HUMAN HEALTH ASSESSMENT

CLARKSON TRANSIT STATION AREA

SLATE ASSET MANAGEMENT L.P.

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WSP
100 COMMERCE VALLEY DRIVE WEST
THORNHILL, ON, CANADA L3T 0A1

WSP.COM

SIGNATURES

PREPARED BY

Ahmed Negm, M.Env.Sc
Risk Assessor

March 14, 2022 (Original Assessment)
Date

Yangfan Chen, M.Sc., E.I.T
Risk Assessor

March 14, 2022 (Original Assessment)
Date

APPROVED[1] BY

Theresa Repaso-Subang, DABT, ERT, QP_{RA}
Senior Technical Lead,
Toxicology & Risk Assessment

March 14, 2022 (Original Assessment)
Date

REVISIONS REVIEWED AND APPROVED[2] BY

Camille Taylor
Project Manager
Air, Noise, Vibration and Climate

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1 INTRODUCTION

1.1 BACKGROUND

The City of Mississauga (the "City") is developing land use policies for the Clarkson Transit Station Area (TSA) to support intensification of the area. It is recognized that with possible redevelopment of this area and introduction of new sensitive land uses, there would be a need to assess air quality impacts on proposed new sensitive developments, especially given the historical state of air quality in the area.

WSP Canada Inc. (WSP) has been retained by Slate Asset Management (Slate) to complete the TSA Air Quality Study (AQS) based on Terms of Reference provided by the City of Mississauga, intended to be used to assess the compatibility of proposed development blocks within the TSA. In support of the Clarkson TSA AQS, a human health assessment (HHA) was completed to assess any acute and chronic risks associated with the cumulative concentrations of chemicals predicted to be above Ambient Air Quality Criteria (AAQC) or federal standards, established by Ontario Ministry of Environment, Conservation and Parks (MECP), and determine appropriate implications and consideration of any mitigation measures for the proposed development/intensification.

The HHA relies on six months of ambient on-site air monitoring data and an air dispersion modelling assessment of identified contaminants of potential concern (COPCs) from the recently completed Clarkson TSA AQS (WSP, 2021). The model results represent the air quality impacts on the proposed development from surrounding land uses, including industrial operations and transportation sources in the Clarkson TSA. Based on the results of the ambient air monitoring and air dispersion modelling, the HHA evaluates the potential health effects from the predicted cumulative impacts from nearby activities on the proposed development.

This HHA predicts the potential health impacts of the proposed development within the Clarkson TSA that will consist of four 25-storey residential buildings.

1.2 OBJECTIVES OF THE HHA

The purpose of the HHA is to assess potential human health risks, if any, associated with predicted cumulative concentrations of identified COPCs from nearby activities on the proposed development.

To achieve this objective, WSP evaluated the source-pathway-receptor linkage based on possible interactions with human receptors within the proposed development. The HHA applied risk assessment approaches and methodology that are endorsed by federal and provincial regulatory agencies including Health Canada, MECP, and other relevant regulatory agencies.

The objectives of the HHA included the following:

- To assess whether the predicted cumulative concentrations of COPCs in ambient air influenced by nearby activities pose a health concern for identified human receptors in the proposed development; and,
- Based on the findings of the HHA, identify controls, mitigation measures, or monitoring programs that could be implemented to prevent or address the potential for health effects.

2 PROBLEM FORMULATION

The problem formulation section of the HHA is the first step in the assessment that lays out the source-pathway-receptor linkage based on possible interactions with human receptors at the proposed development to assess how the predicted cumulative concentrations from nearby sources may affect health. This step identifies the chemical of concern, receptors of concern, and exposure pathways to be evaluated in the assessment.

2.1 CHEMICALS OF POTENTIAL CONCERN

Available air quality data collected during the AQS (WSP, 2021) was used to determine COPCs.

The six months of ambient air monitoring data (Clarkson monitoring program) and dispersion modelling assessment were completed in accordance with the terms of reference (TOR) provided by the City and completed in accordance with the MECP operations manual for air quality monitoring in Ontario. The parameters outlined in the City TOR for monitoring were:

- Total suspended particulate matter (TSP);
- Volatile organic compounds (VOCs) (benzene, dichloromethane, and acrolein);
- Nitrogen oxides (NO_x); and,
- Sulphur dioxide (SO_2).

Particulate matter with aerodynamic diameter of 10 microns (PM_{10}) and 2.5 microns ($PM_{2.5}$) were later added to the list of monitored parameters at the request of the MECP. The monitoring took place from July 8, 2020, to January 10, 2021.

The Clarkson monitoring program was used in combination with air dispersion modelling results to predict cumulative impacts at the Site for benzene, acrolein, PM_{10}, $PM_{2.5}$, TSP, NO_x, SO_2, and dichloromethane.

Several contaminants were not monitored as part of the Clarkson monitoring program, in which case ambient air monitoring concentrations were obtained from the Clarkson Air Shed Industrial Association (CASIA) monitoring program, the National Air Pollution Surveillance (NAPS), and the MECP ambient air quality monitoring program. These contaminants include carbon monoxide (CO), benzene, benzo(a)pyrene, 1,3-butadiene, formaldehyde, acetaldehyde, SO_2, total reduced sulphur [TRS (as H_2S)], xylene, and dichloromethane.

In order to assess the cumulative impacts on the Site, the 90th percentile of ambient air concentrations of each contaminant was calculated for 10-min, 1-hour, and 24-hour averaging periods. For contaminants with annual averaging periods, the annual mean was calculated.

The complete list of contaminants for which monitoring data was collected (as described above) includes: PM_{10}, $PM_{2.5}$, TSP, NO_x (expressed as NO_2), CO, SO_2, acrolein, benzene, 1,3-butadiene, acetaldehyde, formaldehyde, benzo(a)pyrene, methylene chloride, TRS (as H_2S), and xylenes.

Predicted modelled concentrations from stationery and transportation sources within the Clarkson study area (i.e., 1000 m area around the proposed development) were assessed at various heights using the AERMOD air dispersion model. Air dispersion modelling included predicted emission rates from roadways, trains on the Clarkson GO rail corridor, and facilities of concern within the study area. Cumulative concentration impacts from ambient background concentrations and predicted modelled concentrations were then compared to air quality project thresholds [i.e., either the AAQC or Canadian Ambient Air Quality Standards (CAAQS), whichever is more stringent].

For each contaminant with a cumulative concentration that exceeded its air quality project threshold, it was identified as a COPC and assessed for potential human health risks as part of the HHA. These contaminants include **acrolein**, **benzene**, **benzo(a)pyrene**, **NO_2**, and **$PM_{2.5}$**. Although PM_{10} and TSP reported cumulative concentrations that were greater than 80% of their respective air quality project thresholds, they were not considered as part of the assessment as they have no available health-based benchmarks for evaluation. Moreover, given their large particulate size, they are usually trapped in the upper respiratory airways and thus, are not a considered predominant

health concern relative to finer ($PM_{2.5}$) particulate matter. All other contaminants identified in the AQS were below their air quality project thresholds, and thus, were not carried forward as part of this assessment.

Table 3-2 in Section 3 presents the complete list of COPCs, their cumulative concentrations, and their respective air quality project thresholds.

It is important to note that the COVID-19 pandemic resulted in a reduction of traffic in the area, and a reduced train frequency along the Lakeshore West corridor during the monitoring period; therefore, this report assumes that vehicular emissions from nearby parking lots and major roadways were reduced. The ambient air quality monitoring results are used in conjunction with dispersion modelling to conservatively assess the air quality impacts on the proposed development. Dispersion modelling was completed using data from prior to the COVID-19 pandemic. Historical data, including monitoring data from the Clarkson Airshed Industrial Association (CASIA) from 2012 to 2018 was also incorporated into this study for comparative purposes, where applicable. Despite the uncertainties of the effects of COVID-19 on the ambient monitoring data, WSP has confidence in the report and its findings. Further details are found in the AQS (WSP, 2021).

2.2 RECEPTORS OF POTENTIAL CONCERN

The human receptors evaluated in the HHA were identified based on the proposed development within the Clarkson TSA (i.e., four 25-storey residential buildings). The human receptors associated with this identified land use are intended to be inclusive of human populations including sensitive subpopulations such as asthmatics, children, pregnant females, and the elderly. The following two (2) human receptors were considered:

1. Toddler residents who live in the buildings within the proposed development; and
2. Adult residents who live in the buildings within the proposed development.

The exposure modelling, described below in Section 3.0, considered that all of these human receptors may be exposed to maximum impacts associated with cumulative concentrations of COPCs that may be influenced by neighbouring sources. This approach provides maximum flexibility in the interpretation of results but may be overly conservative if the likelihood of human presence is not accounted for in the risk characterization.

2.3 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.

The HHA quantitatively evaluated the following exposure pathways based on the identified human receptors, COPCs [i.e., acrolein, benzene, benzo(a)pyrene, NO_2, and $PM_{2.5}$], and relevant environmental media (i.e., ambient air).

Toddler Residents:

- Exposure to concentrations of COPCs via direct inhalation of ambient air emissions.

Adult Residents:

- Exposure to concentrations of COPCs via direct inhalation of ambient air emissions.

For the purposes of exposure modelling, it was assumed that the predicted cumulative concentrations of COPCs in outdoor air are equal to that in indoor air (i.e., established equilibrium).

It should be noted that maximum COPC concentrations are expected at various heights and locations across the proposed development, depending on the contaminant. More importantly, several studies that investigate vertical difference of chemical concentrations confirm findings from atmospheric measurements and modeling that show concentrations tend to decrease with building height (Stephens *et al*, 2019). Table 2-1 (below) provide adjusted ambient concentrations with increasing building heights. A 10% reduction in chemical concentration with building height is observed at approximately 25 m. As a conservative measure, the worst-case COPC concentrations were used for assessment of all receptor groups.

A more detailed discussion on the exposure pathways for the above-noted receptors is provided in Section 3.0.

Table 2-1 Maximum Model Ambient Air Concentrations for Identified COPCs Adjusted with Increasing Building Height

RECEPTOR HEIGHT (M)	CONTAMINANT (µG/M^3)						
	PM2.5	NOX		ACROLEIN	BENZENE	B(A)P	
	ANNUAL	1 HR	ANNUAL	24 HR	ANNUAL	24 HR	ANNUAL
0	8.20	36.0	16.0	0.63	0.49	5.00E-05	1.00E-05
4.3	8.20	36.0	16.0	0.63	0.49	1.10E-04	1.00E-05
8.6	7.67	35.3	15.5	0.56	0.44	1.10E-04	1.00E-05
12.9	7.04	33.6	15.4	0.45	0.38	1.08E-04	9.81E-06
17.2	6.35	39.0	14.8	0.35	0.33	1.04E-04	9.49E-06
21.5	5.71	41.7	13.4	0.28	0.28	1.15E-04	1.05E-05
25.8	5.15	38.5	11.3	0.20	0.25	1.22E-04	1.11E-05
30.1	4.63	32.7	8.9	0.14	0.22	1.23E-04	1.12E-05
34.4	4.16	26.1	6.6	0.11	0.20	1.11E-04	1.01E-05
38.7	3.71	20.7	4.8	0.08	0.18	9.40E-05	8.54E-06
43	3.29	16.7	3.5	0.07	0.17	7.42E-05	6.74E-06
47.3	2.90	15.8	2.6	0.06	0.16	5.70E-05	5.18E-06
51.6	2.53	15.7	2.1	0.06	0.15	4.65E-05	4.22E-06
55.9	2.19	15.5	1.8	0.05	0.15	3.90E-05	3.55E-06
60.2	1.89	15.5	1.6	0.05	0.14	3.29E-05	2.99E-06
64.5	1.62	15.6	1.5	0.05	0.14	2.81E-05	2.55E-06
68.8	1.38	15.7	1.4	0.05	0.14	2.41E-05	2.19E-06
73.1	1.17	15.8	1.4	0.04	0.14	2.03E-05	1.84E-06
77.4	0.99	16.0	1.4	0.04	0.14	1.75E-05	1.59E-06
81.7	0.84	16.3	1.4	0.04	0.14	1.51E-05	1.38E-06
86	0.71	16.5	1.4	0.04	0.14	1.37E-05	1.25E-06
90.3	0.61	16.7	1.4	0.04	0.14	1.24E-05	1.13E-06
94.6	0.52	17.0	1.4	0.04	0.14	1.14E-05	1.04E-06
98.9	0.45	17.3	1.4	0.04	0.14	1.07E-05	9.70E-07
103.2	0.40	17.5	1.4	0.03	0.14	1.01E-05	1.00E-05
107.5	0.35	18.0	1.5	0.03	0.14	9.41E-06	1.00E-05

Assumes ambient concentrations are collected at a minimum of 2m in height

Estimated ambient concentrations at heights based on % change from ground level

2.4 UNCERTAINTY ANALYSIS

The major sources of uncertainty associated with the problem formulation of the HHA are briefly described below:

- For the purpose of exposure modelling, it has been assumed that the predicted concentrations of COPCs in outdoor air are equal to that in indoor air. 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 is possible that other human receptors may be present at the proposed development for a period of time (e.g., site visitor or indoor worker); however, a resident is assumed to be the most sensitive human receptor to occupy the proposed development. Therefore, assessment of residents is protective of all other human receptors that may occupy the proposed development.

3 EXPOSURE ASSESSMENT

The receptor-specific exposure parameters for toddler residents, and adult residents are summarized in **Table 3-1** and **Table 3-2.**

3.1 EXPOSURE PARAMETERS FOR TODDLER RESIDENTS

It is assumed that toddlers from the ages of 7 months to 4 years old would reside in one of the buildings at the proposed development. The toddler is assumed to spend 24 hours/day, 7 days/week, for 50 weeks/year within their residential unit. It is also assumed that this receptor (whether in an indoor or outdoor environment, or both) will be continuously exposed to COPC concentrations in ambient air throughout the duration of their residence.

3.2 EXPOSURE PARAMETERS FOR ADULT RESIDENTS

It is assumed that an adult (i.e., > 20 years) would reside in one of the buildings at the proposed development. The adult is assumed to spend 24 hours/day, 7 days/week, for 50 weeks/year (assuming a two-week vacation) within their residential unit. It is also assumed that this receptor (whether in an indoor or outdoor environment, or both) will be continuously exposed to COPC concentrations in ambient air throughout the duration of their residence. A pregnant female resident was also evaluated to assess potential exposure to developmental COPCs (i.e., benzo(a)pyrene). A key difference in the evaluation of developmental toxicants is the absence of dose averaging. As such, exposure is assumed to occur for 24 hours/day, 7 days/week, for 52 weeks/year.

The exposure duration assumptions applied were considered reasonable and appropriate given the proposed development and anticipated receptors.

Table 3-1 Exposure Factors for Toddler, Adult, and Pregnant Female Residents

EXPOSURE FACTOR	UNITS	TODDLER (RESIDENT)	ADULT (RESIDENT)	PREGNANT FEMALE (RESIDENT)	REFERENCE
EF (exposure frequency for inhalation) = EFa x EFb x EFc	hrs/yr	8400	8400	8760	MECP, 2011
EFa (daily exposure frequency)	d/wk	7	7	7	MECP, 2011
EFb (weekly exposure frequency)	wk/yr	50	50	52	MECP, 2011
EFc (hourly exposure frequency)	hr/d	24	24	24	MECP, 2011
ED (exposure duration)	yr	4.5	56	56	MECP, 2011
AP (averaging period): non-cancer	yr	4.5	56	56	MECP, 2011
AP (averaging period): cancer	yr	76	76	76	MECP, 2011

3.3 CUMULATIVE EXPOSURES

The AQS (WSP, 2021) determined background ambient COPC concentrations to complete the air dispersion modelling and assess the predicted cumulative impacts from nearby activities on the proposed development.

Selected background ambient concentrations are added to modelled predictions to determine the cumulative impact to air quality. In this context, “background ambient” is defined as concentrations collected as part of the Clarkson monitoring program or the NAPS monitoring program and which represent background air quality.

For this assessment, the 90th percentile of ambient background concentrations of each COPC monitored was calculated for 10-min, 1-hour, and 24-hour averaging periods. For COPCs with annal averaging periods, the annual mean was calculated. Further details on the complete air dispersion modelling methodology applied as part of this assessment can be found in the AQS (WSP, 2021). A discussion on the conservatism applied to generate the cumulative concentrations are provided in Section 3.4.

Predicted modelled concentrations were collected from stationary and transportation sources within the study area. All sources were conservatively assumed to be operating 24 hours/day, 7 days/week, 52 weeks/year in the modelling assessment.

Subsequently, the cumulative impacts at the proposed development are calculated by aggregating the background ambient concentrations with the predicted modelling results (i.e., background ambient + predicted modelled = cumulative).

Table 3-2 below summarizes the COPC cumulative concentrations (including background ambient and predicted modelled concentrations) compared to their respective air quality project thresholds.

Although the AQS modelled concentrations from a total of 18 contaminants (as listed in section 2.1), only those contaminants that exceeded their applicable AAQC were listed in the table below and carried forward as part of the assessment

Table 3-2 Summary of Modelled Concentrations, Ambient Background Concentrations, and Cumulative Concentrations for COPCs against their Air Quality Project Thresholds

COPC	CAS Number	Total Emission Rate (g/s)	Air Dispersion Model Used[1]	Concentration (µg/m³)			Averaging Period	Air Quality Project Threshold (µg/m³)	Threshold Source	Percent of Limit from Background (%)	Percent of Limit from Modelled Conc. (%)	Percent of Limit (%)
				Modelled[2]	Background	Cumulative						
Acrolein	107-02-8	7.26E-06	AERMOD v.19191	0.010	1.6	1.6	1-hr	4.5	AAQC	36%	0. 2%	36%
				0.004	0.63	0.63	24-hr	0.4	AAQC	**158%**	1%	**158%**
Benzene	71-43-2	1.87E-01	AERMOD v.19191	0.03	0.69	0.72	24-hr	2.3	AAQC	30%	2%	31%
				0.009	0.49	0.50	Annual	0.45	AAQC	**109%**	2%	**111%**
Benzo(a)pyrene	50-32-8	6.49E-08	AERMOD v.19191	7.48E-07	0.00011	0.00011	24-hr	0.00005	AAQC	**213%**	1%	**215%**
				0.00E+00	0.000012	0.000012	Annual	0.00001	AAQC	**115%**	0%	**115%**
NO_x (as NO_2)	10102-44-0	1.13E+02	AERMOD v.19191	54	36	90	1-hr	79	CAAQS	46%	68%	**114%**
				32	30	62	24-hr	200	AAQC	15%	16%	31%
				14	16	30	Annual	23	CAAQS	68%	63%	**131%**
$PM_{2.5}$	N/A[1]	2.14E+00	AERMOD v.19191	4.5	15	19	24-hr	27	AAQC	54%	17%	71%
				1.8	8.2	10	Annual	8.8	AAQC	93%	21%	**114%**

Notes:

[1] Predicted modelled concentrations were derived using the MECP identified AERMOD dispersion model, version 19191.

[2] Maximum point of impingement (POI) concentrations are based on AERMOD dispersion modelling results.

Due to rounding, some cumulative values may not correspond with the sum of the background and modelled values

AAQC = Ambient Air Quality Criteria

CAAQC = Canadian Ambient Air Quality Standard

Bolded = concentrations exceed the air quality project threshold

3.4 UNCERTAINTY ANALYSIS

The major sources of uncertainty associated with the exposure assessment of the HHA are briefly described below:

- Worst-case exposure scenarios were evaluated for all human receptors considered. For example, it has been assumed that the predicted concentrations of COPCs in outdoor air are equal to that in indoor air. 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.
- The HHA also assume that predicted concentrations of COPCs are constant with building height. However, several studies that investigate vertical difference of concentrations confirm findings from atmospheric measurements and modeling that PM concentrations tend to decrease with building height, meaning that high-rise housing could experience improved air quality relative to low-rise housing (Stephens *et al*, 2019).
- The maximum point of impingement (MPOI) concentration for each COPC was selected as the predicted modelled concentration to be used for assessment. The MPOI is specific to a certain height and location along the façade of the proposed buildings. For example, the most impacted receptor for 24-hr NO_2 concentrations is located at the northwest property boundary at a height of approximately 21.5 m as a result of this location being near train and road sources. However, all identified human receptors were assumed to be exposed to the COPC-specific MPOI concentrations at all times regardless of their spatial location within the proposed development. This is considered a conservative method of characterization and may overestimate risks.
- For those COPCs which were not part of the Clarkson monitoring program, there is an added level of uncertainty given that ambient background concentrations were collected from monitoring stations outside of the Clarkson TSA; and thus, the data becomes less representative of actual site conditions. For example, ambient background concentrations for benzo(a)pyrene were based on a NAPS station located near Highway 401. As such, higher concentrations were recorded given the close proximity to high volumes of vehicular traffic than in the vicinity of the Clarkson TSA. In this case, this is considered a conservative approach, and may overestimate risks.
- In many cases the ambient background concentrations collected already accounted for some of the sources modelled for the predicted modelled concentrations. In other words, sources captured in the Clarkson monitoring program are then modelled and added again to the results of the background ambient concentrations collected from the monitoring program to calculate the cumulative concentrations; in essence, leading to double counting. This is considered a conservative approach and may overestimate risks.

A series of conservative assumptions and characterization methods (as described above) were applied when obtaining ambient background concentrations and predicted modelled concentrations for COPCs. These assumptions, when used in aggregate, may result in conservative overestimates. Further details about the assumptions, methods, and uncertainties used to predict cumulative COPC concentrations can be found in the AQS (WSP, 2021).

4 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 health effects. This value is called the toxicity reference value (TRV).

4.1 REVIEW OF TOXICOLOGICAL BASIS OF JURISDICTIONAL AMBIENT AIR QUALITY OBJECTIVES OF COPCS

Exposure limits are derived based on the duration of exposure. For this HHA, 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 acrolein, benzene, benzo(a)pyrene, NO_2, and $PM_{2.5}$. Additionally, a comprehensive review of the available short-term (acute) and long-term (chronic) numerical limits was conducted. This review considered the following:

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

Exposure limits used in the HHA 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 HHA, 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], MECP, California Office of Environmental Health Hazard Assessment [Cal OEHHA]), and international organizations (e.g., World Health Organization [WHO]). Human health-based screening criteria from Ontario, Health Canada, and CCME were prioritized.

Scientifically defensible exposure limits applied in the HHA for each COPC and for each duration (acute vs chronic) 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 COPC;
- Protective of sensitive individuals through the use of appropriate uncertainty factors (UFs); 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 and the rationale for the decision is provided in the toxicity profile (Section 4.2). The findings of the jurisdictional review of available AAQOs for acute and chronic exposure and their toxicological basis are described in the sections below for each COPC.

4.1.1 ACROLEIN

Jurisdictional acute (or short-term, expressed as 1-hr and/or 8-hr) and chronic (or long-term, expressed as annual) exposure limits for acrolein are provided in **Table 4-1** and **Table 4-2**, respectively. The studies supporting the available exposure limits are described in detail below.

Table 4-1 Acute Inhalation Exposure Limits for Acrolein

REGULATORY AGENCY	TYPE	VALUE (ppb)	VALUE (µg/m^3)	REFERENCE
BC ENV	1-hour AAQO	-	-	BC ENV, 2020
	8-hour AAQO	-	-	
AENV	1-hour AAQO	1.9	4.5	AENV, 2019
	24-hour AAQO	0.17	0.40	
ATSDR	Acute (1 to 14 days)	3	6.9	ATSDR, 2007
CCME	1-hour CAAQS	-	-	CCME, 2017
ON MECP	1-hour AAQC	-	4.5	Ontario MECP, 2022
US EPA	10 mins to 8 h	-	70	US EPA, 2010
	-	-	7	US EPA, 2008
Cal OEHHA	Acute 1-hour	1.1	2.5	Cal OEHHA, 2014
	8 hour	0.30	0.70	
WHO	1-hour AQG	-	-	WHO, 2000
	8-hour AQG	-	-	
Health Canada Environmental Canada	STEL (1h) REL	17	38	HC and EC, 2000
	LTEL (24h)	0.19	0.44	
Cal EPA	Acute (1h)	1.1	2.5	Cal OEHHA, 2008
ANSES	Acute (1h)	3	6.9	ANSES, 2013
TCEQ	Acute Reference Value (1h)	4.8	11	TCEQ, 2015
	Acute ESL (1h)	1.4	3.2	

AAQO - Ambient Air Quality Objective; AAQC – Ambient Air Quality Criteria; AQG - Air Quality Guideline; CAAQS – Canadian Ambient Air Quality Standard; REL – Reference Exposure Limit, STEL-Short Term Exposure Levels, TLV-Threshold Limit Value, TWA – Total Weighted Average; STEL Short Term Exposure Limit; LTEL – Long term Exposure Limit; AAQG-Ambient Air Quality Guideline

AENV – Alberta Environment, BC ENV – British Columbia Ministry of Environment and Climate Change Strategy; ATSDR-Agency for Toxic Substances and Disease Registry; ANSES - Agence Nationale de Sécurité Sanitaire de L'alimentation ; 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. TCQE - Texas Commission on Environmental Quality.

There are no available acute (short-term) jurisdictional limits from BC MoECCS, CCME or WHO.

Table 4-2 Chronic Inhalation Exposure Limits for Acrolein

REGULATORY AGENCY	TYPE	VALUE (ppb)	VALUE (µg/m^3)	REFERENCE
ATSDR	Chronic MRL	-		ATSDR, 2007
AENV	Annual AAQO	-	-	AENV, 2019
BC ENV	Annual AAQO	-	-	BC ENV, 2020
CCME CAAQS	Annual CAAQS	-	-	CCME 2017
Health Canada Environment Canada	Chronic		0.4	HC and EC, 2000
ON MECP	Annual AAQC	-	-	Ontario MECP 2022
	Chronic (24 h)	0.17	0.4	OMoE, 2009
Cal OEHHA	Chronic	0.15	0.35	Cal OEHHA, 2008
Arizona Department of Health Services		-	-	AESRD, 2013
US EPA	Chronic		0.02 (RfC for Nasal Lesions)	US EPA, 2003
ANSES	Chronic		0.8	ANSES, 2013
WHO (unit risk)		-	-	WHO 2017
TCEQ	Air monitoring comparison Value (Annual)	0.1	0.2	TCEQ, 2015

AAQO - Ambient Air Quality Objective; AAQC – Ambient Air Quality Criteria; AQG - Air Quality Guideline; CAAQS – Canadian Ambient Air Quality Standard; REL – Reference Exposure Level, STEL-Short Term Exposure Levels, TLV-Threshold Limit Value, TWA – Total Weighted Average; STEL Short Term Exposure Limit,

AENV – Alberta Environment, BC ENV – British Columbia Ministry of Environment and Climate Change Strategy; ATSDR-Agency for Toxic Substances and Disease Registry, AESRD - Alberta Environment and Sustainable Resource Development; ANSES - Agence Nationale de Sécurité Sanitaire de L'alimentation 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;OMoE – Ontario Ministry of Environment; US EPA – United States Environmental Protection Agency; WHO – World Health Organization; TCEQ - Texas Commission on Environmental Quality

ATSDR, BC ENV, AENV, CCME, ON MECP, Arizona Department of Health Services, and WHO have not established annual Ambient Air Quality Standards, objectives, criteria, or exposure limits for acrolein.

Environment Canada and Health Canada

In 2000, Environment Canada and Health Canada completed an assessment report for acrolein. The report concluded that acrolein is considered to be "toxic" as defined in Section 64 of the Canadian Environmental Protection Act, 1999. Within the report, Environment Canada and Health Canada developed an inhalation Tolerable Concentration (TC) of 0.4 µg/m^3(Microgram per cubic meter) for acrolein based on a chronic (3-day) exposure study investigating non-neoplastic lesions in the nasal and respiratory epithelium in rats.

Agency for Toxic Substances and Disease Registry

ATSDR (2007) derived an acute (1 to 14 day) minimal risk level of 3 part per billion (ppb) (6.9 µg/m^3), based on a lowest-observed-adverse-effect Level (LOAEL) of 0.3 part per million(ppm) (0.7 mg/m^3) for an increase in eye, nose, and throat irritation, and a decrease in respiration rate in a study of 46 volunteers exposed to acrolein for 60 minutes (Weber-Tschopp *et al.*, 1977). UFs of 10 for the use of a LOAEL and 10 for intraspecies variation were applied, giving a total UF of 100.

California Environmental Protection Agency

For acute exposures, the California Environmental Protection Agency (Cal EPA) (Cal OEHHA) (2008) derived an acute (1 hour) reference exposure level of 2.5 µg/m^3. This reference level is based on the geometric mean of effect levels for eye irritation in humans from the following two studies: a LOAEL of 138 µg/m^3 in a study of 36 volunteers exposed (eye only) to acrolein for 5 minutes, and a LOAEL of 210 µg/m^3 in a study of 53 volunteers exposed to increasing acrolein concentrations for 40 minutes. UFs of 6 for the use of LOAELs and 10 for intraspecies variation were applied, giving a total UF of 60. The revised value is set to protect against nasal lesions however it incorporates new scientific information pertaining to observed histological changes in the upper airways which are relevant to setting an air quality standard.

United States Environmental Protection Agency

The US EPA (2010) derived an acute exposure guideline limit (AEGL-1) of 70 µg/m^3 for non-disabling effects for timeframes of 10 minutes to 8 hours, based on eye irritation at 210 µg/m^3 in humans exposed to increasing acrolein concentrations for 40 minutes. An UF of 3 was applied to account for intraspecies variability.

In their pesticide evaluations, the US EPA (2008) and Health Canada's Pest Management Regulatory Agency (2016) derived a concentration of concern for short-term exposure of 7 µg/m^3, using a LOAEL of 210 µg/m^3 for eye irritation with UFs of 10 for intraspecies sensitivity and 3 or lack of no-observed-adverse-effect level (NOAEL), and a LOAEL of 700 µg/m^3 for nasal and throat irritation with UFs of 10 for intraspecies sensitivity and 10 for lack of NOAEL.

The US EPA (2003b) derived an inhalation reference concentration (RfC) of 0.2 µg/m^3, based on a LOAEL of 0.9 mg/m^3 from a 13-week rat study in 1978. The LOAEL was adjusted for continuous exposure (6 hours/14 hours and 5 days/7 days), and a human equivalent concentration (HEC) was calculated using a regional gas dose ratio (RGDR) conversion factor of 0.13 (HEC = 0.02 mg/m^3). This ratio accounts for pharmacokinetic but not pharmacodynamic differences between animals and humans; an UF of 3 was also applied for pharmacokinetic differences between species. UFs of 10 for sensitive human populations, 10 to account for the use of a subchronic study, and 3 for the use of a LOAEL were also applied, giving a total UF of 1000.

Health Canada and Environment Canada

The Government of Canada (Environment Canada and Health Canada, 2000) derived a tolerable concentration of 0.4 µg/m^3, based on a benchmark concentration producing a 5% response rate (BMC05) of 0.14 mg/m^3 from a 3-day study, which was adjusted for continuous exposure (6 hours/24 hours). UFs of 10 for interspecies extrapolation and 10 for sensitive human populations were applied, giving a total UF of 100.

Ontario Ministry of Environment, Conservation and Parks

Based on an evaluation of the scientific rationale of air guidelines from leading agencies, the following AAQCs are set for acrolein: A one-hour average AAQC of 4.5 µg/m^3, based on the development of irritation following acute exposure to acrolein; a 24-hour average AAQC of 0.4 µg/m^3, based on the development of lesions in the upper airways following chronic exposure to acrolein.

Alberta Environment

Alberta Environment (AENV, 2019) reports a 1-hour AAQO for Acrolein of 4.5 µg/m^3 (1.9 ppb) based on the development of irritation and 24-hour AAQO for 0.40 µg/m^3 (0.17 ppb) based on the development of lesions in upper airways. These values were both adopted from OMoE. According to OMoE, these levels are to protect against or prevent the development of nasal lesions following chronic exposure to acrolein.

California Office of Environmental Health Hazard Assessment

The California Office of Environmental Health Hazard Assessment (Cal OEHHA) is required to develop guidelines for conducting health risk assessments under the Air Toxics Hot Spots Program. Cal OEHHA, 2014 derived an acute Reference exposure level (REL) of 2.5 µg/m^3 (1.1ppb) based on the critical effects of subjective ocular irritation of eyes. The 8-hour REL and chronic REL are 0.70 µg/m^3 (0.30 ppb) and 0.35 ug/m^3 (0.15 ppb), respectively. Both of the above values are based on the critical effects of lesions in respiratory epithelium affecting the respiratory system.

Texas Commission on Environmental Quality

According to TCEQ (2015), a literature review was conducted for acrolein. The Weber-Tschopp et al. (1977) 1-hr study with a LOAEL of 0.3 ppm is selected as the key study because the exposure duration of 60 min corresponds to that desired for derivation of an acute Reference Value (ReV)/ Effects Screening Level (ESL). The experimental procedures and study discussion were more robust than those of the 1960 study and resulted in a LOAEL similar to that from the 40-minute Weber-Tschopp et al. (1970) study; and 1960 study only evaluated eye irritation for a 5-min exposure whereas the Weber-Tschopp study evaluated eye irritation (sensory effects) and effects on the respiratory tract using both qualitative and quantitative measures. The following UFs were applied to the point of departure adjusted for human equivalent concentration (POD_{HEC}) of 0.3 ppm: 10 for intra-human variability (UF_H), 6.3 for extrapolation from a LOAEL to a NOAEL (UF_L), and 1 for database uncertainty (UF_D) for a total UF = 63. Based on the above information, TCEQ derived the acute ReV (1 h) of 4.8 ppb. The acute ReV was multiplied by 0.3 to calculate the acute ESL. Thus, at the target hazard quotient of 0.3, the acute ESL is 1.4 ppb (3.2 µg/m^3).

Agence Nationale de Sécurité Sanitaire de L'alimentation

ANSES (2013) derived a short-term exposure guideline of 6.9 µg/m^3 for a 1-hour time frame, based on a LOAEL of 0.7 mg/m^3 for eye, nose, and throat irritation in volunteers exposed to acrolein for 60 minutes (Weber-Tschopp et al. 1977). UFs of 10 for the use of a LOAEL and 10 for intraspecies variability were applied, giving a total UF of 100.

ANSES (2013) also used the NOAEL of 0.46 mg/m^3 from a 2008 study to derive a long-term exposure guideline of 0.8 µg/m^3. No duration adjustment was made, and a HEC was calculated using an RGDR conversion factor of 0.13 (HEC = 60 µg/m^3). This ratio accounts for pharmacokinetic but not pharmacodynamic differences between animals and humans; an UF of 2.5 was also applied for pharmacokinetics. UFs of 10 for sensitive human populations and 3 to account for the use of a subchronic study were also applied, giving a total UF of 75.

4.1.2 BENZENE

Jurisdictional acute (or short-term, expressed as 1-hr and/or 8-hr) and chronic (or long-term, expressed as annual) exposure limits for benzene are provided in **Table 4-3** and Table 4-4, respectively. The studies supporting the available exposure limits are described in detail below.

Table 4-3 Acute Inhalation Exposure Limits for Benzene

Regulatory Agency	Type	Value (ppb)	Value (µg/m^3)	Reference
BC MoECCS	1-hr AAQO	-	-	BC MoECCS 2020

Regulatory Agency	Type	Value (ppb)	Value (µg/m^3)	Reference
	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	-	-	Ontario MECP 2020
	8-hr AAQC	-	-	
US EPA	1-hr Standard	-	-	US EPA NAAQS Table 2021
	8-hr Standard	-	-	
Cal OEHHA	8-hr REL	0.1	3	California OEHHA 2014
	1-hr REL	8	26	
WHO	1-hr AQG	-	-	WHO 2000
	8-hr AQG	-	-	

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 4-4 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 (IURs) 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	1	3	OEHHA 2014; based on health effects to hematologic system, nervous system, and development 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 IURs that range from 2.2 x 10^{-6} $(\mu g/m^3)^{-1}$ to 7.8 x 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.

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 TCEQ, the basis for the development of short-term and long-term ESLs 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 the air exceed the screening levels, it does not necessarily indicate a problem, rather, 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) modeling 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 comparison analysis based on BMD modeling of haematological data from the Ward *et al*. (1985) subchronic experimental animal inhalation study was also conducted. In addition, comparison analyses using the LOAELfrom 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 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 (IURs) of 2.2 x10^{-6} $(\mu g/m^3)^{-1}$ to 7.8 x 10^{-6} $(\mu g/m^3)^{-1}$ 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 HHA, 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 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 UF 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 vapors 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 UF 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 modeling 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 UF 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 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 the 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, 2001). 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 2001) to 0.45 µg/m^3. For 1 in 100,000 risk, 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 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 ReV and acute acute 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

World Health Organization (WHO) decided to rely on the 1994 risk calculations rather than derive new estimates. 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.

4.1.3 BENZO(A)PYRENE

Jurisdictional acute (or short-term, expressed as 1-hr and/or 8-hr) and chronic (or long-term, expressed as annual) exposure limits for benzo(a)pyrene are provided in **Table 4-5** and **Table 4-7,** respectively. The studies supporting the available exposure limits are described in detail below.

Table 4-5 Acute Inhalation Exposure Limits for Benzo(a)pyrene

Regulatory Agency	Type	Value (ppb)	Value (µg/m^3)	Reference
AENV	1-hour AAQO	-	-	AENV AAQO 2019
	8-hour AAQO	-	-	
ATSDR	Acute MRL	-		ATSDR, 1995
	Intermediate MRL	-		
Arizona Department of Health Services	24-hour	-	0.18	Arizona DHS, 1999
	1-hour	-	0.67	
BC ENV	1-hour AAQO	-	-	BC ENV 2020
	8-hour AAQO	-	-	
Cal EPA	AAQS	-	-	California EPA, 1999
TCEQ	1-hour average ESL		0.03	TNRCC, 2004
MOE	24-hours AAQC		0.00005	MOE 2020
US EPA	Reference Concentration (Developmental Toxicity)	-	0.002	US EPA, 2017
WHO	1-hour AQG	-	-	WHO 2000
	8-hour AQG	-	-	

AAQO - Ambient Air Quality Objective; AQG - Air Quality Guideline; REL – Reference Exposure Level; ESL – Effects Screening Levels; MRL – Minimal Risk Level; TLV-Threshold Limit Value; AAQS – Ambient Air Quality Standard; AAQC – Ambient Air Quality Criteria.

AENV – Alberta Environment; ATSDR-Agency for Toxic Substances and Disease Registry; Arizona DHS – Department of Health Services; BC ENV – British Columbia Ministry of Environment and Climate Change Strategy; Cal EPA – California Environmental Agency; TCEQ – Texas Commission on Environmental Quality; MOE – Ontario Ministry of Environment; US EPA – United States Environmental Protection Agency; WHO – World Health Organization.

AENV, ATSDR, BC ENV, Cal EPA and WHO have not established acute Ambient Air Quality Standards, objectives, criteria or exposure limits for benzo[a]pyrene.

Table 4-6 Chronic Inhalation Exposure Limits for Benzo(a)pyrene

Regulatory Agency	Type	Value (ppb)	Value (µg/m^3)	Reference
ATSDR	Chronic MRL	-		ATSDR 2007
BC ENV	Annual AAQO	-	-	BC ENV2020
AENV	Annual AAQO	2.9*10^{-5}	0.30 ng/m^3	AENV, 2019
MOE	Annual AAQC	-	0.00001	MECP 2020
TCEQ	Annual averaging time	-	0.003	TNRCC, 2004
Arizona DHS	Annual AAQG	-	0.00048	Arizona DHS, 1999
US EPA (unit risk)	Risk-Specific Concentration	-	0.002	US EPA, 2017; Risk-Specific Concentration that corresponds with an IUR of 6 x 10^{-4} (µg/m^3)$^{-1}$ and an excess lifetime risk level of I in 1,000,000.
Cal EPA (unit risk)	Risk-Specific Concentration		0.009	Cal EPA, 1999; Risk-Specific Concentration that corresponds with an IUR of 1 x 10^{-3} (µg/m^3)$^{-1}$ and an excess lifetime risk level of I in 100,000.
WHO (unit risk)	Ambient air guidance value (protection for general population using an IUR of 8.7(10^{-5}) per ng/m3 and corresponding to an excess lifetime risk level pf I in 100,000.	-	0.0012	WHO 2000

AAQO - Ambient Air Quality Objective; AAQC – Ambient Air Quality Criteria; AAQG – Ambient Air Quality Guideline; CAAQS – Canadian Ambient Air Quality Standard; MRL – Minimal Risk Level; IUR – Inhalation Unit Risk.

ATSDR-Agency for Toxic Substances and Disease Registry; BC ENV – British Columbia Ministry of Environment and Climate Change Strategy; AENV – Alberta Environment; MOE-Ontario Ministry of Environment; TCEQ – Texas Commission on Environmental Quality; Arizona DHS - Arizona Department of Health Services; US EPA – United States Environmental Protection Agency; Cal EPA – California Environmental Protection Agency; WHO – World Health Organization.

ATSDR, BC ENV, CCME, and MOE have not established annual Ambient Air Quality Standards, objectives, criteria or exposure limits for Benzo[a]pyrene.

Alberta Environment

Alberta Environment (AENV, 2019) reports an annual AAQO for B[a]P of 0.30 ng/m^3 based on chronic and carcinogenic human health effects. However, the basis for the selection of these thresholds was not specified in this document.

United States Environmental Protection Agency

Developmental toxicity, represented by decreased embryo/fetal survival, was chosen as the basis for the proposed inhalation RfC as the available data indicates that developmental effects represent a sensitive hazard of benzo[a]pyrene exposure. A 2002 developmental inhalation study in rats and the observed decreased embryo/fetal

survival (i.e., increased resorptions) following exposure to benzo[a]pyrene on gestation days 11−20 were used to derive the overall RfC. The LOAEL of 25 μg/m^3 based on decreased embryo/fetal survival was selected as the points of departure (POD). The LOAEL was adjusted to account for the discontinuous daily exposure to derive the POD_{ADJ} and the HEC was calculated from the POD_{ADJ} by multiplying by the regional deposited dose ratio for extra-respiratory (i.e., systemic) effects. These adjustments resulted in a POD_{HEC} of 4.6 μg/m^3, which was used as the POD for RfC derivation

The RfC was calculated by dividing the POD by a composite UF of 3,000 to account for toxicodynamic differences between animals and humans (3), interindividual differences in human susceptibility (10), LOAEL-to- NOAEL extrapolation (10), and deficiencies in the toxicity database (10).

Based on a study in 1981, the inhalation unit risk of 6×10^{-4} per μg/m^3 was calculated by linear extrapolation (slope factor = 0.1/benchmark concentration lower confidence limit ($BMCL_{10}$)) from a $BMCL_{10}$ of 0.16 mg/m^3 for the occurrence of upper respiratory and upper digestive tract tumors in male hamsters chronically exposed by inhalation to benzo[a]pyrene (US EPA, 2017). The corresponding risk-specific concentration from this IUR is 0.002 μg/m^3 based on an excess lifetime cancer risk level of 1 in 1,000,000.

Ontario Ministry of the Environment

The OMoE adopted an AAQC of 0.00005 μg/m^3 and 0.00001 μg/m^3 as a 24-hour and annual guideline, respectively. Note that the 24-hour AAQC is a converted value from the annual AAQC which is based on carcinogenic effects (MECP, 2020).

California Environmental Protection Agency

A risk specific concentration (RsC) of 0.009 μg/m^3 corresponding to 1 in 100,000 risk was used to illustrate a benzo[a]pyrene guideline for the Cal EPA (1999). The RsC corresponding to 1 in 100,000 risk (risk criteria used in Alberta) was derived using respiratory tract tumor data from male hamsters, in which an IUR of 1.1E-03 per (μg/m^3) was calculated using a linearized multistage procedure. It was based on the assumptions of additivity of individual risks posed by other selected PAHs with four or more rings classified as carcinogens.

Arizona Department of Health Services

The annual AAAQG is derived by taking the US EPA oral cancer slope factor of 7.3 [mg/kg/day]$^{-1}$ and an acceptable cancer risk of 1 in 1,000,000 (10^{-6}). The 24-hour AAAQG is derived by multiplying the annual AAAQG by 365. The one-hour AAAQG is derived by multiplying the 24-hour AAAQG by 3.8. The multiplier of 3.8 represents the proportional difference in the LOAEL for 24-hour and 1-hour exposure to a common irritant (SO_2) in human subjects (Arizona DHS, 1999). AAAQGs are not intended to be used as standards. Rather, they are intended to provide health-based guidelines that may be useful in making environmental risk management decisions. AAAQGs consider human health risk from inhalation of contaminants in ambient air. They do not take into account odor thresholds or threats to wildlife (Arizona DHS, 1999).

AAAQGs are residential screening values that are protective of human health, including children. Chemical concentrations in air that exceed AAAQGs may not necessarily represent a health risk. Rather, when contaminant concentrations exceed these guidelines, further evaluation may be necessary to determine whether there is a true threat to human health. Arizona DHS has individual guidelines for other selected PAHs with four or more rings that are classified as carcinogens (commonly present as mixtures of PAHs in the atmosphere with benzo[a]pyrene) (Arizona DHS, 1999).

Texas Commission on Environmental Quality

ESLs are used to evaluate the potential for effects to occur as a result of exposure to concentrations of constituents in air. ESLs are based on data concerning health effects, odor nuisance potential, effects with respect to vegetation, and corrosion effects. They 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, but rather, triggers a more in-depth review (TNRCC, 2004).

World Health Organization

The WHO (2000) recommended an ambient air guidance value of 0.0012 µg/m^3 for the general population using an inhalation unit risk factor of 8.7x 10^{-5} per mg/m^3 and corresponding to an excess lifetime cancer risk level of 1 in 100,000. The guideline is intended to provide background information and guidance to governments in making risk management decisions, particularly in setting standards. It is not stated how other selected PAHs with four or more rings classified as carcinogens are treated by the WHO.

4.1.4 NITROGEN DIOXIDE

Jurisdictional acute (or short-term expressed as 1-hr and/or 8-hr) and chronic (or long-term expressed as annual) exposure limits for NO_2 are provided in Table 4-7 and Table 4-8. Jurisdictions with established values are reviewed and studies supporting these exposure limits are described in detail below.

Table 4-7 Acute Inhalation Exposure Limits for NO2

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

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 Metro Vancouver (2020) revised their acute 1-hour AAQOs for NO_2 to further reduce NO_2 emissions and minimize impacts to public health resulting from increasing population density. Both BC MoECCS and Metro Vancouver adopted the 2020 CAAQS for NO_2 endorsed by the CCME in 2017. The Provincial Framework (2021) 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).

Canadian Council of Ministers of the Environment

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 support either the 2020 or 2025 CAAQS for NO_2.

Health Canada (2016) completed a comprehensive review of relevant health- and exposure-related data during the conduct of a "Human Health Assessment for Ambient Nitrogen Dioxide" to support the development of the CAAQS for NO_2 to replace the previous National Ambient Air Quality Objectives (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; 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) issued a 1-hour AAQO for NO_2 of 159 parts per billion (ppb; 300 $\mu g/m^3$) based on respiratory effects. The previous 24-hour AAQO of 200 $\mu g/m^3$ has been withdrawn by AENV. However, limited information is provided regarding the rationale for the derivation of 300 $\mu g/m^3$ 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, Chronic Obstructive Pulmonary Disease (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 UFs were selected by AENV in the derivation of the 1-hour AAQO of 300 $\mu g/m^3$.

Ontario Ministry of Environment, Conservation and Parks

The Ontario MECP provides a 1-hour AAQC of 200 ppb (400 $\mu g/m^3$) and a 24-hour AAQC of 100 ppb (200 $\mu g/m^3$). While the 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, 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 Cal 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 an 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 World Health Organization (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 exceeding 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 airways responsiveness to a range of constrictor stimuli when asthmatic subjects were exposed to levels of NO_2 > 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 its guideline for NO_2 since 2005.

Table 4-8 Chronic Inhalation Exposure Limits for NO_2

Regulatory Agency	Type	Value (ppb)	Value (µg/m^3)	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	24-hour AAQC	-	-	Ontario MECP 2020
US EPA	Annual Standard	53	100	US EPA 2018
WHO	Annual AQG	-	40	WHO 2005

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 (2021) 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 AAQOs 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 the 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 (MV2019).

Canadian Council of Ministers of Environment

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/m^3) based on its 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 Ontario MECP has not determined an annual AAQC for NO_2.

United States Environmental Protection Agency

The US EPA (2012) has not derived an inhalation RfC for NO_2. In 1971, US EPA derived a NAAQS of 53 ppb (100 µg/m^3) which remains current to date based on a scientific and regulatory review that was completed (US EPA, 2010). Although the 1971 document is not readily available, the scientific reviews conducted in 1993 and 2010 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/m^3) was 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) was 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 23 ppb (40 µg/m^3) 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 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.

4.1.5 FINE PARTICULATE MATTER (<2.5 µm)

Jurisdictional acute (or short-term, expressed as 1-hr and/or 24-hr) and chronic (or long-term, expressed as annual) exposure limits for $PM_{2.5}$ are provided in **Table 4-10** to **Table 4-11**. The studies supporting the available exposure limits are described in detail below.

Table 4-9 Acute Inhalation Exposure Limits for $PM_{2.5}$

REGULATORY AGENCY	TYPE	VALUE (ppb)	VALUE ($\mu g/m^3$)	SOURCE
BC MoECCS	24-hour	-	25	BC MoECCS 2020
AENV	1-hour	-	80	AENV AAQO 2018
	24-hour	-	29	
CCME 2020 (2025)	24-hour	-	27	CCME 2019
ON MECP	24-hour	-	27	Ontario MECP 2020
US EPA	24-hour	-	35	US EPA 2021
Cal OEHHA	-	-	-	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; 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

British Columbia Ministry of Environment and Climate Change Strategy

The new AAQC for $PM_{2.5}$ were adopted by the BC MoECCS (2020) on April 9, 2009 and remains as the current provincial standard. The 24-hour AQO was set to 25 $\mu g/m^3$ and is based on the annual 98th percentile of daily average, over one year. No technical supporting documents detailing the derivation of the AQO were made available.

Alberta Environment

Alberta Environment (AENV, 2019) issued a 1-hour and 24-hour AAQO of 80 $\mu g/m^3$ and 29 $\mu g/m^3$, respectively. The 1-hour value is intended for use in monitoring and reporting of the Ambient Air Quality Index. 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 (ERVs) 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 Ontario MECP (2020) provides a 24-hour AAQC for $PM_{2.5}$ of 27 $\mu g/m^3$. 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 revised from 65 to 35 µg/m^3. This value is identified as a 98th percentile, 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.

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.

World Health Organization

The World Health Organization (WHO, 2005) provided a 24-hour guideline for $PM_{2.5}$ of 25 µg/m^3. This value represents a 99th percentile of the distribution of daily values and are intended to protect against peaks of pollution that would lead to substantial excess morbidity or mortality. The 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 significant reductions in risks from acute exposure health effects such as short-term mortality.

Table 4-10 Chronic Inhalation Exposure Limits for $PM_{2.5}$

REGULATORY AGENCY	TYPE	VALUE (ppb)	VALUE (µg/m^3)	SOURCE
BC MoECCS	Annual	-	8	BC MoECCS 2020
AENV	-	-	-	AENV AAQO 2019
CCME 2020 (2025)	Annual	-	8.8	CCME 2021
ON MECP	Annual	-	8.8	Ontario MECP 2020
US EPA	Annual	-	12	US EPA 2021
Cal OEHHA	Annual	-	12	Cal OEHHA 2016
WHO	Annual	-	10	WHO 2005

Notes:
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

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.

Ontario Ministry of the Environment, Conservation and Parks

The 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 value was derived.

United States Environmental Protection Agency

In 2013, US EPA revised 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 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.

4.2 TOXICOLOGICAL REVIEW OF COPCS

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

4.2.1 ACROLEIN

Acrolein is a colourless or yellowish liquid at 1013 hPa and 20 °C. Acrolein is miscible with lower alcohols, ketones, benzene, diethyl ether, and other common organic solvents (ECHA, 2022). It is a very reactive and volatile α,β-unsaturated aldehyde, which is found in both indoor and outdoor air (HC, 2021) .

Acrolein is ubiquitous throughout the ambient environment. The primary natural source of acrolein is incomplete combustion of organic matter during forest fires. The principal anthropogenic source of atmospheric acrolein is the combustion of organic matter and fuels, with motor vehicles (including aircrafts) generating most of the acrolein

emissions. Industrial processes such as incineration, pulp and paper and oriented-strand board production, and coal electricity generation also contribute to acrolein emissions, though much less than mobile sources.

Acrolein levels in residential indoor air are generally greater than outdoor levels. Some of the sources of acrolein in indoor air are smoking, using gas stoves, wood-burning fireplaces, burning incense, cooking with oils, and secondary formation by oxidation of other VOCs from products and building materials. However, no information is available on the relative contributions of these various sources to the total indoor air concentration of acrolein. (HC, 2021).

4.2.1.1 *SHORT-TERM HEALTH EFFECTS*

Among all acrolein studies, eye irritation was the most sensitive endpoint, occurring at concentrations of 0.14 to 0.23 mg/m^3 for exposure durations as short as 5 minutes (HC, 2021).

Weber-Tschopp *et al.* (1997) conducted three studies. In the first study, 53 volunteers were exposed to continuously increasing acrolein concentrations (up to 1.4 mg/m^3) for 40 minutes; significantly higher incidence of eye irritation was first observed at 0.210 mg/m^3. Reports of nasal irritation was noted starting at 0.35 mg/m^3, throat irritation starting at 1.0 mg/m^3 and respiratory irritation (measured by decreased respiration rate) starting at 0.69 mg/m^3. In the second study, 42 subjects were exposed to acrolein for 1.5 minutes at concentrations of 0.35 to 1.4 mg/m^3. Finally, 46 volunteers were exposed to acrolein for 60 minutes at 0.69 mg/m^3. Eye, nose, and throat irritation increased during the first 10 to 20 minutes, and there was a significant decrease in respiration rate.

Similar effects were observed from other studies. In a 1957 study that observed that exposures of 1.84 mg/m^3 for 10 minutes, or 2.76 mg/m^3 for 5 minutes were “extremely irritating” and caused lacrimation (US EPA, 2003a). Another study in 2016 found that volunteers reported eye irritation starting about 7 minutes into a 15-minute eye-only exposure to 0.36 mg/m^3 acrolein. Irritation continued for 10 minutes after cessation of exposure. No difference in eye irritation was found between control exposures and a 45-minute exposure to 0.16 mg/m^3 or a 60-minute exposure to 0.07 mg/m3. A study in 2015 exposed 18 subjects to 0.12 or 0.23 mg/m^3 acrolein for 2 hours. Subjective eye irritation and blink frequency were slightly increased at 0.23 mg/m^3 but not 0.12 mg/m^3 acrolein. There was no difference between control and exposed subjects in terms of breathing frequency, pulmonary function, or inflammatory markers in blood or sputum.

Several case studies describe the effects of acute exposure to acrolein; however, exposures are often to multiple substances, and acrolein concentrations are generally unknown. A two-year-old boy was hospitalized for acute respiratory failure following exposure for about an hour to acrid smoke from vegetable oil burning. Lung effects were still visible eighteen months following exposure (Cal OEHHA, 2008). A chemical worker was exposed to a sudden release of acrolein in the workplace, causing chemical pneumonia and eye irritation, both of which were resolved with treatment (US EPA 2003a). The Centers for Disease Control and Prevention (CDC) (2013) conducted a review of acute poisonings to acrolein from occupational use of pesticides and identified eight cases in the United States between 1993 and 2009. Symptoms observed included respiratory distress, eye irritation, headache, dyspnea, and skin irritation/burns.

Therefore, eye irritation is the most sensitive endpoint, and the t LOAELs identified for this endpoint were 0.21 mg/m^3 from a study in 1977 and 0.23 mg/m^3 from another study in 2015. As the 1977 study did not identify a NOAEL, the NOAEL of 0.12 mg/m^3 (115 $\mu g/m^3$) for eye irritation from the 2015 study was selected as the POD for the acute RfC. This POD is also below the LOAEL and NOAEL for respiratory effects observed by 1997 study and 2015 study, respectively. An UF of 3 was applied to account for sensitive individuals and is considered sufficient as eye irritation due to contact is not expected to vary greatly across the population (NRC 2001; US EPA 2008). No UF for database deficiencies was applied as the critical study and the database for acute toxicity were adequate. Thus, the acute RfC is 38 $\mu g/m^3$ (HC, 2021).

Adverse health effects reported in well conducted human studies following the acute inhalation of acrolein and the air concentration at which they are predicted to occur are summarized in **Table 4-12** below.

Table 4-11 Acute Effects Following Human Exposure to Acrolein

Acute Effects Following Human Exposure to Acrolein Effect	Exposure Period	Air Concentration ppm (mg/m^3)	Reference
Eye irritation	5 minutes	0.06 (0.14)	HC, 2021
Eye irritation	40 minutes	0.09 (0.21)	Weber-Tschopp *et al*. 1977
Nasal irritation	40 minutes	0.15 (0.35)	Weber-Tschopp *et al*. 1977
Throat irritation	40 minutes	0.43 (1)	Weber-Tschopp *et al*. 1977
A decrease in respiration rate	40 minutes	0.6 (1.4)	Weber-Tschopp *et al*. 1977
Eye irritation	1.5 minutes with recovery period between exposures	0.3 (0.69)	Weber-Tschopp *et al*. 1977
Nasal Irritation	1.5 minutes with recovery period between exposures	0.6 (1.4)	Weber-Tschopp *et al*. 1977
Eye, nose, throat irritation, and decrease in reparation rate	60 minutes	0.3 (0.69)	Weber-Tschopp *et al*. 1977
lacrimation	10 minutes	0.8(1.84)	HC, 2021
lacrimation	5 minutes	1.2 (2.76)	HC, 2021
Eye irritation	7 minutes	(0.36)	HC, 2021
Eye irritation	2 hours	0.1(0.23)	HC, 2021

4.2.1.2 *LONG-TERM HEALTH EFFECTS*

Epidemiological data on the long-term effects in humans are limited to two studies in France. One study showed a positive association between acrolein levels in schools and allergic asthma in the previous year, and between acrolein levels and exercise-induced asthma, but a negative association between acrolein levels and non-allergic asthma. In the other study, no significant relationship was identified between acrolein levels measured in homes and asthma in the previous year. Neither study showed a relationship between acrolein levels and rhinitis (HC, 2021).

A study in 2008 identified a NOAEL of 0.46 mg/m^3 and LOAEL of 1.38 mg/m^3 for degenerative lesions in the respiratory epithelium of the rat nasal cavity. The NOAEL of 0.46 mg/m^3 was selected as the POD because it was the lowest exposure concentration associated with an adverse effect. Toxicokinetic differences between rats and humans were accounted for by applying a regional gas dose ratio of 0.13 for a category 1 gas with extra-thoracic respiratory effects, giving a human equivalent NOAEL of 11 $\mu g/m^3$. UFs of 2.5 for toxicodynamic differences between rats and humans, and 10 for sensitivity in the human population were also applied. Thus, the long-term RfC is 0.44 $\mu g/m^3$.

Regarding acrolein developmental and reproductive toxicity, the existing data do not suggest that inhalation of an extremely reactive and irritating aldehyde like acrolein would present a significant teratogenic or reproductive risk. While the delivered dose of acrolein to the embryo as a consequence of cyclophosphamide or other anticancer drug metabolism can be sufficient to induce developmental toxicity, the absorbed dose of acrolein after inhalation of the compound would be insufficient to produce an increase in acrolein concentrations in tissues distant from initial contact (ACGIH, 2001).

4.2.1.3 CARCINOGENIC EFFECTS

With respect to carcinogenicity, the International Agency for Research on Cancer (IARC) considers acrolein "not classifiable as to its carcinogenicity to humans" (Group 3; IARC 1995) due to inadequate evidence in both humans and experimental animals. The US EPA also considers the acrolein database inadequate for the assessment of its carcinogenicity potential (US EPA, 2003). Conclusions regarding its carcinogenicity potential cannot be drawn from the limited studies available (HC, 2021).

One occupational case-control study in 1989 identified workers exposure to multiple chemicals. Exposure to acrolein was reported for two men who had died with non-Hodgkin's lymphoma, one with multiple myeloma, and three with nonlymphocytic leukaemia. There was no statistically significant increase in cancer cases for workers exposed to acrolein, therefore, the results of this study are insufficient to conclude on the carcinogenic potential of acrolein.

No additional studies on the carcinogenic potential of inhaled acrolein were identified in the literature (HC, 2021).

4.2.2 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.

4.2.2.1 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). Brief exposure (30 minutes) to 300 ppm (978 mg m-3) benzene produced drowsiness, dizziness and headaches in exposed workers (ATSDR).

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).

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 has identified well conducted occupational studies with effects linked to specific benzene exposure concentrations. Adverse health effects reported in well conducted 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 4-12 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

Acute Effects	Exposure Period	Air Concentration ppm (mg m^{-3})	Reference
Neurological: drowsiness, dizziness, headaches	30 min	300 (978)	Flury *et al*. 1928
Neurological: dizziness, headaches, nausea, fatigue (males)	1-21 d, 2.5-8 hr/d	60 (196)	Midzenski *et al*. 1992
Respiratory: mucus membrane irritation and dyspnea (males). Hematological: leucopenia, anemia, and thrombocytopenia (males).	1-21 d, 2.5-8 hr/d	60 (196)	Midzenski *et al*. 1992

4.2.2.2 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.

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. 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.

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 acute myelogenous leukemia, or cancer of the myeloid line of white blood cells (ATSDR).

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). 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). 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).

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 1-21 years (ATSDR). This exposure level was selected by the California Office of Environmental Health Hazard Assessment and adjusted for continuous exposure and variation in human sensitivity to develop a chronic REL of 0.02 ppm or 60 µgm-3 (OEHHA).

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). A chronic MRL of 0.003 ppm

(0.01 mg m-3) was determined using BMD modeling and adjusting from occupational to continuous exposure. A 10-fold UF was also applied to account for variations in human sensitivity (ATSDR).

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 modeling and adjusted for human variability, subchronic-to-chronic exposures, and database deficiencies to arrive at an RfC of 30 µg m-3 for lifetime chronic human exposure to benzene (US EPA, 2002).

The California 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). 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).

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). Paternal exposure to benzene prior to conception was also associated with childhood leukemia (US EPA, 1998).

4.2.2.3 *CARCINOGENIC EFFECTS*

Both the IARC and the 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 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 acute myelogenous leukemia, often referred to as AML. This is a cancer of the bloodforming 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 ANL as well as non-Hodgkin's lymphoma and multiple myeloma (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 has been used for the development of ambient air quality guidelines 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.

4.2.3 BENZO(A)PYRENE

Benzo[a]pyrene is a five-ring polycyclic aromatic hydrocarbon (PAH) (US EPA, 2017). It exists in various crystalline forms when pure, usually as yellowish plates or needles. It is insoluble in water, but very soluble in chloroform and it is also soluble in benzene, toluene, and xylene. In nature, Benzo[a]pyrene is considered an environmental pollutant, usually bound to small particulate matter present in smoke from forest fires, industrial processes, vehicle exhaust, cigarettes, and through the burning of fuel (such as wood, coal, and petroleum products). Benzo[a]pyrene levels are often used as a rough index of air pollution and of total PAHs (ACGIH, 2001).

Although epidemiological and toxicological studies have confirmed that benzo[a]pyrene is a potent carcinogen, benzo[a]pyrene emissions are not controlled in the United States (ACGIH, 2001). The magnitude of human exposure to benzo[a]pyrene depends on factors such as lifestyle (e.g., diet, tobacco smoking), occupation, and living conditions (e.g., urban versus rural setting, domestic heating, and cooking methods) (US EPA, 2017).

Inhalation exposure to single PAH compounds, for example benzo[a]pyrene alone, does not occur without other PAHs being present. Several PAHs with four of more rings are treated as having the potential to cause cancer in addition to benzo[a]pyrene. As a result, benzo[a]pyrene is proposed as an indicator for the carcinogenic fraction of these PAHs which are all present as mixtures in ambient air. Further, a method using factors of 10 to represent the potency of individual PAHs relative to benzo[a]pyrene is recommended to address mixtures of PAHs in ambient air (AENV, 2004).

4.2.3.1 SHORT-TERM HEALTH EFFECTS

There is limited information on non-carcinogenic acute toxicity in humans and animals, and any acute human studies looking at non-carcinogenic effects focus on developmental endpoints, which are discussed below (US EPA, 2017; ATSDR, 1995; WHO, 2000).

4.2.3.2 LONG-TERM HEALTH EFFECTS

The primary route of benzo[a]pyrene exposure is via inhalation, and the majority of epidemiologic studies to date have studied the correlation between mortality from lung cancer and benzo[a]pyrene exposure. Although cigarette smoking, air pollution, and occupational exposure are all significant means of inhalation exposure, it is generally agreed that cigarette smoking is the overwhelming factor in the causation of lung cancer (ACGIH, 2001).

Table 4-13 Chronic Effects Following Human Exposure to Benzo(a)Pyrene

Chronic Effects Following Human Exposure to Benzo[a]pyrene	Exposure Period	Air Concentration ppm (mg/m^3)	Reference[1]
Respiratory: bloody vomit, breathing problems, chest pains, chest irritation, throat irritation and cough (Serious LOAEL)	6 months to <6 years (Occupational study)	9.69×10^{-6} (0.0001)	ATSDR, 1995
Increased rate of mutations in peripheral lymphocytes (LOAEL)	2 to 46 years (Occupational study)	8.7×10^{-5} (0.0005)	ARSDR, 1995
Reduced serum immunoglobins	Average 15 years	$1.9\text{x}10^{-5}$ – 0.048 (0.0002 - 0.50) Szczeklik *et al.*, cited in	ATSDR, 1995

The available human PAH mixtures studies report developmental and reproductive effects that are generally analogous to those observed in animals, and provide qualitative, supportive evidence for the hazards associated with benzo[a]pyrene exposure (US EPA, 2017).

Human and animal studies provide evidence for benzo[a]pyrene-induced male and female reproductive toxicity. Effects on sperm quality and male fertility have been demonstrated in human populations highly exposed to PAH mixtures. The use of internal biomarkers of exposure in humans (e.g., BPDE-DNA adducts) supports associations

between benzo[a]pyrene exposure and these effects. In females, numerous epidemiological studies indicate that cigarette smoking reduces fertility; however, few studies have specifically examined levels of benzo[a]pyrene exposure and female reproductive outcomes. Animal studies demonstrate decrements in sperm quality, changes in testicular histology, and hormone alterations following benzo[a]pyrene exposure in adult male animals, and decreased fertility and ovo-toxic effects in adult females following exposure to benzo[a]pyrene. (US EPA, 2017)

Animal studies demonstrate that exposure to benzo[a]pyrene is associated with developmental (including developmental neurotoxicity), reproductive, and immunological effects. In addition, epidemiology studies involving exposure to PAH mixtures have reported associations between internal biomarkers of exposure to benzo[a]pyrene (benzo[a]pyrene diol epoxide-DNA adducts) and adverse birth outcomes (including reduced birth weight, postnatal body weight, and head circumference), neurobehavioral effects, and decreased fertility (US EPA, 2017).

4.2.3.3 CARCINOGENIC EFFECTS

The strong and extensive experimental evidence for the carcinogenicity of benzo[a] pyrene in many animal species, supported by the consistent and coherent mechanistic evidence from experimental and human studies provide biological plausibility to support the overall classification of benzo[a]pyrene as a human carcinogen (Group 1). (IARC, 2010)

According to US EPA, benzo[a]pyrene is “carcinogenic to humans” based on strong and consistent evidence in animals and humans. The evidence includes an extensive number of studies demonstrating carcinogenicity in multiple animal species exposed via all routes of administration and increased cancer risks, particularly in the lung and skin, in humans exposed to different PAH mixtures containing benzo[a]pyrene. Mechanistic studies provide strong supporting evidence that links the metabolism of benzo[a]pyrene to DNA-reactive agents with key mutational events in genes that can lead to tumor development. These events include formation of specific DNA adducts and characteristic mutations in oncogenes and tumor suppressor genes that have been observed in humans exposed to PAH mixtures. This combination of human, animal, and mechanistic evidence provides the basis for characterizing benzo[a]pyrene as “carcinogenic to humans.” (US EPA, 2017)

4.2.4 NITROGEN DIOXIDE

NO_2 is the main pollutant within a group known as NO_x. NO_2 is produced from nitrogen and oxygen during fuel combustion; as such, ambient NO_2 comes from burning of coal, fuel, oil, diesel, and gasoline. 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 combine 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.

4.2.4.1 SHORT-TERM HEALTH EFFECTS

In support of CAAQS development, Health Canada conducted a comprehensive HHA 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 parts per billion (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 airway hyperresponsiveness , 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 interquartile range 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 and 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 ERV. 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.

4.2.4.2 *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 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 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 49.31ppb.

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 find 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 is several of these associations were more strongly related to particulate matter 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 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.

4.2.4.3 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.

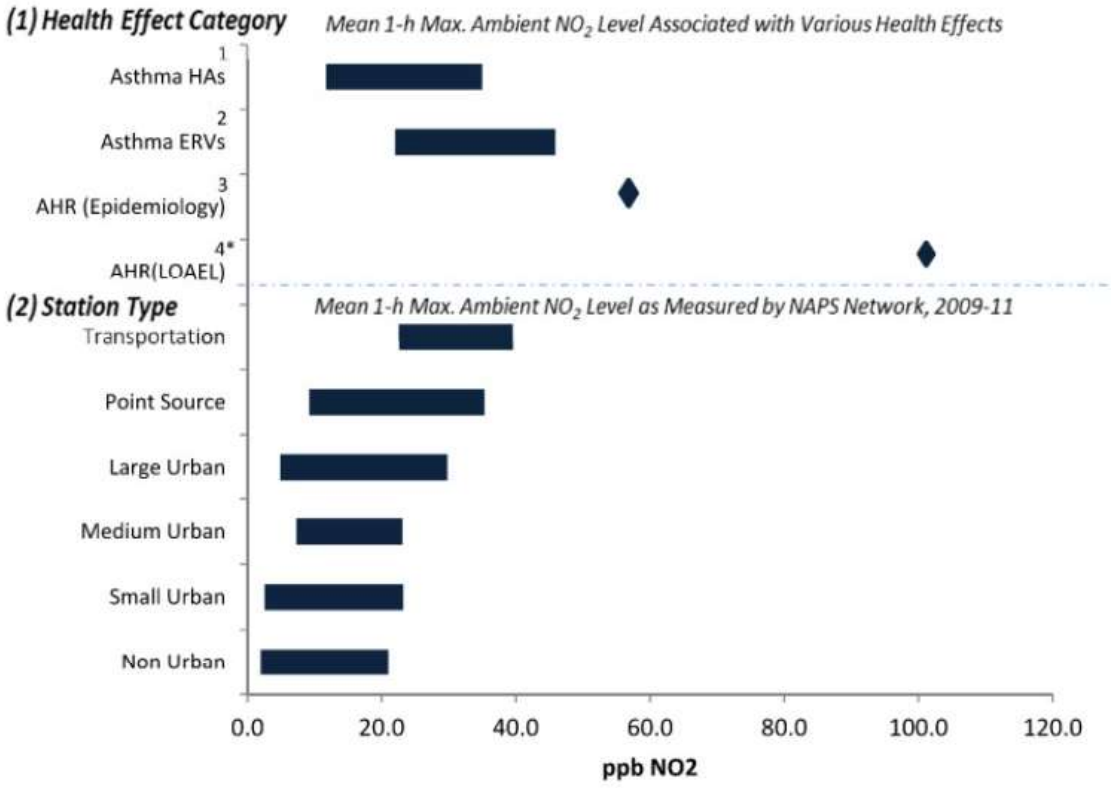
Effects of NO_2 on reproduction in humans are not known. IARC and US EPA have not classified nitrogen oxides for potential carcinogenicity. Nitrogen oxides have caused changes in the genetic material of animal cells, but it is not known if these can cause developmental effects in humans.

4.2.4.4 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.

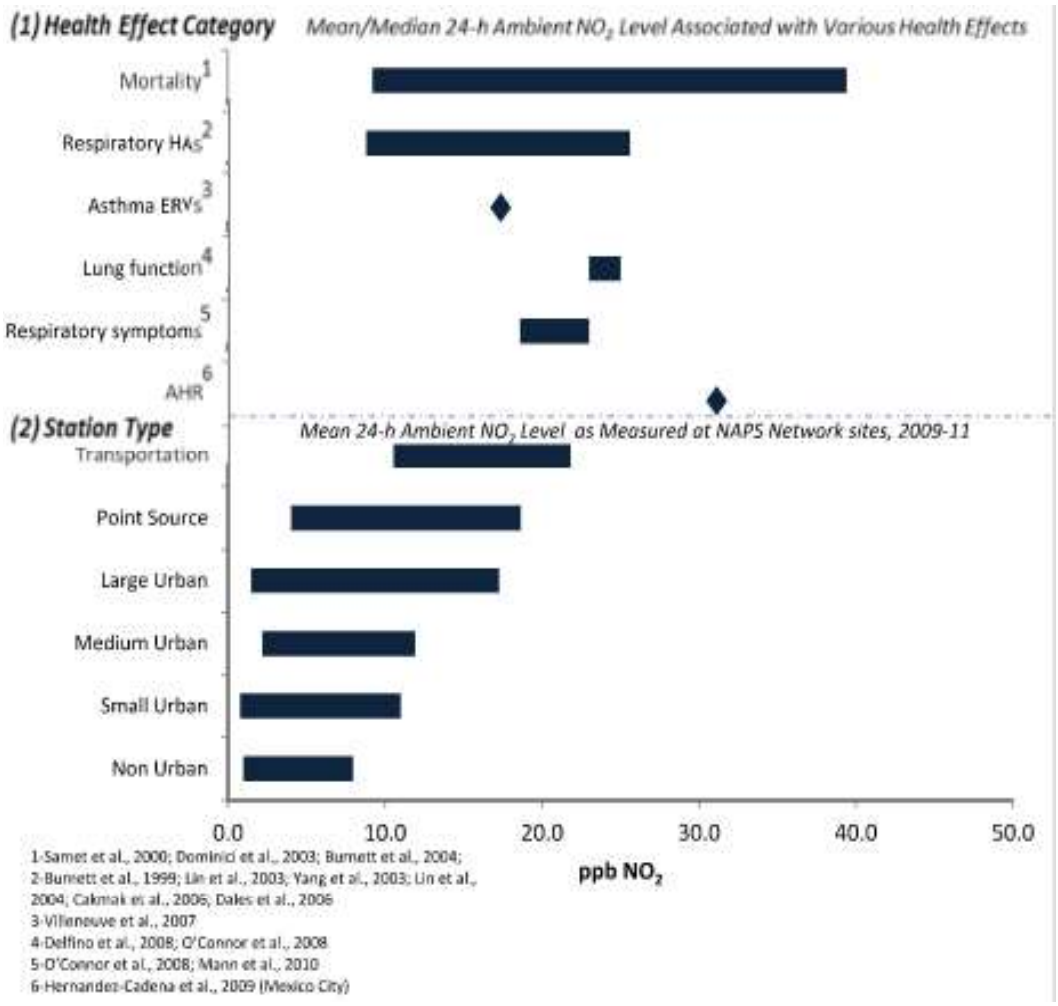
Health Canada (2016) presented the analyses in **Figure 4-1** for the daily 1-hour max NO_2, in **Figure 4-2** for the 24-hour average NO_2, and in **Figure 4-3** for NO_2 as the long-term (annual/multi-year) average. For each figure, the top panel presents the mean or median NO_2 levels associated with 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)

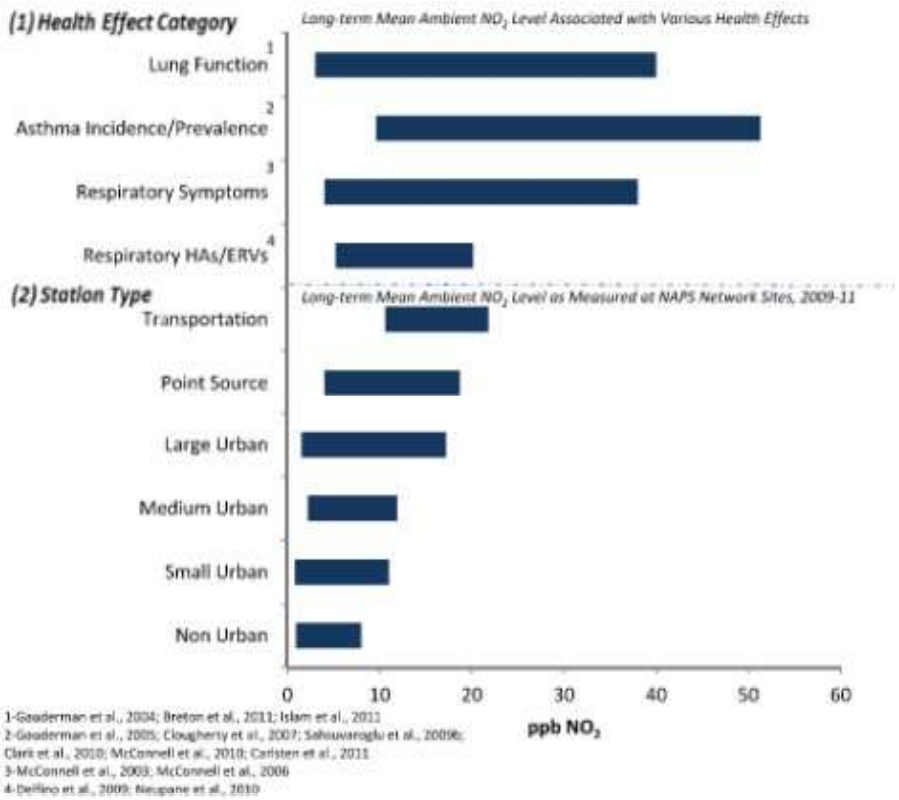
Notes:
HA – hospital admissions
ERV – emergency room visits
AHR – airway hyper-responsiveness

Figure 4-1 **Comparison between daily 1-h max 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 12.1 from Health Canada (2016))**



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

Figure 4-2 **Comparison between mean 24-h avg 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 12.2 from Health Canada (2016))**



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

Figure 4-3 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))

4.2.1 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 PM (US EPA, 2019), which builds upon a previous ISA for PM 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 4-14 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 4-14 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 4-14.

4.2.1.1 *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.

4.2.1.2 *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.

4.2.1.3 *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.

4.3 SELECTED TOXICOLOGICAL REFERENCE VALUES FOR APPLICATION IN THE HHA

Based on review of available jurisdictional health-based standards for selected COPCs, as well as review of health and exposure related data reviewed and discussed in the toxicological summary write-up, this HHA adopted the health-based TRVs shown in **Table 4-15**, below.

Table 4-15 Selected TRVs for the HHA

COPC	Type	TRV	Source	Basis
Acute Exposure Duration				
Benzene	24-hr	30 µg/m^3	US EPA (2003)	**Protection against hematopoietic effects.** This TRV (30 µg/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.

COPC	Type	TRV	Source	Basis
Acrolein	1-hr	2.5 µg/m^3	Cal OEHHA (2014)	**Protection against eye irritation** This 1-hr TRV is based on the geometric mean of effect levels for eye irritation in humans from two studies: a LOAEL of 138 µg/m^3 in a study of 36 volunteers exposed (eye only) to acrolein for 5 minutes, and a LOAEL of 210 µg/m^3 in a study of 53 volunteers exposed to increasing acrolein concentrations for 40 minutes. A total UF of 60 was applied.
Benzo(a)pyrene	24-hr	0.002 µg/m^3	US EPA (2017)	**Protection against decreased embryo/fetal survival** This TRV was chosen given that developmental effects represent a sensitive hazard of benzo(a)pyrene exposure. The TRV is based on a LOAEL of 25 µg/m^3 from a developmental inhalation study in rats which observed decreased embryo/fetal survival. Several adjustments including use of an UF of 3000 were then applied to derive the TRV.
NO_2	1-hr	113 µg/m^3	Health Canada (2016)	**For protection of airway hyper-responsiveness (AHR)** The 1-hour TRV (113 µg/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 max NO_2, with similar decreases in response 0 to 3 days following exposure inhaled corticosteroids.
	1-hr	79 µg/m^3	Health Canada (2016)	**To reduce frequency of asthma ERVs** Asthma ERV is also considered as a health endpoint in this HHA 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 µg/m^3	WHO (2005)	**For protection against excess morbidity or mortality** This 24-hour TRV (25 µg/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 µg/m^3. This value is considered to provide a significant reduction in risks from acute exposure health effects such as short-term mortality.

COPC	Type	TRV	Source	Basis
Chronic Exposure Duration				
Benzene	Annual (carcinogenic); 24-hr (non-carcinogenic)	0.45 µg/m^3 [1] (carcinogenic); 30 µg/m^3 (non-carcinogenic)	Health Canada (2021), TCEQ (2015) and US EPA (2003)	**Carcinogenic** **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. **Non-Carcinogenic** **Protection against hematopoietic effects.** A TRV of 30 µg/m^3 was also used for chronic non-carcinogenic exposures given the 24-hr averaging period. The basis of this value is outlined (above) under the acute exposure duration heading of this table.
Acrolein	Chronic (24-hr)	0.4 µg/m^3	HC and EC (2000); MECP (2009)	**Protection against development of lesions in upper airways** This TRV is derived from a BMC05 of 0.14 mg/m^3 from a 3-day study. Adjustments for continuous exposure and a total UF of 100 was applied derive the TRV.
Benzo(a)pyrene	Annual (carcinogenic);	0.002 µg/m^3[2] (carcinogenic);	US EPA (2017)	**Protection against upper respiratory and digestive tract tumors** This TRV is based on a study in 1981 which calculated an IUR of 6.0E-04 per µg/m^3 by linear extrapolation from a $BMCL_{10}$ of 0.16 mg/m^3 for the occurrence of upper respiratory and upper digestive tract tumors in male hamsters chronically exposed by inhalation.
NO_2	Annual	23 µg/m^3	Health Canada (2016)	**Protection of respiratory morbidity** This TRV (23 µg/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.
$PM_{2.5}$	Annual	10 µg/m^3	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.

Notes:

[1] Value reported in Health Canada is 4.5 µg/m^3 (based on an incremental lifetime cancer risk of 1-in-100,000). This value was converted to 0.45 µg/m^3 to reflect an incremental lifetime cancer risk of 1-in-1,000,000, which was applied as part of this assessment.

[2] IUR was converted to a risk-specific concentration of 0.002 µg/m^3 to reflect an incremental lifetime cancer risk of 1-in-1,000,000, which was applied as part of this assessment.

All chronic TRVs evaluated for benzo(a)pyrene as part of this assessment were based on carcinogenic human health effects; as such, benzo(a)pyrene was assessed as a carcinogen only for chronic exposure.

4.4 UNCERTAINTY ANALYSIS

The major sources of uncertainty associated with the hazard assessment of the HHA are briefly described below

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 in ambient air and potential confounding health effects: 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 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.
- 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 Metro Vancouver, 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.

Fine Particulate Matter (<2.5 µm):

- 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 HHA.
- 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.

Acrolein:

- As only limited data were available on repeated inhalation exposure to acrolein in humans, animal data were used as a POD when deriving the RfC. Although the nature of effects (irritation) is likely to be the same across species, quantitative differences in sensitivity were accounted for using default values for the toxicodynamic UF (rats to humans) and an intraspecies UFs (for sensitive individuals). No studies could be found on the effects of acrolein in sensitive individuals such as asthmatics which would reduce the uncertainty in the RfC.
- Studies on the effects of long-term inhalation exposure to acrolein are limited. There were also significant limitations, as described in section 4 to the few epidemiological studies examining associations between acrolein exposure and asthma or rhinitis. Similarly, most studies in experimental animals did not go beyond a subchronic duration, and those few chronic studies available were inadequate to draw conclusions about the carcinogenicity of acrolein.
- Existing exposure studies have evaluated 24-hour sampling times to give an average daily exposure. Exposures to peak concentrations over shorter durations have not been evaluated. As described in section 3, acrolein is difficult to quantify accurately, and current methods have limitations.

- Exposure to co-pollutants in ambient air and potential confounding health effects: Exposure to co-pollutants remains the major uncertainty in the overall health database for air pollutants including acrolein.

Benzene:

- It is noted that no jurisdictional limits were identified from Ontario MECP 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.

Benzo(a)pyrene:

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

5 RISK CHARACTERIZATION

Risk characterization is the final step in the HHA process, during which the exposure and hazard (toxicity) assessments are integrated. The process of risk characterization conducted in this HHA 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.

5.1 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-carcinogens. Regulatory agencies assume that for non-carcinogens, there is a dose 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 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 toxicity values, 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) versus chronic (annual) exposures.

For acute exposures, the daily maximum concentration (1-hour or 8-hour) is compared directly to the acute TRV to calculate a HQ.

For chronic exposures, EE is defined as the 24-hour or 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)

A HQ benchmark (or “Target HQ”) of 1.0 was applied to acute and chronic exposures for all COPCs and for all human receptors. In the case where contaminant exposure from all potential sources, including ambient exposures

are considered, a HQ benchmark of 1.0 is considered acceptable. This assumption is considered to be met for all identified human receptors (i.e., toddler and adult residents).

5.2 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 health 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 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 4, benzene and benzo(a)pyrene 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 a 76-year lifespan as a result of exposure to a carcinogenic COPC associated with the proposed development (i.e., incremental risk above the typical background risk that exists). The MECP considers that acceptable ILCR to be one-in-one million (1 x 10^{-6}). An ILCR greater than 1 x 10^{-6} is indicative of a potential health concern that should be more closely examined. An ILCR of less than 1 x 10^{-6} is considered essentially negligible.

5.3 RESULTS OF THE QUANTITATIVE ASSESSMENT

In this section, the contribution of overall risk from each source-receptor pathway is discussed. The predicted exposure estimates, ILCRs, and HQs for acute and chronic exposures for each of the identified receptors and COPCs are provided in **Table 5-1** to **Table 5-12**.

5.3.1 ACROLEIN

Table 5-1 Predicted Non-Carcinogenic Health Risks Associated with Acute Exposure to Acrolein for Toddler and Adult Residents

1-Hr Acute TRV (µg/m^3)	Background Conc. (µg/m^3)	HQ (Background)	Modelled Conc. (µg/m^3)	HQ (Modelled -Only)	Cumulative Conc. (µg/m^3)	HQ (Cumulative)	% Background HQ Attributable to Cumulative
Toddler Resident							
2.5	1.6	6.4E-01	0.01	4.0E-03	1.6	6.4E-01	99%
Adult Resident							
2.5	1.6	6.4E-01	0.01	4.0E-03	1.6	6.4E-01	99%

Notes:
Cumulative Concentration = Background concentration + Modelled concentration

Target HQ = 1
HQs presented in **bold** if exceeding Target HQ

Table 5-2 Predicted Non-Carcinogenic Health Risks Associated with Chronic Exposure to Acrolein for Toddler and Adult Residents

24-hr Chronic TRV (µg/m³)	Background Conc. (µg/m³)	HQ (Background)	Modelled Conc. (µg/m³)	HQ (Modelled -Only)	Cumulative Conc. (µg/m³)	HQ (Cumulative)	% Background HQ Attributable to Cumulative
Toddler Resident							
0.4	0.63	**1.5E+00**	4.0E-03	9.59E-03	0.63	**1.5E+00**	99%
Adult Resident							
0.4	0.63	**1.5E+00**	4.0E-03	9.59E-03	0.63	**1.5E+00**	99%

Notes:
Cumulative Concentration = Background concentration + Modelled concentration
Target HQ = 1
HQs presented in **bold** if exceeding Target HQ

The results presented above in **Table 5-1** and **Table 5-2** indicate the following:

- A predicted cumulative 24-hr acrolein concentration of 0.63 µg/m³ results in HQs that are greater than 1.0, and thus may result in the potential for increased development of lesions in upper airways for toddler and adult residents.

It should be noted that predicted health risks are associated almost entirely with background acrolein concentrations, which represent approximately 99% of cumulative concentrations, and thus, are a significant driver of predicted health risks. For acrolein, the results of this HHA are consistent with the results derived by the City of Toronto and Toronto Public Health wherein a comparable HQ of 1.6 was presented in a report entitled: "Traffic-Related Air Pollution (TRAP) in Toronto and Options for Reducing Exposure" (City of Toronto and Toronto Public Health, 2017).

A major reason for the elevated HQ reported in this HHA as well as in the city-wide study is the adoption of an updated, more stringent threshold for health effects based on information from MECP. That is, while the concentrations of acrolein across the greater Toronto area are not much different from what was modelled in previous studies, our understanding of the risk associated with acrolein has changed.

While the HQs for acrolein appear to be elevated, monitoring data suggests that the levels predicted by the modelling are not unusual. Data collected by Canada's NAPS network between 2009 and 2013 suggests that acrolein concentrations are routinely above guideline levels at sites across Canada, and indicated concentrations could commonly be in the range of 0.1 to 1 µg/m³ or greater (Galarneau *et al.,* 2016). For comparison, the modelling for the City of Toronto predicted concentrations ranging from 0.02 µg/m³ – 0.05 µg/m³. Acrolein is primarily emitted by transportation sources, and the highest risks are predicted to be along the busy highways and congested areas of greater Toronto area. However, given that acrolein is transportation-related and given previous studies, there is evidence to suggest that these concentrations could also diminish as you move above ground level.

5.3.2 BENZENE

Table 5-3 Predicted Non-Carcinogenic Health Risks Associated with Acute Exposure to Benzene for Toddler and Adult Residents

24-Hr Acute TRV (µg/m³)	Background Conc. (µg/m³)	HQ (Background)	Modelled Conc. (µg/m³)	HQ (Modelled -Only)	Cumulative Conc. (µg/m³)	HQ (Cumulative)	% Background HQ Attributable to Cumulative
Toddler Resident							
30	0.69	2.3E-02	0.03	1.0E-03	0.72	2.4E-02	96%
Adult Resident							
30	0.69	2.3E-02	0.03	1.0E-03	0.72	2.4E-02	96%

Notes:
Cumulative Concentration = Background concentration + Modelled concentration
Target HQ = 1
HQs presented in **bold** if exceeding Target HQ

Table 5-4 Predicted Non-Carcinogenic Health Risks Associated with Chronic Exposure to Benzene for Toddler and Adult Residents

Annual Chronic TRV (µg/m³)	Background Conc. (µg/m³)	HQ (Background)	Modelled Conc. (µg/m³)	HQ (Modelled -Only)	Cumulative Conc. (µg/m³)	HQ (Cumulative)	% Background HQ Attributable to Cumulative
Toddler Resident							
30	0.49	1.6E-02	0.009	2.9E-04	0.50	1.6E-02	98%
Adult Resident							
30	0.49	1.6E-02	0.009	2.9E-04	0.50	1.6E-02	98%

Notes:
Cumulative Concentration = Background concentration + Modelled concentration
Target HQ = 1
HQs presented in **bold** if exceeding Target HQ

Table 5-5 Predicted Carcinogenic Health Risks Associated with Chronic Exposure to Benzene for Adult Residents

Annual Chronic TRV (µg/m³)	Background Conc. (µg/m³)	ILCR (Background)	Modelled Conc. (µg/m³)	ILCR (Modelled -Only)	Cumulative Conc. (µg/m³)	ILCR (Cumulative)	% Background ILCR Attributable to Cumulative
Adult Resident							
0.45	0.49	7.7E-01	0.009	1.4E-02	0.50	7.9E-01	97%

Notes:
Cumulative Concentration = Background concentration + Modelled concentration
ILCRs per 1-in-1,000,000 presented in **bold** if > 1

The findings of the HHA indicated that background concentrations of benzene account for 96-98% of the cumulative concentrations, HQ, and ILCR, which suggests that background concentrations are a significant driver of the cumulative concentrations and predicted health risks.

Air quality studies in the City of Toronto have identified benzene as an important vehicle emission exceeding the annual average and 24-hr average health benchmarks (City of Toronto, 2017). **Figure 5-1** below, obtained from the report entitled: "Avoiding the TRAP: Traffic-Related Air Pollution in Toronto and Options for Reducing Exposure" shows modelled annual average concentrations of benzene based on 2012 collected data.

The influence of transportation emissions is clear along Highway 401 in **Figure 5-1** as well as along other major highways, including the additional traffic on ramps and at highway crossings and interchanges. Benzene levels are also elevated in the congested downtown area. While the provincial annual AAQC for benzene is 0.45 µg/m^3 (0.14 ppb), the modelled concentration is predicted at 0.004 µg/m^3. Depending on the exact location within Toronto; the City does not achieve the annual AAQC guideline, with most areas exceeding the AAQC.

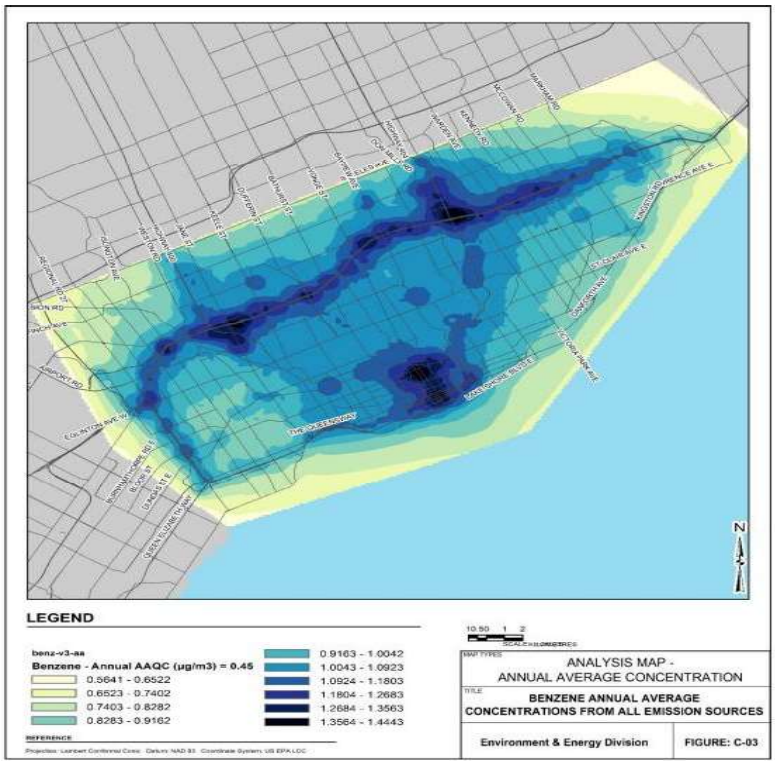


Figure 5-1 **Toronto Modelled Annual Benzene Concentrations (2012), from City of Toronto, 2017**

5.3.3 BENZO(A)PYRENE

Table 5-6 **Predicted Non-Carcinogenic Health Risks Associated with Acute Exposure to Benzo(a)pyrene for Toddler and Adult Residents**

24-Hr Acute TRV (µg/m^3)	Background Conc. (µg/m^3)	HQ (Background)	Modelled Conc. (µg/m^3)	HQ (Modelled -Only)	Cumulative Conc. (µg/m^3)	HQ (Cumulative)	% Background HQ Attributable to Cumulative
Toddler Resident							
0.002	1.1E-04	5.5E-02	7.48E-07	1.5E-02	1.1E-04	5.5E-02	100%
Adult Resident							

24-Hr Acute TRV (µg/m³)	Background Conc. (µg/m³)	HQ (Background)	Modelled Conc. (µg/m³)	HQ (Modelled -Only)	Cumulative Conc. (µg/m³)	HQ (Cumulative)	% Background HQ Attributable to Cumulative
0.002	1.1E-04	5.5E-02	7.48E-07	1.5E-02	1.1E-04	5.5E-02	100%
Pregnant Resident							
0.002	1.1E-04	5.5E-02	7.48E-07	1.5E-02	1.1E-04	5.5E-02	100%

Notes:
Cumulative Concentration = Background concentration + Modelled concentration
Target HQ = 1
HQs presented in **bold** if exceeding Target HQ

Table 5-7 Predicted Carcinogenic Health Risks Associated with Chronic Exposure to Benzo(a)pyrene for Adult Residents

Annual Chronic TRV (µg/m³)	Background Conc. (µg/m³)	ILCR (Background)	Modelled Conc. (µg/m³)	ILCR (Modelled -Only)	Cumulative Conc. (µg/m³)	ILCR (Cumulative)	% Background ILCR Attributable to Cumulative
Adult Resident							
0.002	1.2E-05	0.006	0	0	1.2E-05	0.006	100%

Notes:
Cumulative Concentration = Background concentration + Modelled concentration
ILCRs per 1-in-1,000,000 presented in **bold** if > 1

The results presented above in **Table 5-5** to **Figure 5-6** Average PM2.5 concentrations in selected Canadian urban areas (From Environment Canada and Climate Change (ECCC), Air quality - Canada.ca) indicate that no unacceptable health risks from acute or chronic exposure to benzo(a)pyrene were predicted for any of the identified human receptors, given that cumulative concentrations did not exceed a target HQ of 1 or an ILCR of 1 x 10^{-6}.

It should be noted that background concentrations of benzo(a)pyrene account for approximately 100% of the cumulative concentrations, HQ, and ILCR, which suggests that background concentrations are a significant driver of the cumulative concentrations.

Additionally, benzo(a)pyrene present in the atmosphere is primarily bound to particulate matter and as such, is already accounted for in the $PM_{2.5}$ assessment shown in section 5.3.4

National anthropogenic PAH emissions reported through Canada's Air Pollutant Emissions Inventory have declined by a factor of three since 1990, and are now dominated by residential wood combustion (RWC) (Tevlin et al, 2020). The most recent contributions from motor vehicle exhaust are comparatively small at 8% of the anthropogenic total when accounting is conducted at the national scale. When assessed at the local scale, vehicles contribute more to PAH burdens in ambient air (Tevlin et al, 2020). Air in the Greater Toronto Area has vehicle contributions up to 50%, and smaller municipalities that are near major highways but otherwise have few PAH sources can have vehicle contributions up to 90% (Tevlin et al, 2020).

Figure 5-2 provided below illustrates ambient concentrations of benzo(a)pyrene in comparison with guidelines (Tevlin et al, 2020). The benzo(a)pyrene concentrations reported at the Clarkson Study Area are within the range reported in Ontario and in Canada.

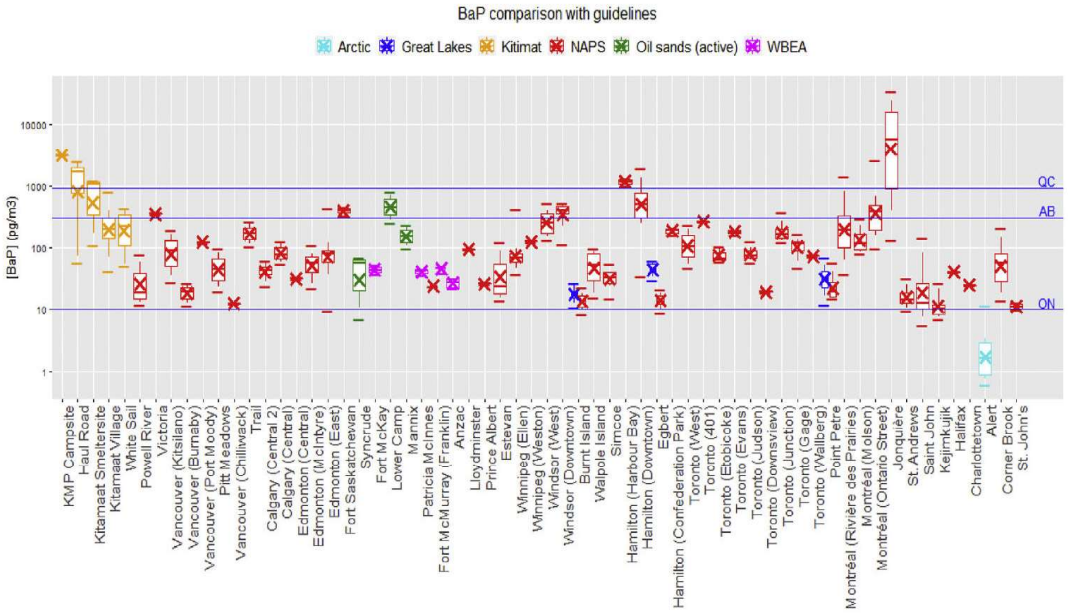


Figure 5-2 **Measured range of annual average benzo(a)pyrene concentrations (pg m^{-3}). Annual average ambient air guidelines from the provinces of Ontario, Alberta, and Quebec are depicted as horizontal blue lines.**

5.3.4 NITROGEN DIOXIDE

Table 5-8 **Predicted Health Risks Associated with Acute Exposure (Airway Hyper-Responsiveness) to Nitrogen Dioxide for Toddler and Adult Residents**

1-Hr Acute TRV (µg/m³)	Background Conc. (µg/m³)	HQ (Background)	Modelled Conc. (µg/m³)	HQ (Modelled -Only)	Cumulative Conc. (µg/m³)	HQ (Cumulative)	% Background HQ Attributable to Cumulative
Toddler Resident							
113	36	3.2E-01	54	4.8E-01	90	8.0E-01	40%
Adult Resident							
113	36	3.2E-01	54	4.8E-01	90	8.0E-01	40%

Notes:
Cumulative Concentration = Background concentration + Modelled concentration
Target HQ = 1
HQs presented in **bold** if exceeding Target HQ

Table 5-9 Predicted Health Risks Associated with Acute Exposure (Asthma Emergency Room Visits) to Nitrogen Dioxide for Toddler and Adult Residents

1-Hr Acute TRV (µg/m³)	Background Conc. (µg/m³)	HQ (Background)	Modelled Conc. (µg/m³)	HQ (Modelled -Only)	Cumulative Conc. (µg/m³)	HQ (Cumulative)	% Background HQ Attributable to Cumulative
Toddler Resident							
79	36	4.6E-01	54	6.8E-01	90	**1.1E+00**	42%
Adult Resident							
79	36	4.6E-01	54	6.8E-01	90	**1.1E+00**	42%

Notes:
Cumulative Concentration = Background concentration + Modelled concentration
Target HQ = 1
HQs presented in **bold** if exceeding Target HQ

Table 5-10 Predicted Health Risks Associated with Chronic Exposure to Nitrogen Dioxide for Toddler and Adult Residents

Annual Chronic TRV (µg/m³)	Background Conc. (µg/m³)	HQ (Background)	Modelled Conc. (µg/m³)	HQ (Modelled -Only)	Cumulative Conc. (µg/m³)	HQ (Cumulative)	% Background HQ Attributable to Cumulative
Toddler Resident							
23	16	6.7E-01	14	5.8E-01	30	**1.3E+00**	54%
Adult Resident							
23	16	6.7E-01	14	5.8E-01	30	**1.3E+00**	54%

Notes:
Cumulative Concentration = Background concentration + Modelled concentration
Target HQ = 1
HQs presented in **bold** if exceeding Target HQ

The results presented above in **Table 5-8** to **Table 5-10** indicate the following:

- A predicted cumulative 1-hr NO_2 concentration of 90 µg/m³ is greater than the TRV of 79 µg/m³ and results in HQs that are marginally greater than 1.0, and thus may result in the potential for increased asthma ERVs for toddler and adult residents; and,
- A predicted cumulative annual NO_2 concentration of 30 µg/m³ results in HQs that are greater than 1.0, and thus may result in the potential for increased respiratory morbidity for toddler and adult residents.

However, it should be noted that a significant portion of the predicted health risks are associated with background NO_2 concentrations, which represent between 42-54% of cumulative concentrations, and thus, are a significant driver of predicted health risks.

Canadian emission estimates for numerous pollutants including NO_x/NO_2 are compiled in the National Pollutant Release Inventory (NPRI). It comprises facility-reported data collected under the authority of CEPA 1999. The NPRI also presents emission summaries and trends for key air pollutants, including NO_x/NO_2, based upon facility-reported data and emission estimates for such other sources as motor vehicles, residential heating, forest fires and agriculture.

Figure 5-3 below, provides a breakdown of the 2011 Canadian NO_x emissions by the broadest NPRI categories. At a national level mobile sources (transportation) are the dominant NO_x source, at 50% of the total, with industrial sources contributing a further 30%. Non-industrial (e.g. electrical power generation, commercial fuel combustion) and natural sources combined contributed slightly less than 20% of 2011 NO_x emissions, with incineration, miscellaneous and open sources together contributing the remaining 1%. Mobile sources are even more important from at human health perspective than this breakdown would suggest, considering that most of the Canadian population lives in urban areas where the bulk of NO_x emissions are from transportation and to a lesser extent consumer/residential sources (e.g. residential fuel combustion, residential wood combustion); such areas tend to be removed from natural and industrial sources.

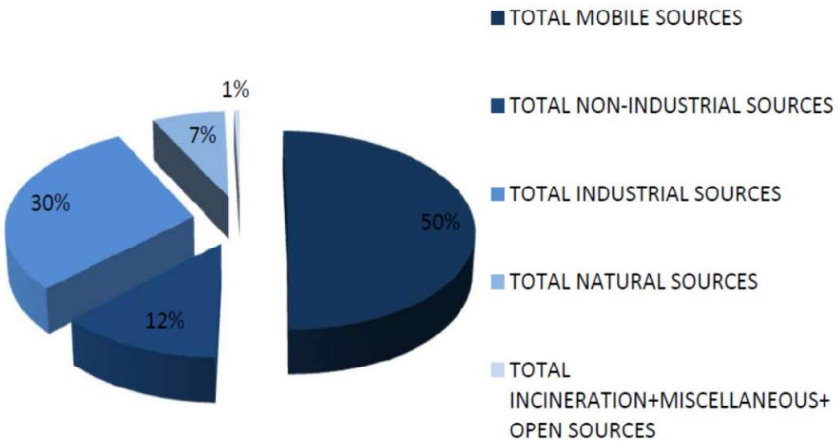


Figure 5-3 2011 NPRI NO_x emissions by broad source category (From NPRI, https://www.ec.gc.ca/inrp-npri/)

NPRI reports generally decreasing trends from 1985 to 2011, particularly from three of the dominant sources including mobile sources, natural sources and non-industrial sources. Industrial NO_x emissions have increased over the same time period by approximately 13%, largely because of increased emissions from the upstream petroleum sector. Due to the importance of the mobile sources for NO_x emissions, there has been an overall decrease in emissions nationally, though this is not true in all regions as a result of the differing importance of various sectors.

In 2016 among the selected urban areas, concentrations of NO_2 were the highest in Calgary, Toronto, Windsor, Vancouver and Hamilton, while Charlottetown, Yellowknife and Fredericton had the lowest concentrations. **Figure 5-4** presents the average annual ambient concentration (in ppb) and **Figure 5-5** presents the peak annual ambient concentrations (in ppb) for NO_2 in selected Canadian urban areas.

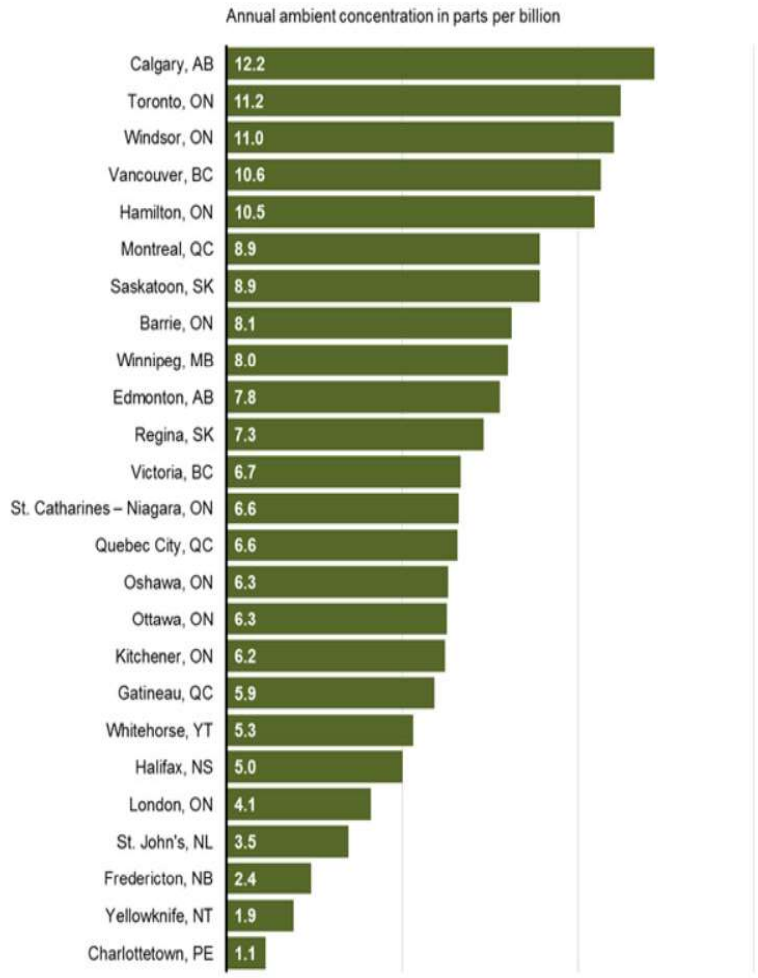


Figure 5-4 **Average NO_2 concentrations in selected Canadian urban areas (2016) (From Environment Canada and Climate Change (ECCC), https://www.canada.ca/en/environment-climate-change/services/environmental-indicators/air-quality.html#NO2-average)**

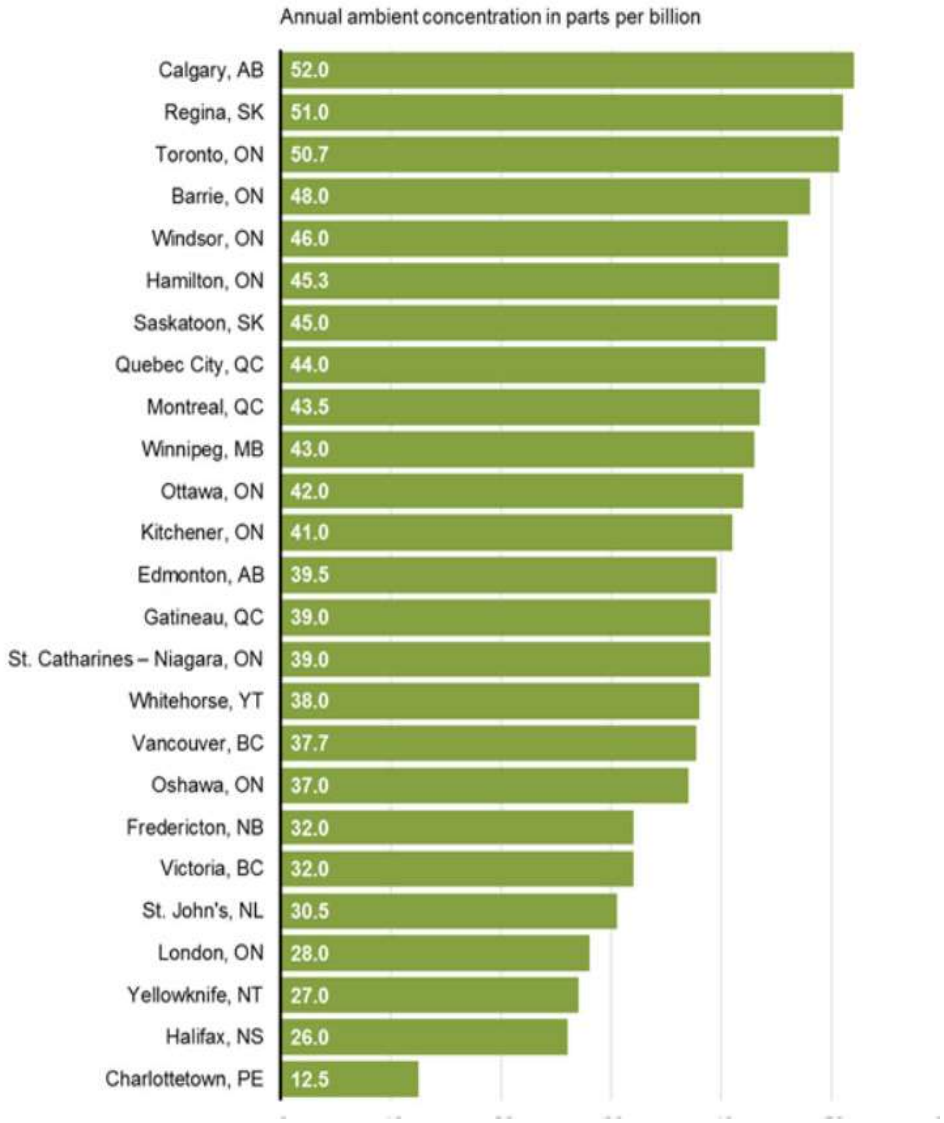


Figure 5-5 **Peak NO_2 concentrations in selected Canadian urban areas (From Environment Canada and Climate Change (ECCC), https://www.canada.ca/en/environment-climate-change/services/environmental-indicators/air-quality.html#NO2-average)**

The influence of transportation emissions for NO_2 is significant. The NO_2 annual cumulative concentrations for the proposed development within Clarkson TSA (30 µg/m^3 or 16 ppb) are within the ranges reported in Toronto and in Canadian urban areas (as shown in **Figure 5-4** and **Figure 5-5**, above).

5.3.5 PARTICULATE MATTER (2.5 µm)

Table 5-11 Predicted Non-Carcinogenic Health Risks Associated with Acute Exposure to PM2.5 for Toddler and Adult Residents

24-Hr Acute TRV (µg/m³)	Background Conc. (µg/m³)	HQ (Background)	Modelled Conc. (µg/m³)	HQ (Modelled -Only)	Cumulative Conc. (µg/m³)	HQ (Cumulative)	% Background HQ Attributable to Cumulative
Toddler Resident							
25	15	6.0E-01	4.5	1.8E-01	19	7.6E-01	79%
Adult Resident							
25	15	6.0E-01	4.5	1.8E-01	19	7.6E-01	79%

Notes:
Cumulative Concentration = Background concentration + Modelled concentration
Target HQ = 1
HQs presented in **bold** if exceeding Target HQ

Table 5-12 Predicted Non-Carcinogenic Health Risks Associated with Chronic Exposure to PM2.5 for Toddler and Adult Residents

Annual Chronic TRV (µg/m³)	Background Conc. (µg/m³)	HQ (Background)	Modelled Conc. (µg/m³)	HQ (Modelled -Only)	Cumulative Conc. (µg/m³)	HQ (Cumulative)	% Background HQ Attributable to Cumulative
Toddler Resident							
10	8.2	7.9E-01	1.8	1.7E-01	10	9.6E-01	82%
Adult Resident							
10	8.2	7.9E-01	1.8	1.7E-01	10	9.6E-01	82%

Notes:
Cumulative Concentration = Background concentration + Modelled concentration
Target HQ = 1
HQs presented in **bold** if exceeding Target HQ

The results of the HHA indicated that background concentration accounts for a significant portion of the predicted health risk given that background concentrations account for approximately 80% of cumulative concentrations for toddler and adult residents.

In 2016, among the selected urban areas, concentrations of $PM_{2.5}$ were the highest in Windsor, Quebec City, and Regina. Whitehorse, Charlottetown and Victoria had the lowest concentrations. **Figure 5-6** presents the average annual ambient concentration (in µg/m³) and **Figure 5-7** presents the peak annual ambient concentrations (in µg/m³) for $PM_{2.5}$ in selected Canadian urban areas.

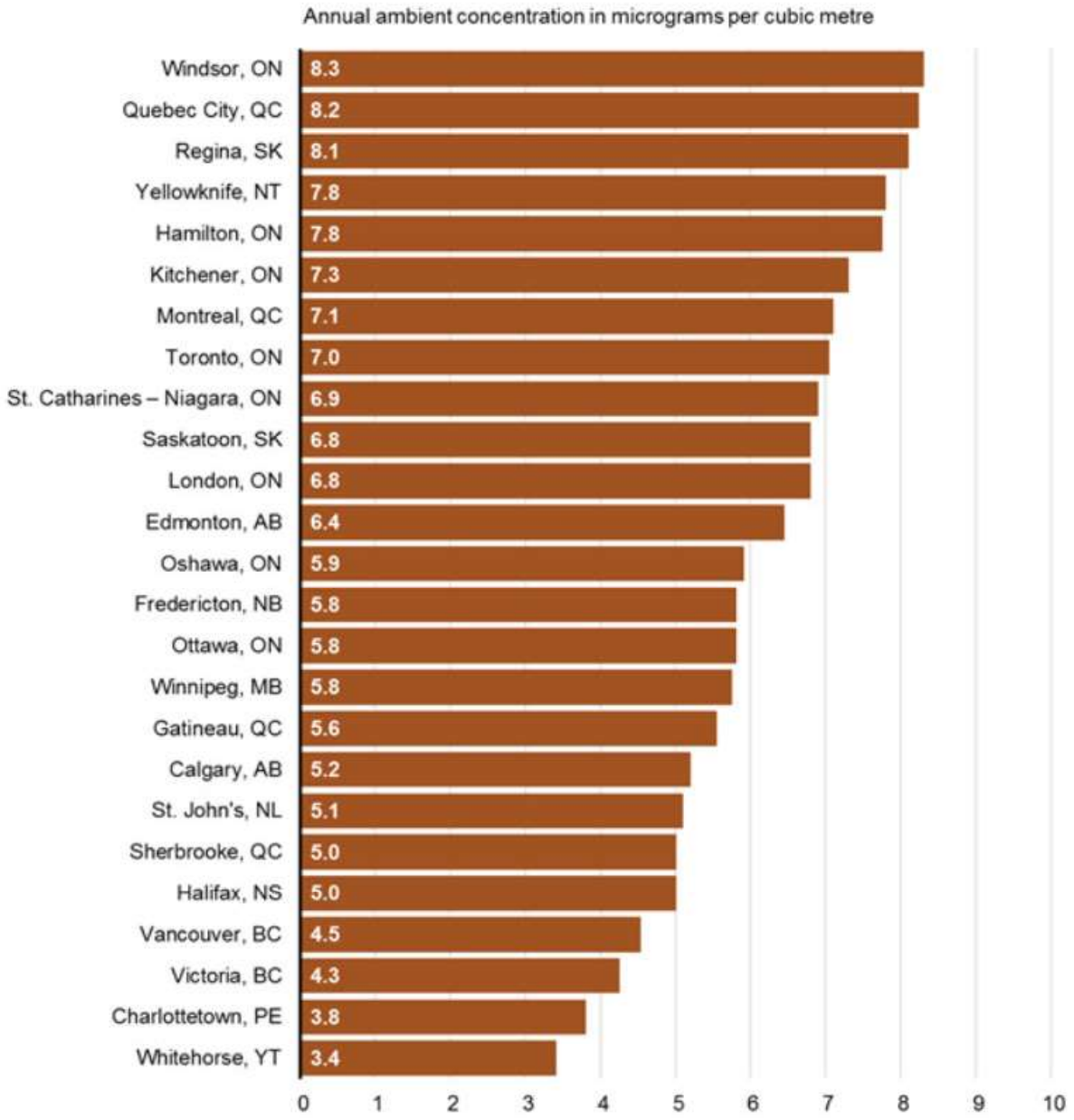


Figure 5-6 **Average $PM_{2.5}$ concentrations in selected Canadian urban areas (From Environment Canada and Climate Change (ECCC), Air quality - Canada.ca)**

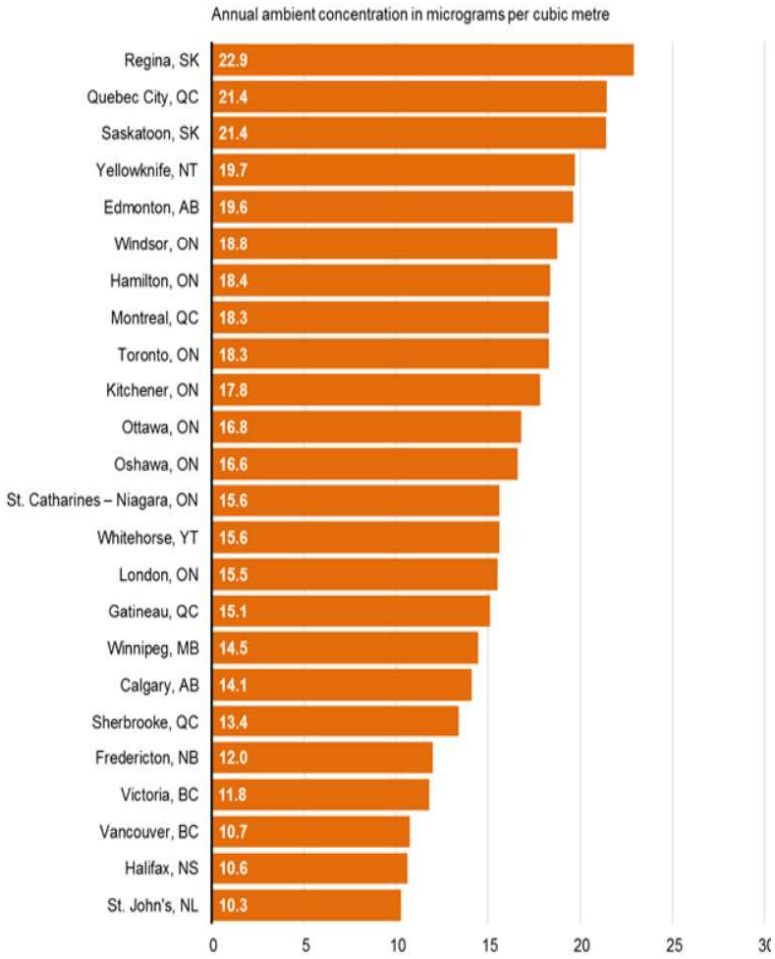


Figure 5-7 **Peak $PM_{2.5}$ concentrations in selected Canadian urban areas (From Environment Canada and Climate Change (ECCC), Air quality - Canada.ca)**

The influence of transportation emissions for $PM_{2.5}$ is significant. The $PM_{2.5}$ annual cumulative concentration for the proposed development at Clarkson TSA (9.5 µg/m^3) is within the ranges reported in Canadian urban cities (as shown in **Figure 5-6** and **Figure 5-7**).

5.4 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, these assumptions are considered to be conservative and result in an overestimation of the true risk. A summary of the major assumptions made in the HHA and resulting uncertainties are provided below:

- Exposure Point Concentrations: The HHA relies largely on output from predictive air dispersion modelling rather than measured values. A detailed discussion of the assumptions and uncertainties related to the air quality modelling is provided in the AQS (WSP, 2021) and include:
    - The air dispersion modelling exercise assumed that all sources would emit continuously (i.e., 24 hrs/day, 7 days/week, 52 weeks/year) and simultaneously. In reality, this scenario is not likely to occur as it is not representative of a typical real-world scenario and only acts as a highly conservative upper bounding case.

- Background ambient concentrations collected from surrounding monitoring stations (such as those captured in the Clarkson monitoring program) in many cases already account for some of the sources modelled for predicted modelled concentrations. Given that modelled concentrations are being added to background concentrations, this double count in concentrations results in a conservative assessment.

- Confounding exposures by co-pollutants and synergistic effects:
  - As discussed in Section 4, 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 solely due to a specific contaminant of interest as it is challenging to separate the effect of each air pollutant.
  - Environmental air pollutants are typically inhaled as complex mixtures; despite this, it is difficult to quantify or evaluate potential synergistic effects between individual contaminants. Although some scientific literatures (largely laboratory experiments) have demonstrated synergism among certain co-pollutants, our understanding on a public health scale remains uncertain given the limited ability to address this issue in epidemiological studies. This is largely because it is difficult to investigate synergism outside of a laboratory environment as there is no control over spatial-temporal variations, exposure concentrations, and population size, among other variables. Thus, it is difficult to characterize the true synergistic effects of co-pollutants in the environment. It is important to note that although synergistic effects of co-pollutants cannot be characterized, all the COPCs identified in the HHA act via different modes of action and elicit different toxicological effects (see Table 4-15); as such, additive effects of co-pollutants are not expected.

- TRVs: The TRVs used in this HHA (and in general) are typically based on the most sensitive endpoints, with the application of safety factors to protect sensitive subpopulations. The uncertainty associated with TRVs is highly dependent on the number of studies available, and whether the key study was based on humans (higher certainty) or animals (lower certainty). When few studies are available, and the studies available are conducted using animals as test organisms under laboratory-testing conditions, several types of safety factors must be applied to account for this uncertainty (e.g., factors for inter- and intraspecies sensitivity).

Significance of background ambient concentrations:

- Background ambient concentrations contribute a significant portion of the cumulative (short-term and long-term) concentrations for all COPCs. For instance, the cumulative concentration (24-hr) for acrolein is 0.63 $\mu g/m^3$ which results in a HQ of 1.5 for toddler and adult residents. However, background ambient concentrations also recorded a concentration of approximately 0.63 $\mu g/m^3$ whereas the predicted modelled concentration is only 4×10^{-3} $\mu g/m^3$. This results in the background ambient concentration comprising of approximately 99% of the cumulative concentration and HQ, which highlights the significance of background ambient concentrations in contributing to the overall cumulative acrolein concentrations and predicted health effects. The significance of ambient background concentrations is further demonstrated for all other COPCs, with the contribution of background concentrations to cumulative concentrations and predicted health risks ranging from 40% to 100%.

The risks identified in Section 5.3, 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).

6 SUMMARY AND CONCLUSIONS

The City of Mississauga is developing land use policies for the Clarkson TSA to support intensification of the area. It is recognized that with the possible redevelopment of this area and introduction of new sensitive land uses, there would be a need to assess air quality impacts on proposed new sensitive developments, especially given the historical state of air quality in the area.

The HHA relies on six months of ambient air monitoring data and an air dispersion modelling assessment of identified COPCs from the recently completed Clarkson TSA AQS (WSP, 2021). The model results represent the air quality impacts on the proposed development from surrounding land uses, including industrial operations and transportation sources in the Clarkson TSA. Based on the results of the ambient air monitoring and air dispersion modelling, the HHA evaluates the potential health effects from the predicted cumulative impacts from nearby activities on the proposed development.

The human receptors evaluated in the HHA were identified based on the proposed development within the Clarkson TSA (i.e., four 25-storey residential buildings). The human receptors associated with this identified land use are intended to be inclusive of human populations including sensitive subpopulations such as asthmatics, children, pregnant females, and the elderly. The following two (2) human receptors were considered:

1. Toddler residents who live in the buildings within the proposed development; and
2. Adult residents who live in the buildings within the proposed development.

A review of health outcomes related to COPC exposures following short- and long-term exposures were completed as well as a jurisdictional review of available ambient air exposure limits. Based on review of available jurisdictional health-based standards for COPCs, as well as toxicological review of health and exposure-related data, this HHA evaluated whether the predicted cumulative concentrations of COPCs in ambient air influenced by nearby activities pose a public health concern in the proposed development for identified human receptors.

A list of the final TRVs used for the assessment can be found in **Section 4.3** (**Table 4-15**).

The findings of the HHA for identified short-term and long-term health endpoints are summarized below.

Acrolein:

- A predicted cumulative 24-hr acrolein concentration of 0.63 µg/m^3 results in HQs that are greater than 1.0, and thus may result in the potential for increased development of lesions in upper airways for toddler, and adult residents.
- It should be noted that predicted health risks are associated almost entirely with background acrolein concentrations, which represent approximately 99% of cumulative concentrations, and thus, are a significant driver of predicted health risks.
- The results of this HHA are consistent with the results derived by the City of Toronto and Toronto Public Health wherein a comparable HQ of 1.6 was presented in a report entitled: “Traffic-Related Air Pollution (TRAP) in Toronto and Options for Reducing Exposure” (the City of Toronto and Toronto Public Health, 2017).
- Monitoring data suggests that the levels predicted by the modelling are not unusual. Data collected by Canada’s NAPS network between 2009 and 2013 suggest that acrolein concentrations are routinely above guideline levels at sites across Canada and indicated concentrations could commonly be in the range of 0.1 to 1 µg/m^3 or greater (Galarneau *et al.,* 2016).

Benzene:

- No unacceptable health risks following acute or chronic exposure to benzene were predicted for any of the identified human receptors, given that cumulative concentrations did not exceed a target HQ of 1 or an ILCR of 1 x 10-6.

- It should be noted that background concentrations of benzene account for 96-98% of the cumulative concentrations, HQ, and ILCR, which suggests that background concentrations are a significant driver of the cumulative concentrations and predicted health risks.

Benzo(a)pyrene:

- No unacceptable health risks from acute or chronic exposure to benzo(a)pyrene were predicted for any of the identified human receptors, given that cumulative concentrations did not exceed a target HQ of 1 or an ILCR of 1×10^{-6}.
- It should be noted that background concentrations of benzo(a)pyrene account for approximately 100% of the cumulative concentrations, HQ, and ILCR, which suggests that background concentrations are a significant driver of the cumulative concentrations.

Nitrogen Dioxide:

- A predicted cumulative 1-hr NO_2 concentration of 90 $\mu g/m^3$ is greater than the TRV of 79 $\mu g/m^3$ and results in HQs that are marginally greater than 1.0, and thus may result in the potential for increased asthma ERVs for toddler and adult residents.
- A predicted cumulative annual NO_2 concentration of 30 $\mu g/m^3$ results in HQs that are greater than 1.0, and thus may result in the potential for increased respiratory morbidity for toddler and adult residents.
- It should be noted that a significant portion of the predicted health risks are associated with background NO_2 concentrations, which represent between 42-54% of cumulative concentrations, and thus, are a significant driver of predicted health risks.

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

- No unacceptable health risks from acute or chronic exposure to $PM_{2.5}$ were predicted for any of the identified human receptors, given that cumulative concentrations did not exceed a target HQ of 1.
- It should be noted that background concentrations of $PM_{2.5}$ account for approximately 80% of cumulative concentrations and HQs for toddler and adult residents and therefore comprise a significant portion of the predicted health risks.

It is emphasized that while the HHA identifies exceedances of the TRVs for certain COPCs and exposure durations, there are uncertainties associated with these predicted health outcomes. The major points of uncertainty include:

- The HHA relied on stringent predicted air dispersion modelling which applies highly conservative scenarios to generate predicted modelled values (e.g., assuming all sources are continuously emitting at 24 hrs/day, 5 days/week, 52 weeks/year);
- Double counting of predicted modelled concentrations as in many cases the modelled sources are already accounted for in the background ambient concentration measurements;
- Worst-case exposure scenarios were evaluated for all human receptors considered. For example, it has been assumed that the predicted concentrations of COPCs in outdoor air are equal to that in indoor air. 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.
- The HHA also assume that predicted concentrations of COPCs are constant with building height. However, several studies that investigate vertical difference of concentrations confirm findings from atmospheric measurements and modeling that PM concentrations tend to decrease with building height (Stephens *et al*, 2019).
- Background ambient concentrations make up a significant portion of the cumulative concentrations for all COPCs, ranging from 40% to 100% of cumulative concentrations. This indicates that generally, background concentrations, relative to modelled concentrations, are the drivers and major contributors to predicted health risks.

Further discussion on the uncertainties applied in this HHA including the conservatism inherent in developing TRVs can be found in Sections 2.4, 4.4, and 5.4.

Based on the findings of the AQS and the HHA, WSP is of the opinion that air quality in the study area is not expected to adversely impact high density residential development. Elevated concentrations of contaminants reported (i.e., above health-based thresholds) which could lead to potential health risks (see Section 5.3), are not unique to the Clarkson TSA and are expected throughout urban areas in Ontario (i.e., the Greater Toronto Area) and Canada. Transit-oriented development within the Clarkson TSA is expected to reduce reliance on passenger vehicle trips as the community shifts to alternative modes of transportation such as public transit and active transportation. This transition is expected to reduce emissions of TRAP contaminants within the Clarkson TSA and likely will result in improved air quality in the community. Full details regarding the mitigation recommendations as well as potential air quality improvements at Clarkson TSA are included in the Mitigations Recommendations Memo provided as Appendix L of the AQS (WSP, 2022).

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