Baseline
Reserve Lands *Infant* *Toddler* *Child* *Teen* *Adult*	10	4.8	0.3	0.03	Isopleth 2	0.2	0.01	0.001	5.0	0.30	0.03	96%
					S3	0.3	0.02	0.002	5.1	0.30	0.03	94%
					S2 Isopleth 1	0.4	0.02	0.002	5.2	0.31	0.03	92%

Notes:
Cumulative Concentration/HQ = Baseline + Refinery Contribution
Target HQ=0.2
HQs presented in **bold** and shaded if Target HQ is exceeded.

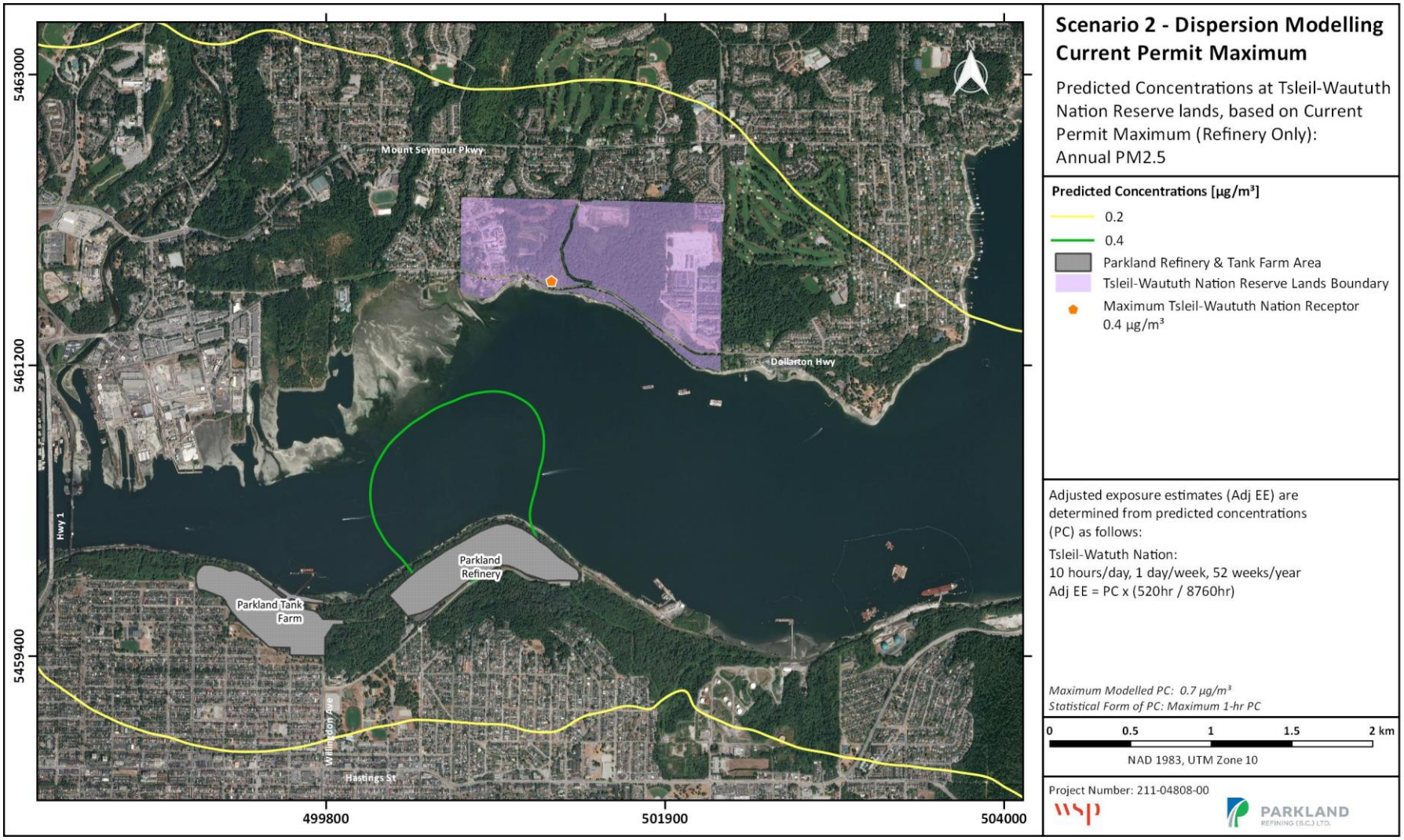


Figure 6-53: Scenario 2 – Predicted Health Risks at TWN Reserve Lands Based on Current Permit Maximum (Refinery-Only) Annual $PM_{2.5}$ Concentrations

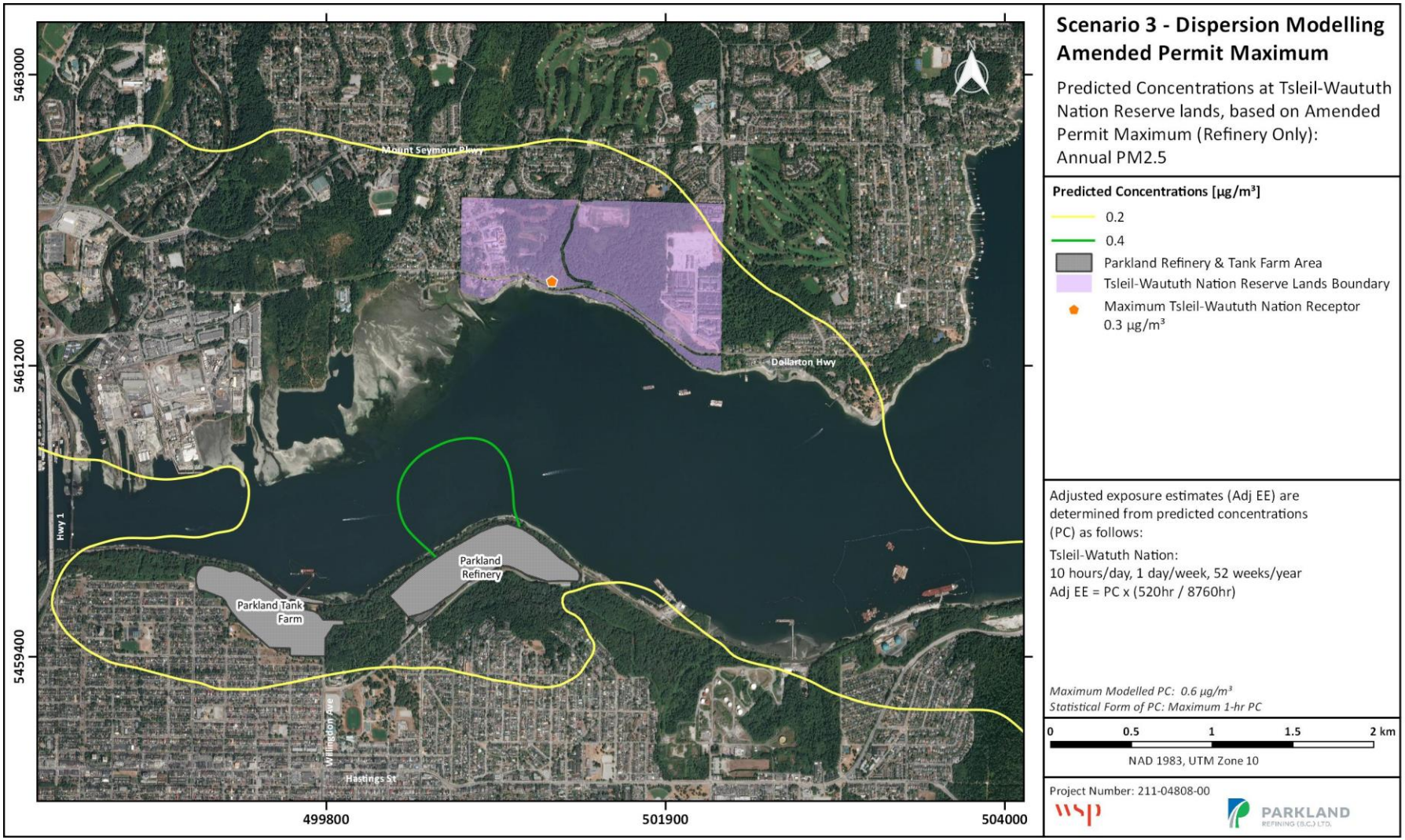


Figure 6-54: Scenario 3 – Predicted Health Risks at TWN Reserve Lands Based on Amended Permit Maximum (Refinery-Only) Annual $PM_{2.5}$ Concentrations

6.3.7 BENZENE AND 1,3-BUTADIENE

Figure 6-55 and **Figure 6-57** below present the predicted exposure estimates and HQs for benzene and 1,3-butadiene based on MVRD ambient monitoring data from stations located within the HHRA study area (i.e., Scenario 1). There are no predicted health risks to any receptors based on maximum daily 24-hour concentrations for either contaminant. Concentrations measured at MVRD monitoring stations T9 and T24 result in a HQ less than 1 suggesting that the potential for health risks is negligible.

With respect to long-term (annual) exposures, predicted cancer risk to residents or seniors in LTC based on benzene and 1,3-butadiene ambient air concentrations (**Figure 6-56**, **Figure 6-58**) are considered negligible. Ambient air concentrations measured at MVRD monitoring stations T9 and T24 result in less than a 1 in 100,000 ILCR for both contaminants, which is the threshold used by both B.C. Ministry of Health and Health Canada.

Appendix B presents additional detail related to historical trends in levels of these COPCs at MVRD monitoring stations nearest to the refinery.

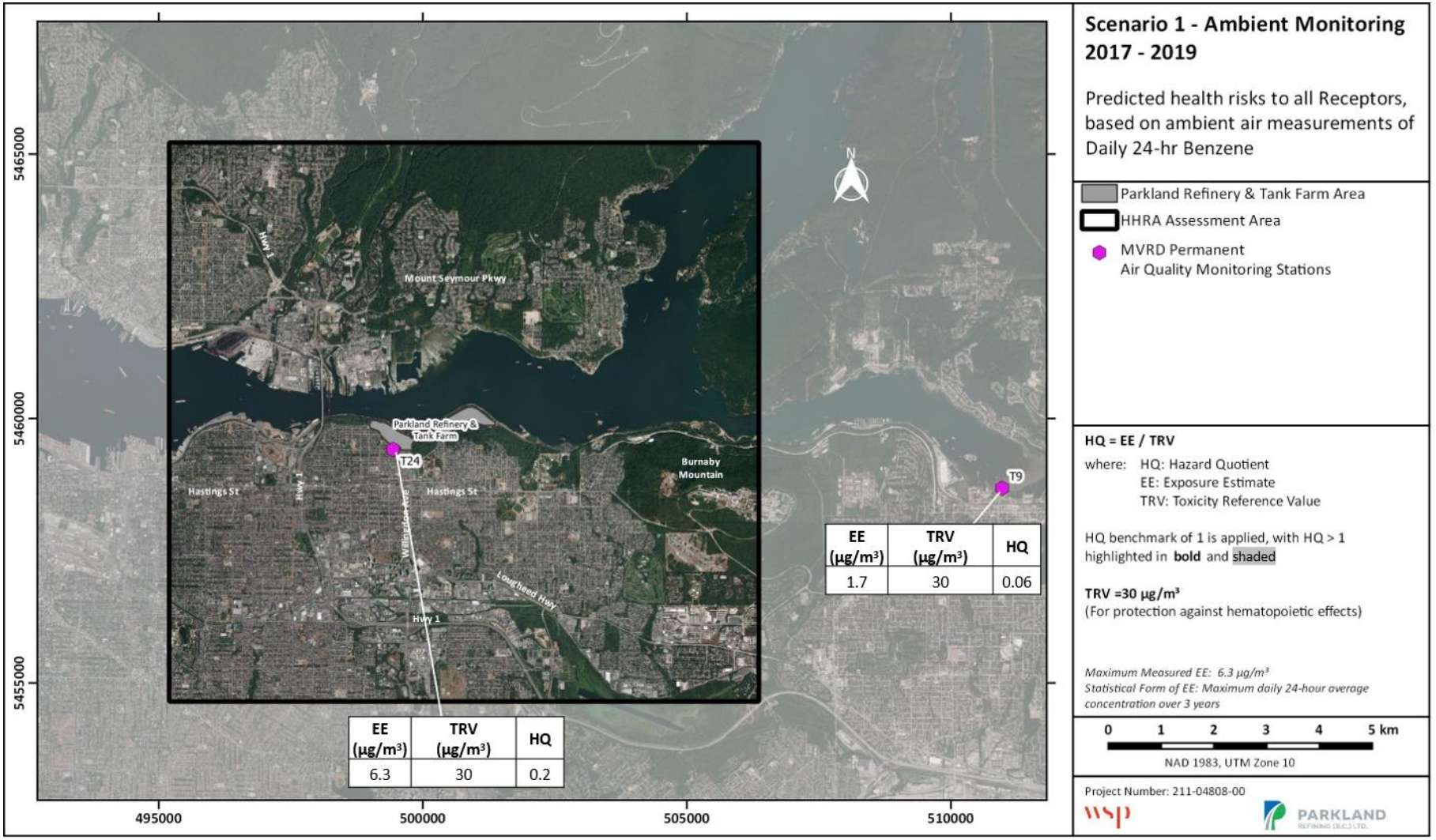


Figure 6-55: Scenario 1 – Predicted Health Risks to All Receptors Based on Ambient Air Measurements of Daily 24-hr Benzene

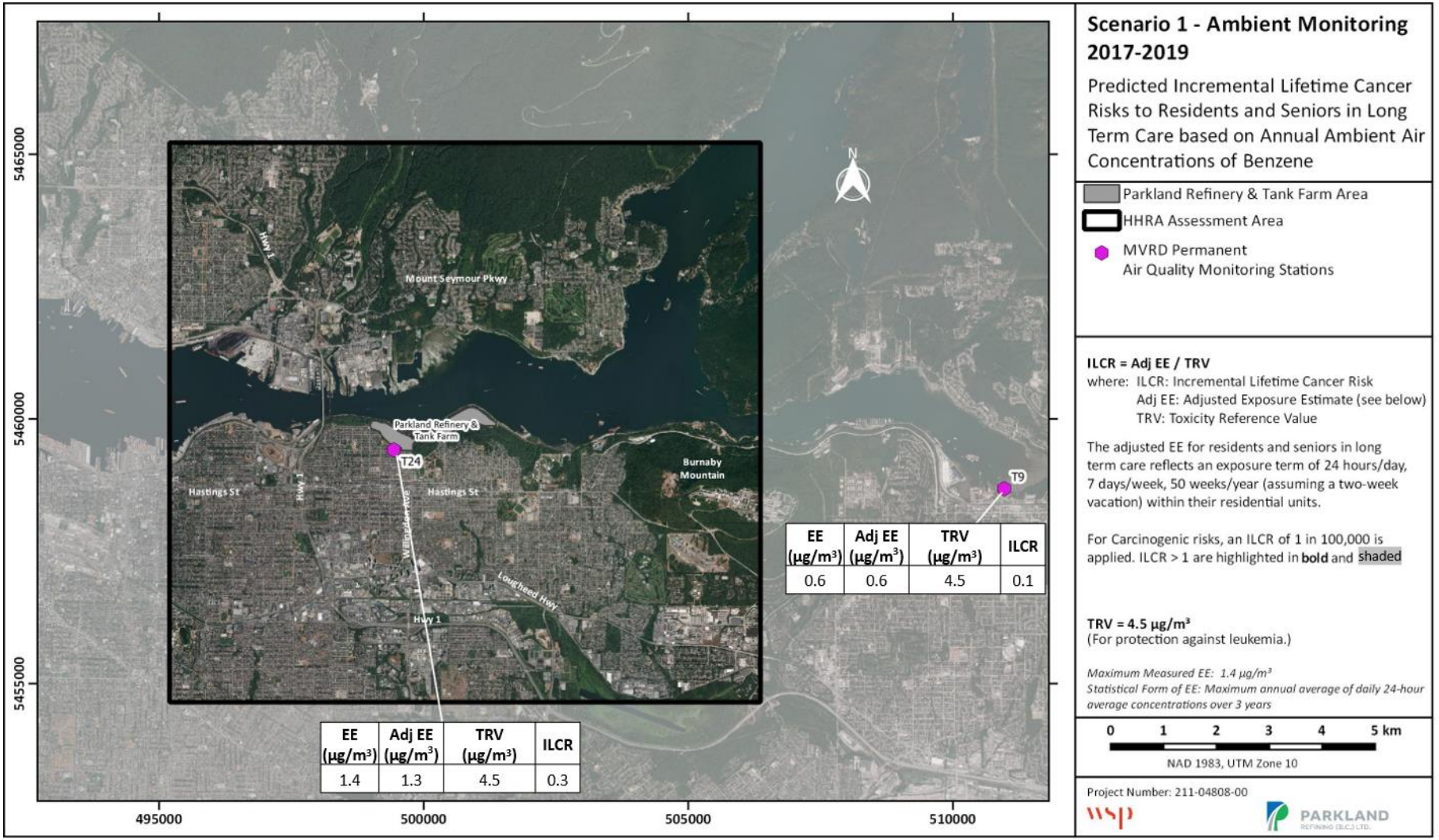


Figure 6-56: Scenario 1 – Predicted Incremental Lifetime Cancer Risk to Residents and Seniors in Long Term Care Based on Annual Ambient Air Concentrations of Benzene

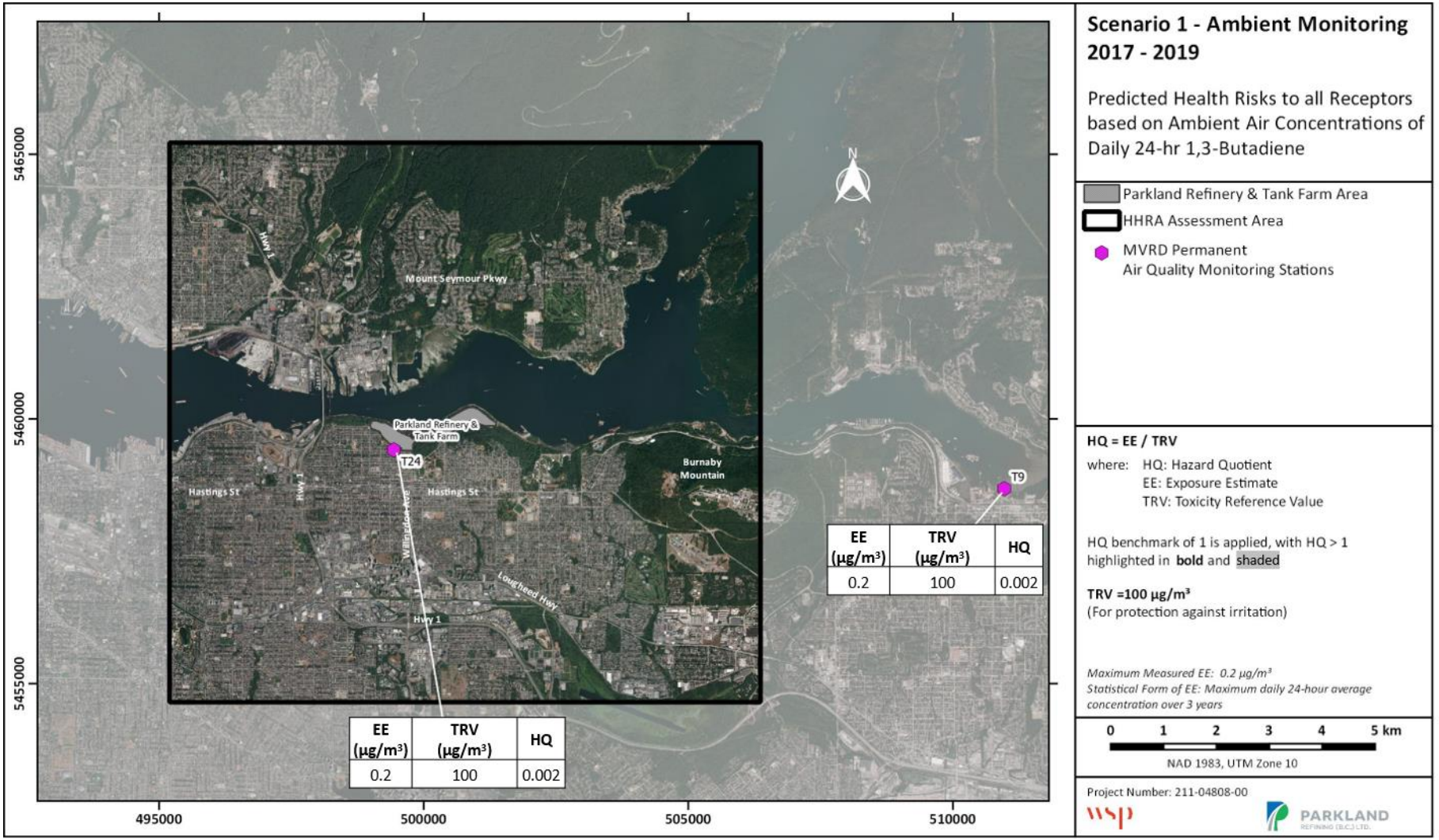


Figure 6-57: Scenario 1 – Predicted Health Risks to All Receptors Based on Ambient Air Concentrations of Daily 24-hr 1,3-Butadiene

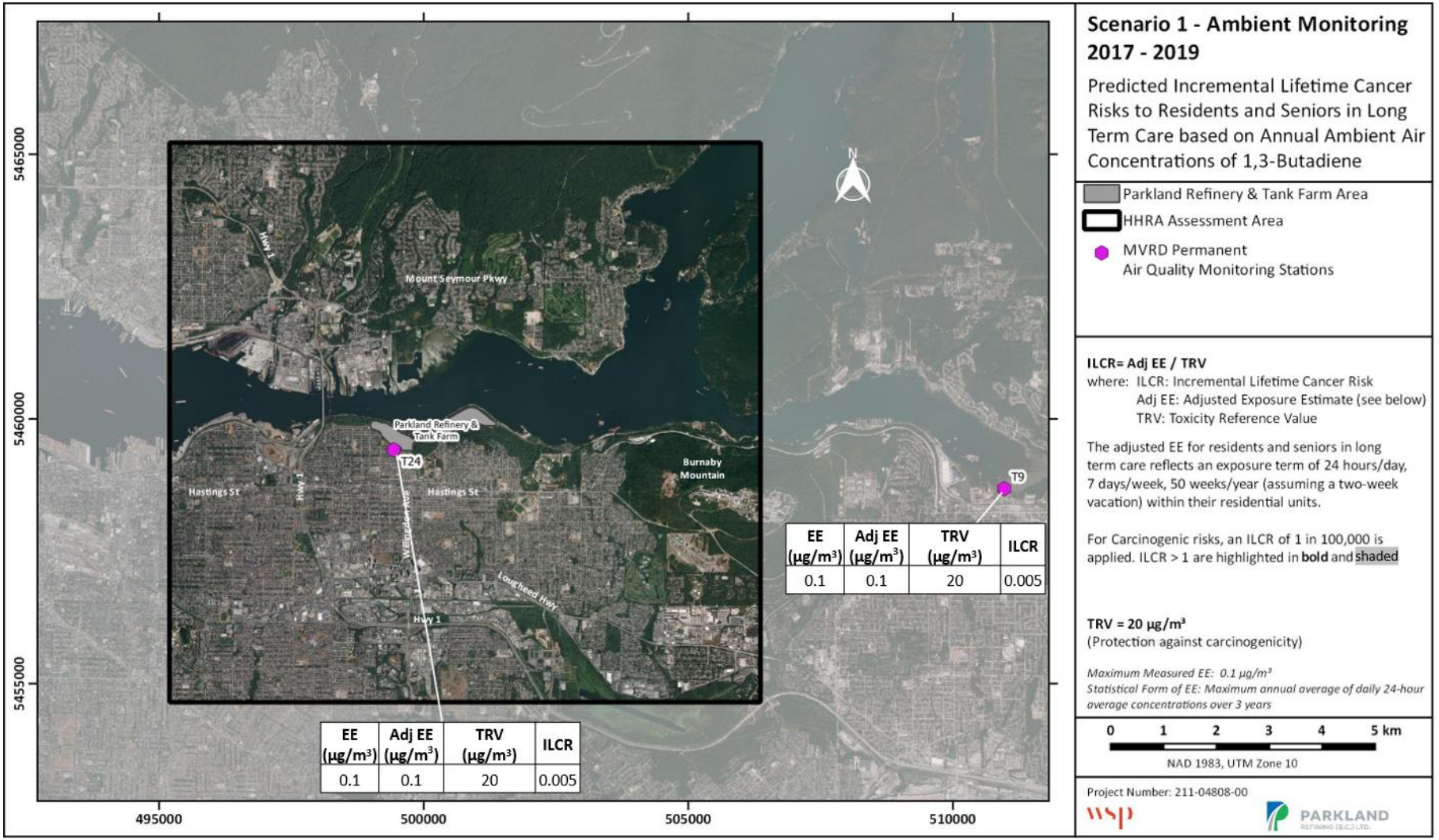


Figure 6-58: Scenario 1 – Predicted Incremental Lifetime Cancer Risks to Residents and Seniors in Long Term Care Based on Annual Ambient Air Concentrations for 1,3-Butadiene

6.4 CHANGE IN AIR QUALITY WITHIN STUDY AREA

Predicted air quality within the study area will benefit from an overall reduction in NO_2, SO_2, and $PM_{2.5}$ refinery emissions compared to the current permitted maximums (Scenario 2) as a result of the planned operational and capital upgrades to the refinery. This change results in a net benefit with respect to the health of the community as well as individual human receptors residing or spending time within the HHRA study area.

Below, **Table 6-15** to **Table 6-20** present the dispersion model results and net decrease for short-term (1-hr or 24-hr) and annual predicted concentrations of SO_2, NO_2, and $PM_{2.5}$ at the maximum point of impingement location.

Table 6-15 Percent Change in Maximum Predicted 1-hour SO_2 Concentrations Relative to Current Permit Maximum for Amended Permit Scenarios

Scenario	Predicted Maximum Conc. From Refinery (µg/m³)	% Change from Current Permit Maximum	Maximum Cumulative Conc. (µg/m³)	% Change from Current Permit Maximum
2 - Current Permit Maximum	488.2	-	495.6	-
3 - Amended Permit Maximum	237.2	**51% decrease**	244.6	**51% decrease**
4 - Amended Permit Normal	107.4	**78% decrease**	114.7	**77% decrease**

Table 6-16 Percent Change in Maximum Annual SO_2 Concentrations Relative to Current Permit Maximum for Amended Permit Scenarios

Scenario	Predicted Maximum Conc. From Refinery (µg/m³)	% Change from Current Permit Maximum	Maximum Cumulative Conc. (µg/m³)	% Change from Current Permit Maximum
2 - Current Permit Maximum	10.1	-	11.3	-
3 - Amended Permit Maximum	3.2	**68% decrease**	4.5	**60% decrease**
4 - Amended Permit Normal	2.2	**78% decrease**	3.4	**70% decrease**

Table 6-17 Percent Change in Maximum Predicted 1-hour NO_2 Concentrations Relative to Current Permit Maximum for Amended Permit Scenarios

Scenario	Predicted Maximum Conc. From Refinery (µg/m³)	% Change from Current Permit Maximum	Maximum Cumulative Conc. (µg/m³)	% Change from Current Permit Maximum
2 - Current Permit Maximum	105.6	-	180.3	-
3 - Amended Permit Maximum	103.1	**2% decrease**	177.8	**1% decrease**
4 - Amended Permit Normal	95.2	**10% decrease**	169.9	**6% decrease**

Table 6-18 Percent Change in Maximum Annual NO_2 Concentrations Relative to Current Permit Maximum for Amended Permit Scenarios

Scenario	Predicted Maximum Conc. From Refinery (µg/m³)	% Change from Current Permit Maximum	Maximum Cumulative Conc. (µg/m³)	% Change from Current Permit Maximum
2 - Current Permit Maximum	3.9	-	25.8	-
3 - Amended Permit Maximum	3.1	**20% decrease**	25.1	**3% decrease**
4 - Amended Permit Normal	2.0	**50% decrease**	24.0	**7% decrease**

Table 6-19 Percent Change in Maximum Predicted 24-hour $PM_{2.5}$ Concentrations Relative to Current Permit Maximum for Amended Permit Scenarios

Scenario	Predicted Maximum Conc. From Refinery (µg/m³)	% Change from Current Permit Maximum	Maximum Cumulative Conc. (µg/m³)	% Change from Current Permit Maximum
2 - Current Permit Maximum	8.0	-	19.7	-
3 - Amended Permit Maximum	6.4	**20% decrease**	18.1	**8% decrease**
4 - Amended Permit Normal	2.0	**75% decrease**	13.7	**30% decrease**

Table 6-20 Percent Change in Maximum Annual $PM_{2.5}$ Concentrations Relative to Current Permit Maximum for Amended Permit Scenarios

Scenario	Predicted Maximum Conc. From Refinery (µg/m³)	% Change from Current Permit Maximum	Maximum Cumulative Conc. (µg/m³)	% Change from Current Permit Maximum
2 - Current Permit Maximum	0.7	-	5.5	-
3 - Amended Permit Maximum	0.6	**14% decrease**	5.4	**2% decrease**
4 - Amended Permit Normal	0.3	**57% decrease**	5.1	**7% decrease**

6.5 UNCERTAINTY ANALYSIS

Conducting a risk assessment involves many steps within the process and assumptions are made at each stage to account for the lack of scientific data pertaining to the given project. Due to the application of these assumptions, uncertainty is inherently involved in the process. However, as discussed above in **Sections 3.4, 4.4,** and **5.4** these assumptions are considered to be conservative and result in an overestimation of the true risk.

The following sources of uncertainty in the HHRA are noted:

- The general conservativeness of regulatory dispersion modelling predictions is discussed in the AQA report (WSP, 2021).
  - Ambient air concentrations recorded at MVRD stations in effect capture current operational contributions of the refinery's emissions. In developing the baseline values for the HHRA Scenarios 2, 3, and 4, WSP endeavored to select stations that show a minimum direct impact of refinery emissions. However, some refinery influence on baseline stations is still likely, and given that modelled/predicted refinery contributions are being added to baseline values in HHRA Scenarios 2, 3, and 4, this potential double count of refinery contributions likely results in a conservative assessment.
  - Typical operation of the refinery at fully permitted emission rates for all sources simultaneously is not viable and thus very unlikely to occur. This "maximum" scenario acts as a conservative upper bounding case that is not representative of how the refinery operates. The use of conservative exposure estimates further compounds the conservative nature of the predicted risks. As such, predicted risks based on a maximum modelled scenario are likely to overestimate actual risks to human receptors.
- Human exposure to co-pollutants remains the major source of uncertainty in the overall health database for air pollutants.

  With respect to SO_2:
  - The potential confounding health effects by co-pollutants such as $PM_{2.5}$ in epidemiology studies remains a major uncertainty; for this reason, the LOAEC for lung function decrements was solely based on controlled human exposure studies.

- While Health Canada (2016) examined long-term epidemiological studies, a chronic (annual) TRV was not adopted largely due to the inconsistency across studies and inability to distinguish potential confounding by co-pollutants, as well as uncertainties regarding geographic scale of analysis. As such, an assessment of risk was not completed for long-term exposures.
- Health based 1-hour AAQOs are available from other jurisdictions that are higher than the value adopted as part of this assessment (40 ppb/106 µg/m^3); however, these exposure limits are either dated and/or documentation describing the technical basis or derivation of the standards are lacking. As such, it is not possible to confirm whether exposure limits from other jurisdictions are adequately protective of human health.

With respect to NO_2:

- As discussed in **Section 5.2**, human epidemiology studies are observational rather than experimental, and hence there can be uncertainty as to whether the effects reported in the epidemiology studies are in fact due to ambient NO_2 alone (Health Canada, 2016).
- This uncertainty also applies to hospital admissions and emergency room visits as health endpoints because it is challenging to separate the effect of each air pollutant. Based on the supporting science behind the asthma emergency room visit endpoint (TRV of 79 µg/m^3), risk estimates may be over-conservative and may "double-count" impacts of other pollutants.
- This same uncertainty also applies to long-term exposure to NO_2 levels from traffic-related exposures as co-pollutant models adjusting for other key traffic-related air pollutants such as carbon monoxide or ultrafine particulates have not been performed.

With respect to $PM_{2.5}$:

- Considerable uncertainty remains as to which of the PM fractions are responsible for eliciting certain health effects. For instance, the extent to which $PM_{2.5}$ may also contribute to the health effects observed as a result of exposure to coarse PM is an important source of uncertainty.
- Some acute- and chronic- health based standards from other jurisdictions are higher than the values adopted as part of this assessment; however, these exposure limits are either dated and/or documentation describing the technical basis or derivation of the standards are lacking. As such, it is not possible to confirm whether exposure limits from other jurisdictions are adequately protective of human health.

With respect to benzene and 1,3-butadiene:

- Modelled data were not utilized for these COPCs as the estimation of risk was based solely on ambient air measurements. Confounding exposures to other chemicals, and potential for significant exposures via indoor sources and/or cigarette smoke (particularly for 1,3-butadiene) as detailed in **Section 5.2**, are important sources of uncertainty.

The risks identified in **Section 6.3** are therefore, considered theoretical (i.e., there is the potential for risk, but there is some uncertainty as to whether adverse effects would be evident in the human receptors when exposed to the predicted concentrations).

7 DEVELOPMENT AND IMPLEMENTATION OF MITIGATION MEASURES

Based on previous air quality assessments conducted by WSP for Parkland Refinery, as well as the results of this HHRA, a key focus area for the mitigation of refinery related air quality impacts and their associated health risks is the reduction of SO_2 emissions. The refinery is the primary source of SO_2 within the HHRA study area and is the dominant influence on ambient SO_2 levels throughout the study area. As such, key mitigation measures and monitoring actions are as follows:

- Continued reduction of SO_2 emissions from key sources including the FCC and SRU. The emissions reductions incorporated into Scenarios 3 and 4 (45% reduction for amended permit maximum relative to current permit maximum) in the HHRA will lead to significant reductions in the extent and frequency of elevated SO_2 levels, and their associated respiratory health risks.
- Improved SO_2 monitoring coverage, particularly on the North Shore of Burrard Inlet. Parkland is working with Metro Vancouver on the installation of a new permanent SO_2 monitoring station there, as well as the addition of routine SO_2 monitoring to Metro Vancouver's Burnaby Mountain (T14) monitoring station.
- Continued utilization of the existing SO_X Curtailment Event provision in Parkland's air permit (GVA0117), whereby Parkland is required to increase the use of FCC sulphur scavenging catalyst during periods of elevated SO_2 levels (190 ppb 10 min average, 70 ppb 1 hr average) at monitoring stations near the refinery.
- Continued proactive increase of FCC sulphur scavenging catalyst during temperature inversion events to help avoid elevated ambient levels that may lead to the triggering of a SO_X Curtailment Event.

The HHRA also indicates that there is the potential to exceed short term NO_2 air quality objectives for a limited area immediately beside the refinery when considering the impact of predicted refinery emissions alone, and over a larger area around the refinery when considering the cumulative impact of refinery emissions together with background NO_2 levels from all regional emission sources. Because baseline / non-refinery sources are the driver of concentrations and associated health risks throughout the HHRA study area, the potential for refinery NO_x emissions reductions to affect broad reductions in NO_2-related to health risks is limited at this time. It is noted that NO_2 is considered to be a non-threshold contaminant, and as such, key mitigation measures and monitoring actions at the refinery related to NO_2 are as follows:

- Continued reduction of NO_x emissions from key sources including the FCC and COB. The reductions incorporated into Scenarios 3 and 4 (18% reduction for amended permit maximum relative to current permit maximum) in the HHRA will lead to modest reductions in the extent and frequency of elevated NO_2 levels very near the refinery, along with their associated respiratory health risks.
- Improved NO_2 monitoring coverage, particularly on the North Shore of Burrard Inlet. Parkland is working with Metro Vancouver on the installation of a new permanent NO_2 monitoring station there, as well as the additional NO_2 monitoring at Metro Vancouver's Burnaby North McGill Park (T24) and Burnaby Capitol Hill (T23) monitoring stations.

The HHRA indicates that health-based objective levels for $PM_{2.5}$ were not exceeded at any of the ambient monitoring stations, or anywhere throughout the HHRA study area for $PM_{2.5}$ levels modelled under Scenarios 2 to 4. However, $PM_{2.5}$ is considered to be a non-threshold contaminant, which means that there may be some level of health risk associated with any level of exposure. As such, further mitigation measures and monitoring actions are justified, and key actions are as follows:

- Continued reduction of $PM_{2.5}$ emissions from the key refinery source: the FCC. The reductions incorporated into Scenarios 3 and 4 (23% reduction for amended permit maximum relative to current permit maximum) in the HHRA will lead to modest reductions in $PM_{2.5}$ levels very near the refinery, along with their associated health risks.

- Improved $PM_{2.5}$ monitoring coverage, particularly on the North Shore of Burrard Inlet. Parkland is working with Metro Vancouver on the installation of a new permanent $PM_{2.5}$ monitoring station there, as well as the additional $PM_{2.5}$ monitoring at Metro Vancouver's North Burnaby McGill Park (T24) and Burnaby Capitol Hill (T23) monitoring stations.

The HHRA indicates that health based objective levels for 1,3-butadiene and benzene were not exceeded at any of the ambient monitoring stations included in the HHRA. However, these COPCs are considered to be non-threshold contaminants, which means that there may be some level of health risk associated with any level of exposure. As such, further mitigation measures and monitoring actions are justified, and key actions are as follows:

- Ongoing operation of existing fugitive VOC management programs, including the existing refinery Leak Detection and Repair Program ("LDAR").
- Expansion of the existing LDAR program in line with ECCC regulations, increasing leak surveys to 3 times per year, with an approximately 25% increase in components being monitored, and significant capital investment to improve equipment seals such as compressors and pressure relief valves.
- Implementation of a fenceline VOC monitoring program compliant with ECCC regulations. This will provide significant additional detail regarding the spatial variability in VOC levels around the refinery fenceline, enhancing the understanding of how these levels may vary relative to the levels measured at the existing VOC monitoring site at Metro Vancouver's Burnaby North McGill Park (T24) station. This monitoring may also be useful for identifying abnormal emissions of VOCs.
- Future revision of the existing HHRA to incorporate fenceline VOC monitoring data once at least two years of monitoring has been completed and validated.

In addition to the COPC-specific mitigation measures detailed above, the following general key mitigation measures and monitoring actions are also underway or planned for future implementation:

- Ongoing engagement with community stakeholders, including the Community Advisory Panel ("CAP"), on questions and concerns related to refinery air quality and human health impacts.
- Ongoing engagement with Tsleil-Waututh Nation, on questions and concerns related to refinery air quality and human health impacts.

8 SUMMARY AND CONCLUSIONS

The HHRA evaluated the potential health risks associated with short-term and long-term exposures to ambient concentrations of identified COPCs that may be influenced by emissions from the refinery. To achieve this objective, WSP evaluated the source-pathway-receptor linkage based on possible interactions with human receptors within a 10 km x 10 km study area centered on the refinery (**Figure 1-2**). Exposure concentrations were provided by both air quality monitoring data (Scenario 1) and AQA dispersion modelling outputs (Scenarios 2-4).

The COPCs evaluated in the HHRA included criteria air contaminants: SO_2, NO_2, and $PM_{2.5}$ as well as two VOCs (benzene and 1,3-butadiene) that were previously identified as posing potential health risks near the refinery. The identified COPCs in this HHRA are consistent with those evaluated in previous 2002 and 2013 health assessments.

The human receptors evaluated in the HHRA were identified based on land uses within the HHRA study area and included the following receptor groups:

- Residents, including Tsleil-Waututh Nation residential communities;
- Elderly residents in long-term care facilities;
- Young children and toddlers in childcare facilities;
- Children and teens in school;
- Adult patients in hospital facilities;
- Adult workers at workplaces near the refinery;
- Individuals who use the nearby recreational areas (including trails) or otherwise visit the study area for other short-term trips, and;
- Members of Tsleil-Waututh Nation who make use of Reserve Lands near the facility for outdoor cultural activities.

The HHRA assessed the short-term (acute) and long-term (chronic) health effects associated with each COPC and determined the health-protective dose for each averaging duration (i.e., acute and chronic) that a receptor can be exposed to without experiencing harmful health effects (i.e., the TRV). To establish a COPC-specific TRV, a comprehensive review of available ambient air exposure limits established by regulatory and health agencies was completed for all identified COPCs. Health-based TRVs were selected for each COPC and averaging period, if available, based on information obtained during this review.

The HHRA also summarised existing community health information based on publicly available data as provided by the BC Centre for Disease Control (see **Section 3.2.1**). Health profiles for communities near the refinery including Burnaby Northwest and North Vancouver DM - East were reviewed. Key health information for 2015 to 2018, for above noted communities, indicated that crude incidences rates, and age-standardized incidence and prevalence rates for cancer (including leukemia) and various chronic diseases including asthma and COPD were below provincial averages.

The findings of the HHRA for identified short-term (acute) and long-term (chronic) health endpoints are summarized below.

SO_2:

- The short-term health risks associated with lung function decrement (TRV of 106 µg/m^3) were evaluated in the HHRA. Long-term health risks were not evaluated as there was insufficient evidence supporting a causal relationship between long-term exposures to SO_2 and respiratory effects.
- The refinery is currently the largest source of regional SO_2 emissions and is the driver of associated human exposures in the study area.
    - Air quality monitoring data for 2017-2019 (Scenario 1) indicates that only the Burnaby Capitol Hill (T23) monitoring station near the refinery shows a HQ greater than 1.0, with a maximum measured HQ of 3.05. Analysis of the underlying hourly data indicates that over the 3-year monitoring period, the HQ exceeded 1.0 for a total of 25 hours, with only 2 hours exceeding an HQ of 2. The number of

hours exceeding decreased from 18 in 2017 to 1 in 2019, corresponding with Parkland's increased usage of SO_2 reduction additive starting in 2018.

- For Scenario 2 - Current Permit Maximum, health risks are above acceptable levels (HQ>1.0) for a very small number of short duration events. The total number of hours predicted to exceed the Target HQ of 1.0 at each of the maximally exposed receptors ranged from 0 hours (maximum hospital receptor and maximum senior care receptor) to 9 hours (maximum recreational receptor near the refinery). Note that 9 hours represents a very small proportion of the year (0.1%).
- For Scenario 3 - Amended Permit Maximum, health risks are below acceptable levels (HQ<1.0) for all receptors but a single recreational location very near the refinery. Only 2 hours per year are predicted to exceed the Target HQ of 1.0 at this location.
- For Scenario 4 - Amended Permit Normal, health risks are below acceptable levels (HQ<1.0) for all receptors.
- Based on the dispersion modelling results, the significant reduction in refinery SO_2 emissions due to the permit amendment (45% reduction for Scenario 3 relative to Scenario 2) lead to a similar significant reduction in SO_2 concentrations, virtually eliminating the spatial extent of cumulative concentrations resulting in a HQ of greater than 1.0 under the amended permit scenarios.

NO_2:

- Two health endpoints were evaluated in the HHRA: asthma emergency room visit (TRV of 79 µg/m^3) and airway hyper-responsiveness (TRV of 113 µg/m^3). Based on the supporting science behind the asthma emergency room visit endpoint, risk estimates may be over-conservative and potentially "double-count" impacts of other pollutants.
- Within the 10 km x 10 km study area surrounding the refinery, background or non-refinery sources are the largest contributors to NO_x emissions and associated human exposures.
- Air quality monitoring data (Scenario 1) indicates that none of the monitoring stations included in the study area shows an HQ greater than 1.0 for either of the acute TRV, and a single station relatively distant from the refinery (Port Moody – T9) shows a HQ slightly greater than 1.0 for the chronic TRV. It is important to note that the HQs are similarly high across the MVRD monitoring stations within the HHRA study area, indicating consistent maximum NO_2 concentrations throughout the study area, regardless of the distance from the refinery to the monitoring stations. This suggests that baseline / non-refinery NO_2 sources such as on road vehicles, space heating and marine sources are the driver of concentrations and associated NO_2 health risks throughout the study domain.
- Air quality modelling results (Scenarios 2, 3 and 4) indicate that baseline "non-refinery" NO_2 health risks are below the target HQ of 1.0 for acute exposures, but account for 50 to 96% of the estimated cumulative risk for maximum sensitive receptors. When "refinery + background" air concentrations are considered, health risks are above acceptable levels (HQ>1.0) for 8-15% of the year over a relatively broad area, driven largely by background concentrations. For chronic exposures, baseline "non-refinery" health risks exceed the target HQ of 0.2 for two receptor groups (daycare and workers).
- These significant baseline risks identified are beyond the control of the refinery and means that the 18% refinery NO_x emissions reduction from Scenario 2 to Scenario 3 only results in a modest reduction in NO_2 exposures and associated health risks in the HHRA study area.

$PM_{2.5}$:

- The health risks associated with excess morbidity or mortality (TRV of 25 µg/m^3) and excess mortality (TRV of 10 µg/m^3) following short-term and long-term exposures to $PM_{2.5}$, respectively, were evaluated in the HHRA.
- Health risks associated with acute 24-hr and long-term $PM_{2.5}$ exposures are below acceptable levels (HQ<1.0) for all scenarios for all receptors. Given that none of the cumulative HQs exceeded target levels, no risks to sensitive receptors beyond acceptable levels are anticipated as a result of exposure to maximum predicted 24-hour $PM_{2.5}$ concentrations

- As for NO_2, baseline $PM_{2.5}$ concentrations account for the majority of the cumulative acute and chronic risk within the HHRA study area for all receptors evaluated. Baseline contributions to the cumulative risk range from 67 to 97% within the HHRA study area based on modelled results.

BENZENE

- The health risks associated with hematopoietic (blood) effects (TRV of 30 µg/m^3) and leukemia (TRV of 4.5 µg/m^3) following short-term and long-term exposures to benzene, respectively, were evaluated in the HHRA.
- Air quality monitoring data for 2017-2019 (Scenario 1) indicate that short term exposures do not result in a HQ greater than 1.0, and long-term exposures do not result in an ILCR greater than 1 in 100,000. As a result, there are no health risks beyond acceptable levels associated with acute 24-hr exposures and long-term exposures to benzene.

1,3-BUTADIENE

- The health risks associated with irritation effects (TRV of 100 µg/m^3) and cancer (TRV of 20 µg/m^3) following short-term and long-term exposures to 1,3-butadiene, respectively, were evaluated in the HHRA.
- Air quality monitoring data for 2017-2019 (Scenario 1) indicate that short term exposures do not result in a HQ greater than 1.0, and long-term exposures do not result in an ILCR greater than 1 in 100,000. As a result, there are no health risks beyond acceptable levels associated with acute 24-hr exposures and long-term exposures to 1,3-butadiene for all scenarios for all receptors.

UNCERTAINTY

Conducting a risk assessment involves many steps within the process and assumptions are made at each stage to account for the lack of scientific data pertaining to the given assessment. Due to the application of these assumptions, uncertainty is inherently involved in the process, but risk assessment frameworks are inherently conservative, so the impact of assumptions typically results in an overestimation of the true risk by design.

Key sources of uncertainty that result in overestimates of risk for this study are as follows:

- Ambient air concentrations recorded at MVRD stations in effect capture current operational contributions of the refinery's emissions. In developing the baseline values for the AQA and HHRA, WSP endeavored to select stations that show a minimum direct impact of refinery emissions. However, some refinery influence on baseline stations is still likely, and given that modelled/predicted refinery contributions are being added to baseline values in HHRA Scenarios 2, 3, and 4, this potential double count of refinery contributions is expected to result in a conservative assessment.
- Typical operation of the refinery at fully permitted emission rates for all sources simultaneously (Scenarios 2 and 3) is not operationally viable and thus very unlikely to occur. These "maximum" scenarios act as a conservative upper bounding case that are not representative of how the refinery operates. The use of conservative exposure estimates further compounds the conservative nature of the predicted risks. As such, predicted risks based on these maximum modelled scenarios are likely to overestimate actual risks to human receptors.
- Human exposure to co-pollutants remains the major source of uncertainty in the development of TRVs for air pollutants. As s result, the TRVs for NO_2, SO_2 and $PM_{2.5}$ are all likely conservative.

MITIGATION MEASURES & RECOMMENDATIONS

Based on previous air quality assessments conducted by WSP for Parkland, as well as the results of this HHRA, key mitigation actions are as follows:

- Continued reduction of SO_2 emissions from key sources including the FCC and SRU. The emissions reductions incorporated into Scenarios 3 and 4 (45% reduction for amended permit maximum relative to current permit maximum) in the HHRA will lead to significant reductions in the extent and frequency of elevated SO_2 levels, and their associated respiratory health risks.
- Continued reduction of NO_x emissions from key sources including the FCC and COB. The reductions incorporated into Scenarios 3 and 4 (18% reduction for amended permit maximum relative to current permit

maximum) in the HHRA will lead to modest reductions in the extent and frequency of elevated NO_2 levels very near the refinery, along with their associated respiratory health risks.

- Continued reduction of $PM_{2.5}$ emissions from the key refinery source: the FCC. The reductions incorporated into Scenarios 3 and 4 (23% reduction for amended permit maximum relative to current permit maximum) in the HHRA will lead to modest reductions in $PM_{2.5}$ levels very near the refinery, along with their associated health risks.
- Improved monitoring coverage for SO_2, NO_2 and $PM_{2.5}$ throughout the HHRA study area with the addition of a new Parkland-funded permanent MVRD monitoring location on the north shore of Burrard Inlet and addition to monitors to existing MVRD stations. For VOCs, leverage the fenceline VOC monitoring installed in early 2022 to better characterize near-site VOC levels.
- Continued utilization of operation and maintenance programs focused on emissions control, including the SO_X Curtailment Event procedure, FCC sulphur scavenging catalyst inversion event procedure, and VOC LDAR program.
- Ongoing engagement with community stakeholders, including the CAP, on questions and concerns related to refinery air quality and human health impacts.
- Ongoing engagement with Tsleil-Waututh Nation, on questions and concerns related to refinery air quality and human health impacts.

In addition to these mitigation measures, WSP recommends updates of this HHRA in support of future permit amendments for the refinery that result in significant changes to emissions.

REFERENCES

- Alberta Health. 2018. Inventory and Analysis of Exposure Factors for Alberta (January 2018). Edmonton, AB: Public Health and Compliance, Alberta Health Retrieved from https://open.alberta.ca/publications/9781460135914
- BC Cancer Registry, Provincial Health Services Authority. 2021a. Cancer Incidence Rates (per 100,000 population), British Columbia, 2018. http://www.bccancer.bc.ca/statistics-and-reports-site/Documents/Crude_Incidence_Rates_Report_2018_20210204.pdf
- BC Cancer Registry, Provincial Health Services Authority. 2021b. Cancer Mortality Rates (per 100,000 population), British Columbia, 2018. http://www.bccancer.bc.ca/statistics-and-reports-site/Documents/Crude_Mortality_Rates_Report_2018_20210304.pdf
- BC Centre for Disease Control. 2020a. British Columbia Community Health Service Area Health Profile (Version 1.0). 2221 Burnaby Northwest. http://communityhealth.phsa.ca/CHSAHealthProfiles/PdfGenerator/Burnaby%20Northwest
- BC Centre for Disease Control. 2020b. British Columbia Community Health Service Area Health Profile (Version 1.0). 3314 North Vancouver DM – East. http://communityhealth.phsa.ca/CHSAHealthProfiles/PdfGenerator/North%20Vancouver%20DM%20-%20East
- British Columbia Ministry of Environment & Climate Change Strategy. 2020. Dustfall Monitoring and Pollution Control Objectives. Technical Guidance. Regional Operations Branch. GUI-TEC-04.1. June 2020.
- British Columbia Ministry of Environment & Climate Change Strategy. 2021. British Columbia Air Quality Dispersion Modelling Guideline. https://www2.gov.bc.ca/assets/gov/environment/air-land-water/air/reports-pub/bc_dispersion_modelling_guideline_2021.pdf
- British Columbia Ministry of Health. 2021. British Columbia Guidance for Prospective Human Health Risk Assessment, Version 1.0. April 2021.
- Canadian Medical Association Journal (CMAJ) 2020 June 29; 192:E694-701.
- Fraser Health Authority & Metro Vancouver. 2013. Air Quality & Health Impact Assessment Update – Chevron CAP
- Health Canada. 2012. Federal Contaminated Site Risk Assessment in Canada, Part I: Guidance on Human Health Preliminary Quantitative Risk Assessment (PQRA), Version 2.0. Ottawa, ON: Contaminated Sites Division, Safe Environments Directorate, Health Canada Retrieved from http://publications.gc.ca/collections/collection_2018/sc-hc/H128-1-11-632-eng.pdf
- Kennedy, S.M. et al. 2002. Air Emissions from the Chevron North Burnaby Refinery: Human Health Impact Assessment: Final Report
- Richardson, G. M., & Stantec Consulting Ltd. 2013. 2013 Canadian Exposure Factors Handbook. In. Saskatoon, SK: Toxicology Centre, University of Saskatchewan.
- WSP, 2021. Refined Technology Assessment for Non-Capital Solutions – Supporting Air Quality Assessment for Parkland Refining (B.C.) Ltd. August 2021.

Sulphur Dioxide (SO_2):

- Alberta Environment (AENV). 2011. Alberta Ambient Air Quality Objectives. Ambient Air Quality Objectives | Alberta.ca
- Alberta Health and Wellness (AHW). 2006. Health Effects Associated with Short-term Exposure to Low Levels of Sulphur Dioxide (SO_2) – A Technical Review.
- B.C. Ministry of Environment and Climate Change Strategy (BC MoECCS). 2020. Provincial Air Quality Objective Information Sheet. https://www2.gov.bc.ca/assets/gov/environment/air-land-water/air/reports-pub/prov_aqo_fact_sheet.pdf
- California Office of Environmental Health Hazard Assessment (Cal OEHHA). 2008. Appendix D.2 Acute RELs and toxicity summaries using the previous version of the Hot Spots Risk Assessment guidelines.

- Canadian Council of Ministers of the Environment (CCME). 2017. Canada's Air. https://ccme.ca/en/air-quality-report#slide-1
- CCME. 2020. Guidance Document on Achieving Determination for Canadian Ambient Air Quality Standards for Sulphur Dioxide.
- CCME. 2021. Air Quality Report. https://www.ccme.ca/en/air-quality-report
- Health Canada. 2016. Human Health Risk Assessment for Sulphur Dioxide. CAS RN: 7446-09-05.
- International Agency for Research on Cancer (IARC). 1992. IARC Monographs on the Evaluation of Carcinogenic Risks to Humans. Volume 54 Occupational Exposures to Mists and Vapours from Strong Inorganic Acids; and Other Industrial Chemicals.
- Metro Vancouver Ambient Air Quality Objectives, updated January 2020. http://www.metrovancouver.org/services/air-quality/AirQualityPublications/CurrentAmbientAirQualityObjectives.pdf
- Ontario Ministry of the Environment, Conservation and Parks (MECP). 2020. Ambient Air Quality Criteria. Human Toxicology and Air Standards Section. Technical Assessment and Standards Development Branch.
- United States Environmental Protection Act (US EPA). 2008. Integrated Science Assessment (ISA) for Sulfur Oxides-Health Criteria. https://www.epa.gov/isa/integrated-science-assessment-isa-sulfur-oxides-health-criteria
- US EPA. 2010. Primary National Ambient Air Quality Standard for Sulfur Dioxide; Final Rule. Federal Register / Vol. 75, No. 119.
- US EPA. 2019. Review of the Primary National Ambient Air Quality Standards for Sulfur Oxides. Federal Register / Vol. 84, No. 52.
- World Health Organization (WHO). 2005. Air Quality Guidelines. Global Update.

Nitrogen Dioxide (NO_2):

- Alberta Environment (AENV). 2007. Assessment Report on Nitrogen Dioxide for Developing Ambient Air Quality Objectives. https://open.alberta.ca/dataset/c1d93f22-b949-4b7e-9504-d6f87dc7b891/resource/862c1d97-38bd-4c27-a5e2-d003f7a6b4f8/download/2007-assessmentreport-nitrogendioxide-oct2007.pdf.
- AENV. 2011. Alberta Ambient Air Quality Objectives. https://open.alberta.ca/dataset/836cdc25-935a-426b-8f95-d89506679ff1/resource/c6c530d5-c03a-4d8d-932f-b3198eaaff48/download/2011-AAQO-NitrogenDioxide-Jun2011.pdf
- AENV. 2019. Alberta Ambient Air Quality Objectives and Guidelines Summary. AEP, Air Policy, 2016, No. 2. January 2019. https://open.alberta.ca/publications/9781460134856
- B.C. Ministry of Environment and Climate Change Strategy (BC MoECCS). 2020. Provincial Framework for Developing Provincial Air Quality Objectives. https://www2.gov.bc.ca/assets/gov/environment/air-land-water/air/reports-pub/provincial_framework_for_developing_provincial_air_quality_objectives_-_info_sheet.pdf.
- BC MoECCS. 2021. Provincial Air Quality Objectives for Nitrogen Dioxide. Policy Intentions Paper 2021. https://www2.gov.bc.ca/assets/gov/environment/air-land-water/air/reports-pub/env_no2_intentionpaper.pdf
- California Air Resources Board (CARB). 1992. Review of the one-hour ambient air quality standard for nitrogen dioxide technical support document. Sacramento: State of California Air Resources Board Technical Support Division; December 1992.
- California Office of Environmental Health Hazard Assessment (Cal OEHHA). 2008. Appendix D.2 Acute RELs and toxicity summaries using the previous version of the Hot Spots Risk Assessment guidelines.
- Canadian Council of Ministers of the Environment (CCME). 2017. Canada's Air. https://ccme.ca/en/air-quality-report#slide-1
- Health Canada (Health Canada) 2016. Human Health Risk Assessment for Ambient Nitrogen Dioxide, Water and Air Quality Bureau, Safe Environments Directorate, Healthy Environments and Consumer Safety Branch, Health Canada Available At: https://www.canada.ca/en/health-canada/services/publications/healthy-living/human-health-risk-assessment-ambient-nitrogen-dioxide.html

- Metro Vancouver Ambient Air Quality Objectives, updated January 2020. http://www.metrovancouver.org/services/air-quality/AirQualityPublications/CurrentAmbientAirQualityObjectives.pdf
- Ontario Ministry of the Environment, Conservation and Parks (MECP). 2020. Ambient Air Quality Criteria. Human Toxicology and Air Standards Section. Technical Assessment and Standards Development Branch.
- US EPA (United States Environmental Protection Agency). 1993. Air Quality Criteria for Oxides of Nitrogen. EPA 600/8-91-049Bf. August 1993.
- US EPA. 1996. 40 CFR Part 50. Vol 61. NO. 196. 52852. October 8, 1996. National Ambient Air Quality Standards for Nitrogen Dioxide: Final Decision.
- US EPA. 2008. Integrated Science Assessment (ISA) for Nitrogen Dioxide – Health Criteria. https://www.epa.gov/isa/integrated-science-assessment-isa-nitrogen-dioxide-health-criteria.
- US EPA. 2012. Integrated Risk Information System (IRIS) database on-line search. Cincinnati, OH: United States Environmental Protection Agency. Available at: http://cfpub.epa.gov/ncea/iris/index.cfm?fuseaction=iris.showSubstanceList&list_type=alpha&view
- US EPA. 2018. National Ambient Air Quality Standards for Nitrogen Dioxide: Final Decision. https://www.govinfo.gov/content/pkg/FR-2018-04-18/pdf/2018-07741.pdf
- WHO (World Health Organization). 2005. WHO Air Quality Guidelines for particulate matter, ozone, nitrogen dioxide and sulphur dioxide. Global update 2005. Summary of risk assessment. World Health Organization. Available At: https://www.euro.who.int/en/health-topics/environment-and-health/air-quality/publications/pre2009/air-quality-guidelines.-global-update-2005.-particulate-matter,-ozone,-nitrogen-dioxide-and-sulfur-dioxide

Fine Particulate Matter ($PM_{2.5}$):

- Alberta Environment (AENV). 2018. Alberta Ambient Air Quality Objectives and Guidelines – Fine Particulate Matter ($PM_{2.5}$). AEP, Air Policy, 2018, No. 1.
- AENV. 2019. Alberta Ambient Air Quality Objectives and Guidelines Summary. AEP, Air Policy, 2016, No. 2.
- B.C. Ministry of Environment and Climate Change Strategy (BC MoECCS). 2020. Provincial Air Quality Objective. Information Sheet.
- California Office of Environmental Health Hazard Assessment (Cal OEHHA). 2001. Health Effects of Particulate Matter. Public Review Draft.
- Cal OEHHA. 2016. Ambient Air Quality Standards. https://ww2.arb.ca.gov/resources/california-ambient-air-quality-standards
- Canadian Council of Ministers of the Environment (CCME). 2012. Guidance Document on Achievement Determination Canadian Ambient Air Quality Standards for Fine Particulate Matter and Ozone.
- CCME. 2017. Canada's Air. https://ccme.ca/en/air-quality-report#slide-1
- CCME. 2020. Guidance Document on Achievement Determination Canadian Ambient Air Quality Standards for Fine Particulate Matter and Ozone.
- CCME. 2021. Air Quality Report. https://www.ccme.ca/en/air-quality-report
- Health Canada (HC). 2016. Human Health Risk Assessment for Coarse Particulate Matter.
- International Agency for Research on Cancer (IARC). 2013. IARC Monographs on the Evaluation of Carcinogenic Risks to Humans Volume 109: Outdoor Air Pollution
- Metro Vancouver Ambient Air Quality Objectives, updated January 2020. http://www.metrovancouver.org/services/air-quality/AirQualityPublications/CurrentAmbientAirQualityObjectives.pdf
- Ontario Ministry of the Environment, Conservation and Parks (MECP). 2020. Ambient Air Quality Criteria. Human Toxicology and Air Standards Section. Technical Assessment and Standards Development Branch.
- United States Environmental Protection Act (US EPA). 1997. National Ambient Air Quality Standards for Particulate Matter. Federal Agency / Vol. 62, No. 138.

— US EPA. 2006. National Ambient Air Quality Standards for Particulate Matter; Final Rule. Federal Register / Vol. 71, No. 200.
— US EPA. 2009. U.S. EPA. Integrated Science Assessment (ISA) for Particulate Matter (Final Report, Dec 2009). U.S. Environmental Protection Agency, Washington, DC, EPA/600/R-08/139F.
— US EPA. 2013. National Ambient Air Quality Standards for Particulate Matter; Final Rule. Federal Register / Vol. 78, No. 10.
— US EPA. 2019. Integrated Science Assessment (ISA) for Particulate Matter (Final Report, Dec 2019). U.S. Environmental Protection Agency, Washington, DC, EPA/600/R-19/188.
— US EPA. 2021. Timeline of Particulate Matter (PM) National Ambient Air Quality Standards. https://www.epa.gov/pm-pollution/timeline-particulate-matter-pm-national-ambient-air-quality-standards-naaqs
— World Health Organization (WHO). 2005. Air Quality Guidelines. Global Update.

Benzene:

– Agency for Toxic Substances and Disease Registry (ATSDR). 2007. Toxicological Profile for Benzene. https://www.atsdr.cdc.gov/toxprofiles/tp3.pdf
– Alberta Environment (AENV). 2019. Alberta Ambient Air Quality Objectives and Guidelines Summary. AEP, Air Policy, 2016, No. 2. January 2019. https://open.alberta.ca/publications/9781460134856
– B.C. Ministry of Environment and Climate Change Strategy (BC MoECCS). 2020. B.C. Ambient Air Quality Objectives. February 28, 2020. https://www2.gov.bc.ca/assets/gov/environment/air-land-water/air/reports-pub/prov_aqo_fact_sheet.pdf
– California Office of Environmental Health Hazard Assessment (Cal OEHHA). 2014. Appendix D.1 Summaries using this version of the Hot Spots Risk Assessment guidelines. Appendix D. Individual Acute, 8-Hour, and Chronic Reference Exposure Level Summaries (ca.gov)
– Canadian Council of Ministers of the Environment (CCME). 2017. Canada's Air. https://ccme.ca/en/air-quality-report#slide-1
– Environment Canada/Health Canada. 2021. Residential Indoor Air Quality Guidelines. https://www.canada.ca/en/health-canada/services/air-quality/residential-indoor-air-quality-guidelines.html
– Flury F. 1928. [II. Toxicities in modern industry. IIa. Pharmacological-toxicological aspects of intoxicants in modern industry.] Arch Exp Pathol Pharmakol 138:65-82. (German)
– Health Canada. 2021. Federal Contaminated Site Risk Assessment in Canada, Toxicological Reference Values (TRVs), Version 3.0. Ottawa, ON.
– Metro Vancouver Ambient Air Quality Objectives, updated January 2020. http://www.metrovancouver.org/services/air-quality/AirQualityPublications/CurrentAmbientAirQualityObjectives.pdf
– Midzenski MA, McDiarmid MA, Rothman N, et al. 1992. Acute high dose exposure to benzene in shipyard workers. Am J Ind Med 22:553-565.
– Ontario Ministry of the Environment, Conservation and Parks (MECP). 2020. Ambient Air Quality Criteria. Human Toxicology and Air Standards Section. Technical Assessment and Standards Development Branch. https://www.ontario.ca/page/ontarios-ambient-air-quality-criteria#section-4
– Rothman N, Li Gl, Dosemeci M, et al. 1996. Hematotoxicity among Chinese workers heavily exposed to benzene. Am J Med 29(3):236-246.
– Texas Commission on Environmental Quality (TCEQ). 2015. Benzene, Development Support Document. Revised September 2015. https://www.tceq.texas.gov/assets/public/implementation/tox/dsd/final/benzene.pdf
– United States Environmental Protection Act (US EPA). 1998. Carcinogenic effects of benzene: An update. Washington, DC: U.S. Environmental Protection Agency. EPA600P97001F. PB99101420.

- US EPA. 2002 Toxicological Review of Benzene (Non-Cancer Effects) CAS No. 71-43-1 In Support of Summary Information on the Integrated Risk Information System. https://cfpub.epa.gov/ncea/iris/iris_documents/documents/toxreviews/0276tr.pdf
- US EPA. 2003. Integrated Risk Information System (IRIS) Chemical Assessment Summary of Benzene. https://iris.epa.gov/ChemicalLanding/&substance_nmbr=276
- US EPA. 2021. National Ambient Air Quality Standards, NAAQS Table. https://www.epa.gov/criteria-air-pollutants/naaqs-table
- Ward CO, Kuna RA, Snyder NK, et al. 1985. Subchronic inhalation toxicity of benzene in rats and mice. Am J Ind Med 7:457-473
- World Health Organization (WHO). 2000. Air Quality Guidelines – Chapter 5.2, Benzene. https://www.euro.who.int/__data/assets/pdf_file/0017/123056/AQG2ndEd_5_2benzene.pdf
- WHO. 2017. Evolution of WHO air quality guidelines. https://www.euro.who.int/__data/assets/pdf_file/0019/331660/Evolution-air-quality.pdf

1,3-Butadiene:

- Alberta Environment (AENV). 2019. Alberta Ambient Air Quality Objectives and Guidelines Summary. AEP, Air Policy, 2016, No. 2. January 2019. https://open.alberta.ca/publications/9781460134856
- Agency for Toxic Substances and Disease Registry (ATSDR). September 2012a. Toxicological Profile for 1,3-Butadiene. https://www.atsdr.cdc.gov/ToxProfiles/tp28.pdf
- ATSDR. October 2012b. ToxGuide™ for 1,3-Butadiene. https://www.atsdr.cdc.gov/toxguides/toxguide-28.pdf
- B.C. Ministry of Environment and Climate Change Strategy (BC MoECCS). 2020. B.C. Ambient Air Quality Objectives. February 28, 2020. https://www2.gov.bc.ca/assets/gov/environment/air-land-water/air/reports-pub/prov_aqo_fact_sheet.pdf
- Canadian Council of Ministers of the Environment (CCME). 2017. Canada's Air. https://ccme.ca/en/air-quality-report#slide-1
- California Office of Environmental Health Hazard Assessment (Cal OEHHA). 2008. Appendix D.2 Acute, 8-Hour and Chronic REL Summaries. https://oehha.ca.gov/media/downloads/crnr/appendixd1final.pdf
- Cal OEHHA. 2014. Appendix D.2 Acute, 8-Hour and Chronic REL Summaries. https://oehha.ca.gov/media/downloads/crnr/appendixd1final.pdf
- Environment Canada/Health Canada May 2000. Priority Substance List Assessment Report-1,3 Butadiene, Canadian Environmental Protection Act, 1999. https://publications.gc.ca/collections/Collection/En40-215-52E.pdf
- Environment Canada/Health Canada. 2021. Residential Indoor Air Quality Guidelines. https://www.canada.ca/en/health-canada/services/air-quality/residential-indoor-air-quality-guidelines.html
- Metro Vancouver Ambient Air Quality Objectives, updated January 2020. http://www.metrovancouver.org/services/air-quality/AirQualityPublications/CurrentAmbientAirQualityObjectives.pdf
- Ontario Ministry of the Environment. 2011. Ontario Air Standards for 1,3-Butadiene. Standards Development Branch. June 2011.
- Ontario Ministry of the Environment, Conservation and Parks (MECP). 2020. Ambient Air Quality Criteria. Human Toxicology and Air Standards Section. Technical Assessment and Standards Development Branch. https://www.ontario.ca/page/ontarios-ambient-air-quality-criteria#section-4

- United States Environmental Protection Agency (US EPA). 2002. Integrated Risk Information System (IRIS), Chemical Assessment Summary, 1,3-Butadiene; CASRN 106-99-0.
- World Health Organization (WHO). 2000. Air Quality Guidelines for Europe, 2nd Edition, Chapter 5.3, 1,3 Butadiene. https://www.euro.who.int/__data/assets/pdf_file/0018/123057/AQG2ndEd_5_3butadiene.pdf
- WHO. 2001, Concise International Chemical Assessment Document, 1,3-BUTADIENE: Human Health Aspects. https://www.who.int/ipcs/publications/cicad/en/cicad30.pdf?ua=1
- WHO. 2005. Air Quality Guidelines. Global Update.
- IARC 2008, IARC Monographs = 1,3 Butadiene. https://monographs.iarc.who.int/wp-content/uploads/2018/06/mono100F-26.pdf
- US EPA. 2021. National Ambient Air Quality Standards, NAAQS Table. https://www.epa.gov/criteria-air-pollutants/naaqs-table