SLATE ASSET MANAGEMENT L.P.

CLARKSON TRANSIT STATION AREA AIR QUALITY STUDY

MONITORING AND DISPERSION MODELLING REPORT

February 16, 2023

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SLATE ASSET MANAGEMENT L.P.

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WSP
UNIT 2
126 DON HILLOCK DRIVE
AURORA, ON, CANADA L4G 0G9

T: +1 905 750-3080
F: +1 905 727-0463

Signatures

Prepared By

Stephanie Clarke, B.A.
Environmental Consultant

Katelyn Sysiuk, B. Eng., E.I.T.
Environmental Consultant

Reviewed By

Camille Taylor, P. Eng.
Director and Sr. Air Quality Engineer

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EXECUTIVE SUMMARY

The purpose of this study is to assess the air quality impacts from surrounding land uses, including industrial operations and transportation sources in the Clarkson Transit Station Area (TSA). WSP Canada Inc. (WSP) was retained by Slate Asset Management (Slate) to complete an Air Quality Study including six months of ambient monitoring and an air dispersion modelling assessment for the proposed development located at 2077, 2087, 2097, and 2105 Royal Windsor Drive in Mississauga, Ontario. The City of Mississauga (the City) requires an updated study to determine the compatibility of additional sensitive land uses within the area and will also use this report to inform their Master Plan. The City will have this final report peer reviewed. The City and their peer reviewer have been following the process since the beginning and have provided feedback on this study.

The six months of ambient air monitoring and dispersion modelling assessment were completed in accordance with the Terms of Reference provided by the City of Mississauga on June 23, 2020 (TOR). The ambient air quality monitoring was conducted at the Slate lands located at 2105 Royal Windsor Drive in Mississauga, Ontario from July 2020 to January 2021.

For baseline, the Ministry of the Environment, Conservation, and Parks (MECP) conducted an air quality study in 2007 which found elevated concentrations of various contaminants; benzene, dichloromethane (methylene chloride) and acrolein were identified as air contaminants that were greater than their respective Ambient Air Quality Criteria (AAQC). The AAQC values are not enforceable through regulatory actions, they are concentrations of individual contaminants in air that are determined to be protective against adverse effects on health and/or the environment. AAQC values are used to assess ambient air quality resulting from all sources of a contaminant to air and are commonly used to determine impacts from projects on the ambient air quality. It was expected that there was general improvement of the air quality in the area since 2007 due to improvements in vehicle emissions and industrial practices.

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. The following report outlines all timelines, methodologies, and relevant guidelines.

Based on the results of the ambient air quality monitoring and the dispersion modelling assessment there is no reason to exclude high density residential land use and other sensitive land uses in the study area.

Relevant results are summarized here:

- All significant contaminants included in this assessment, except for acrolein, benzene, benzo(a)pyrene, NO_X, $PM_{2.5}$, and PM_{10} were predicted to be below their respective AAQC;
- Acrolein concentrations recorded at the monitoring station had a 90th percentile concentration that was elevated compared to the 24-hour AAQC. The 90th percentile acrolein concentrations recorded during the six months of monitoring were 67 % lower than the 90th percentile recorded during the 2007 MECP study showing a downward general trend;
- The ambient baseline concentration of acrolein is significantly contributing to the AAQC exceedance for acrolein, with the modelled concentration being only 1% of the cumulative concentration. The background concentration is comparable to reported acrolein concentrations in Ontario;
- Benzo(a)pyrene was not part of the ambient monitoring program; the modelling results show concentrations elevated compared to the AAQC for both 24-hour and annual concentrations. This analysis is based on cumulative concentrations using the NAPS station located near Highway 401, which has higher concentrations given the close proximity to high volumes of vehicular traffic than in the vicinity of the Clarkson TSA;
- The ambient baseline concentration of benzo(a)pyrene is significantly contributing to the AAQC exceedance, with modelled concentration being only 1% of the cumulative concentration for the 24-hour average and 0% for the annual average. The baseline concentration is comparable to reported benzo(a)pyrene concentrations in Ontario and Canada;
- Based on the NPRI data both acrolein and benzo(a)pyrene are not emitted from the surrounding industrial facilities. The main source of anthropogenic acrolein and benzo(a)pyrene in the area is expected to be traffic and locomotive sources. Emissions are expected to decrease as older vehicles are removed from service and vehicle emission controls become more efficient as well as through eventual electrification of the Lakeshore West GO corridor; Both acrolein and benzo(a)pyrene are listed as Traffic Related Air Pollutants and are often elevated compared to the AAQC in urban areas and near highways and roadways;

- Benzene concentrations recorded at the monitoring station had a 90th percentile concentration that was elevated compared to the 24-hour AAQC. The modelled concentration of benzene only contributed 2% to the cumulative concentration. The ambient baseline concentration recorded is within the range reported in Ontario and in Canada.
- The 90th percentile 24-hour concentration of NO_2 recorded at the monitoring station was below the AAQC threshold. The cumulative concentration calculated from the dispersion modelling was above the annual Canadian Ambient Air Quality Standard (CAAQS) of 12 ppb which may be attributable to the addition of sources to the baseline ambient data which already includes the nearby sources. It should also be noted that the CAAQS is based on the average over a single calendar year of all 1-hour average concentrations, not 90th percentiles. The average of all one hour NO_2 concentrations collected at the monitoring station was 6.9 ppb.
- The modelled concentration of NO_2 and baseline concentration have similar contribution to the cumulative concentrations. The NO_2 annual cumulative concentration for the Clarkson TSA is within the range reported in Toronto and in urban areas of Canada.
- Concentrations of $PM_{2.5}$ and PM_{10} at the Site property boundary were reported as elevated compared to the annual air quality threshold and 24-hour air quality threshold respectively; however, reported concentrations have been conservatively combined with ambient air monitoring data which would have already captured $PM_{2.5}$ and PM_{10} concentrations in ambient air and the resulting cumulative concentration was not significantly altered. The cumulative impacts at the proposed development showed a minor increase from existing conditions likely as a result of expected traffic growth in the study area. The $PM_{2.5}$ annual cumulative concentrations and PM_{10} 24-hour cumulative concentration for the Clarkson TSA are within the range reported in Canadian urban cities.
- By examining receptors at various heights at the property boundary and adding the modelled concentration and the ambient concentration it was determined that for the contaminants of concern ($PM_{2.5}$, PM_{10}, NO_X, acrolein, and benzene) there are no concentrations elevated compared to the AAQC above 30.1 m except for benzo(a)pyrene.
- Background concentrations of acrolein and benzo(a)pyrene are elevated compared to the AAQC values; however, B(a)P is elevated anywhere a development were to proceed in an urban area.
- Air quality mitigation is not required at the proposed development; however, mitigation recommendations have been included to improve indoor air quality.
- If air intakes are designed to be located in each suite, then for any suites below the fourth floor (estimated at 12.9 m) filters to control particulate matter ($PM_{2.5}$ and PM_{10}) impregnated with

carbon to control benzene could be utilized to improve indoor air quality. Percent reductions required can be calculated from Table 3 attached to the memorandum located in Appendix L. Since Table 3 represents a very conservative approach then it is recommended that a method of ambient monitoring be incorporated to ensure the controls of a local air intake design are working, or even required. An alternative to filtering local air intakes and monitoring could be to have a centralized air intake system ducted from above 12.9 m for any suites located below this level.

- Based on the air quality study, air quality in the study area is not expected to adversely impact high density residential development nor the existing local industrial sites level of compliance to existing standards. Elevated concentrations of contaminants reported (i.e., above health-based thresholds) which could lead to health risks are not unique to the Clarkson TSA and are expected throughout urban areas in Ontario (i.e., Greater Toronto Area and Hamilton) 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.

Tables

Figures

Appendices

1 Introduction

WSP Canada Inc. (WSP) was retained by Slate Asset Management (Slate) to complete an Air Quality Study including six months of ambient monitoring and an air dispersion modelling assessment for the proposed development located at 2077, 2087, 2097, and 2105 Royal Windsor Drive in Mississauga, Ontario (the Site). The ambient air quality monitoring was conducted at the Slate lands located at 2105 Royal Windsor Drive in Mississauga, Ontario.

The six months of ambient air monitoring and dispersion modelling assessment were completed in accordance with the Terms of Reference provided by the City of Mississauga (the City) on June 23, 2020 (TOR) and completed in accordance with the Ontario Ministry of the Environment, Conservation and Parks (MECP) Operations Manual for Air Quality Monitoring in Ontario, 2018 (the Operations Manual). The monitoring was carried out to identify any potential ambient air quality effects on the proposed development area from nearby industrial sources, transit, and vehicular traffic. The parameters outlined in the TOR for monitoring were:

— Total suspended particulate (TSP);

— Volatile organic compounds (benzene, dichloromethane, and acrolein);

— Nitrogen oxides (NO_X); and

— Sulphur dioxide (SO_2).

PM_{10} and $PM_{2.5}$ 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. This report outlines the results of the monitoring program.

This report outlines the specific modelling approach and input data used to complete the air dispersion modelling for the proposed development and assesses the predicted cumulative impacts from the nearby activities on the Site.

The proposed development is located within the Clarkson Transit Station Area (TSA) and would introduce sensitive land uses. As a result, the City requires an Air Quality Assessment to be completed to assess air quality impacts on the proposed development from surrounding land uses, including industrial operations and transportation sources. The results of the dispersion modelling were combined with ambient air monitoring results to assess the predicted cumulative concentrations of each contaminant.

The Site is located on the west side of the Royal Windsor Drive and Southdown Road intersection in Mississauga, Ontario. The Site is currently zoned as employment (E2-108) and

is surrounded by residential, commercial, and employment zones. In the City of Mississauga's Official Plan, the lands are designated as Mixed Use within the Southdown Employment Area and currently do not permit residential uses. A rail corridor is located to the northwest of the Site and includes the Clarkson GO Station located at 1110 Southdown Road. Lands to the north, east, and northwest consist of predominately residential developments while lands to the west through southeast are predominately commercial and industrial developments. The location of the Slate proposed development is shown in **Figure 1-1**. The location of the proposed development, Clarkson TSA monitoring station, and Study Area are shown in **Figure 1-2**. The development is proposed to include four 25-storey residential buildings.

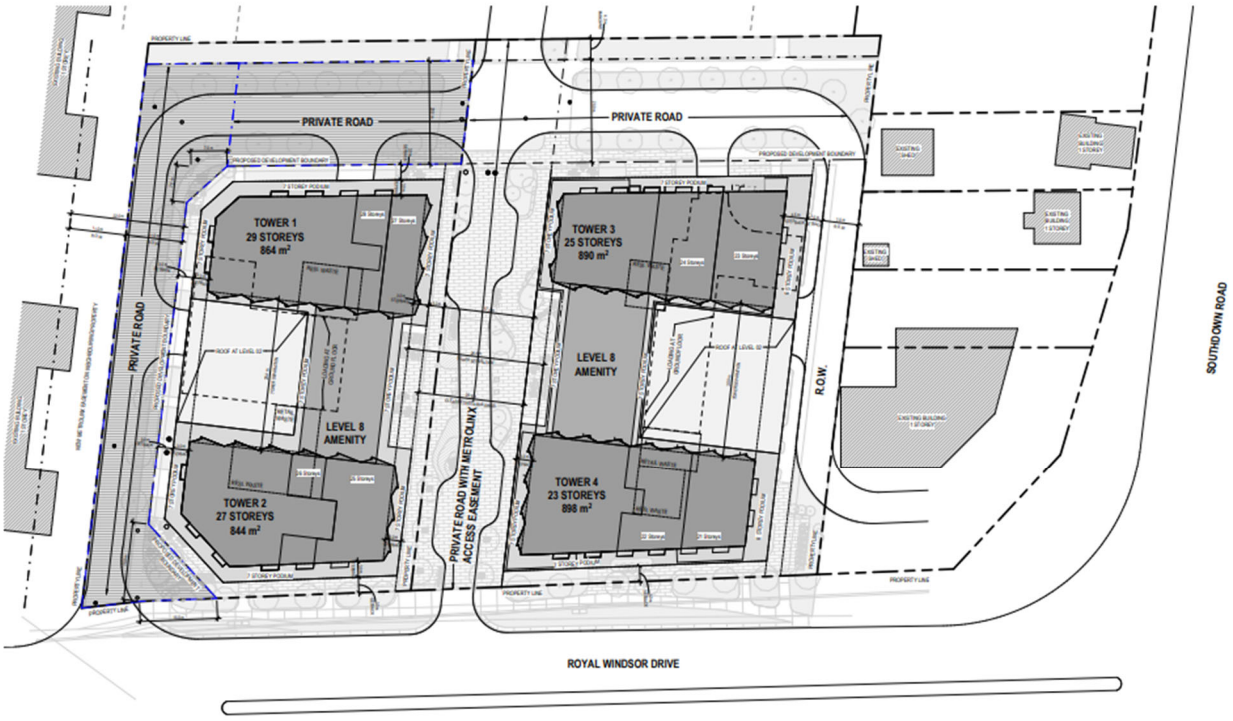

Figure 1-1 Slate Proposed Development

The location of the proposed development, Clarkson TSA monitoring station, and Study Area are shown in **Figure 1-2**.

Figure 1-2 Air Quality Assessment Study Area

1.1 COVID-19 Influences

The current COVID-19 situation has resulted in the reduction of roadway traffic and a change to train operating schedules along the GO corridor. Nearby industrial activities that may have an impact on air quality may also have altered emission rates during the COVID-19 pandemic. Vehicular emissions from the nearby parking lots and major roadways are expected to be reduced during this time period. As such, the results presented from the ambient air quality monitoring may represent atypical conditions. Monitoring data from the Clarkson Airshed Industrial Association (CASIA) was provided by the participating industries to be incorporated into the Air Quality 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 finding. While there are still unknown possible effects of COVID-19 on the ambient monitoring data, several data set comparisons have been undertaken and included in this report to ensure the dependability of the information. The possible effects of COVID-19 on the ambient monitoring study are further discussed in **Section 5** of this report.

2 Monitoring Summary

2.1 Methodology

After receiving approval from the City, the ambient air quality monitoring station was installed on July 8, 2020 at the Site in order to ensure the summer months were captured in the monitoring program. The continuous analyzers were operating since the installation on July 8, 2020. The first round of discrete sampling was completed on July 14, 2020, aligning with the North American schedule. Monitoring was carried through to completion on January 10, 2021, to fulfill the requirements of the City's Terms of Reference.

Following the MECP Operations Manual for Air Quality Monitoring in Ontario (the Operations Manual) and the Terms of Reference provided by the City, the following instruments and sampling methods were used:

— Total Suspended Particulate (TSP): TSP filter media and TSP gravimetric analysis using a Tisch TE-5170 Mass Flow Controlled TSP Sampler (Hi-Vol). Sampling was conducted on a one-in-six-day schedule and ran for 24 hours (00:00 – 23:59) per sample. An exhaust hose was used to direct sampled air away from the intake.

— Particulate Matter <10 µm (PM_{10}) and <2.5 µm ($PM_{2.5}$) in diameter: PQ200 discrete samplers. Sampling was conducted on a one-in-six-day schedule and ran for 24 hours (00:00 – 23:59) per sample.

— VOCs (Benzene, dichloromethane (methylene chloride), and acrolein): US EPA Compendium Method TO-15 using vacuum canisters (concurrent sample collection). Sampling was conducted on a one-in-six-day schedule and with samples collected for 24 hours (00:00 – 23:59). A programmable timer/regulator was used on the canisters to trigger sampling. Since acrolein is highly reactive, the VOC samples were delivered to the laboratory for analysis as soon as reasonably possible.

— Sulphur dioxide (SO_2): Thermo Scientific 43i SO_2 analyzer housed in a temperature-controlled weatherproof enclosure. Sampling was continuous with a resolution of five minutes.

— Nitrogen Oxides (NO_X): Thermo Scientific 42i $NO/NO_2/NO_X$ analyzer housed in a temperature-controlled weatherproof enclosure. Sampling was continuous with a resolution of five minutes.

Sample probe siting for all sampling equipment was completed in accordance with the

Operations Manual. All monitoring equipment was distanced from walls or structures at least twice the height of the wall or structure. The SO_2 and NO_X continuous analyzers were installed to have an inlet height of at least three meters. The TSP, PM_{10}, and $PM_{2.5}$ inlets were installed to be a minimum of two meters above the ground and more than 20 m from any trees. The VOC inlet was installed to be a minimum of three meters above the ground. All other requirements of the Operations Manual related to probe siting were followed, including Table 3: Sample Probe Siting Criteria.

Monitoring results have been summarized for sampling data collected between July 8, 2020 and January 10, 2021 (the monitoring period). The location of the monitoring station is shown in **Table 2-1** and **Figure 1-1.**

Table 2-1 Monitoring Station Location

Location/Address	Zone	UTM-X Coordinates	UTM-Y Coordinates
2105 Royal Windsor Dr., Mississauga, ON	17T	610529	4818409

2.2 Equipment Calibration and Record Keeping

A site logbook was maintained and a record of each site visit including the purpose of visit, work performed on each instrument, and observations while on site were recorded. Any equipment malfunctions, repairs, and maintenance were properly logged per the Operations Manual. The logbook was kept up to date for each site visit. All site logs were reviewed monthly by the Senior Air Quality Engineer.

Calibrations of sampling equipment completed during the monitoring period were conducted in accordance with the Operations Manual, the Terms of Reference provided by the City and manufacturer recommendations. The following equipment calibrations were completed during the monitoring period:

— The Tisch TE-5170 was calibrated upon installation, and after three months of sampling;

— The PQ200 discrete samplers were calibrated bimonthly;

— VOC sampling unit leak test calibration was completed bimonthly;

— The Thermo Scientific 43i SO_2 analyzer was calibrated monthly; and

— The Thermo Scientific 42i $NO/NO_2/NO_X$ analyzer was calibrated monthly.

All equipment Calibration Certificates that were completed during the monitoring period are presented in **Appendix A**.

The SO_2 and NO_X analyzers were equipped with a data logger and remote communication to ensure data was recorded and that field staff were alerted to equipment downtime in a timely manner. The analyzers were remotely checked for normal operations a minimum of once per day.

Power to the monitoring station was hardwired (via extension cords to the adjacent building on Site) for the duration of the monitoring period to ensure consistent monitoring with no electrical background noise impacting data measurements or communication. Power draw for all sampling equipment was metered and recorded regularly in the Site's logbook during site visits.

2.3 Laboratory Analysis and Data Validation

The discrete samples that required laboratory analyses included TSP, VOCs, PM_{10}, and $PM_{2.5}$. Laboratory analysis for all discrete samples collected was completed by ALS Environmental, a laboratory whose analytical methods, as required by the monitoring program, have Canadian Association for Laboratory Accreditation (CALA) accreditation. Sample media for the discrete samplers was sampled, collected, transported and stored in accordance with the Operations Manual, Reference Methods, and laboratory requirements.

The procedure for data validation for continuous and discrete data has been completed in accordance with the Operations Manual. The discrete sampling followed a one day of every six days frequency, per the North American schedule. All laboratory analysis and continuous NO_X and SO_2 data have gone through internal review by the Senior Air Quality Engineer to ensure sampling was conducted per the Operations Manual and all data presented within this report is valid.

2.4 Uncertainties of Air Quality Monitoring

WSP followed the Operations Manual for Air Quality Monitoring in Ontario and industry best practice to ensure that uncertainties were minimized. There is some uncertainty when sampling acrolein, considering factors such as how canisters are cleaned in preparation for sample collection and the gas standards used to calibrate analytical equipment. Historically, the method typically used for sampling acrolein in ambient air was by collection on a DNPH-coated silica gel cartridge, followed by high performance liquid chromatography (HPLC) analysis, per the United States Environmental Protection Agency (USEPA) Method TO-11A. This changed in 2000 when the USEPA amended the "Compendium of Methods for the Determination of Toxic Organic Compounds in Ambient Air – Second Edition", which removed

acrolein due to significant data quality concerns. Air samples collected in canisters and analyzed by gas chromatography/mass spectrometry (GC/MS) per USEPA method TO-15 later became the industry standard for sampling acrolein in ambient air. As this approach was being tested, it became a concern that there may be formation of acrolein in the canisters, resulting in a reading higher than actual (i.e., high bias). In 2010, the Office of Air Quality Planning and Standards (OAQPS) conducted a study to assess how the canister cleaning process might result in increased acrolein concentration during analysis. The study showed that acrolein could be elevated even in clean canisters. The study also showed that there were variables when it came to the acrolein gas standards used to calibrate the analytical systems for different laboratories. To reduce the likelihood of uncertainties, the USEPA recommended that collection canisters be heated to a minimum of 80 °C while being cleaned. ALS Environmental follows this USEPA recommended practice of heating canisters while cleaning.

The USEPA also recommended analyzing the cleaned canisters for acrolein by GC/MS immediately after cleaning and once a week for two to three weeks to determine whether acrolein was likely to form in the canister over time. The canisters from ALS are proofed after sitting for 24 hours under pressure with humidified nitrogen. ALS also conducts method blanks to confirm the limit of reporting (LOR) is lower than 0.2 ppbv.

The calibration gas standards that laboratories use to calibrate their GC/MS analytical system can also cause variation in analysis. The 2010 study completed by OAQPS indicated that laboratories using higher concentration acrolein standards and diluting to target range provided more consistent analytical results. The gas standards that ALS Environmental uses have an analytical accuracy of ±10 %. ALS Environmental also uses a stock standard that is 1 000 ppbv and diluted to 1 ppbv. The USEPA also recommended analyzing the canister as soon as reasonably possible after collection. WSP submitted the canisters to the laboratory the following workday after each 24-hour sample.

The uncertainties for benzene and methylene chloride analysis are not as significant as acrolein. The analysis provided by ALS Environmental would be reasonably accurate based on Reference Methodology. Further, the uncertainties in particulate sampling (TSP, PM_{10}, and $PM_{2.5}$) are also minimal; however, there were some noted issues with the 47 mm PM_{10} and $PM_{2.5}$ filters at the start of the sampling program, which were later resolved by switching to more durable polytetrafluoroethylene (PTFE) filters. Uncertainties relating to NOx and SO_2 analysis are minimized as WSP maintained calibrations on the analyzers per the Operations Manual.

3 Summary of Monitoring Results

3.1 Discrete Sampling Results

Discrete sampling events were completed on a one-in-six-day schedule and ran for 24 hours (00:00 – 23:59) per sample. All discrete sampling results have been compared to the 24-hour and annual Ambient Air Quality Criteria (AAQC) guidelines for each respective sample parameter. The comparison to annual AAQC guidelines is for informational purposes only; six months of data should not be held to the annual guidelines, which account for seasonal variations. Since acrolein and PM_{10} do not have annual AAQC guidelines, only the 24-hour guidelines were used for these parameters.

$PM_{2.5}$ and PM_{10} were added to the monitoring parameters at a later date as requested by the MECP, as a result, WSP was unable to obtain the 47 mm filters in time for the July 14, 2020 sample event. At the onset of the monitoring program, there were issues with the $PM_{2.5}$ and PM_{10} sampling that occurred due to visually unobservable damage to sampling media during the sampling events. WSP was not aware of this issue until laboratory results were made available weeks after the sampling events occurred. The 47 mm filters used for $PM_{2.5}$ and PM_{10} were reported by the laboratory as showing signs of damage sustained during the sampling event. This was noted on $PM_{2.5}$ samples from July 20, August 1, August 13, and August 19, 2020. This was noted on PM_{10} samples from July 26, August 13 and August 19, 2020. Data from these sample events were not included in any average calculations as they would underestimate the levels of $PM_{2.5}$ and PM_{10} due to the damage. Despite WSP's best effort to keep the 47 mm filters intact, the issue remained. WSP investigated alternative types of 47 mm filters and decided to use the 47 mm PTFE-filters. After receiving better results on the August 25, 2020 sample event more PTFE-filters were ordered; however, they did not arrive in time for the September 6, 2020 sample event. The PTFE-filters were used for every sampling event following and did not show any signs of damage for the remainder of the ambient sampling program. All other samples were collected without any observable issues. There was an error with the flow controller on November 17, 2020 that resulted in the VOC canister's final pressure being positive. For this reason, these results were not included in the report.

A summary of the individual discrete sampling results compared to the AAQC 24-hour threshold guidelines is presented in **Table 3-1.** The Certificates of Analysis from each sampling event are located in Appendix B.

Table 3-1 Summary of 24-Hour Discrete Sampling Results

MEASURED CONTAMINANT (µg/m³)	ACROLEIN	BENZENE	METHYLENE CHLORIDE	TSP	$PM_{2.5}$	PM_{10}
24-HOUR AAQC (µg/m³)	**0.4**	**2.3**	**220**	**120**	**27**	**50**
SAMPLE DATE						
14-Jul	0.5	0.69	1.27	30.2	--	--
20-Jul	0.63	<0.32	<0.69	35.7	<0.62 [A]	<0.62
26-Jul	0.68	0.47	<0.69	51.4	1.37	<0.63 [A]
01-Aug	0.53	<0.32	<0.69	<15	<0.62 [A]	<0.63
07-Aug	0.4	0.5	0.75	45.6	2.25 [C]	0.63 [C]
13-Aug	0.63	0.45	1.22	44.9	<0.62 [A]	<0.63 [A]
19-Aug	0.45	0.69	4.42	26.1	<0.62 [A]	<0.63 [A]
25-Aug	0.53	0.49	<0.69	32.4	8.58	16.8
31-Aug	0.67	0.68	<0.69	25.3	4.7	11.1
06-Sep	0.26	<0.32	1.33	16.5	NA [B]	NA [B]
12-Sep	0.58	0.75	1.27	20.7	2.17 [C]	1 [C]
18-Sep	<0.23	<0.32	<0.69	30.1	2.5	10.1
24-Sep	0.28	0.94	1.67	96.3	22.4	58.2
30-Sep	<0.23	0.37	<0.69	27.2 [D]	10.3	22.8
06-Oct	<0.23	0.37	<0.69	89.3	4.5	37.7

MEASURED CONTAMINANT (µg/m³)	ACROLEIN	BENZENE	METHYLENE CHLORIDE	TSP	$PM_{2.5}$	PM_{10}
24-HOUR AAQC (µg/m³)	**0.4**	**2.3**	**220**	**120**	**27**	**50**
SAMPLE DATE						
12-Oct	<0.23	0.32	<0.69	14.2	2.12	3.46
18-Oct	<0.23	0.39	<0.69	25.8	5.75	14.30
24-Oct	<0.23	0.32	<0.69	14.7	0.79	4.09
30-Oct	<0.23	0.34	<0.69	19.5	4.09	10.10
05-Nov	<0.23	0.44	<0.69	10.9 [C]	7.90	47.10 [C]
11-Nov	<0.23	0.35	<0.69	34.8	6.71	14.50
17-Nov	NA	NA	NA	22.2	4.33	8.50
23-Nov	<0.23	0.49	<0.69	32.5	5.29	8.17
29-Nov	<0.23	0.48	<0.69	31.7	5.79	16.20
05-Dec	<0.23	0.34	<0.69	16.4 [C]	3.58	20.70 [C]
11-Dec	<0.23	1.79	1.91	120	28.20	84.90
17-Dec	<0.23	0.67	<0.69	94	9.37	27.20
23-Dec	<0.23	0.47	<0.69	25.3	5.75	21.50
29-Dec	<0.23	0.39	<0.69	20.9	6.66	8.75
04-Jan	<0.23	0.51	<0.69	20.2	<0.62	14.80
10-Jan	<0.23	0.58	<0.69	24.5	9.37	12.9

Note: A Filter samples in this submission show obvious signs of damage, sustained during the sampling event. Data is expected to be biased low as a result of matrix loss. Data from these samples is not included in the average calculations.

B Sample media was not available from the laboratory for Sep 6, 2020.

C Discrepancies in concentrations (TSP<PM_{10}, or PM_{10}<$PM_{2.5}$)

D Power was lost due to the extension cord being disconnected by a pedestrian, sampled October 2, 2020 instead.

'--' Requirement for PM_{10} and $PM_{2.5}$ discrete sampling was introduced after the sampling event occurred. PQ200 discrete samplers were not yet installed and ready to sample.

'<' Indicates that the sampling result was below the laboratory detection limit.

'NA' Indicates missing data.

Red text indicates measurement is above the respective 24-hour AAQC guideline.

When comparing individual sampling events to the AAQC, a total of nine acrolein samples collected during the monitoring period were elevated compared to the 24-hour AAQC guideline of 0.4 µg/m^3.

When comparing individual sampling events to the AAQC, there were no benzene, methylene chloride, or TSP samples collected during the monitoring period that were elevated compared to their respective 24-hour AAQC guidelines.

When comparing individual sampling events to the AAQC, there was one $PM_{2.5}$ measurement collected on December 11, 2020 that was elevated compared to the AAQC guideline of 27 µg/m^3. There were two PM_{10} measurements collected during the monitoring period that were elevated compared to the 24-hour AAQC guideline of 50 µg/m^3. The PM_{10} elevated levels occurred on September 24, 2020 and December 11, 2020. On December 11, 2020, PM_{10}, $PM_{2.5}$, TSP and benzene concentrations were all greater than the typical ranges seen during the monitoring period; the reason for these elevated concentrations is currently unknown. Wind direction on this day was blowing from the north northeast, so it is likely not due to the industry activities located to the south of the Site.

There were four days when discrepancies in measured TSP and PM fractions were identified where the smaller $PM_{2.5}$ size fraction was larger than the PM_{10} fraction, or TSP was less than PM_{10}. On these days no errors in sampling methodology were identified and samples were deemed valid by ALS Environmental. As a result, TSP and PM fraction results were included in the analysis.

When the benzene concentration from all sampling events is averaged over the six-month program it is elevated compared to the AAQC annual threshold limit of 0.45 µg/m^3. The average six-month concentrations for all other sample parameters with annual AAQC guidelines were below their respective AAQC guidelines. A summary of the contaminants' average concentrations compared to the AAQC annual guidelines is presented in **Table 3-2**, a reminder that this comparison is for informational purposes only and that six months of data is not a valid data set to compare to annual guidelines due to seasonal variations. The Certificates of Analysis from each sampling event can be found in **Appendix B**. The collected data represents six months of monitoring and meets the City's requirements set forth in the Project's Terms of Reference.

Table 3-2 Summary of the Discrete Monitoring Results

Contaminant	Annual AAQC Threshold ($\mu g/m^3$)[1]	Average Concentration ($\mu g/m^3$)	90th Percentile Concentrations
Acrolein	**--**	0.27	0.63
Benzene	**0.45**	0.49	0.70
Methylene Chloride	**44**	0.71	1.36
TSP	**60**	35.7	89.3
PM_{10}	**--**	18.3	42.4
$PM_{2.5}$	**8.8**	6.6	9.93

Note: Average concentrations for each contaminant were calculated by calculating the mean value across all sampling events that occurred in the monitoring period. Mean calculations presented above excluded missing or invalid sampling events.

Red text indicates a contaminant six-month average is above the Annual AAQC guideline.

Missing data or invalid data was not included in the average concentrations.

Non-detectable concentrations were assumed to be half the detection limit.

[1] Annual AAQC Threshold included for reference, average concentration from WSP sampling is not annualized, so seasonal variations have not been accounted for.

3.2 Continuous Monitoring Results

Continuous monitoring for SO_2 and NO_X was completed for the duration of the monitoring period, with a five-minute resolution in accordance with the Operations Manual. Results of continuous monitoring were compared to the corresponding AAQC guidelines. The AAQC for SO_2 was compared to the unpublished MECP changes; the old 24-hour average was removed and the new 10-minute and one-hour averages were included. As a result, SO_2 data collected was evaluated on a running average for both one-hour and 10-minute averages over the monitoring period. The one-hour and 24-hour AAQCs for NO_2 were used to compare monitoring data, per the Operations Manual. As a result, NO_2 data collected was evaluated on a running average for both one-hour and 24-hour averages over the monitoring period.

For one-hour and 10-minute running averages of SO_2 data, there were no elevated levels during the monitoring period when compared to the AAQC. For one-hour and 24-hour running averages of NO_2 data there were no elevated levels compared to the AAQC during the

monitoring period. A summary of all SO_2 and NO_X data collected over the monitoring period is presented in **Appendix C**. The maximum concentrations of NO_2 and SO_2 measured during the six-month monitoring period relative to each AAQC averaging period are presented in **Table 3-3**.

Table 3-3 Summary of the Continuous Monitoring Results – Maximum Concentrations

Contaminant	Averaging Period	Applicable AAQC Threshold (ppb)	Maximum Concentration (ppb)
Nitrogen Dioxide	1-hour	200	50
	24-hour	100	29
Sulphur Dioxide	10-minute	67	43
	1-hour	40	27
	Annual [1]	4	0.47

Note: [1] Annual AAQC Threshold included for reference, maximum concentration from WSP sampling is not annualized, so seasonal variations have not been accounted for.

4 Ambient Data Comparison

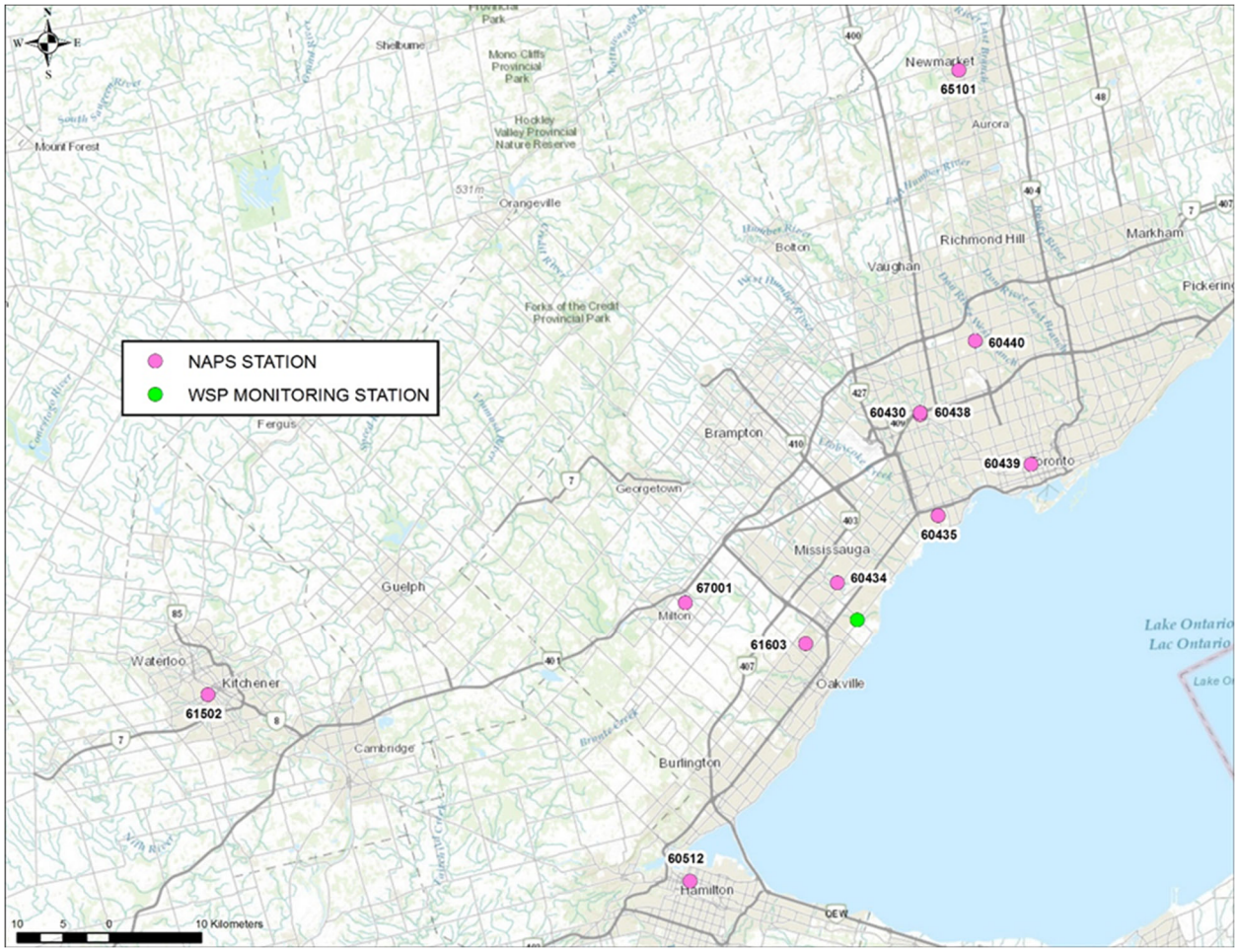

Figure 4-1 National Air Pollution Surveillance Station Location

Data comparisons were completed using the most recent validated data available from the nearest government-operated ambient air quality monitoring stations. Data from the closest National Air Pollution Surveillance (NAPS) stations were used and calculations were made based on data from July to December for each year. The location of each NAPS station used in this report can be found in **Figure 4-1**.

4.1 Discrete Monitoring

Monitoring was conducted following the North American six-day schedule to allow for comparison to local ambient air quality stations upwind and downwind of the Site. At this point

in time, current data across all sample parameters are not available from nearby MECP stations. As a result, data collected at the Site were compared to data collected from local Environment and Climate Change Canada (ECCC) monitoring stations governed by the NAPS Air Toxics Program. The NAPS stations used for data comparison had available data and were representative of the study area. Data comparisons were made with the most recent published data for the NAPS stations (past five years), which was used to compare pollutant trends to the monitoring results.

4.1.1 Acrolein Data Comparison

The ambient acrolein data was not compared to any NAPS stations due to the difference in methodology. The NAPS stations use a model 926 Two Channel Carbonyl Sampler to obtain their acrolein sample. The samples are collected on a DNPH cartridge and analyzed via high performance liquid chromatography (HPLC). The NAPS stations used 24-hour samples with a flow rate of 1 L/min resulting in a volume of approximately 1.44 m^3 over the sampling duration. The lab would need a detection limit of 0.0043 µg for acrolein per sample with a 1.44 m^3 sample to obtain the NAPS reported detection limit of 0.003 $µg/m^3$. Based on discussions with commercial laboratories the lowest detection limit for acrolein is on the order of 1 µg, over 300 times higher than what was calculated from the NAPS results. Commercial laboratories also warned of the potential risk of the high flow rate associated with the NAPS methodology and acrolein not having enough contact time with the DNPH tube to be effectively captured, resulting in the breakthrough of acrolein.

Commercial laboratories instead use evacuated canisters to get acrolein data in ambient air. This analysis is performed using procedures adapted from USEPA Method TO-15, as previously discussed in Section 2.4. Commercial laboratories do not use the ECCC high-volume DNPH methodology as it is not a published Reference Method. Due to the difference in methodology, it is not possible to compare the ambient acrolein data to the NAPS station data.

In the summer of 2007, the MECP completed an Air Quality Monitoring Program for the Clarkson and Oakville area (Report #PIBS 7074e). The monitoring program was completed to determine acrolein, acrylonitrile, and dichloromethane (methylene chloride) concentrations and the potential sources in the area. Since this data was collected from the same area using the same methodology, it was used for comparison purposes. For the MECP study, sampling was completed at three locations to attempt to triangulate a likely source. The MECP study spatially occurred within three kilometers of the WSP ambient monitoring station. MECP sampling in 2007 was completed following USEPA TO-15 methodology. MECP sampling was completed on June 14, June 26, August 28, and September 20 of 2007. Due to the variation in wind direction, the MECP could not identify a point source of elevated acrolein concentrations. The

MECP Air Quality Monitoring Report is attached in **Appendix D**. A comparison of Site data and MECP 2007 data is included in

Table 4-1.

Table 4-1 Acrolein Monitoring Results Comparison with Clarkson Airshed Study

	WSP Sample Results (2020) - µg/m³	MECP Clarkson Airshed Study (2007) - µg/m³	Percent Change
90th Percentile	0.696	2.12	-67 %

The results obtained in 2020 are lower than the baseline data collected by the MECP in 2007 as part of the Clarkson Airshed Study. The 90th percentile concentrations decreased 67 % when compared to the results collected in the 2007 Clarkson Airshed Study. It should be noted that this comparison is done with limited data and taken during different conditions (both spatially and temporally). It is also noted that 2020 data may have been reduced due to COVID-19 impacted operations or traffic. It can be assumed that the proposed development will not further degrade the air quality with respect to acrolein, as will be discussed further in the air dispersion modelling assessment.

4.1.2 Benzene Data Comparisons

Benzene data collected was compared to the closest NAPS stations with benzene data available. The following table shows the NAPS stations used and their location.

Table 4-2 NAPS Station Locations - Benzene

	NAPS Station 60435	NAPS Station 60438	NAPS Station 60440	NAPS Station 60512	NAPS Station 61502	NAPS Station 65101
Location	Etobicoke South, 461 Kipling Ave.	Etobicoke, 401W – 125 Resource Rd.	Toronto North - Downsview, 4905 Dufferin St	Hamilton, Elgin St. & Kelly St. - Beasley Park	Kitchener, West Ave. and Homewood	Newmarket, Eagle St. and McCaffrey Rd.
Distance from WSP's Station	14 km northeast	23 km northeast	33 km north east	34 km southwest	70 km west	60 km northeast

The most recent NAPS data available (2015-2019) was summarized over the same six-month sampling period (July – December) for comparison. When comparing benzene sampling

results to historical data collected at nearby NAPS stations benzene concentrations were comparable. The average benzene concentration from the monitoring program was lower than the average benzene concentrations collected at NAPS stations 60512, 60440, and 60438. The results of this comparison are shown in **Table 4-3.**

Table 4-3 Benzene Monitoring Results Comparison (July – December)

	WSP Sample Result Average	NAPS Station 60435 (2015-2016)	NAPS Station 60438 (2017-2019)	NAPS Station 60440 (2017-2019)	NAPS Station 60512 (2015-2019)	NAPS Station 61502 (2015-2019)	NAPS Station 65101 (2017-2019)	Annual AAQC[1]
	µg/m³	µg/m³	µg/m³	µg/m³	µg/m³	µg/m³	µg/m³	µg/m³
Six Month Mean	0.49	0.45	0.56	0.55	0.67	0.39	0.33	0.45
Six Month 90th Percentile	0.7	0.63	0.78	0.98	1.26	0.63	0.52	0.45

Note: [1] Annual AAQC Threshold included for reference, other concentrations from WSP and NAPS sampling are not annualized, so seasonal variations have not been accounted for.

The NAPS stations were also assessed for the number of 24-hour concentrations with elevated levels compared to the annual AAQC for benzene, the following table shows the summary.

Table 4-4 Benzene Monitoring Results Comparison – Percentage of Daily Concentrations Greater Than The Annual AAQC For Benzene (July – December)

WSP	NAPS Station 60435 (2015-2016)	NAPS Station 60438 (2017-2019)	NAPS Station 60440 (2017-2019)	NAPS Station 60512 (2015-2019)	NAPS Station 61502 (2015-2019)	NAPS Station 65101 (2017-2019)
50 %	48 %	70 %	58 %	54 %	29 %	22 %

The tables above indicate that it is already historically common for benzene to have elevated levels compared to the annual AAQC in similarly developed areas. The NAPS stations 60438 (Etobicoke 401W), 60440 (Toronto North), and 60512 (Hamilton) all have greater concentrations than WSP's monitoring station and NAPS station 60435 (Etobicoke South) had similar concentrations. NAPS station 61502 (Kitchener) and 65101 (Newmarket) have lower concentrations as expected since these areas are less developed and more rural.

The available data collected by NAPS for VOCs is limited, for this reason, the Stations in Kitchener and Newmarket were added for additional comparison, although these locations are a significant distance from the Site. It is difficult to determine the proportion of decrease related to COVID-19 restrictions on benzene concentrations; however, it can be demonstrated that the Site is within typical ranges seen historically throughout Ontario.

It can be assumed that the proposed development will not further degrade ambient air quality within the Clarkson airshed with respect to benzene, as will be discussed further in the air dispersion modelling assessment.

4.1.3 Methylene Chloride (Dichloromethane) Data Comparisons

Methylene chloride data collected was compared to the closest NAPS stations with methylene chloride data available. The following table shows the NAPS stations used and their location.

Table 4-5 NAPS Station Locations – Methylene Chloride

	NAPS Station 60435	NAPS Station 60438	NAPS Station 60440	NAPS Station 60512	NAPS Station 61502	NAPS Station 65101
Location	Etobicoke South, 461 Kipling Ave.	Etobicoke, 401W – 125 Resource Rd.	Toronto North - Downsview, 4905 Dufferin St	Hamilton, Elgin St. & Kelly St. - Beasley Park	Kitchener, West Ave. and Homewood	Newmarket, Eagle St. and McCaffrey Rd.
Distance from WSP's Station	14 km northeast	23 km northeast	33 km north east	34 km southwest	70 km west	60 km northeast

The most recent NAPS data available (2015-2019) was summarized over the same six-month sampling period for comparison (July – December).

When comparing methylene chloride sampling results to historical data collected at nearby NAPS stations methylene chloride concentrations were comparable. The average methylene chloride concentration from the monitoring program was within the typical range of concentrations collected at the NAPS stations. The results of this comparison are shown in **Table 4-6.**

Table 4-6 Methylene Chloride Monitoring Results Comparison

	WSP Sample Results	NAPS Station 60435 (2015-2016)	NAPS Station 60438 (2017-2019)	NAPS Station 60440 (2017-2019)	NAPS Station 60512 (2015-2019)	NAPS Station 61502 (2015-2019)	NAPS Station 65101 (2017-2019)	Annual AAQC[1]
	µg/m³	µg/m³	µg/m³	µg/m³	µg/m³	µg/m³	µg/m³	µg/m³
Six Month Mean	0.71	0.62	0.61	1.15	0.39	0.46	0.35	44
Six Month 90th Percentile	1.36	0.87	1.06	2.29	0.58	0.63	0.50	44

Note: [1] Annual AAQC Threshold included for reference, other concentrations from WSP and NAPS sampling are not annualized, so seasonal variations have not been accounted for.

Methylene chloride concentrations are within the typical ranges seen at the surrounding NAPS stations. Methylene chloride samples were mostly non-detectable in the laboratory reports and were below the annual AAQC of 44 µg/m³.

4.1.4 PM Data Comparison

PM_{10} and $PM_{2.5}$ data collected was compared to the closest NAPS stations with data available. The following table shows the NAPS stations used for PM_{10} and $PM_{2.5}$ data.

Table 4-7 NAPS Station Locations – PM_{10} and $PM_{2.5}$

	NAPS Station 60435	NAPS Station 60438	NAPS Station 60439	NAPS Station 60440	NAPS Station 60512
Location	Etobicoke South, 461 Kipling Ave.	Etobicoke, 401W – 125 Resource Rd.	Toronto, 200 College St.	Toronto North - Downsview, 4905 Dufferin St	Hamilton, Elgin St. & Kelly St. - Beasley Park
Distance from WSP's Station	14 km northeast	23 km northeast	25 km north east	33 km north east	34 km southwest

The most recent NAPS data available (2015-2019) was summarized over the same six-month sampling period for comparison (July – December).

Overall, PM_{10} concentrations recorded during the monitoring period were greater than the historical PM_{10} concentrations recorded at the nearby NAPS stations; however, there is no

annual AAQC guideline for PM_{10} and the six-month average concentration was below the 24-hour AAQC. The results of this comparison are shown in

Table 4-8.

Table 4-8 PM_{10} Monitoring Results Comparison

	WSP Sample Result	NAPS Station 60435 (2015)	NAPS Station 60438 (2017-2019)	NAPS Station 60439 (2015-2016)	NAPS Station 60440 (2017-2019)	NAPS Station 60512 (2015-2019)
	µg/m³	µg/m³	µg/m³	µg/m³	µg/m³	µg/m³
Six Month Average	18.3	19.3	18.2	13.5	11.8	12.4
Six Month 90th Percentile	42.4	30.9	29.2	25.0	20.3	20.6

Overall, $PM_{2.5}$ concentrations recorded during the monitoring period were generally lower than $PM_{2.5}$ historical concentrations recorded at the nearby NAPS stations. The six-month average was lower than the Annual AAQC. The results of this comparison are shown in **Table 4-9.**

Table 4-9 $PM_{2.5}$ Monitoring Results Comparison

	WSP Sample Result	NAPS Station 60435 (2015)	NAPS Station 60438 (2017-2019)	NAPS Station 60439 (2015-2016)	NAPS Station 60440 (2017-2019)	NAPS Station 60512 (2015-2019)	Annual AAQC[1]
	µg/m³	µg/m³	µg/m³	µg/m³	µg/m³	µg/m³	µg/m³
Six Month Average	6.6	8.9	8.6	6.9	6.5	7.5	8.8
Six Month 90th Percentile	9.9	19.0	14.5	12.6	12.0	13.1	8.8

Note: [1] Annual AAQC Threshold included for reference, other concentrations from WSP and NAPS sampling are not annualized, so seasonal variations have not been accounted for.

4.1.5 TSP Data Comparison

At this time, no representative MECP or NAPS Station data was available to compare TSP monitoring results.

4.2 Continuous Monitoring

Comparable ambient data for SO_2 and NO_X was not yet validated from nearby MECP stations; as a result, data collected at the Site were compared to data collected from local ECCC monitoring stations governed by the NAPS Air Toxics Program. NAPS stations used for data comparison were stations that had available data and were representative of the study area. The most recent NAPS data available (2015-2019) was summarized over the same six-month sampling period and compared to the WSP sampling data.

4.2.1 SO_2 Data Comparison

Continuous SO_2 data collected from the ambient program was compared to the closest NAPS stations with SO_2 data available. The following table shows the NAPS stations used and their location.

Table 4-10 Naps Station Locations - SO_2

	NAPS Station 60430	NAPS Station 60434	NAPS Station 60438	NAPS Station 60440	NAPS Station 60512	NAPS Station 67001
Location	Etobicoke, 401 W and Resources Rd.	Mississauga, 3359 Mississauga Rd. N. - UofT Campus	Etobicoke, 401W – 125 Resource Rd.	North York, 4905 Dufferin St,	Hamilton, Elgin St. & Kelly St. - Beasley Park	Milton, Main St. E. and Harris Blvd.
Distance to WSP's Station	23 km northeast	4.5 km northwest	23 km northeast	33 km north east	34 km southwest	19 km west

Continuous SO_2 data collected during the monitoring period was below the AAQC for SO_2. Overall, SO_2 concentrations recorded during the monitoring period were comparable to SO_2 concentrations recorded at the nearby NAPS stations over the past five years. It should be noted that NAPS station 60512 (Hamilton) had much higher levels of SO_2 compared to other stations as it measures the impacts of the heavily industrialized areas of Hamilton on the hospital/downtown core. The results of this comparison are shown below in **Table 4-11**.

Table 4-11 SO2 Monitoring Results Comparison – Six Month 90th Percentile

	WSP Sample Result	NAPS Station 60430 (2015-2019)	NAPS Station 60434 (2019)	NAPS Station 60438 (2017-2019)	NAPS Station 60440 (2017-2019)	NAPS Station 60512 (2015-2019)	NAPS Station 67001 (2019)
	ppb	ppb	ppb	ppb	ppb	ppb	ppb
Six Month Mean	0.47	0.47	0.42	0.21	0.16	3.30	0.90
Six Month 90th Percentile	1.0	0.70	1.0	0.63	0.27	9.4	1.9

4.2.2 NO_X DATA Comparison

Continuous NO_X data collected as part of the ambient program was compared to the closest NAPS stations with NO_X data available. The following table shows the NAPS stations used and their location.

Table 4-12 NAPS Station Locations - NO_x

	NAPS Station 60434	NAPS Station 60435	NAPS Station 60438	NAPS Station 60512	NAPS Station 61603	NAPS Station 67001
Location	Mississauga, 3359 Mississauga Rd. N. - UofT Campus	Etobicoke, 461 Kipling Ave.	Etobicoke, 401W – 125 Resource Rd.	Hamilton, Elgin St. & Kelly St. - Beasley Park	Oakville, 8th Line & Glenashton Dr.	Milton, Main St. E. and Harris Blvd.
Distance to WSP's Station	4.5 km northwest	14 km northeast	23 km northeast	34 km southwest	6 km southwest	19 km west

Continuous NO_X data collected during the monitoring period was below the AAQC for NO_2. Overall, NO_X concentrations recorded during the monitoring period were less than NO_X concentrations recorded at the nearby NAPS stations over the past five years. The results of this comparison are shown below in **Table 4-13**.

Table 4-13 NO_x Monitoring Results Comparison

	WSP Sample Result	NAPS Station 60434 (2015 – 2019)	NAPS Station 60435 (2015 – 2019)	NAPS Station 60438 (2017-2019)	NAPS Station 60512 (2015-2019)	NAPS Station 61603 (2015 – 2019)	NAPS Station 67001 (2019)
	ppb	ppb	ppb	ppb	ppb	ppb	ppb
Six Month Mean	10.5	10.9	21.3	39.8	15.2	9.8	12.8
Six Month 90th Percentile	23.6	23.6	44.4	86.3	31.0	21.2	28.0

5 Baseline Concentrations

Ambient air monitoring data collected as part of the Clarkson TSA ambient air quality monitoring program (Clarkson monitoring program) was used in combination with air dispersion modelling results to predict cumulative impacts of air contaminants at the Site for benzene, acrolein, $PM_{2.5}$, PM_{10}, TSP, NO_X, SO_2, and methylene chloride. In order to assess the cumulative impact on the Site, the 90th percentile of ambient concentrations of each contaminant monitored as part of the Clarkson monitoring program was calculated for 10-min, 1-hour, and 24-hour averaging periods. The 90th percentile of the available monitoring data is typically considered a conservative estimate of baseline air quality (CEA Agency and CNSC, 2009).

Ambient air monitoring data collected as part of the Clarkson Air Shed Industrial Association (CASIA) ambient air quality monitoring program (CASIA monitoring program) was used in combination with air dispersion modelling results to predict cumulative impacts of air contaminants at the Site for carbon monoxide. In order to assess the cumulative impact on the Site, the 90th percentile of ambient concentrations of carbon monoxide was calculated for 1-hour and 8-hour averaging periods. NAPS monitoring data collected in 2019 was used to supplement Clarkson monitoring data collected by WSP to allow for a full year of data to be used to calculate ambient NO_2 and $PM_{2.5}$ concentrations. There was only NO_x data available from CASIA so NAPS data with NO_2 was used instead. The $PM_{2.5}$ data from CASIA was collected continuously using a different methodology so NAPS data was used instead since the methodology was similar to WSP's ambient program.

Ambient air monitoring data collected as part of the NAPS ambient air quality monitoring program (NAPS monitoring program) and Ontario Ministry of Environment, Conservation, and Parks (MECP) ambient air quality monitoring program (MECP monitoring program) was used to obtain ambient concentrations of contaminants which are not part of the Clarkson or CASIA monitoring program. NAPS data was also used to supplement Clarkson monitoring data collected by WSP to allow for a full year of data to be used to calculate ambient contaminant concentrations. The NAPS monitoring station closest to the study area with the most recent data available was used to supplement Clarkson monitoring data. These contaminants include benzene, benzo(a)pyrene, 1,3-butadiene, formaldehyde, acetaldehyde, SO_2, NO_2, $PM_{2.5}$, xylene, and methylene chloride. In order to assess the cumulative impact on the Site, the 90th percentile of ambient concentrations of these contaminants was calculated for 10-min, ½-hour, 1-hour and 24-hour. For contaminants with annual averaging periods, the annual mean was calculated.

A summary of ambient air monitoring data and sources is shown in **Table 5-1** . Impacts from contaminants which have not been retained for the monitoring and modelling assessment will be discussed; however, these impacts will only include existing conditions.

Table 5-1 Summary of Ambient Baseline Concentrations

Contaminant	Averaging Period	Baseline Concentration (µg/m³)	Air Quality Threshold (µg/m³)	% of Threshold	Data Source
PM_{10} [A]	24 h	47	50	94%	Clarkson Air Monitoring and NAPS #60438 (Toronto)
$PM_{2.5}$ [A]	24 h	15	27	54%	Clarkson Air Monitoring and NAPS #60438 (Toronto)
	Annual	8.2	8.8	93%	
TSP [B]	24 h	89	120	74%	Clarkson Air Monitoring
	Annual	36	60	60%	
NO_x (expressed as NO_2) [A]	1 h	36	79	46%	Clarkson Air Monitoring and NAPS #60434 (Mississauga)
	24 h	30	200	15%	
	Annual	16	22.6	68%	
CO	1 h	298	36200	1%	CASIA
	8 h	279	15700	2%	
SO_2 [A]	10 min	3	175.6	2%	Clarkson Air Monitoring
	1 h	2	104.8	2%	Clarkson Air Monitoring and NAPS #60438 (Toronto)
	Annual	0.98	10.5	9%	
Acrolein [B]	1 h	1.6 [C]	4.5	36%	

Contaminant	Averaging Period	Baseline Concentration (µg/m³)	Air Quality Threshold (µg/m³)	% of Threshold	Data Source
	24 h	0.6	0.4	158%	Clarkson Air Monitoring
Benzene [A]	24 h	0.69	2.3	30%	Clarkson Air Monitoring and NAPS #60438 (Toronto)
	Annual	0.49	0.45	109%	
1,3-Butadiene	24 h	0.1	10	1%	NAPS #60435 (Etobicoke)
	Annual	0.01 [C]	2	0.5%	
Acetaldehyde	30 min	5 [C]	500	1%	NAPS #60211 (Windsor West)
	24 h	2	500	0.3%	
Formaldehyde	24 h	3	65	5%	NAPS #60211 (Windsor West)
Benzo(a)pyrene	24 h	0.0001	5.00E-05	213%	NAPS #60430 (Toronto West) NAPS # 60438 (Toronto) NAPS #60439 (Toronto Downtown)
	Annual	0.00001 [C]	1.00E-05	115%	
Methylene Chloride [A]	24 h	1.3	220	1%	Clarkson Air Monitoring and NAPS #60438 (Toronto)
	Annual	0.6	44	1.4%	
Total Reduced Sulphur (as H_2S)	10 min	1.4 [D]	13	11%	MECP #29000 (Hamilton)
	24 h	0.3	7	5%	

Contaminant	Averaging Period	Baseline Concentration (µg/m³)	Air Quality Threshold (µg/m³)	% of Threshold	Data Source
Xylenes	10 min	6.2 [D]	3000	0.2%	NAPS #60435 (Etobicoke)
	24 h	1.5	730	0.2%	

Notes:

[A] Clarkson air monitoring data supplemented with NAPS or CASIA data

[B] Ambient concentration calculated based on 6-months of Clarkson monitoring data

[C] Concentration was converted from the 24-hour concentration. Reference: Ontario Ministry of the Environment, Conservation, and Parks, 2018 (“Procedure for Preparing an Emission Summary and Dispersion Modelling Report”)

[D] The 10-minute concentration was converted from the 24-hour concentration. Reference: Ontario Ministry of the Environment, Conservation, and Parks, 2018 (“Procedure for Preparing an Emission Summary and Dispersion Modelling Report”)

As shown in **Table 5-1,** ambient concentrations of benzo(a)pyrene are greater than the 24-hour and annual air quality thresholds. Benzo(a)pyrene was not monitored by WSP, the nearest monitoring station that was used for baseline concentrations is situated next to Highway 401 as there are not many monitoring stations that monitor benzo(a)pyrene in the surrounding area. Using this location for the baseline concentration is conservative as it likely has higher concentrations of benzo(a)pyrene than at the Clarkson TSA due to the higher volume of traffic experienced on Highway 401.

Benzo(a)pyrene and other polycyclic aromatic hydrocarbons (PAHs) are widespread environmental contaminants formed during incomplete combustion or pyrolysis of organic material. These substances are found in air, water, soils and sediments, generally at trace levels except near their sources. Benzo(a)pyrene is released to the atmosphere from a wide variety of anthropogenic and natural sources including wildfires (ACGIH, 2019). Biomass burning is the most important category of PAH emissions in Canada given that wildfires and residential wood combustion are the largest reported natural and anthropogenic sources, respectively (Tevlin et al, 2020). Residential wood combustion (RWC) is also used for recreational purposes in winter (wood-burning fireplaces) and summer (fire pits, chimineas, and outdoor ovens and smokers) (Tevlin et al, 2020).

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 (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). The benzo(a)pyrene concentrations reported at the Site fall within the ranges reported in Ontario and Canada and are to be expected in urban areas.

The figure provided below illustrates ambient concentrations of benzo(a)pyrene in comparison with guidelines (Tevlin et al, 2020). Annual average ambient air guidelines from the provinces of Ontario (ON), Alberta (AB) and Quebec (QC) are depicted as horizontal blue lines.

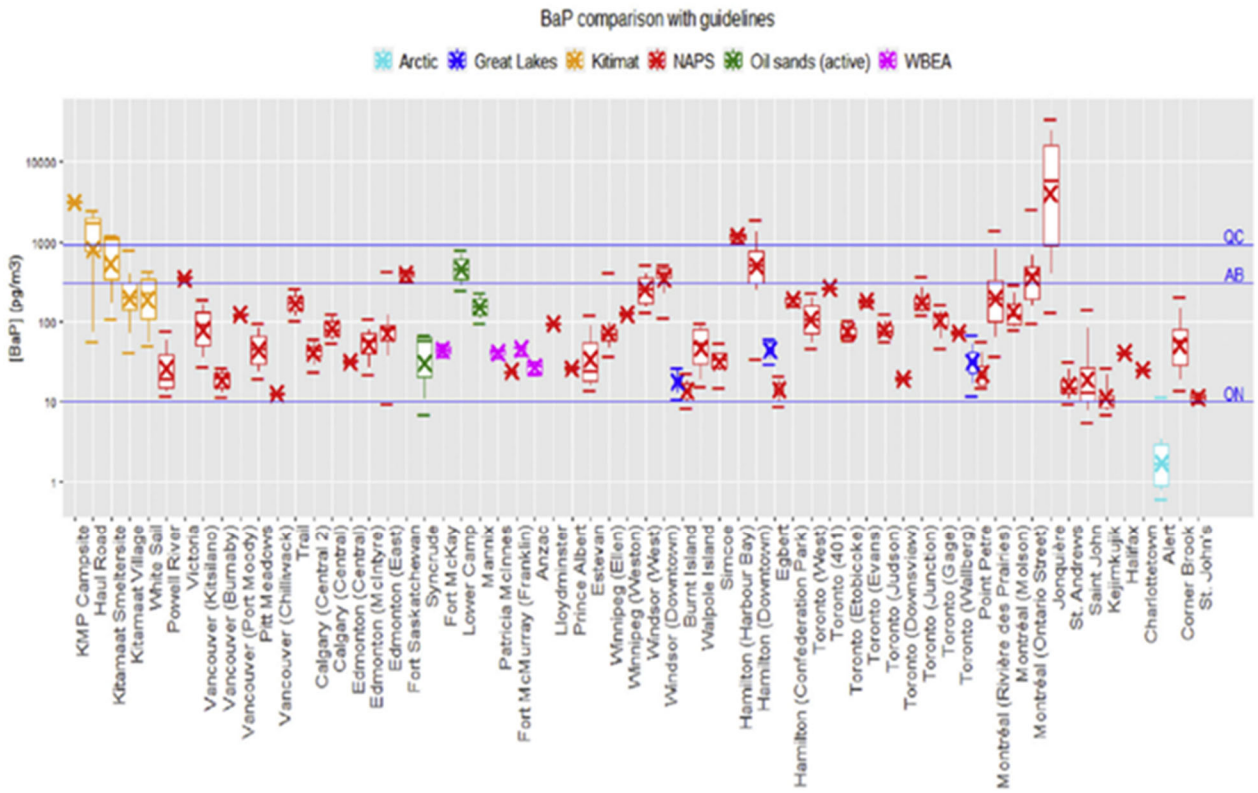

Figure 5-1 Measured Range of Annual Average Benzo(A)Pyrene Concentrations (pg/m^3)

Ambient concentrations of acrolein are also greater than the 24-hour air quality threshold. Acrolein is released to the atmosphere from a wide variety of anthropogenic and natural sources including forest, crop and grassland fires (MOE, 2009). Man-made sources of acrolein

include industrial emissions from manufacturing facilities that make or use acrolein, fossil fuel combustion, motor vehicle exhaust, tobacco smoke, burning of animal and vegetable fats, heating of lubrication oils, burning of wood and plastics and aquatic and terrestrial pesticide uses (MOE, 2009). Forest product manufacturing processes that release VOCs are also known to emit significant amounts of acrolein to the air (MOE, 2009).

From 1996 to 1998, acrolein concentrations in three urban locations in Ontario ranged from 0.14 to 0.25 µg/m^3 with a range of maximum concentrations from 0.56 to 0.71 µg/m^3 (MOE, 2009). From 1989 to 1996, the ECCC NAPS program reported acrolein levels in major urban areas across Canada ranging from 0.05 µg/m^3 to 2.47 µg/m3 with a mean of 0.18 µg/m^3. The highest level in a suburban area was 1.85 µg/m^3 and in a rural area was 0.33 µg/m^3. The acrolein concentrations reported at the Site fall within the ranges reported in Ontario and across Canada and are to be expected in urban areas.

As shown in **Table 5-1**, ambient concentrations of benzene are greater than the annual air quality threshold. Benzene was monitored by WSP for six months; therefore, WSP monitoring data was supplemented with NAPS monitoring data to provide a more representative annual baseline concentration. The nearest monitoring station that was used for baseline concentrations is situated next to Highway 401 as there are not many monitoring stations that monitor benzene in the surrounding area. Using this location for the baseline concentration is conservative as it likely has higher concentrations of benzene than at the Clarkson TSA due to the higher volume of traffic experienced on Highway 401.

All other contaminants of concern are below ambient air quality thresholds.

5.1 Impacts of COVID-19 on Ambient Air Quality

5.1.1 Metrolinx Train Data

Metrolinx has reported ridership on GO Trains being down to less than 10 % of the pre-pandemic levels from April to September 2020. Due to the decreased ridership, Metrolinx reduced the number of trains. WSP evaluated the train schedules as changes were made and determined the actual decrease in train activity for the GO Trains that stop at the Clarkson GO Station. Based on the schedule updates provided to the public by Metrolinx, the following changes were made to the Lakeshore West line since the start of the pandemic. On March 30, 2020 the express rush-hour trips were no longer running. There were further reductions on April 14, 2020 and again on April 27, 2020. On June 9 most of the trains on the Lakeshore West line were reduced from twelve to six coaches. There were still select rush hour trains which had twelve coaches. There were further reductions in the number of coaches per train that began on June 22, 2020. Sampling began on July 8, 2020, when train activity had already been reduced. On September 5, 2020, as the lockdown restrictions were being removed, the

rush hour service was resumed, providing trains every 15 to 30 minutes during rush hours and hourly or better in the midday, evenings and weekends. Most of the trains were still reduced to six cars per train. There were no further updates provided by Metrolinx until after the monitoring was completed in January 2021. Based on the available historic train schedules for the Lakeshore West line, there was a significant decrease in train activity. The following table, **Table 5-2** shows the number of train stops at the Clarkson GO Station.

Table 5-2 Number of Train Stops at Clarkson GO Station

	WEEKDAY		WEEKEND		
Schedule Date	**Eastbound**	**westbound**	**eastbound**	**westbound**	**Weekly total**
05-Jan-19	56	51	35	37	**893**
12-Apr-20	21	21	18	19	**368**
05-Sep-20	34	34	19	19	**552**
Percent Reduction in April 2020	63 %	59 %	49 %	49 %	**59 %**
Percent Reduction in September 2020	39 %	33 %	46 %	49 %	**38 %**

The total weekly stops at the Clarkson GO Station saw a percent decrease of 59 % when comparing the 2019 schedule with the April 2020 schedule. On September 5, 2020, when the schedule was increased there was still 38 % fewer train stops than during pre-COVID conditions. The reduction in train activity in the area likely contributed to reductions in nitrogen oxides, sulfur dioxide, and particulate matter that were being monitored by WSP.

5.1.2 Roadway Traffic

Official traffic data was unavailable to WSP at the time of preparing this report. There was some data available through TomTom's satellite navigation devices that show a decrease in rush hour traffic, between 33 % and 62 %, as shown in **Table 5-3**.

Table 5-3 Percent Reduction in Traffic Due to Covid-19

Month	AM Rush Hour Congestion (% Reduction)	PM Rush Hour Congestion (% Reduction)
July	62 %	43 %
August	51 %	33 %
September	59 %	37 %
October	53 %	43 %
November	61 %	46 %
December	58 %	45 %
Average	**57 %**	**41 %**

Without valid traffic data specific to the area (Royal Windsor Drive and Southdown Road), it is impossible to know the exact reduction in traffic around the Site; however, it can be assumed that it was reduced by approximately 50 %.

5.1.3 Ambient Data Comparison

In order to assess the potential impacts of the COVID-19 pandemic and associated provincial shut-downs on local air quality, the CASIA data over five years (2014 – 2018) during the same six-month period (July – December) was compared to the data collected at WSP's ambient air monitoring station for $PM_{2.5}$ and NO_X. A comparison of monitoring data is presented in **Table 5-4 and Table 5-5.**

Table 5-4 SP and Historical CASIA Data Comparison – $PM_{2.5}$

	Station ID	24-Hour 90th Percentile (µg/m³)	Six Month Mean (µg/m³)
CASIA 2014	STN46118	18.3	10.2
	STN44086	17.0	9.8
CASIA 2015	STN46118	18.5	9.5
	STN44086	19.0	9.8
CASIA 2016	STN46118	15.4	9.3
	STN44086	14.4	8.9
CASIA 2017	STN46118	15.3	10.2
	STN44086	15.4	10.3
CASIA 2018	STN46118	15.8	9.7
	STN44086	17.1	10.9
CASIA Average		16.6	9.9
WSP		15.1	6.6
Percent Change		**-9.0 %**	**-33.3 %**

Table 5-5 WSP and Historical CASIA Data Comparison – NO_X

	Station ID	24-Hr 90th Percentile (ppb)	24 Hr 98th Percentile (ppb)	1 Hr 90th Percentile (ppb)	1 Hr 98th Percentile (ppb)	Six Month Mean (ppb)
CASIA 2014	STN46118	15.3	28.9	20.7	42.0	9.4
	STN44086	20.3	34.9	24.0	52.0	11.1
CASIA 2015	STN46118	19.6	30.3	21.0	47.0	9.6
	STN44086	24.1	48.7	28.0	64.0	12.2
CASIA 2016	STN46118	20.1	38.7	23.0	48.0	10.7
	STN44086	21.4	53.9	23.0	71.0	11.4
CASIA 2017	STN46118	21.3	42.1	27.0	56.0	12.6

	Station ID	24-Hr 90th Percentile (ppb)	24 Hr 98th Percentile (ppb)	1 Hr 90th Percentile (ppb)	1 Hr 98th Percentile (ppb)	Six Month Mean (ppb)
	STN44086	23.8	46.1	28.0	65.9	12.2
CASIA 2018	STN46118	13.2	29.6	16.0	38.0	7.5
	STN44086	18.4	36.6	20.0	51.0	10.0
CASIA Average		19.7	39.0	23.1	53.5	10.7
WSP		24.7	36.1	23.6	55.4	10.5
Percent Change		**25.2 %**	**-7.4 %**	**2.1 %**	**3.5 %**	**-1.3 %**

Based on the six-month mean data comparisons presented in **Table 5-4** and **Table 5-5**, there was a 1.3 % decrease in NO_x concentrations and a 33.3 % decrease in $PM_{2.5}$ concentrations which may have been due to reduced vehicle traffic in the area, or could also be attributed to the difference in station locations or methodology. It should be noted that there is a difference in location and direct comparison between the two data sets has unknown variables. This data comparison demonstrates the reduction in $PM_{2.5}$ being 33.3 % less than the 6-month mean from the CASIA data. The 6-month mean for NOx was only reduced by 1.3 %; however, the 90th percentile increased by 25.2 %. In order to better quantify potential bias adjustment factors for COVID-related impacts on air quality recent data from MECP monitoring stations were assessed. The results are presented in the following section of the report.

Dispersion modelling was completed using supplemented data from January to July to account for the first half of the year when ambient concentrations were not monitored. The baseline concentrations for $PM_{2.5}$, NO_X, PM_{10}, SO_2, benzene and methylene chloride were supplemented with NAPS data from January - July 2019 which helps to adjust to pre-COVID-19 conditions.

5.1.4 MECP Bias Adjustment Factors

MECP air quality data was used to determine bias adjustment factors for WSP's data collected in 2020. MECP air quality data was selected for comparison and development of a bias factor over CASIA data because the MECP monitoring program uses the same sampling methodology and type of equipment. The CASIA should not be compared directly as the sampling methodology and the type of equipment which was used to conduct the sampling are not equivalent to the ones used by the MECP and WSP. MECP data for NO_2, $PM_{2.5}$, and SO_2 were analyzed to determine the percent change from 2019 to 2020. Since the majority of

WSP's sampling took place from July – December 2020, the same period was used when calculating the percent change in the MECP data.
The following table includes a list of MECP monitoring stations used to determine the bias adjustment factors.

Table 5-6 MECP Monitoring Stations Used for Bias Adjustment Factor

Station Name	Contaminants
Mississauga	NO2, PM2.5
Toronto West	NO2, PM2.5, SO2
Toronto North	NO2, PM2.5, SO2
Hamilton Downtown	SO2
Hamilton Mountain	SO2

The following tables include the percent change from 2019 (July-December) to 2020 (July-December).

Table 5-7 NO_2 Bias Adjustment Factor

	Percent Change 2019 – 2020	Average Percent Change per Year (5 Year Average)
Mississauga	-24%	3%
Toronto West	-18%	-1%
Toronto North	-24%	-6%
Average	-22%	-1%

Based on the table above it can be concluded that an approximate percent change for NO_2 concentrations from July – December (monitoring period) due to COVID-19 influences would be -21%. WSP's data set was multiplied by the bias adjustment factor of 1.266 to account for the 21% decrease from 2019. This data was then incorporated into supplementary data to obtain a baseline concentration.

Table 5-8 $PM_{2.5}$ Bias Adjustment Factor

	Percent Change 2019 – 2020	Average Percent Change (5 Year Average)
Mississauga	-2%	-4%
Toronto West	1%	-2%
Toronto North	-11%	-7%
Average	-4%	-4%

The table above demonstrates that $PM_{2.5}$ has been decreasing by approximately 4% each year since 2015. The average decrease as a result of COVID-19 lockdowns is also 4%, so it can be concluded that no bias adjustment factor is required. Further to this, $PM_{2.5}$ decreased less in 2020 when compared to the average percent change over the previous five years at the Mississauga MECP monitoring station.

Table 5-9 SO_2 Bias Adjustment Factor

	Percent Change 2019 – 2020	Percent Change 2018 - 2020	Percent Change 2018 - 2019
Toronto West	-25%	1%	34%
Toronto North	7%	-39%	-43%
Hamilton Downtown	-22%	-13%	11%
Hamilton Mountain	-6%	21%	29%
Average	-12%	-8%	8%

The data quality for SO_2 from MECP is not ideal for these purposes. The data collected from 2015 – 2018 does not include a decimal place, resulting in rounding errors when calculating the mean. There is also no station located in Mississauga that records SO_2 so two stations in

Hamilton were included. Since there does not appear to be any clear trend in the dataset, the average percent change from 2019-2020 and 2018-2020 was used. The average percent change is -10% in 2020, due to the impact of COVID-19 lockdowns. WSP's data set was multiplied by the bias adjustment factor of 1.111 to account for the 10% decrease as a result of COVID-19 lockdowns. This is a conservative approach considering that the average percent change from 2018-2019 (no COVID-19 impact) was an 8% increase, meaning the average decrease is only -2%. This data was then combined with supplementary data from NAPS to obtain a baseline concentration.

5.1.5 COVID-19 Correction Recommendations

Assuming a worst-case scenario based on the MECP data comparison, where NO_2 concentrations were reduced by 22 % due to the reduction in traffic and train activity, the NO_2 concentrations may have been as high as 13.5 ppb, which is still below the 24-hour AAQC for nitrogen dioxide of 100 ppb. Based on the MECP data comparisons for 2019 and 2020 there was no significant change in $PM_{2.5}$ concentrations as a result of COVID-19. The average for the three (3) MECP monitoring stations was a 4% decrease, which is the same as the average decrease per year over the past 5 years. As an absolute worst-case scenario, $PM_{2.5}$ can be assumed to have been reduced by 4 % and the actual concentration may have been 6.9 µg/m³, which is below the annual AAQC and below the 24-hour AAQC threshold.

When assessing the reduction in nearby industrial activity, WSP has concluded that the WWTP most likely would have seen no impact, since the stay-at-home orders and business closures would not have impacted throughput. Petro Canada Lubricants confirmed verbally that their boilers did not slow down throughout 2020 when compared to 2019. Since their boilers are the primary source of the contaminants of concern evaluated in this study, it can be assumed that there were no significant changes due to the pandemic. There was likely some reduction in production at CRH; however, the data required to quantify the reduction was not available at the time this report was prepared. The emission factors used for the dispersion modelling for CRH are based on public NPRI data and working hours.

WSP determined the baseline concentrations using WSP's monitoring data from approximately July – December 2020 combined with supplementary data from the most appropriate source (CASIA, NAPS or MECP). The bias adjustment factors determined from the MECP data were applied to WSP's monitoring data (NO_2, $PM_{2.5}$ and SO_2) to account for the effects of COVID-19 lockdowns on the surrounding air quality.

At the time of this report submission, there are no full datasets for 2020 for the other contaminants monitored as part of this study (benzene, acrolein, methylene chloride, PM_{10}, TSP).

6 Ambient Air Monitoring Conclusions

Based on the ambient monitoring completed over the six-month monitoring period, the following conclusions have been made:

— Data collected since 2015 from NAPS ambient air quality monitoring stations were used to compare with monitoring results. Only data available from NAPS stations closest to the study area and generally similar in surroundings were used to allow for a representative comparison;

— Acrolein concentrations during the monitoring period were higher when compared to representative NAPS stations (2015 - 2019); however, the difference in analytical methodologies does not allow for a reasonable comparison, as such the 2007 data from the MECP Clarkson Airshed Study was used;

— Acrolein concentrations during the monitoring period were lower than the 2007 MECP air quality study. Sources of elevated acrolein concentrations could not be identified in the MECP study due to the variation in wind direction during sampling events, the same is true based on an examination of wind patterns over the six-month study just completed. No wind direction aligned with a single producer/traffic source when acrolein levels were recorded elevated compared to the AAQC;

— More than half of the acrolein samples analyzed in the six-month study were below the laboratory detection limit of 0.23 µg/m^3;

— There were no benzene samples analyzed that were greater than the 24-hour AAQC of 2.3 µg/m^3;

— $PM_{2.5}$ concentrations collected during the monitoring period were comparable or less than $PM_{2.5}$ concentrations of historic nearby NAPS stations. There was one sample that had an elevated concentration compared to the 24-hour AAQC limit for $PM_{2.5}$;

— PM_{10} concentrations collected during the monitoring period were comparable to PM_{10} concentrations of historic nearby NAPS stations. There were two sample days that had measured levels elevated compared to the 24-hour AAQC for PM_{10} of 50 µg/m^3;

— No representative TSP data was available to compare TSP sampling results; there were no 24-hour concentrations elevated compared to the 24-hour AAQC;

- Continuous SO_2 and NO_X data collected during the monitoring period were below the respective AAQC guidelines;
- The 90th percentile concentration of NO_2 was greater than the CAAQS annual concentration (2025). This standard is meant to be based on the average over a single calendar year of all 1-hour average concentrations, not 90th percentiles. The 6-month mean for NO_2 was 18.1 µg/m^3, assuming there was a 21% decrease due to COVID-19 lockdowns this becomes 22.9 µg/m^3, within the conservative 2025 CAAQS. The cumulative concentrations meet the 2020 CAAQS limits and the AAQC limits.
- Meteorological data from Petro Canada Lubricants was received and ambient data analysis for trends was completed as part of air quality dispersion modelling assessment; and
- Although monitoring data shows elevated concentrations compared to the annual AAQC for benzene, it should be noted that an AAQC guideline is a concentration of a contaminant in the air that is protective against adverse effects on health and/or the environment. Benzene exceedances are common across Ontario near sensitive receptors containing high-density residential areas; the magnitude and potential source contribution of elevated benzene will be examined as part of the air quality dispersion modelling assessment.

7 Prevailing Wind Directions

Figure 7-1 illustrates the expected prevailing wind directions at the proposed development. Wind data was obtained from the Clarkson Air Shed Monthly Columnar Data Set (Station ID# 44666) provided by Petro Canada Lubricants Inc. The data from this station was selected to best represent meteorological conditions at the proposed development due to its proximity to the proposed development, data availability over five years, and similar surrounding land uses. Data from January 1, 2016 to December 31, 2020 was used to determine prevailing winds at the Site. Based on the data, prevailing winds are expected to be blowing from the west-southwest and east-northeast. A wind rose diagram with data covering the monitoring period and each sample day can be found in **Appendix E**. When comparing the wind speed and direction for each sample date there was no clear trend indicating where sources of the sampling parameters may have been located.

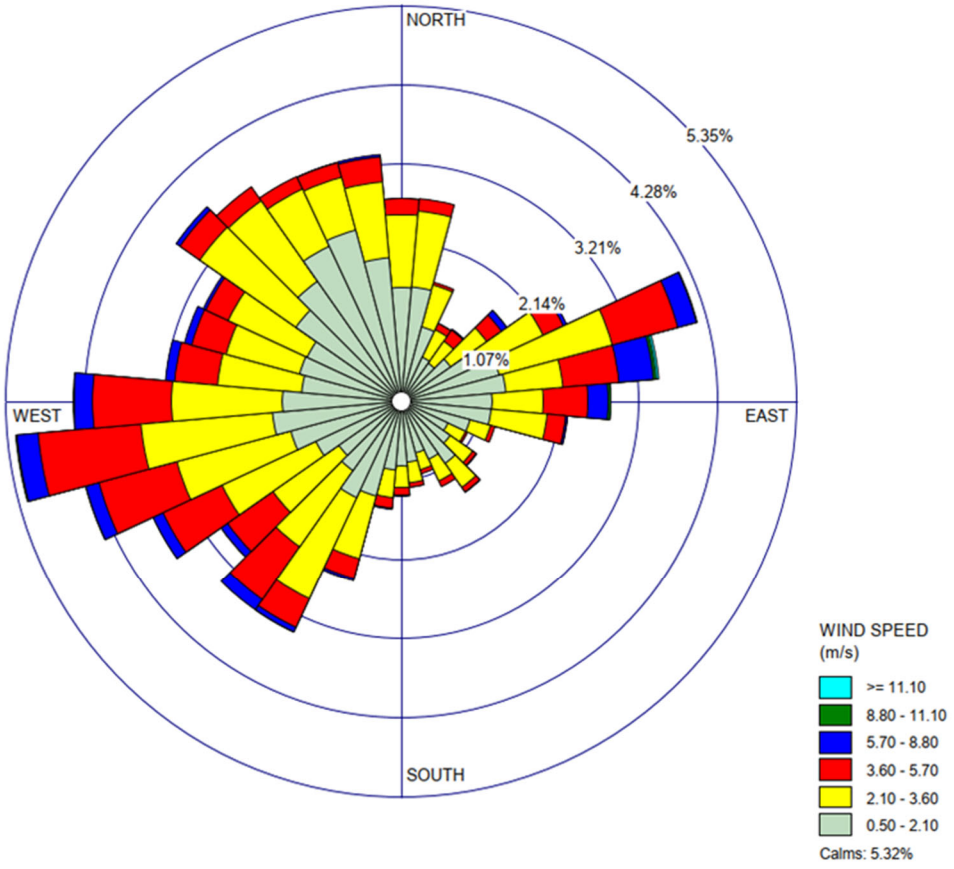

Figure 7-1 Clarkson Prevailing Wind Directions

8 Evaluation of Surrounding Land Uses

Based on the D-6 Guideline, a study area of 1 000 m around the Site was established. The D-6 Guideline outlines a recommended minimum separation distance and potential influence area between industrial facilities and sensitive land uses for three classes of industrial use. The recommended minimum separation distance is the distance (property line to property line) between the incompatible land uses, where industrial use has the potential to cause an adverse effect. The potential influence area is a greater distance in which the industrial operations may have the potential to cause an adverse effect, depending on site operations and meteorological conditions. Additionally, the facilities that are outside of their respective recommended minimum separation distance and potential influence area are expected to have no potential for creating nuisance issues that would give rise to complaints.

In this assessment, facilities of potential concern were assessed based on facility provided emission data, the National Pollutant Release Inventory (NPRI), the Environmental Activity and Sector Registry (EASR) or the Environmental Compliance Approval (ECA) data published online in the Environment Registry of Ontario, aerial photography, and other publicly available data.

8.1 D-6 Guideline

The objective of the D-6 Guideline is to prevent or minimize the encroachment of sensitive land uses upon industrial land uses and vice versa. These two land uses are normally incompatible due to possible adverse effects on sensitive land uses created by industrial operations. For the purpose of this study, a commercial or employment land use is considered an industrial operation in terms of the potential to adversely impact a sensitive land use. The D-6 Guideline categorizes industrial facilities into three classes according to their size, the volume of operations, and nature of their emissions and defines what a sensitive land use is.

The D-6 Guideline provides definitions and examples to illustrate the three industrial classes, provided in **Appendix F**. Facilities that do not meet the definition of any one of the three industrial classes have little to no potential for creating nuisance issues that would give rise to complaints. The definitions and examples in the D-6 Guideline relevant to air quality concerns were used to characterize the nearby facilities. The D-6 Guideline defines a recommended minimum separation distance and potential influence area between industrial facilities and sensitive land uses for each industrial classification, presented in **Table 8-1**.

Table 8-1 Guideline D-6 Recommended Minimum Separation Distance And Potential Influence Areas For Industrial Land Uses

Industrial Classification	Recommended Minimum Separation Distance (m)	Potential Influence Area (m)
Class I – Light Industrial	20	70
Class II – Medium Industrial	70	300
Class III – Heavy Industrial	300	1,000

8.2 Facilities Within Potential Influence Area

A total of 55 industrial facilities surrounding the proposed development were qualitatively assessed for the potential for adverse air quality impacts at the proposed development, as shown in **Table F-1** of **Appendix F**. The locations of industrial facilities identified surrounding the proposed development are shown in **Figure 8-1**. A summary of facilities located within the potential influence area or recommended minimum separation distance is shown in **Table 8-2**. There are 16 facilities located within the potential influence area and six facilities located within the recommended minimum separation as shown. The remaining facilities identified are located outside the potential influence area and are shown in **Table 8-3**.

Figure 8-1 Surrounding Industrial Facilities

Table 8-2 Summary of Industrial Facilities Within the Recommended Minimum Separation Distance or Potential Influence Area

Facility ID	Facility	Address	Industrial Classification	Approximate Distance From Site (m)	Public Reporting	Facility Within Recommended Minimum Separation Distance	Facility Within Potential Influence Area	Quantitative Air Quality Assessment Required	Comments/Rationale
A2	H.L. Blachford Limited [A]	2323 Royal Windsor Drive	III	620	Yes	No	Yes	Yes	Located within potential influence area, public air emission data available
A4	All Tank (1342131 Ontario Limited) [A]	2460 Royal Windsor Drive	III	988	Yes	No	Yes	Yes	Located within potential influence area, public air emission data available
A10	Greater Toronto Transit Authority (Clarkson Go Station) [A]	1110 Southdown Road	I	7	No	Yes	Yes	No	Located within the minimum separation distance, however, the Clarkson GO Station has an ECA for a standby diesel generator to be used during emergency situations and periodic testing. Is it expected, given its purpose, that the diesel generator will be located near a building. The nearest building to the Site is the parking garage approximately 118 m northwest of the Site. The diesel generator will be used infrequently and is expected to be located outside the recommended minimum separation distance and potential area of influence. Any additional emissions from the facility would have been captured in ambient data.
A11	ICS Universal Drum Reconditioning Limited [A]	2460 Royal Windsor Drive	III	988	Yes	No	Yes	Yes	See All Tank (Facility ID A4)
A12	IPEX Inc. [A]	2441 Royal Windsor Drive	III	882	Yes	No	Yes	Yes	Located within potential influence area, public air emission data available
A14	Petro Canada Lubricants Inc [A]	385 Southdown Road	III	887	Yes	No	Yes	Yes	Located within potential influence area, public air emission data available
A15	CRH Canada Group [A]	2391 Lakeshore Rd West	III	990	Yes	No	Yes	Yes	Located within potential influence area, public air emission data available
A16	Stackpole International Powder Metal [A]	2430 Royal Windsor Drive	III	796	Yes	No	Yes	Yes	Located within potential influence area, public air emission data available
A17	Stackpole Powertrain International [A]	2400 Royal Windsor Drive	III	884	Yes	No	Yes	Yes	Located within potential influence area, public air emission data available
A18	Trans-Northern Pipelines Inc [A]	385 Southdown Road	III	887	Yes	No	Yes	Yes	Located within potential influence area, public air emission data available
A22	Musket Transportation Ltd	2215 Royal Windsor Drive	II	223	No	No	Yes	No	Located within the potential influence area, however expected emissions associated with the facility (road dust) would be captured in ambient data

Facility ID	Facility	Address	Industrial Classification	Approximate Distance From Site (m)	Public Reporting	Facility Within Recommended Minimum Separation Distance	Facility Within Potential Influence Area	Quantitative Air Quality Assessment Required	Comments/Rationale
A27	Ritcey Custom Cabinetry	2133 Royal Windsor Drive	I	0	No	Yes	Yes	No	Located within the minimum separation distance; however, expected emissions associated with the facility (dust) would be captured in ambient data.
A29	WaySide Auto Service	2133 Royal Windsor Drive	I	0	No	Yes	Yes	No	Located within the minimum separation distance; however, expected emissions associated with the facility would be captured in ambient data.
A30	Audi Repair Mississauga - Lorne Park Car Centre	2133 Royal Windsor Drive	I	0	No	Yes	Yes	No	Located within the minimum separation distance; however, expected emissions associated with the facility would be captured in ambient data.
A48	Caruso's Service Centre Inc.	2133 Royal Windsor Drive	I	0	No	Yes	Yes	No	Located within the minimum separation distance; however, expected emissions associated with the facility would be captured in ambient data.
A55	Mississauga BMW Repair	2133 Royal Windsor Drive	I	0	No	Yes	Yes	No	Located within the minimum separation distance; however, expected emissions associated with the facility would be captured in ambient data.

Notes: [A] Facility operates under Section 9 approval (ECA/EASR).

Table 8-3 Summary of Industrial Facilities Outside the Potential Influence Area

Facility ID	Facility	Address	Industrial Classification	Approximate Distance from Site (m)	Public Reporting	Facility Within Recommended Minimum Separation Distance	Facility within Potential Influence Area	Quantitative Air Quality Assessment Required	Comments/Rationale
A1	Longlac Wood Industries Inc. [A]	2311 Royal Windsor Drive	II	420	No	No	No	No	Located outside potential influence area for applicable facility Class (300 m)
A3	1375 Southdown Road Ltd [A]	1375 Southdown Road	I	995	No	No	No	No	Located outside potential influence area for applicable facility Class (70 m)
A5	Autobody shop [A]	8-2355 Royal Windsor Drive	I	705	No	No	No	No	Located outside potential influence area for applicable facility Class (70 m)
A6	Bruckmann Manufacturing Inc. [A]	2265 Royal Windsor Drive	II	408	No	No	No	No	Located outside potential influence area for applicable facility Class (300 m)
A7	Corporation of the City of Mississauga - Fire Station #103 [A]	2035 Lushes Lane	I	140	No	No	No	No	Located outside potential influence area for applicable facility Class (70 m)

Facility ID	Facility	Address	Industrial Classification	Approximate Distance from Site (m)	Public Reporting	Facility Within Recommended Minimum Separation Distance	Facility within Potential Influence Area	Quantitative Air Quality Assessment Required	Comments/Rationale
A8	Clarkson Wastewater Treatment Plan [A]	2307 Lakeshore Road West	III	1600	Yes	No	No	Yes	Located outside potential influence area for applicable facility Class (1000 m); however, the facility has the potential for significant air emissions and public air emission data is available
A9	FMK Holdings Inc. [A]	2355 Royal Windsor Drive	II	705	No	No	No	No	Located outside potential influence area for applicable facility Class (300 m)
A13	The Peel District School Board [A]	1290 Kelly Drive	I	937	No	No	No	No	Located outside potential influence area for applicable facility Class (70 m)
A19	Trimac Transportation Services [A]	474 Southdown Road	II	1450	No	No	No	No	Located outside potential influence area for applicable facility Class (300 m)
A20	Wawel Villa Incorporated [A]	880 Clarkson Road South	I	690	No	No	No	No	Located outside potential influence area for applicable facility Class (70 m)
A21	Bernardi Building Supply	2235 Royal Windsor Drive	II	330	No	No	No	No	Located outside potential influence area for applicable facility Class (300 m)
A23	Car Pride Auto Spa	2380 Royal Windsor Drive	I	645	No	No	No	No	Located outside potential influence area for applicable facility Class (70 m)
A24	Canada Fruit	885 Avonhead Rd	II	653	No	No	No	No	Located outside potential influence area for applicable facility Class (300 m)
A25	Praxair Canada Inc. - CO2 Plan	566 Southdown Road	II	1300	No	No	No	No	Located outside potential influence area for applicable facility Class (300 m)
A26	Cleanharbors Canada [A]	551 Avonhead Road	III	1200	Yes	No	No	Yes	Located outside potential influence area (1000 m); however, the facility has the potential for significant air emissions and public air emission data is available
A28	AGT Products Inc.	2311 Royal Windsor Drive	II	420	No	No	No	No	Located outside potential influence area for applicable facility Class (300 m)
A31	Midas	2175 Royal Windsor Drive	I	226	No	No	No	No	Located outside potential influence area for applicable facility Class (70 m)
A32	City of Mississauga - Clarkson Yard	2167 Royal Windsor Drive	I	132	No	No	No	No	Located outside potential influence area for applicable facility Class (70 m)
A33	ShipShape Marine LTD	2265 Royal Windsor Drive	II	408	No	No	No	No	Located outside potential influence area for applicable facility Class (300 m)
A34	Victoria Strong	2463 Royal Windsor Drive	II	1015	No	No	No	No	Located outside potential influence area for applicable facility Class (300 m)
A35	Cam Tech Automotive Services	2355 Royal Windsor Drive	I	705	No	No	No	No	Located outside potential influence area for applicable facility Class (70 m)
A36	Nestle Purina Petcare [A]	2500 Royal Windsor Drive	III	1160	Yes	No	No	Yes	Located outside potential influence area for applicable facility Class (1000 m); however, the facility has the potential for significant air emissions and public air emission data is available

Facility ID	Facility	Address	Industrial Classification	Approximate Distance from Site (m)	Public Reporting	Facility Within Recommended Minimum Separation Distance	Facility within Potential Influence Area	Quantitative Air Quality Assessment Required	Comments/Rationale
A37	UBA Inc.	2605 Royal Windsor Drive	III	1410	Yes	No	No	Yes	Located outside potential influence area for applicable facility Class (1000 m); however, the facility has the potential for significant air emissions and public air emission data is available
A38	Total Ready Mix Limited (2159978 Ontario Limited) [A]	1040 Winston Churchill Boulevard	II	1850	No	No	No	No	Located outside potential influence area for applicable facility Class (300 m)
A39	Mancor Canada Inc. [A]	2481 Royal Windsor Drive	II	1860	No	No	No	No	Located outside potential influence area for applicable facility Class (300 m)
A40	Ashland Canada Corp. [A]	2620 Royal Windsor Drive	III	1600	No	No	No	No	Located outside potential influence area for applicable facility Class (1000 m)
A41	Nexeo Solutions [A]	2620 Royal Windsor Drive	III	1600	No	No	No	No	Located outside potential influence area for applicable facility Class (1000 m), public air emission data available however the facility operates with an environmental permit and there are no tall stacks or sources of emissions greater than 50 m in height, so it is assumed that emissions are compliant at the property boundary. Fugitive emissions would have been captured in ambient data.
A42	Tri-Phase Environmental Inc. [A]	446 Hazelhurst Rd	II	2190	No	No	No	No	Located outside potential influence area for applicable facility Class (300 m)
A43	The Corporation of the Regional Municipality of Peel	1201 Walden Circle	I	178	No	No	No	No	Located outside potential influence area for applicable facility Class (70 m)
A44	Interim Place	735 Southdown Road	I	750	No	No	No	No	Located outside potential influence area for applicable facility Class (70 m)
A45	ORTECH Consulting Inc.	804 Southdown Road	I	510	No	No	No	No	Located outside potential influence area for applicable facility Class (70 m)
A46	Bosch Service	1806 Lakeshore Rd West	I	770	No	No	No	No	Located outside potential influence area for applicable facility Class (70 m)
A47	Mississauga Auto Centre	1800 Lakeshore Rd West	I	770	No	No	No	No	Located outside potential influence area for applicable facility Class (70 m)
A49	Canadian Tire Auto Parts & Service	900 Southdown Road	I	80	No	No	No	No	Located outside potential influence area for applicable facility Class (70 m)
A50	Davey Tree Expert Co. of Canada, Limited	2265 Royal Windsor Drive	II	408	No	No	No	No	Located outside potential influence area for applicable facility Class (300 m)

Facility ID	Facility	Address	Industrial Classification	Approximate Distance from Site (m)	Public Reporting	Facility Within Recommended Minimum Separation Distance	Facility within Potential Influence Area	Quantitative Air Quality Assessment Required	Comments/Rationale
A51	Canadian Home Granite & Tiles	2265 Royal Windsor Drive	I	408	No	No	No	No	Located outside potential influence area for applicable facility Class (70 m)
A52	Tech Reset	2301 Royal Windsor Drive	I	520	No	No	No	No	Located outside potential influence area for applicable facility Class (70 m)
A53	PPG Automotive Refinish Canada Inc.	2301 Royal Windsor Drive	II	520	No	No	No	No	Located outside potential influence area for applicable facility Class (300 m)
A54	Canadian Automotive Refinish	2355 Royal Windsor Drive	I	705	No	No	No	No	Located outside potential influence area for applicable facility Class (70 m)

9 Sources and Contaminants

9.1 Stationary Sources

Industrial facilities within the Study Area were assessed per the MECP's D-Series of Guidelines, specifically the D-6 Guideline "Compatibility Between Industrial Facilities" (D-6 Guideline). A total of 9 facilities were identified as requiring further assessment based on their expected or known operations, proximity to the Site, publicly available air emission data, and ECAs. An additional four facilities were identified to require further assessment due to known operations, emissions reporting, and the presence of tall stacks greater than 50 m in height.

9.2 Facility Provided Emission Data

Facility air emission data was provided by H.L. Blachford, Stackpole International Powder Metal (Stackpole), and Clarkson Wastewater Treatment Plant (WWTP) in the form of the Emission Summary and Dispersion Modelling (ESDM) tables, which outline the facility emission rates for contaminants emitted to air from the facility as part of the ECA application process. Contaminants included in the facility ESDM reports which are also emitted by other facilities or which were included in ambient air monitoring were further assessed. A summary of shared contaminants emitted from these facilities is provided in **Table 9-1** and was quantitatively assessed for their potential to impact air quality at the proposed development. It should be noted that all contaminants included in H.L Blachford, Stackpole International Powder Metal, and Clarkson WWTP ESDM tables were below applicable air quality criteria at the facility's property boundary.

Table 9-1 Facility ESDM Contaminant Summary

Facility ID	Facility	Contaminants Reported in ESDM Report
A2	H.L. Blachford Limited	Diethanolamine, NO_X, TSP
A8	Clarkson Wastewater Treatment Plant	Ammonia, NO_X, SO_2, TRS, TSP,
A16	Stackpole International Powder Metal	Benzene, benzo(a)pyrene, cadmium, carbon monoxide, cobalt, manganese, nickel, nitrogen oxide, particulate matter, zinc

9.3 Facilities Reporting Emissions to NPRI

Facilities surrounding the Site were also qualitatively assessed for their potential to impact air quality through a review of the National Pollutant Release Inventory (NPRI) databases from 2016 to 2018 which correspond to the most recent publicly available data. A total of 13 facilities listed in **Table 8-2** and **Table 8-3** reported emissions to air in the NPRI from 2016 to 2018. A summary of NPRI reporting facilities is presented in **Table 9-2**.

Table 9-2 NPRI Reporting Facilities Within the Study Area

FACILITY ID	FACILITY	CONTAMINANTS REPORTED [A]	STACKS > 50 M
A2	H.L. Blachford Limited	Chlorinated alkanes, diethanolamine, zinc	No
A4	All Tank (1342131 Ontario Limited)	$PM_{2.5}$, PM_{10}, methyl ethyl ketone, isopropyl alcohol, toluene, xylene, hydrotreated light distillate, heptane, naphthalene, ethyl acetate, methyl isobutyl ketone, hydrochloric acid	No
A8	Clarkson Wastewater Treatment Plan	Ammonia, phenanthrene, carbon monoxide, sulphur dioxide, total particulates, hexane, toluene, NOx (as NO_2), TRS (as H_2S), hydrogen sulphide, fluorene, acenaphthylene, benzene, naphthalene, anthracene, formaldehyde, fluoranthene, benz(a)anthracene, benzo(j)fluoranthene, acenaphthene, dibenz(a,h)anthracene, 7,12-dimethylbenz(a)anthracene, benzo(a)pyrene, benzo(b)fluoranthene, indeno(1,2,3-c,d)pyrene, benzo(g,h,i)perylene, 3-methylchloranthrene, pyrene, mercury, lead, cobalt, arsenic, vanadium, manganese, copper, cadmium, chromium, selenium, nickel, zinc	No

FACILITY ID	FACILITY	CONTAMINANTS REPORTED [A]	STACKS > 50 M
A11	ICS Universal Drum Reconditioning Limited	See All Tank	No
A12	IPEX Inc. [A]	$PM_{2.5}$, PM_{10}	No
A14	Petro Canada Lubricants Inc	Pentane, butane, propane, propylene, carbon monoxide, methanol, TSP, sulphur dioxide, $PM_{2.5}$, PM_{10}, methyl ethyl ketone, hexane, isopropyl alcohol, sulphuric acid, toluene, NO_x (as NO_2), total reduced sulphur (as H_2S)	Yes
A15	CRH (CRH Canada Group)	Ammonia, phenanthrene, hexachlorobenzene, carbon monoxide, sulphur dioxide, $PM_{2.5}$, PM_{10}, total PM, methyl ethyl ketone, hexane, toluene, NO_X (as NO_2), xylene, heptane, fluorene, acenaphylene, benzene, 1,2,4-trimethylbenzene, mercury, selenium, hydrochloric acid	Yes
A16	Stackpole International Powder Metal	$PM_{2.5}$, PM_{10}, nickel	No
A17	Stackpole Powertrain International	See Stackpole International Powder Metal	No
A18	Trans-Northern Pipelines Inc (TNPI)	Naphthalene, MTBE, ethyl alcohol, benzene, cumene (isopropyl benzene), cyclohexane, ethyl benzene, hexane, toluene, xylenes [B]	No

FACILITY ID	FACILITY	CONTAMINANTS REPORTED [A]	STACKS > 50 M
A26	Cleanharbors Canada	Carbon monoxide, methanol, isopropyl alcohol, toluene, NO_X (as NO_2), xylene, methyl isobutyl ketone, dichloromethane, formaldehyde, tetrachloroethylene, ethylene glycol	No
A36	Nestle Purina Petcare	$PM_{2.5}$, PM_{10}	No
A37	UBA Inc.	Sulphuric acid, nitric acid, hydrochloric acid	No

Notes: [A] Based on National Pollutant Release Inventory data from 2016 to 2018.
[B] Emission data provided in the TNPI Facility EASR

9.4 Stationary Sources Contaminant Emission Rates

Contaminant emission rates for stationary sources were conservatively estimated using facility ESDM emission data and NPRI reported data from 2016 to 2018 when facility data was not provided. The maximum reported concentration for each contaminant was used to allow for a conservative estimate of emissions from the facility. Facility operating hours reported to NPRI were also used to determine emission rates. If a facility did not report operating hours to NPRI, it was assumed that the facility operates 5 days a week and 12 hours per day, unless otherwise communicated by the facility. Facilities which noted significant shutdown periods in the NPRI reported data were corrected to represent the total working hours of the facility per year. This includes CRH who reported shutdown periods of up to 50 days. Facility operating hours used to determine emission rates are summarized in **Table 9-3**.

Table 9-3 Facility Operating Hours

Facility	Hours Per Day	Days Per Week
H.L. Blachford Limited [B]	-	-
All Tank (1342131 Ontario Limited)	8	5
Clarkson Wastewater Treatment Plan [B]	-	-
Petro Canada Lubricants Inc	24	7

Facility	Hours Per Day	Days Per Week
CRH (CRH Canada Group)	24	6 [A]
Stackpole International Powder Metal/Powertrain [B]	-	-
Cleanharbors Canada	12	5
Nestle Purina Petcare	24	5
UBA Inc.	12	5
TransNorthern Pipeline [B]	-	-
IPEX Inc.	12	5

Notes: [A] Accounts for annual shut down periods up to 50 days
[B] Emission rates provided in ESDM table

Emissions reported to NPRI are generally in tonnes per year. Based on the facility operating hours, these rates were converted to a grams per second emission rate to be used in the air dispersion model, as shown in **Table G-1** of **Appendix G**. Contaminant emission rates for Trans-Northern Pipelines Inc. were estimated based on emission data provided in the facility's EASR. Contaminant emission rates for H.L Blachford, Stackpole, and Clarkson WWTP were estimated based on emission data provided in facility ESDM data. An example emission rate calculation is provided below.

$$\textbf{Petro Canada TSP Emission Rate } \left(\frac{\mathbf{g}}{\mathbf{s}}\right) = \left(\textbf{Maximum Reported NPRI Concentration } \left(\frac{\textbf{tonnes}}{\textbf{year}}\right) \times \mathbf{1\,000\,000} \frac{\mathbf{g}}{\textbf{tonne}}\right) \times \frac{\textbf{year}}{\textbf{364 days}} \times \frac{\textbf{day}}{\textbf{86400}} \textbf{seconds}$$

$$\textbf{Petro Canada TSP Emission Rate } \left(\frac{\mathbf{g}}{\mathbf{s}}\right) = \left(\mathbf{41.6219} \left(\frac{\textbf{tonnes}}{\textbf{year}}\right) \times \mathbf{1\,000\,000} \frac{\mathbf{g}}{\textbf{tonne}}\right) \times \frac{\textbf{year}}{\textbf{364 days}} \times \frac{\textbf{day}}{\textbf{86400}} \textbf{seconds}$$

$$\textbf{Petro Canada TSP Emission Rate } \left(\frac{\mathbf{g}}{\mathbf{s}}\right) = \mathbf{1.32}$$

9.5 Contaminant Negligibility Assessment

A contaminant negligibility assessment was completed to determine which contaminants were to be included in the air dispersion modelling assessment. The negligibility assessment was

based on the procedures outlined in the Air Dispersion Modelling Guideline for Ontario (ADMGO). The negligibility assessment was completed for each facility of concern outlined in **Table 9-4** to determine which contaminants required air dispersion modelling. All contaminants associated with each facility which have the potential to impact the proposed development were screened for negligibility, as shown in **Appendix G**. Contaminants deemed negligible were not incorporated into the modelling assessment; however, their impacts at the Site would have been captured in ambient air monitoring and baseline conditions. Dispersion factors were determined based on the distance of the facility property line to the nearest property boundary of the proposed development. If a contaminant was deemed negligible from a single facility, it was not included in the air dispersion modelling assessment. If a contaminant was deemed negligible from all facilities which emit that contaminant, the combined emissions of that contaminant was assessed for negligibility based on the emission threshold for the nearest facility. Contaminants and facilities included in the negligibility assessment are presented in **Table G-1** of **Appendix G**. Based on the negligibility assessment, a total of 13 contaminants were determined to be significant, as shown below:

— Sulphur dioxide (SO_2);

— Particulate matter less than 2.5 µm in diameter ($PM_{2.5}$);

— Particulate matter less than 10 µm in diameter (PM_{10});

— Nitrogen oxides (as NO_2);

— Sulphuric acid;

— Total reduced sulphur (as H_2S);

— Carbon monoxide (CO);

— Total suspended particulate (TSP);

— Benzene;

— Ammonia;

— Phenanthrene (as benzo(a)pyrene);

— Hydrochloric acid; and,

— Xylene.

It should be noted that phenanthrene which is emitted from the Clarkson WWTP and CRH was not deemed negligible but was not retained for the assessment as it does not have a threshold

value to use for the assessment. Benzo(a)pyrene is the polycyclic aromatic hydrocarbon (PAH) with the most stringent limit, benzo(a)pyrene was used as a surrogate for all PAHs.

9.6 Transportation Sources

Based on the "Ministry of Transportation Environmental Guide for Assessing and Mitigating the Air Quality Impacts and Greenhouse Gas Emissions of Provincial Transportation Projects" (MTO Guide), dated May 2020, and the MECP "Mitigation Strategies and Municipal Road Class Environmental Assessment Air Quality Impact Protocol", dated July 25, 2017, roadway and railway sources within 500 m of the proposed development were assessed for their potential to impact air quality at the Site. **Table 9-4** lists the road and rail sources that have been identified within 500 m of the Site which were included in the air quality assessment.

Table 9-4 Transportation Sources Identified Within the Study Area

Source	Source Type	Approximate Length of Segment Within Study Area (m)	Expected Contaminants
Clarkson GO Station Rail Corridor (travel and idling)	Rail (GO, CN, VIA)	1000	Products of diesel combustion: CO, NO_2, $PM_{2.5}$, PM_{10}, TSP, formaldehyde, benzo(a)pyrene, acetaldehyde, acrolein, benzene
Royal Windsor Drive	Road	703	Products of fuel combustion: CO, NO_2, TSP, PM_{10}, $PM_{2.5}$, and VOCs and common air toxics from mobile-sources: benzene, benzo(a)pyrene, 1,3-butadiene, formaldehyde, acetaldehyde, acrolein
Lakeshore Road West	Road	425	Products of fuel combustion: CO, NO_2, TSP, PM_{10}, $PM_{2.5}$, and VOCs and common air toxics from mobile-sources: benzene, benzo(a)pyrene, 1,3-butadiene, formaldehyde, acetaldehyde, acrolein

Source	Source Type	Approximate Length of Segment Within Study Area (m)	Expected Contaminants
Southdown Road (North of Royal Windsor/Lakeshore)	Road	588	Products of fuel combustion: CO, NO_2, TSP, PM_{10}, $PM_{2.5}$, and VOCs and common air toxics from mobile-sources: benzene, benzo(a)pyrene, 1,3-butadiene, formaldehyde, acetaldehyde, acrolein
Southdown Road (South of Royal Windsor/Lakeshore)	Road	488	Products of fuel combustion: CO, NO_2, TSP, PM_{10}, $PM_{2.5}$, and VOCs and common air toxics from mobile-sources: benzene, benzo(a)pyrene, 1,3-butadiene, formaldehyde, acetaldehyde, acrolein
Notes: Limited published information is available for 1,3-butadiene emission factors for trains, therefore emissions of 1,3-butadiene from trains were not included in the assessment.			

9.7 Transportation Contaminant Emission Rates

9.7.1 Passenger Vehicles and Trucks

Vehicle emission rates for the future conditions (2024) were estimated using the USEPA Motor Vehicle Emission Simulator (MOVES), version MOVES3, released November 10, 2020, which is the latest motor vehicle emission estimate model, and which has replaced the Canadian version of MOBILE6.2C and is approved and recommended for use by the MTO and the MECP. The MOVES model allows for coverage of multiple geographic scales and can generate emission estimates for various time periods (hour, day, month, and year). Emission rates for the assessment were estimated using Annual Average Daily Traffic (AADT) data provided by the City of Mississauga and default highway vehicle fleet (age and vehicles type distribution), emissions inspection and maintenance, and fuel properties were adjusted to reflect the geographic area of the Project (Ontario). AADT values were projected to 2024 using an annual growth rate of 1 %, as outlined in the City of Mississauga Transportation Master Plan dated May 2019. Emission rates for particulate matter included resuspension emissions. MOVES option selections are presented in **Table H-1** in **Appendix H**.

WSP did not include buses as a separate vehicle category as no traffic data was provided for buses. Traffic volume data for buses was assumed to be included in medium/heavy truck volumes. Freight emissions are included in emissions from trucks (single unit short haul and combination long haul).

9.7.2 Trains

Emission rates from trains, including GO, VIA, and CN were estimated using USEPA exhaust emission standards for Tier 2 line-haul and switch locomotives for TSP, PM_{10}, $PM_{2.5}$, NOx, and CO. Line-haul emission factors were used to estimate emission rates during travel while switch emission factors were used to estimate emission rates during idling. Emission rates for benzene, formaldehyde, acetaldehyde, acrolein, and benzo(a)pyrene were estimated using USEPA Large Uncontrolled Stationary Diesel Engines emission standards for both travel and idling. The emission rates for trains were estimated using diesel train frequency (maximum trips per day) projected to 2024 without GO electrification, average train speed, and average engine power data for the Lakeshore West rail corridor on the Port Credit to Clarkson Station segment found in the GO Rail Network Electrification TPAP Air Quality Impact Assessment Report (August 2017), as well as VIA and CN train schedules with train volumes prior to the COVID-19 pandemic. An example emission rate calculation is provided below. Emission calculation tables can be found in **Appendix I**.

$$\boldsymbol{NOx\ Emission\ Rate} = [(\boldsymbol{Travel\ Length} \div \boldsymbol{Average\ Train\ Speed}) \times \boldsymbol{Trips\ Per\ Day}] \times \boldsymbol{Average\ Engine\ Power} \times \boldsymbol{Emission\ Factor} \times \boldsymbol{Conversion}$$

$$\boldsymbol{NOx\ Emission\ Rate} = \left[\left(1\ km \div 63\frac{km}{hr}\right) \times 10\frac{Trips}{hr}\right] \times 2526\ bhp-h \times 5.5\frac{g}{bhp-hr} \times \frac{1hr}{3600s}$$

$$\boldsymbol{NOx\ Emission\ Rate} = 0.613\frac{g}{s}$$

9.8 Assessment of Contaminants

Contaminants outlined in Section 8.5 were assessed for the potential cumulative impact of air contaminants at the Site using ambient monitoring and air dispersion modelling data. Predicted cumulative concentrations of each contaminant were compared to the AAQC guideline, Canadian Ambient Air Quality Standards (CAAQS), or Ontario's Air Contaminants Benchmarks (ACB) lists for each contaminant of concern. Cumulative impacts for contaminants for which there are no existing baseline concentrations will not be presented; however, the predicted concentrations from the modelling assessment were provided. **Table 9-5** outlines the applicable air quality limit for each contaminant of concern in this assessment. The project

threshold will be selected based on the most stringent AAQC or CAAQS guideline for each contaminant. For contaminants which do not have an AAQC or CAAQS, predicted concentrations will be compared to the limit found in Ontario's ACB list.

Table 9-5 Air Quality Limits for Contaminants of Concern

Contaminant	Averaging Period	AAQC (µg/m^3)	CAAQS [A] (µg/m^3 or ppb)	Project Threshold (µg/m^3 Unless Otherwise Stated)
Benzene	Annual	0.45	-	0.45
	24-hr	2.3	-	2.3
Acrolein	1-hr	4.5	-	4.5
	24-hr	0.4	-	0.4
Particulate Matter less than 2.5 µm ($PM_{2.5}$)	24-hr	27	27 µg/m^3 [B]	27
	Annual	8.8	8.8 µg/m^3 [C]	8.8
Particulate Matter less than 10 µm (PM_{10})	24-hr	50	-	50
Total Suspended Particulates (TSP)	Annual	60	-	60
	24-hr	120	-	120
Nitrogen oxides (NO_x)	1-hr	400	2020: 60 ppb [D] 2025: 42 ppb [D] (79 µg/m^3)	79
	24-hr	200	-	200
	Annual	-	2020: 17 ppb [E] 2025: 12 ppb [E] (23 µg/m^3)	23
CO	1-hr	36200	-	36200
	8-hr	15700	-	15700
Benzo(a)pyrene	24-hr	0.00005	-	0.00005
	Annual	0.00001	-	0.00001
1,3-Butadiene	24-hr	10	-	10
	Annual	2	-	2
Formaldehyde	24-hr	65	-	65
Acetaldehyde	0.5-hr	500	-	500

Contaminant	Averaging Period	AAQC (µg/m³)	CAAQS [A] (µg/m³ or ppb)	Project Threshold (µg/m³ Unless Otherwise Stated)
	24-hr	500	-	500
Sulphur dioxide (SO_2)	10-min	178 (67 ppb)	-	178
	1-hr	106 (40 ppb)	2020: 70 ppb [F] 2025: 65 ppb [F]	106
	Annual	11 (4 ppb)	2020: 5 ppb [G] 2025: 4 ppb [G]	11
Sulphuric Acid	24-hr	5	-	5
TRS (as H_2S)	10-min	13	-	13
	24-hr	7	-	7
Ammonia	24-hr	100	-	100
Hydrochloric Acid	0.5-hr	-	-	60[H]
	24-hr	20	-	20
Xylene	10-min	3000	-	3000
	24-hr	730	-	730
Methylene chloride	Annual	44	-	44
	24-hour	220	-	220

Notes: [A] CAAQS as ppb should assume 10°C and 760 mmHg when converting to µg/m³ consistent with the approach for converting AAQCs

[B] The 3-year average of the annual 98th percentile of the daily 24-hour average concentrations

[C] The 3-year average of the annual average concentrations

[D] The 3-year average of the annual 98th percentile daily maximum 1-hour average concentrations

[E] The average over a single calendar year of all the 1-hour average concentrations

[F] The 3-year average of the annual 99th percentile of the SO_2 daily maximum 1-hour average concentrations

[G] The average over a single calendar year of all the 1-hour average SO_2 concentrations

[H] Air Contaminants Benchmarks (ACB) List

10 Dispersion Modelling

The dispersion modelling was conducted in accordance with MECP's Guideline A11: "Air Dispersion Modelling Guideline for Ontario" (ADMGO), the Ministry of Transportation Environmental Guide for Assessing and Mitigating the Air Quality Impacts and Greenhouse Gas Emissions of Provincial Transportation Projects (MTO Guide), dated May 2020, and best practices from the Air Quality Practitioners Group in Ontario, where applicable to each source.

10.1 Dispersion Modelling Input Summary

As per Section 4.5 of the ADMGO, stationary sources were characterized as point or volume sources. Volume sources were sized to cover the main emission sources at a facility and heights were estimated based on average building height. The height of the material piles at CRH was conservatively estimated at 50 m. Where stack data was available, emissions from tall stacks (> 50 m) from CRH and Petro Canada Lubricants Inc. were modelled as point sources. Stack parameters for CRH and Petro Canada Lubricants Inc. were obtained from the NPRI reported data.

Emission data for each point source was not provided within NPRI data; therefore, WSP assigned emissions to point sources based on the maximum estimated facility emission rate, the percent of stack versus fugitive emissions reported to NPRI, and the percentage of the total flow rate for each stack.

For conservatism, when publicly available data was not available to parameterize the emissions sources, WSP conducted the modelling using volume sources to provide conservative results. As a result, emissions from all other facilities were modelled as volume sources as their emissions were assumed to be fugitive in nature.

Transportation sources were characterized as line volume sources and sized to correspond to the width of the road or rail corridor and the expected average height of the vehicles that may be travelling along the roads or rail corridor. The source data required for each road source was calculated using the road type, width of the road and height of the vehicle according to the procedures provided in the ADMGO. Train idling at Clarkson GO Station was characterized as a volume source and sized to correspond to train length, height, and the approximate location at the station.

A detailed summary of dispersion modelling inputs is provided in **Appendix I**.

10.1.1 Dispersion Model Used

The AERMOD dispersion model, version 19191, predicts concentrations at points of impingement (POI) along the property line and beyond. The MECP identified AERMOD as an approved dispersion model under O. Reg. 419/05 which includes the Plume Rise Model Enhancements (PRIME) algorithms for assessing the effects of buildings on air dispersion. AERMOD is applicable for assessing dispersion accommodating rural and urban areas, flat and complex terrain, surface and elevated releases as well as multiple source types (including point, area, and volume sources). The AERMOD modelling system consists of the AERMOD dispersion model, the AERMET meteorological pre-processor and the AERMAP terrain pre-processor.

An assessment of the applicability and potential impacts of shoreline fumigation for the proposed development was also conducted. The initial assessment was completed using the SCREEN3 dispersion modelling for the point sources greater than 50 m in height with available stack information to assess the impact on the project. SCREEN3 is a highly conservative model to assess fumigation as it uses the stability class F (which is an infrequent meteorological stability class) and also a thermal inversion boundary layer factor of six (6) which is conservative. The SCREEN3 results indicate that there is potential for shoreline fumigation effects associated with the Petro Canada Lubricants Inc. sources identified as PCLI2, PCLI3, PCLI4 and the CRH Canada Group source identified as CRH5, to impact predicted concentrations at the proposed development. WSP conducted additional modelling using the Shoreline Dispersion Model (SDM) to identify the hours when fumigation could occur and to confirm whether further assessment is required for those hours. Of the 5 years of hourly meteorological data assessed for sources PCLI2, PCLI3 and CRH5, only 0.06% (approximately 26 hours) were identified where fumigation could occur; and for source PCLI4 0.11% (approximately 49 hours) were identified where fumigation could occur. The potential increase in concentration presented with fumigation would range from a factor of 1.09 to 2.84; however, the contribution to the maximum from these sources is small for all sources and contaminants except for SO_2 on an hourly basis from CRH5 (50% contribution to maximum). To estimate the potential concentration with fumigation for the worst-case hour, assuming fumigation could occur on this hour which is highly unlikely, we can apply the respective applicable factors of 1.09 to 2.84 to each of the sources (PCLI2, PCLI3, PCLI4 and CRH5). By adding this impact to the existing results we can estimate a concentration of 73 ug/m^3 for SO_2 on an hourly basis (with background) which will remain below the 106 ug/m^3 SO_2 AAQC threshold. This estimate would be highly conservative (and unrealistic) as fumigation occurs for so few hours and does not occur for all sources during the same hours, nor at the same time as maximum concentrations were predicted at the Proposed Development. Given this very

small frequency of hours when fumigation impacts could occur at the Proposed Development, and the level of conservatism already included in the assessment methodology, the emission rates, and the modelling, it was identified that no additional assessment of potential fumigation impacts was required as it would not alter the outcome of the assessment. Therefore, an assessment of predicted concentrations resulting from fumigation impacts for hours with the potential for fumigation to occur is not presented as part of this assessment.

10.1.2 Meteorological Conditions and Land Use Data

The site-specific meteorological data file was developed based on guidance in the ADMGO and USEPA AERMET User's Guide.

WSP received a five-year meteorological dataset from Petro Canada Lubricants Inc. containing data from January 2016 to December 2020. Parameters included in the dataset were wind speed, wind direction, temperature, relative humidity, solar radiation, and precipitation. Additional meteorological parameters were required to develop the meteorological dataset for AERMOD, including pressure and cloud cover. Pressure data for January 2016 to December 2020 was obtained from the Toronto City Centre station (Station ID# 48549) located at Billy Bishop Airport and operated by NAV Canada, located approximately 23 km northeast of the Site. The data from this station was selected to best represent meteorological conditions at the proposed development due to its proximity to Lake Ontario, data availability over five years, and similar surrounding land uses. Land use within three kilometres of the meteorological station was set to "Urban" and "Fresh Water" to determine albedo, Bowen ratio, and surface roughness. Cloud cover data was not available; therefore 5/10 (50 %) assumed cloud cover was used to account for the missing data as outlined in the AERMET User's Guide for AERMOD 19191. Upper air data was obtained from the Buffalo, NY upper air station located at the Greater Buffalo International Airport.

The meteorological data required to execute the MOVES emissions model consists of the temperature and pressure for the month of January and July which are considered the worst-case months. The temperature data required to run the model was obtained from Petro Canada Lubricants Inc. while pressure data were obtained from Billy Bishop Airport.

The meteorological input data was processed using AERMET to develop a site-specific data file for the Site. Only one site-specific data site was created as the project area is not large enough to warrant the development of multiple datasets.

10.1.3 Receptors and Area of Modelling Coverage

The area of modelling coverage was centered around the Site and covered a 5 km square area (25 km²). Receptors were placed along the proposed development boundary at a

minimum of 10 m intervals. Discrete receptors were placed at various heights up to 25 storeys at the property boundary to account for balconies, outdoor spaces, and operable windows. The location of discrete receptors for each model was determined based on the location of the maximum POI concentration for each contaminant. The placement of discrete receptors at various heights is considered conservative as these were placed along the property boundary and did not account for building setback distances. The modelling area and boundary receptor placement are shown in **Figure 10-1** and **Figure 10-2**, respectively.

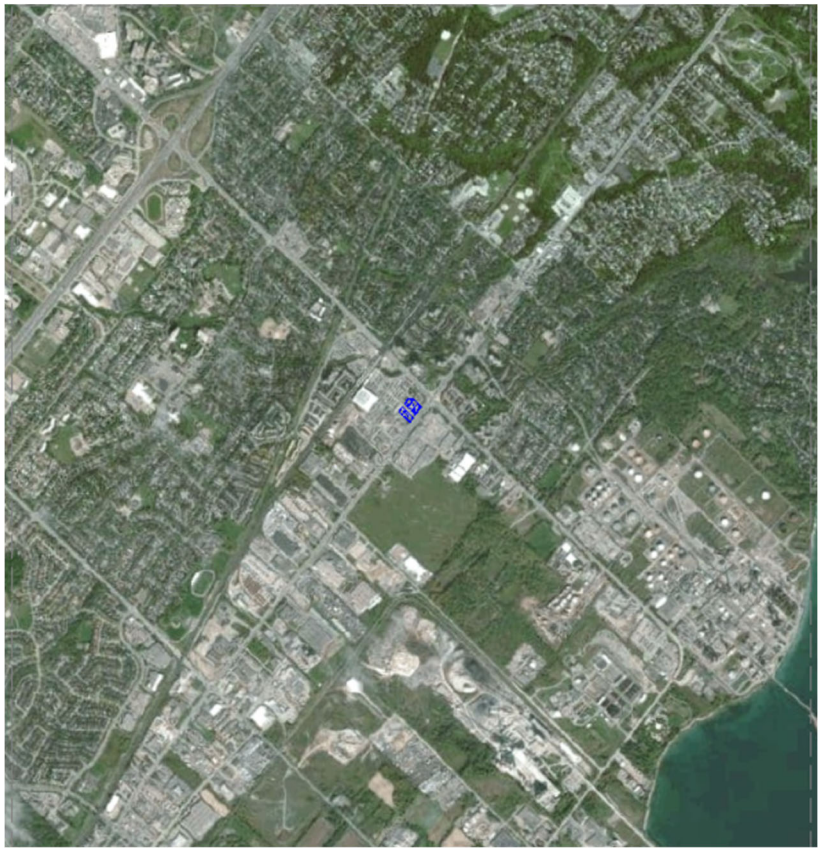

Figure 10-1 Modelling Area Receptors

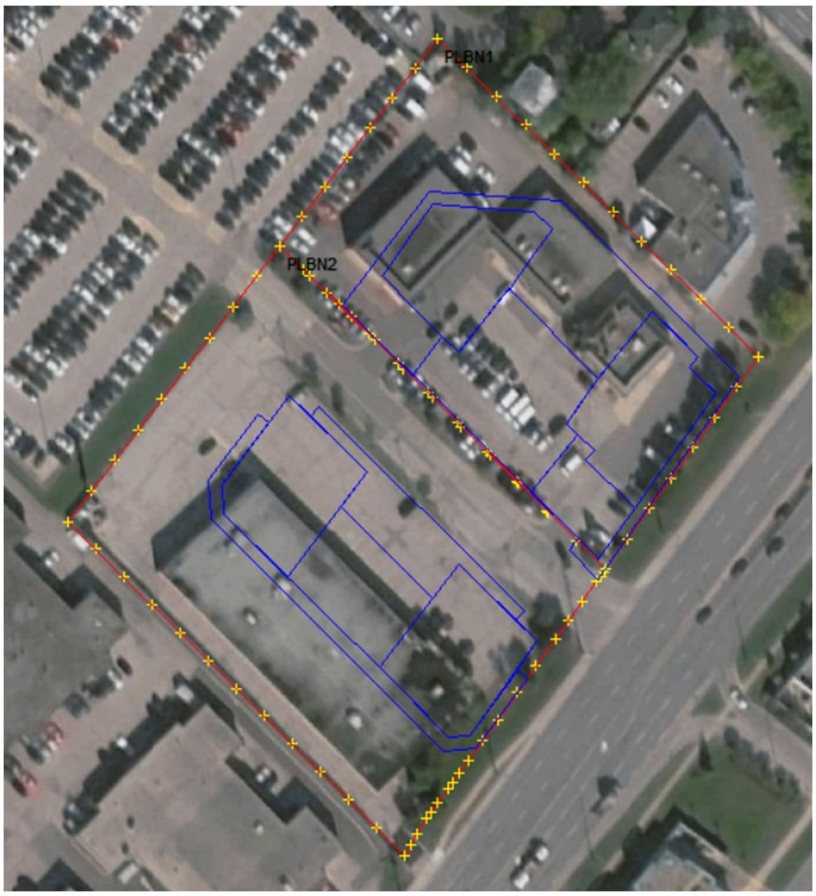

Figure 10-2 Modelling Area and Terrain

10.1.4 Building Downwash

Building wake effects are considered using the USEPA's Building Profile Input Program (BPIP-PRIME), a pre-processor to AERMOD. The inputs into this pre-processor include the coordinates and heights of the relevant buildings and stacks. The output data from BPIP-PRIME is used in the AERMOD building wake effect calculations. For the assessment, no sources of emissions were included on the proposed buildings; therefore, building downwash effects do not apply to the Proposed Development. A preliminary assessment of building downwash effects was completed for industrial sources; however, there were no building downwash effects from the industrial sources on the proposed development modelling area and therefore as a result, building downwash effects were not included in the modelling assessment.

10.1.5 Terrain Data

Terrain information for the area surrounding the Site was obtained from the MECP Ontario Digital Elevation Model data website. The terrain data is based on the North American Datum

1983 (NAD83) horizontal reference datum, cdem_dem_030M.tif, Mississauga, UTM Zone 17. This data was run through the AERMAP terrain pre-processor to estimate base elevations for the buildings, sources and receptors in order to help the model account for changes in the elevations of the surrounding terrain. The modelling area as well as terrain contours are shown in **Figure 10-3**.

Figure 10-3 Modelling Area and Terrain Contours

10.1.6 Averaging Periods Used

Many of the contaminant standards and guidelines are based on 1-hour and 24-hour averaging times, which are averaging times that are provided by AERMOD. In cases where a standard and/or guideline has an averaging period that AERMOD is not designed to predict (e.g. ½-hr or 30-day), a conversion to the appropriate averaging period was completed using the Ministry recommended conversion factors, as documented in the ADMGO and the Ministry Technical Bulletin Methodology for Modelling Assessments with 10-Minute Average Standards and Guidelines under O. Reg. 419/05, dated September 2016.

10.1.7 Dispersion Model Options

A summary of AERMOD dispersion model options is provided in **Table 10-1**.

Table 10-1 AERMOD Model Options

Model Option	Input Selected
Regulatory Options	Default
Dispersion Factor	Urban
Pollutant Models	1,3-butadiene, benzo(a)pyrene, benzene, acrolein, Base model, methylene chloride, NOx, NO_2, TSP, PM_{10}, $PM_{2.5}$, SO_2
Averaging Times	1-hour, 8-hour, 24-hour, annual
Terrain	Elevated
Emission Rate Output Units	$\mu g/m^3$
Source Operating Hours	24 hours/day and 52 weeks/year

10.1.8 Dispersion Modelling Method

Sources were modelled as point sources, volume sources, or line volume sources. All sources were set to be operating 24 hours/day, 7 days/week, 52 weeks/year in the modelling assessment.

Due to the number of sources and contaminant emissions, WSP prepared a simplified modelling approach in a “Base” model. A base emission rate of 1 g/s was entered into each AERMOD source which were then modelled as source groups. The resulting maximum POI concentration from all sources was evaluated and the contribution from each source to the maximum POI concentration was extracted to provide a dispersion factor. The dispersion factor was then used for each applicable source, and the emission rate of each contaminant was multiplied by its corresponding dispersion factor. This allows for a very conservative approach, as the maximum POI concentration from each source will not realistically occur at the same time and place along the property boundary.

Variable emissions were used for train travel and idling to account for hours which do not experience train traffic. Variable emissions were assigned based on GO, VIA and CN train schedules and data. For hours which GO, VIA, and CN are expected to operate, an emission factor of 1 was assigned.

Variable emissions were used for road sources to account for hourly traffic patterns. Midblock hourly traffic counts were provided by the City of Mississauga and used to calculate an emission factor for each hour of the day to account for peak hours.

Contaminant specific models were run for benzo(a)pyrene, benzene, acrolein, TSP, PM_{10}, $PM_{2.5}$, NO_X and NO_2, 1,3-butadiene, SO_2, and methylene chloride given that most of these contaminants are associated with road and rail sources which are expected to have the most impact at the Site. Some of these contaminants also have low air quality thresholds and the existing conditions are above the air quality threshold. This allowed for an assessment of the impact of the proposed project and cumulative impacts.

11 Modelling Results

The air dispersion modelling results for the contaminants of concern are reported in this section. The most impacted property boundary receptor for the Base model was located at the west corner of the site. Air dispersion model results for contaminants included in the modelling assessment are presented for the most impacted receptor. The cumulative impacts at the Site were calculated by aggregating the modelling results with the baseline ambient concentrations. The cumulative impacts at the most impacted receptor were compared to air quality thresholds and are presented in **Table 11-1**.

Table 11-1 Summary of Cumulative Impacts at the Site Property Boundary

Contaminant	Baseline Concentration (µg/m³)	Model Concentration (µg/m³)[1]	Cumulative Concentration (µg/m³)	Averaging Period	Air Quality Threshold (µg/m³)	Percent of Limit From Baseline (%)	Percent of Limit From Model (%)	Percent of Threshold (%)
Benzene	0.69	0.03	0.72	24-hr	2.3	30%	2%	31%
	0.49	0.009	**0.50**	Annual	0.45	**109%**	2%	**111%**
Acrolein	1.6	0.010	1.6	1-hr	4.5	36%	0.2%	36%
	0.63	0.004	**0.63**	24-hr	0.4	**158%**	1%	**158%**
$PM_{2.5}$	15	4.5	19	24-hr	27	54%	17%	71%
	8.2	1.8	**10**	Annual	8.8	93%	21%	**114%**
PM_{10}	47	6.8	**54**	24-hr	50	94%	14%	**108%**
TSP	89	15	104	24-hr	120	74%	12%	87%
	36	6	42	Annual	60	60%	10%	70%
NOx (as NO_2)	36	54	**90**	1-hr	79	46%	68%	**114%**
	30	32	62	24-hr	200	15%	16%	31%
	16	14	**30**	Annual	23	68%	63%	**131%**
CO	298	183	481	1-hr	36200	0.8%	1%	1%
	279	125	404	8-hr	15700	2%	1%	3%
Benzo(a)pyrene	**0.00011**	7.48E-07	**0.00011**	24-hr	0.00005	**213%**	1%	**215%**
	0.000012	N/A[2]	**0.000012**	Annual	0.00001	**115%**	0.0%	**115%**
1,3-Butadiene	0.06	0.001	0.06	24-hr	10	1%	0.01%	1%
	0.01	0.001	0.01	Annual	2	0.5%	0.03%	1%
Formaldehyde	3.1	0.05	3.1	24-hr	65	5%	0.08%	5%
Acetaldehyde	5.0	0.09	5.1	0.5-hr	500	1%	0.02%	1%
	1.7	0.03	1.7	24-hr	500	0.3%	0.01%	0.3%
SO_2	3	88	91	10-min	178	2%	50%	52%
	2	53	55	1-hr	106	2%	50%	52%

Contaminant	Baseline Concentration (µg/m³)	Model Concentration (µg/m³)[1]	Cumulative Concentration (µg/m³)	Averaging Period	Air Quality Threshold (µg/m³)	Percent of Limit From Baseline (%)	Percent of Limit From Model (%)	Percent of Threshold (%)
	1	1.6	2.6	Annual	11	9%	14%	23%
Sulphuric Acid	-	0.06	0.06	24-hr	5	-	1.3%	1.3%
TRS (as H_2S)	1.4	0.1	1.5	10-min	13	11%	1%	12%
	0.3	0.02	0.3	24-hr	7	5%	0.2%	5%
Ammonia	-	0.02	0.02	24-hr	100	-	0.02%	0.02%
Hydrochloric Acid	-	0.02	0.02	0.5-hr	60	-	0.03%	0.03%
	-	0.01	0.01	24-hr	20	-	0.05%	0.05%
Xylene	6	58	64	10-min	3000	0.2%	1.9%	2.1%
	1.5	11	12.5	24-hr	730	0.2%	1.5%	1.7%
Methylene Chloride	1.3	0.3	1.6	24-hr	220	0.6%	0.1%	0.7%
	0.6	0.07	0.67	Annual	44	1.4%	0.2%	1.6%

Notes: Red text indicates concentrations that are elevated compared to the air quality threshold value.

[1] Some modelling results were rounded up for ease of presentation.

[2] Not available – the concentration value is too small to be extracted from the results.

Contaminant concentrations were assessed at various heights where the most impacted property boundary receptor was located to determine where the worst-case contaminant concentrations would be expected along the expected façade of the proposed buildings. A summary of the location of maximum POI concentrations for each contaminant is presented in **Table 11-2**.

Table 11-2 Summary of Maximum POI Concentrations and Location

Contaminant	UTM-E	UTM-N	Model Conc. (µg/m³) [1]	Air Quality Threshold (µg/m³)	Avg. Period	Receptor Height (m)
Benzene	610676.36	4818432.78	0.03	2.3	24-hr	107.5
	610676.36	4818432.78	0.009	0.45	Annual	0
Acrolein	610676.36	4818432.78	0.010	4.5	1-hr	0
	610676.36	4818432.48	0.004	0.4	24-hr	0
$PM_{2.5}$	610520.39	4818401.39	4.5	27	24-hr	21.5
	610676.36	4818432.78	1.8	8.8	Annual	0
PM_{10}	610676.36	4818432.78	6.8	50	24-hr	0
TSP	610598.77	4818323.52	15	120	24-hr	21.5
	610676.36	4818432.78	6	60	Annual	0
NOx (as NO_2)	610585.53	4818486.42	54	79	1-hr	21.5
	610606.67	4818514.03	32	200	24-hr	0
	610606.67	4818514.03	14	23	Annual	0
CO [A]	610676.36	4818432.78	183	36200	1-hr	0
	610676.36	4818432.78	125	15700	8-hr	0
Benzo(a)pyrene	610596.10	4818500.22	7.48E-07	0.00005	24-hr	0
	610676.36	4818432.78	N/A[2]	0.00001	Annual	4.3
1,3-Butadiene	610676.36	4818432.78	0.001	10	24-hr	0
	610676.36	4818432.78	0.001	2	Annual	0
Formaldehyde [A]	610676.36	4818432.78	0.05	65	24-hr	0

Contaminant	UTM-E	UTM-N	Model Conc. ($\mu g/m^3$) [1]	Air Quality Threshold ($\mu g/m^3$)	Avg. Period	Receptor Height (m)
Acetaldehyde [A]	610676.36	4818432.78	0.09	500	0.5-hr	0
	610676.36	4818432.78	0.03	500	24-hr	0
SO_2	610681.47	4818439.89	88	178	10-min	60.2
	610681.47	4818439.89	53	106	1-hr	60.2
	610598.77	4818323.52	1.6	11	Annual	107.5
Sulphuric Acid [A]	610606.67	4818514.03	0.06	5	24-hr	4.3
TRS (as H_2S) [A]	610606.67	4818514.03	0.1	13	10-min	4.3
	610606.67	4818514.03	0.02	7	24-hr	4.3
Ammonia [A]	610606.67	4818514.03	0.02	100	24-hr	4.3
Hydrochloric Acid [A]	610606.67	4818514.03	0.02	60	0.5-hr	4.3
	610606.67	4818514.03	0.01	20	24-hr	4.3
Xylene [A]	610606.67	4818514.03	58	3000	10-min	4.3
	610606.67	4818514.03	11	730	24-hr	4.3
Methylene Chloride	610598.77	4818323.52	0.3	220	24-hr	25.8
	610598.77	4818323.52	0.07	44	Annual	0

Notes: [A] Maximum POI location retrieved from Base model

[1] Some modelling results were rounded up for ease of presentation.[2] N/A - Not available as the concentration is too small to be extracted from the results.

12 Dispersion Modelling Discussion

Emission rates for roadways were predicted using the USEPA's MOVES model. Emission rates for trains on the Clarkson GO rail corridor were predicted using emission standards for Tier 2 diesel locomotives and large diesel engines. Emission rates for facilities of concern were calculated using publicly available facility emission data. Cumulative concentration impacts from the baseline concentrations and the predicted modelled concentration from the stationary and transportation sources within the Clarkson study area were assessed at the Site property boundary and various heights using the AERMOD air dispersion model.

The results presented in **Table 11-1** indicate that the cumulative concentration of acrolein at the most impacted receptor is elevated compared to the 24-hour air quality threshold. It should be noted that ambient concentrations of acrolein collected during the Clarkson Air Monitoring Program are already elevated compared to the 24-hour air quality threshold. Modelled concentrations were combined with ambient data to determine the cumulative impacts; however, this approach is considered conservative as acrolein concentrations from surrounding sources would have already been captured in the Clarkson Air Monitoring Program. The predominant source of acrolein in the study area is transportation sources. As a reminder, baseline concentrations already account for some of the sources modelled for the predicted model concentration; therefore, results are conservative as they include some double counting (i.e., sources captured in the Clarkson Air Monitoring Program are then modelled and added to the results of the Clarkson Air Monitoring Program again). Acrolein has also been identified as a Transportation Related Air Pollutant (TRAP) which is generally elevated near highways and busy roads, often elevated compared to MECP guidelines. Although acrolein was shown to be elevated for the 24-hour air quality threshold in the area, emission rates for acrolein from vehicles are expected to decrease as vehicles become more efficient. To illustrate this, WSP calculated the emissions rates from MOVES for acrolein for a fleet in 2007 (MECP ambient study year) and compared the value to the 2021 and 2024 modelled emission rates. The results are presented in **Table 12-1**.

Table 12-1 Acrolein Emission Rates 2007, 2021, and 2024

Contaminant	Vehicle Type	2007 Emission Rate (g/VMT)	2021 Emission Rate (g/VMT)	2024 Emission Rate (g/VMT)	2007-2021 Change (%)	2021-2024 Change (%)
Acrolein	Passenger Car	3.77E-04	2.15E-05	1.52E-05	-94%	-29%
	Passenger Truck	4.67E-04	5.67E-05	2.95E-05	-88%	-48%

Contaminant	Vehicle Type	2007 Emission Rate (g/VMT)	2021 Emission Rate (g/VMT)	2024 Emission Rate (g/VMT)	2007-2021 Change (%)	2021-2024 Change (%)
	Medium Truck	5.79E-03	7.22E-04	3.91E-04	-88%	-46%
	Heavy Truck	4.40E-03	1.45E-03	9.97E-04	-67%	-31%

Notes: Vehicle Mile Travelled (VMT)

The results presented in **Table 11-1** indicate that cumulative concentrations of benzo(a)pyrene at the most impacted receptor are elevated compared to the 24-hour and annual air quality thresholds. It should be noted that ambient concentrations of benzo(a)pyrene collected as part of the NAPS Air Monitoring Program were already elevated compared to the 24-hour and annual air quality thresholds. Modelled concentrations were combined with ambient data to determine cumulative impacts; however, this approach is considered conservative as benzo(a)pyrene concentrations from surrounding sources would have already been captured in the ambient data, as discussed in the previous paragraph with acrolein. The predominant source of benzo(a)pyrene in the study area is transportation sources. Benzo(a)pyrene has also been identified as a TRAP which is generally elevated near highways and busy roads, often elevated compared to MECP guidelines. Emission rates of benzo(a)pyrene are expected to decrease over time as vehicles become more efficient, similar to acrolein.

The results presented in **Table 11-1** indicate that cumulative concentrations of NO_X at the most impacted receptor are elevated compared to the 1-hour and annual air quality thresholds. Modelled concentrations were combined with ambient data to determine cumulative impacts; however, this approach is considered conservative as NO_X concentrations from surrounding sources would have already been captured in the ambient data, as previously discussed. NOx has also been identified as a TRAP which is generally elevated near highways and busy roads, often elevated compared to MECP guidelines. The predominant source of NOx impacts at the Site is transportation sources; however, emission rates of NOx are also expected to decrease over time as vehicles become more efficient.

The results presented in **Table 11-1** indicate that cumulative concentrations of benzene at the most impacted receptor are elevated compared to the annual air quality thresholds. Modelled concentrations were combined with ambient data to determine cumulative impacts; however, this approach is considered conservative as benzene concentrations from surrounding sources would have already been captured in the ambient data, as previously discussed. Benzene has also been identified as a TRAP which is generally elevated near highways and busy roads, often elevated compared to MECP guidelines. The predominant source of benzene in the

study area is transportation sources; however, emission rates of benzene are also expected to decrease over time as vehicles become more efficient.

The results presented in **Table 11-1** indicate that cumulative concentrations of $PM_{2.5}$ and PM_{10} at the most impacted receptor are elevated compared to the annual air quality thresholds and the 24-hour air quality thresholds respectively. Modelled concentrations were combined with ambient data to determine cumulative impacts; however, this approach is considered conservative as $PM_{2.5}$ and PM_{10} concentrations from surrounding sources would have already been captured in the ambient data, as previously discussed. $PM_{2.5}$ and PM_{10} have also been identified as TRAP which are generally elevated near highways and busy roads, often elevated compared to MECP guidelines. The predominant source of $PM_{2.5}$ and PM_{10} impacts at the Site is transportation sources.

All other significant contaminants included in this assessment were predicted to be below air quality thresholds. The results presented in **Table 11-2** indicate that the maximum contaminant concentration is expected at various heights, depending on the contaminant. When assessing the maximum concentration at the Site from all sources in the Base model, the model indicated that the west corner of the property would experience the highest impact at approximately 0 m for 24-hr, 1-hr and 8-hr averaging periods. Contaminant specific models indicated that the maximum concentrations could occur at various heights depending on the location of sources. For example, the most impacted receptor for 24-hr NOx concentrations is located at the northwest property boundary at a height of approximately 0 m as a result of this location being near train and road sources. In comparison, the most impacted receptor for 1-hr SO_2 concentrations is located at the south property boundary at a height of approximately 60.2 m as a result of this location being near industrial sources of SO_2.

12.1 Nuisance Dust and Odour Impacts

The potential for nuisance dust and odour impacts on the proposed development has been assessed as part of this study. Dust was assessed as part of the dispersion modelling, where emission data was available, and ambient air monitoring. The predominant source or dust impacts on the proposed development are related to traffic and not industrial emissions. PM_{10} and TSP are expected to be below the AAQC thresholds and are not expected to be an issue with respect to nuisance impacts. Facilities within the minimum separation distance and potential influence area are not expected to produce nuisance dust that would impact the proposed development.

Odour may be present from the surrounding facilitates, including the following:

- Clarkson Wastewater Treatment Plant;
- Petro Canada Lubricants Inc; and,
- Ritcey Custom Cabinetry.

The Clarkson Wastewater Treatment Plant (CWWTP) is located approximately 1,600 m from the proposed development and emits some odorous contaminants such as TRS; however, the facility is outside the potential influence area for a Class III facility. The facility uses odour control systems to manage odour from operations to ensure that existing and future operations do not adversely impact offsite receptors. As a result, CWWTP is not expected to cause odour nuisance at the Site.

Petro Canada Lubricants Inc. emits some odorous contaminants such as TRS and is approximately 887 m from the proposed development which is within the potential influence area. The modelled concentrations of contaminants from Petro Canada Lubricants Inc. are low and do not indicate that nuisance odour would be perceivable at the proposed development.

Ritcey Custom Cabinetry is a cabinet manufacturer, the facility building is located approximately 60 m from the proposed development, within the 70 m potential area of influence, but outside the 20 m minimum separation distance for a Class I facility. This facility is small in scale and there are no visible stacks or other emission sources. All products associated with the manufacturing process are expected to be contained inside the facility with minimal potential for fugitive emissions and nuisance. There were no dust and odours were observed onsite during over thirty site visits to install and/or collect sample media. Any potential nuisance dust would have been captured by the air monitoring station on site, which was located approximately 85 m to the northeast of the facility. As a result, Ritcey Custom Cabinetry is not expected to produce any significant odours or dust that would impact the proposed development.

There are 12 auto repair shop facilities within the study area including:

- Mississauga BMW Repair
- WaySide Auto Service;
- Audi Repair Mississauga - Lorne Park Car Centre;
- Caruso's Service Centre Inc.;
- Autobody shop;
- Midas;
- Car Pride Auto Spa;
- Cam Tech Automotive Service;
- Mississauga Auto Centre;
- Canadian Tire Auto Parts & Service;
- PPG Automotive Refinish Canada Inc.; and,
- Canadian Automotive Refinish.

When the distance from the Site is adjusted to account for the distance to the facility building, most of the auto repair shops are located outside potential influence area for applicable facility Class, 70 m for Class I and 300 m for Class II. There are four automotive repair facilities on the property adjacent to the proposed development. Mississauga BMW Repair is within the 20 m

minimum separation distance. WaySide Auto Service is within the 70 m potential area of influence, but outside the 20 m minimum separation distance. Audi Repair Mississauga - Lorne Park Car Centre is within the 70 m potential area of influence but outside the 20 m minimum separation distance. Caruso's Service Centre Inc. is outside the 70 m potential area of influence. These four facilities only conduct repairs and maintenance of vehicles and there is no evidence of paint booths as no environmental permits were found. Any odour generated from operations is expected to be contained within the facility; therefore, there is little potential for nuisance odour. It should also be noted that again no dust or odours were observed in the vicinity of these facilities during over thirty site visits to install and/or collect sample media. All other automotive facilities are well outside the potential influence area and would not be expected to have any odour impacts on the proposed development.

12.2 Summary of Cumulative Human Health Assessment

The Cumulative Human Health Risk Assessment Report (HHRA) can be found in **Appendix K**. Results for each contaminant with a cumulative concentration that exceeded the AAQC and/or CAAQS were provided to the WSP human-health risk assessment team in order to determine appropriate implications and consideration of any mitigation measures for the Proposed Development. Analysis of the frequency and magnitude of exceedances was considered; however, the concentrations presented were primarily a result of existing ambient baseline concentrations due to transportation sources within the study area. As a result, a qualitative assessment of human-health risks was completed.

It was determined that the exceedances of AAQCs are related to significant contribution from ambient baseline sources, with minimal contribution from modelled concentrations. Modelled concentrations for acrolein, benzene, and benzo(a)pyrene contribute ≤2% to cumulative concentrations. The ambient background levels of acrolein, benzene and benzo(a)pyrene are comparable to reported concentrations in Ontario and Canada. Modelled concentrations for $PM_{2.5}$ and PM_{10} concentrations contribute 21% and 14%, respectively. The cumulative concentration of $PM_{2.5}$ is within the range reported in Canadian urban cities. For nitrogen oxides, modelled concentrations and baseline concentrations have similar contributions at approximately 50% to the cumulative concentrations. The NO_2 annual cumulative concentrations for the Clarkson TSA (27 µg/m3 or 15 ppb) are within the range reported in Toronto and Canadian urban areas.

A toxicological review was completed of available jurisdictional ambient air quality objectives (AAQOs) for acrolein, benzene, benzo(a)pyrene, NO_2, PM_{10} and $PM_{2.5}$. Additionally, a comprehensive review of the available short-term (acute) and long-term (chronic) numerical limits was conducted in the HHRA.

Given the ongoing sources of identified chemicals of concern from mobile vehicular and industrial sources, mitigation measures could be developed for implementation in land use planning to improve indoor air quality.

12.3 Mitigation Measures

Air quality mitigation is not required at the proposed development; however, mitigation recommendations have been included to improve indoor air quality. A memorandum with discussion of the recommended mitigation measures to improve indoor air quality can be found in Appendix L. The recommended mitigation measures were determined based on the cumulative concentrations (baseline and modelling) at various heights for each of the COCs that exceeded their respective AAQC threshold value. The cumulative impacts show that except for B(a)P and acrolein, there are no concentrations elevated compared to the AAQC at 30.1 m and above. The Mitigation Recommendations Memorandum presented that background concentrations of acrolein and B(a)P are elevated when compared to the AAQC values; however, B(a)P is elevated anywhere a development were to proceed in an urban area.

For all other COCs, excluding acrolein and B(a)P, based on the data assessed in this memo, the following recommendations are presented:

- Local Air Intakes: If air intakes are designed to be located in each suite, then for any suites below the fourth floor (12.9 m) filters to control $PM_{2.5}$ and PM_{10} impregnated with carbon to control benzene could be utilized. Percent reductions required can be calculated from Table 3. Filters require ongoing maintenance and monitoring per manufacturer specifications, which generally require replacement after a specified duration of time.
- Monitoring: Since Table 3 represents a very conservative approach then it is recommended that a method of ambient monitoring be incorporated to ensure the controls of a local air intake design are working, or even required.
- Ducted Air Intakes: An alternative to filtering local air intakes and monitoring could be to have a centralized air intake system ducted from above 43 m for any suites located below this level.
- Since NO_X is being compared to the CAAQS Annual threshold for NO_2 (12 ppb), it should be based on the same criteria which is the average over a single calendar year of all 1-hour average concentrations. The 6-month average of NO_2 measured by WSP was 6.9 ppb, when adjusted based on the bias adjustment factor (21% decrease due to COVID-19 lockdowns) it becomes 8.7 ppb. At 8.7 ppb the NO_2 concentration for the area is well within the CAAQS annual threshold. The cumulative concentrations include both measured and modelled concentrations for NO_X which is very conservative when assessing the need for mitigation.

With the recommendations presented above and detailed design of mitigation to be conducted by the proponent as part of the Design Process, WSP does not see any further requirements to fulfil a development application at this time.

13 Conclusions

Based on the air dispersion modelling assessment, the following conclusions can be made:

— Benzene, acrolein, PM_{10}, $PM_{2.5}$, NOx, and benzo(a)pyrene were predicted to be above air quality thresholds. All other significant contaminants included in this assessment were predicted to be below air quality thresholds;

— Prevailing wind direction is blowing from the west southwest and east northeast, and not from significant stationary sources of air emissions such as large facilities and tall stacks. As a result, the most significant sources of air impacts at the Site are expected to be transportation sources (road and rail);

— It should be noted that impacts from the Clarkson GO Rail Corridor are expected to decrease over time as Metrolinx electrifies their transportation network, though not included in this assessment as diesel GO trains would continue to operate and pass by until the entirety of the corridor was electrified;

— Modelled maximum air quality impacts were predicted at the most impacted receptor (property boundary or flagpole receptor);

— Concentrations of acrolein at the Site were reported as elevated compared to the 24-hour air quality threshold; however, the proposed development and the cumulative concentration from the nearby sources will not contribute to increasing the existing concentration (i.e., the development is not a source of acrolein);

— Concentrations of benzo(a)pyrene at the Site property boundary were reported as elevated compared to the 24-hour and annual air quality thresholds; however, reported concentrations have been conservatively combined with ambient air monitoring data which would have already captured benzo(a)pyrene concentrations in ambient air and the resulting cumulative concentration was not altered - the cumulative impacts at the proposed development remain unchanged from existing conditions;

— Concentrations of $PM_{2.5}$ and PM_{10} at the Site property boundary were reported as elevated compared to the annual air quality threshold; however, reported concentrations have been conservatively combined with ambient air monitoring data which would have already captured $PM_{2.5}$ concentrations in ambient air and the resulting cumulative concentration was not significantly altered. The cumulative impacts at the proposed development showed a minor increase from existing conditions likely as a result of expected traffic growth in the study area;

- Concentrations of PM_{10} at the Site property boundary were reported as elevated compared to the 24-hour air quality threshold; however, reported concentrations have been conservatively combined with ambient air monitoring data which would have already captured PM_{10} concentrations in ambient air and the resulting cumulative concentration was not significantly altered. The cumulative impacts at the proposed development showed a minor increase from existing conditions likely as a result of expected traffic growth in the study area;
- Concentrations of NOx at the Site property boundary were reported as elevated compared to the 1-hour and annual air quality thresholds; however, reported concentrations have been conservatively combined with ambient air monitoring data which would have already captured NOx concentrations in ambient air. The cumulative impacts at the proposed development showed an increase from existing conditions likely as a result of expected traffic growth in the study area;
- The 90th percentile 24-hour concentration of NO_2 recorded at the monitoring station was below the AAQC threshold. The cumulative concentration calculated from the dispersion modelling was above the annual Canadian Ambient Air Quality Standard (CAAQS) of 12 ppb which may be attributable to the addition of sources to the baseline ambient data which already includes the nearby sources. It should also be noted that the CAAQS is based on the average over a single calendar year of all 1-hour average concentrations, not 90th percentiles. The average of all 1-hour NO_2 concentration collected at the monitoring station was 6.9 ppb.
- Acrolein, PM_{10}, $PM_{2.5}$, benzene, NOx, and benzo(a)pyrene have been identified as Traffic Related Air Pollutants and are identified as often elevated compared to the air quality thresholds in urban areas and near highways and roadways. Elevated concentrations of these contaminants are not unique to the Clarkson TSA and are expected throughout urban areas in Ontario (i.e., Greater Toronto Area and Hamilton) and Canada;
- Based on publicly available data, acrolein and benzo(a)pyrene are not emitted by surrounding industrial facilities in significant amounts; therefore, it is expected that air quality impacts from these contaminants at the proposed development are predominantly associated with transportation emissions;
- Ambient concentrations of acrolein, benzene, NOx, and benzo(a)pyrene are expected to decrease as older vehicles are removed from service and vehicle emission controls become more efficient;

- The proposed development is expected to introduce stationary sources of air emissions associated with comfort heating equipment. These sources would emit contaminants from the stationary combustion and would not alter the results presented as these sources will be very small compared to the transportation emissions. It is unlikely that the introduction of the stationary sources would alter the outcome of the assessment which is dominated by transportation emissions and is conservative;
- Based on the air dispersion assessment, the potential for nuisance odour impacts at the proposed development is not expected based on modelled and cumulative ammonia and TRS concentrations. Ammonia concentrations are well below the 24-hour air quality threshold. Cumulative TRS concentrations are below the 10-minute and 24-hour air quality thresholds, and the majority of TRS concentrations are attributable to baseline conditions which were obtained from Hamilton, Ontario. Based on the model concentrations, there are no significant impacts from surrounding facilities to the proposed development;
- Based on the air dispersion assessment, the potential for nuisance dust impacts at the proposed development is not expected based on cumulative PM_{10} and TSP concentrations. The concentration of TSP is below the air quality threshold. The maximum 24-hour PM_{10} concentration is elevated compared to the air quality threshold; however, reported concentrations have been conservatively combined with ambient air monitoring data which would have already captured PM_{10} and TSP concentrations in ambient air. $PM_{2.5}$ concentrations were elevated compared to the annual air quality threshold; however, $PM_{2.5}$ impacts are predominately from transportation sources that would not give rise to nuisance complaints;
- The Health Assessment, located in Appendix K, determined that the exceedances of AAQCs are related to a significant contribution from ambient baseline sources, with minimal contribution from modelled concentrations. Modelled concentrations for acrolein, benzene and benzo(a)pyrene contribute ≤2% to cumulative concentrations. The ambient background levels of acrolein, benzene and benzo(a)pyrene are comparable to reported concentrations in Ontario and Canada. Modelled concentrations for $PM_{2.5}$ and PM_{10} concentrations contribute 21% and 14%, respectively. The cumulative concentration of $PM_{2.5}$ is within the range reported in Canadian urban cities. For nitrogen oxides, modelled concentrations and baseline concentrations have similar contribution at approximately 50% to the cumulative concentrations. The NO_2 annual cumulative concentrations for the Clarkson TSA (29 $\mu g/m^3$) are within the range reported in Toronto and in Canadian urban areas.
- Air quality mitigation is not required at the proposed development; however, mitigation recommendations have been included to improve indoor air quality.

- The Mitigation Recommendations Memorandum, located in Appendix L, determined that background concentrations of acrolein and B(a)P are elevated when compared to the AAQC values; however, B(a)P is elevated anywhere a development were to proceed in an urban area.

- If air intakes are designed to in each suite, then for any suites below the fourth floor (12.9 m) filters to control PM_{10} and $PM_{2.5}$ impregnated with carbon to control benzene could be utilized to improve indoor air quality. It is recommended that a method of ambient monitoring be incorporated to ensure the controls of a local air intake design are working, or even required. An alternative to filtering local air intakes and monitoring could be to have a centralized air intake system ducted from above 12.9 m for any suites located below this level. A detailed design of mitigation will be conducted by the proponent as part of the Design Process;

- Based on the air quality study, air quality in the study area is not expected to adversely impact high density residential development nor the existing local industrial sites level of compliance to existing standards. Elevated concentrations of contaminants reported (i.e., above health-based thresholds) which could lead to health risks are not unique to the Clarkson TSA and are expected throughout urban areas in Ontario (i.e., Greater Toronto Area and Hamilton) 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.

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H.L. Blachford. Table 4: Emission Summary Table

APPENDIX

D MECP CLARKSON AIRSHED STUDY

Clarkson

Airshed Study

A Scientific Approach to Improving Air Quality

Addendum to Part II – The Ambient Air Monitoring Program: South Mississauga (Clarkson) and Oakville Sampling Results for Acrolein, Acrylonitrile and Dichloromethane in Ambient Air, Summer 2007

April 2009

PIBS 7074e

Protecting our environment. Ontario

Ministry of the Environment

Central Region
Technical Support Section

5775 Yonge Street, 8th Floor
North York, Ontario M2M 4J1
Tel.: (416) 326-6700
Fax: (416) 325-6345

Ministère de l'Environnement

Région du Centre
Section d'appui technique

5775, rue Yonge, 8ième étage
North York, Ontario M2M 4J1
Tél. : (416) 326-6700
Téléc. : (416) 325-6347

April 1, 2009

MEMORANDUM

TO: Dan Orr
Technical Support Manager (A)
Central Region

FROM: Susanne Edwards
Air Quality Analyst
Technical Support Section, Central Region

RE: South Mississauga (Clarkson) and Oakville Sampling Results for Acrolein, Acrylonitrile and Dichloromethane in Ambient Air, Summer 2007

During the implementation of the Clarkson Airshed Part II - Ambient Air Monitoring Program, detectable concentrations of acrolein in ambient air were occasionally measured at three monitoring stations in south Mississauga. Two of these stations (Site No. 44075 and 44080, also called Station QEW West and Station QEW East) were sited west and east, respectively, on the verge of the Queen Elizabeth Way and Highway 403 interchange. The third station (Site No. 46128, also called Station Industrial Centre) was located east of Winston Churchill Boulevard off of Royal Windsor Drive in Mississauga.

Similarly, detectable concentrations of acrylonitrile in ambient air were occasionally measured at five monitoring stations in south Mississauga and Oakville, with elevated concentrations observed at 2 of these monitoring stations. The five monitoring stations were Site No. 44075, 44080, 46117 (also called Station Industrial East), 44083 (also called Station Ford Drive, Oakville), and 44086 (also called Station Residential, Oakville). Elevated acrylonitrile in ambient air was observed at both Oakville locations, namely stations 44083 and 44086.

One June 2004 sample result for dichloromethane, an industrial solvent and paint thinner, exceeded the Ministry 24-hour Ambient Air Quality Criteria (AAQC) and the Ontario Regulation 419/05 Schedule 3 Standard, scheduled to take effect in 2010.

Acrolein is typically emitted into the atmosphere from the combustion and breakdown of petroleum products. For the acrolein results observed in the Clarkson Airshed Part II - Ambient Air Monitoring

Program, this attribution would be consistent with contributions primarily from vehicle emissions, with secondary contributions from the Clarkson industrial complex. Acrylonitrile is used in the manufacture of synthetic polymers or materials. For the acrylonitrile results observed in the Clarkson Airshed Part II - Ambient Air Monitoring Program, this attribution would be consistent with contributions primarily from the Clarkson industrial complex and the vehicle manufacturing facility in Oakville.

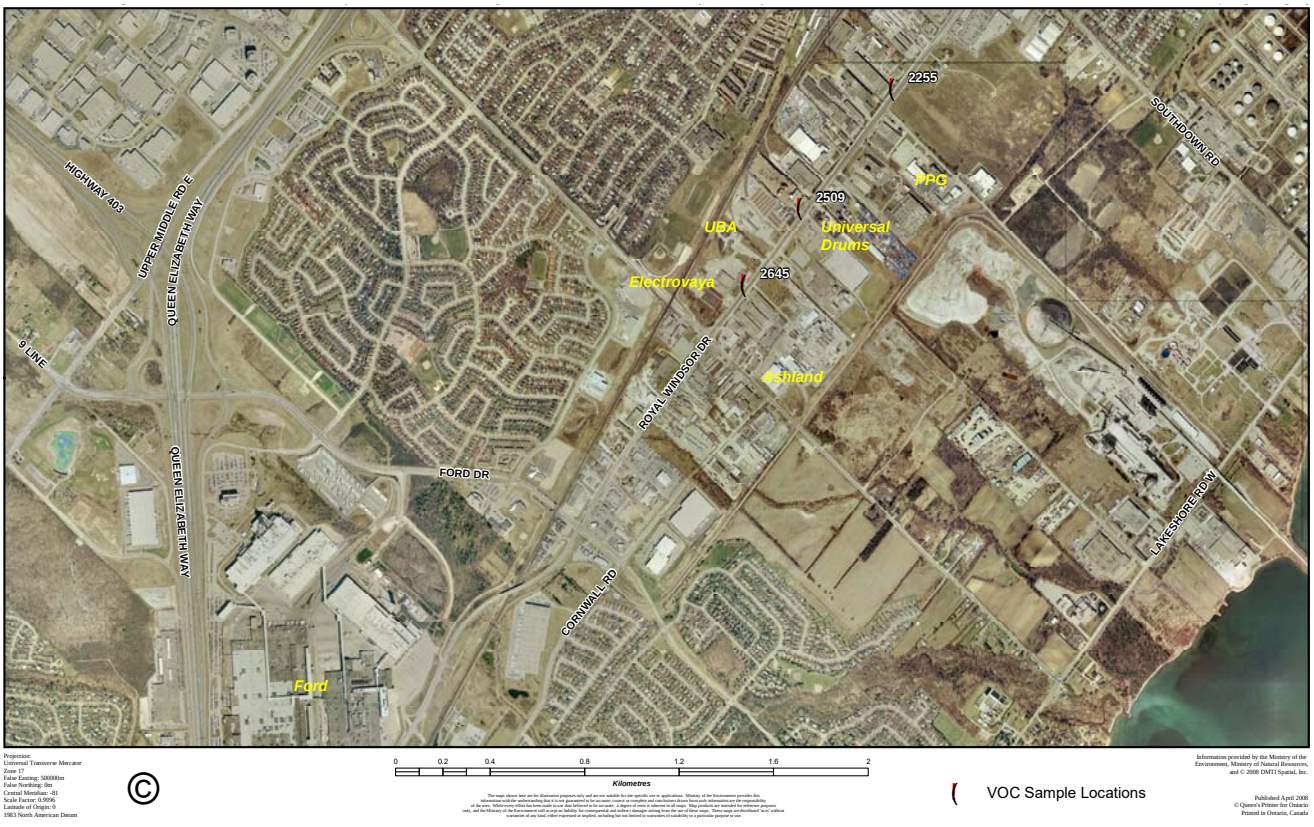
Based upon the preliminary results observed, surmising the source contribution areas, and knowing that both compounds are linked to known or suspected health effects, the Halton-Peel District Office requested that further ambient air monitoring for volatile organic compounds (VOC), particularly for acrolein, acrylonitrile and dichloromethane, be undertaken in the summer of 2007 to expand the VOC database for the south Mississauga-Oakville area.

This report focuses on the results for acrolein, acrylonitrile and dichloromethane sampling in ambient air the vicinity of industrial sources near Winston Churchill Boulevard on Royal Windsor Drive only. Detailed information of these sampling conditions are presented in Appendix 1. Competing monitoring priorities limited the number and duration of VOC sampling during 2007.

A total of three monitoring sites were installed, as shown in Figure 1, at the following locations:

1. 2255 Royal Windsor Drive (in the proximity of Station No. 46128),
2. 2509 Royal Windsor Drive (close to Universal Drum), and
3. 2645 Royal Windsor Drive (Electrovaya Inc.).

Figure 1: VOCs in Ambient Air Sampling Locations – Clarkson, Summer 2007



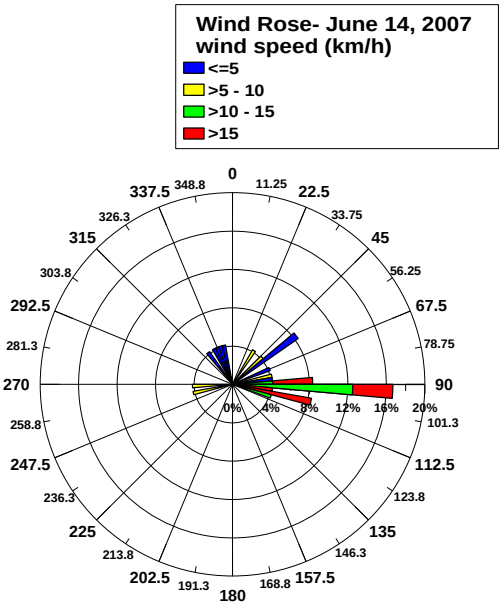
Air sampling for VOC determination was conducted using evacuated stainless steel 2-litre canisters with 24-hour calibrated orifices. Four samples were collected at each of the sampling locations, resulting in twelve samples in total. The samples were collected and submitted to Environment Canada for analysis according to the US EPA TO-14A/TO-15 methodologies.

The four daily (24 h) sampling events were collected on June 14-15, June 26-27, August 28-29, and September 20-21, 2007. Three of the four sampling events were conducted during smog advisories called for the Greater Toronto Area (GTA); June 14-15th, June 26th, and August 29.

Meteorological data from the closest AQI station in Oakville (Station 44017) was used to determine wind speed and direction during the sampling periods.

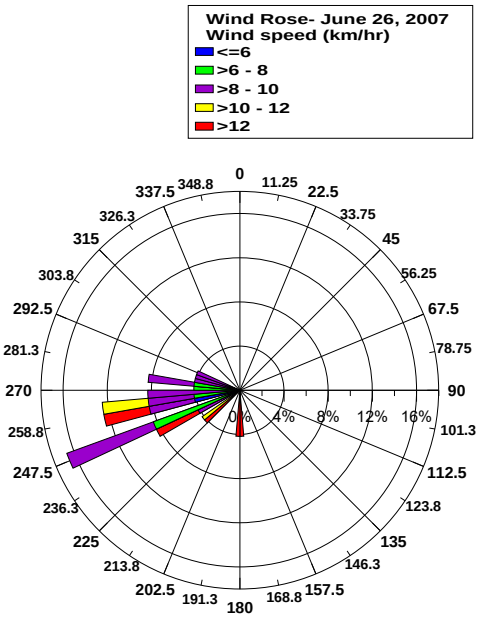
On June 14-15, 2007 the predominant winds were from the East quadrant where 62.5% of the time the wind direction was from 50 degrees to 110 degrees. The wind speeds ranged from 10 to 15 km per hour as illustrated in Figure 2.

Figure 2: Windrose plot for Station 44017 on June 14-15 Sampling Event



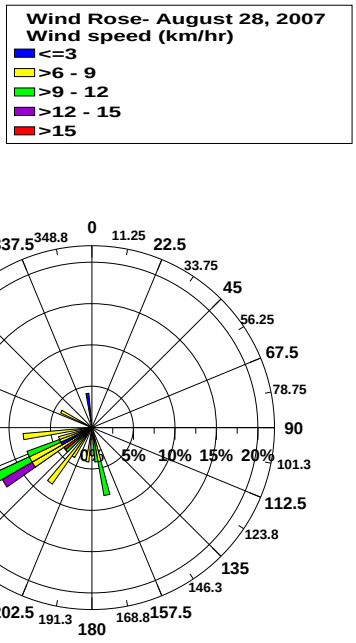
During the June 26-27, 2007 sampling event, the predominant winds were from the South West quadrant where 67% of the time the winds were blowing from 225 to 270 degrees. The wind speeds ranged from 6 to 12 km per hour as illustrated in Figure 3.

Figure 3 Windrose Plot for Station 44017 on June 26-27 Sampling Event



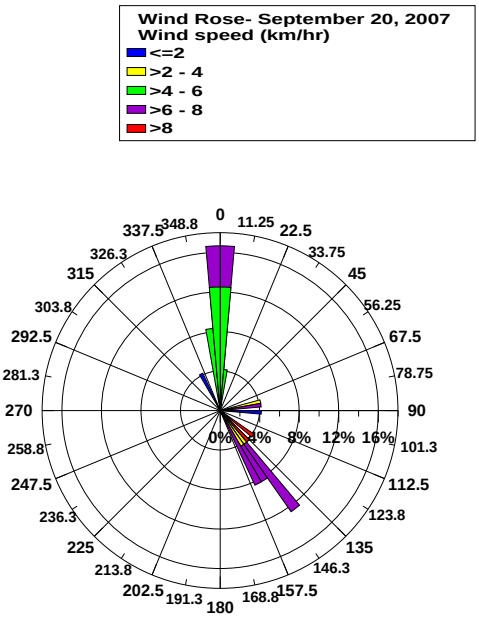
On August 28-29, 2007 the predominant winds were also from South-West quadrant where 58% of the time the winds were from 230 to 270 degrees. The wind speeds ranged from 3 to 15 km per hour as illustrated in Figure 4.

Figure 4 Windrose Plot for Station 44017 on August 28-29 Sampling Event



The fourth sampling event was conducted on September 20-21, 2007. On this day, the predominant winds were blowing from the North 49% of the time (330 degrees to 10 degrees) and later in the day (37.5% of the time) the winds changed to the South-East (125 – 155 degrees). The wind speeds ranged from 2 to 8 km per hour as illustrated in Figure 5.

Figure 5 Windrose Plot for Station 44017 on September 20-21 Sampling Event



The industries situated along Royal Windsor Drive between Winston Churchill and Southdown that potentially contribute to the Volatile Organic Compounds (VOCs) are:

1. UBA Chemicals (2605 Royal Windsor Drive),
2. Ashland Chemicals (2620 Royal Windsor Drive),
3. Universal Drums (2460 Royal Windsor Drive),
4. Stackpole (2400 Royal Windsor Drive),
5. PPG Canada Inc. (2301 Royal Windsor Drive),and
6. Blachford Ltd. (2323 Royal Windsor Drive).

The above industries are mainly involved in the manufacture of chemicals such as adhesives, lubricants, and synthetic polymers, or use a wide range of solvents in their process.

Other potential sources of VOCs, particularly acrolein emissions, are from vehicular traffic and other industries situated west of Winston Churchill, such as Ford Canada, and east of Southdown Road, such as Petro Canada Lubricants.

The sampling results for acrolein, acrylonitrile and dichloromethane are summarized in Table 1.

The daily (24h) concentrations for acrolein were all greater than the Reg 419/05 Schedule 3 standard of 0.08 µg/m^3, The maximum 24-hour average concentration obtained was 3.94 µg/m^3 during the August 28-29 sampling event at the 2645 Royal Windsor Drive station.

During Phase II of the Clarkson Airshed Study, the maximum 24-hour average result obtained was 0.51 µg/m^3 at the Industrial Centre station. In addition, the acrolein levels

Table 1 Daily (24 h) Acrolein, Acrylonitrile and Dichloromethane Concentrations (ug/m^3) in Ambient Air in the vicinity of Royal Windsor Drive, south Mississauga, Summer 2007.

Sampling Dates	Acrolein [1]	Acrylonitrile [2]	Dichloromethane [3]	Location	Station ID. 44017 WD (deg)	Station ID 44017 WS(km/h)
June 14-15, 2007	0.58	<MDL	1.07	2255Royal Windsor	51	4
	1.37	<MDL	8.59	2509Royal Windsor		
	1.70	<MDL	40.5	2645Royal Windsor		
June 26-27, 2007	1.78	<MDL	1.40	2255 Royal Windsor	246	7
	1.69	<MDL	0.75	2509 Royal Windsor		
	1.21	<MDL	0.71	2645 Royal Windsor		
August 28-29, 2007	1.85	<MDL	16.3	2255 Royal Windsor	237	7
	2.14	<MDL	1.81	2509 Royal Windsor		
	3.94	<MDL	2.25	2645 Royal Windsor		
September 20-21, 2007	1.51	<MDL	1.55	2255 Royal Windsor	7	5
	1.93	<MDL	126	2509 Royal Windsor		
	1.08	<MDL	9.57	2645 Royal Windsor		

Notes: The MDL (method detection limit) for acrylonitrile and acrolein is 0.031 ug/m^3 and 0.027 ug/m^3, respectively.

1. O. Reg 419/05 Schedule 3 24-hour standard is 0.08 ug/m^3
2. O. Reg 419/05 Schedule 3 24-hour standard is 0.6 ug/m^3
3. O. Reg 419/05 Schedule 3 24-hour standard is 220 ug/m^3

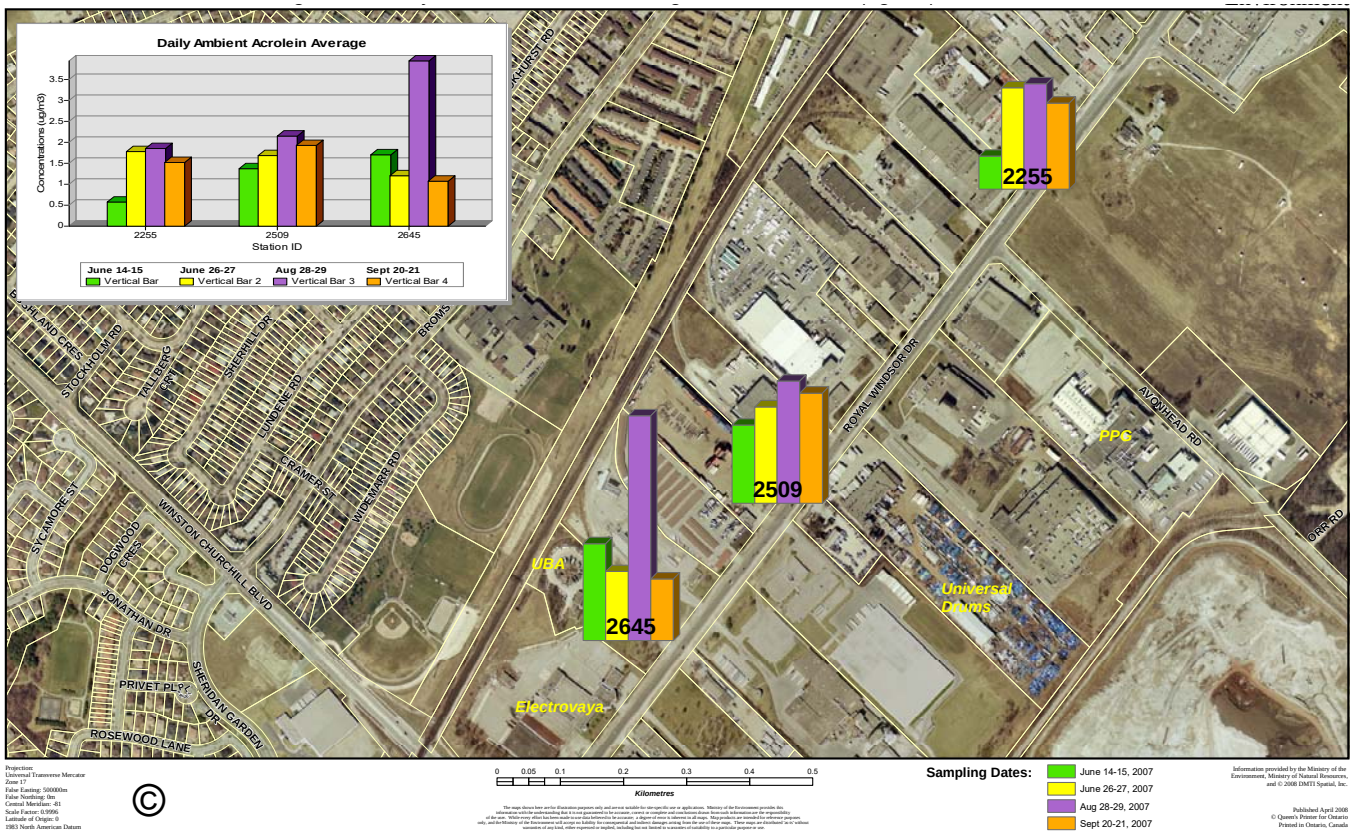
also exceed the O. Reg. 419/05 Upper Risk Threshold (Schedule 6) of 0.8 µg/m^3. However, it is important to note that these standards are based on point of impingement (POI), single source releases, and not the cumulative impacts from all the industries and other potential sources in the area.

Figure 6 shows the spatial variation between the three monitoring stations. Referring to the windrose patterns associated with the sampling events, it is not possible to infer any consistent correlation between wind direction and acrolein concentrations measured. Individual point sources may contribute significantly to maximum 24-hour acrolein concentrations measured, but it is more likely that all industrial point sources and area sources (vehicle emissions) cumulatively contribute to these maximum values.

As shown in Table 1, acrylonitrile levels at all stations were recorded below the detection limit and did not exceed the O. Reg. 419/05 Schedule 3 24-hour standard of 0.6 µg/m^3. During Phase II of the Clarkson Airshed study, the maximum 24-hour acrylonitrile average result obtained was 18.31 µg/m^3 at the Ford Drive station.

Dichloromethane levels are also presented in Table 1. This compound exceeded the AAQC 24-hour standard of 220 µg/m^3 during the Phase II of the Clarkson Airshed Study by 12%. Based on the four sampling events, all the daily dichloromethane levels were below the O. Reg. 419/05 24-hour Schedule 3 standard of 220 µg/m^3. The maximum 24-hour average concentration of 126 µg/m^3 was obtained on September 20-21, 2007 at the 2509 Royal Windsor Drive station.

Figure 6 Daily (24 h) Average Ambient Acrolein Concentrations (µg/m^3) in south Mississauga, Summer 2007



Conclusions

Based on 2007 measurements, and its potential to exceed O. Reg. 419/05 Schedule 3 air standards, which come into effect in 2010, acrolein remains an air quality parameter of concern in the south Mississauga area. Acrylonitrile and dichloromethane concentrations appear to be reduced from Phase II levels

The results from the three sampling locations and the twelve samples, coupled with Phase II results, are statistically insufficient to determine the source(s) contributing to the acrolein exceedances. Based upon poor correlation with wind direction, no one particular point source can be attributed to exclusively contributing to the elevated acrolein concentrations measured. Rather, all industrial point sources and vehicle emissions in the area emitting acrolein likely contribute to the levels measured.

Accordingly, the Ministry of the Environment has decided to undertake additional measurements in the same vicinity of sampling undertaken in 2007 to better characterize VOC concentrations and to identify likely sources. To isolate individual sources of the VOCs of concern, particularly acrolein sources, it will be necessary for future monitoring to undertake short-term sampling (½ hour) at the point of impingement under conditions when wind speed and direction remain relatively constant. Although 2007 levels of

acrylonitrile and dichloromethane appear to be reduced from 2004 levels, these compounds should continue to be monitored to discount the possibility of missing possible higher concentrations by random sampling or being an artifact of the sampling locations chosen.

The results of this study have been shared with the Region of Halton and Region of Peel Public Health Units for their information.

Appendix 1: Summary of sampling conditions for Acrolein, Acrylonitrile and Dichloromethane in Ambient Air – south Mississauga, Summer 2007.

June 14 2007 - Acrolein/Acrylonitrile

					GPS Locations		Electrovaya Meteorological			Sampling Time			
Sample #	**Sample Location**	**Canister ID**	**Initial Vacuum (inch Hg)**	**Final Vacuum (inch Hg)**	**X**	**Y**	**MET (WS) m/s**	**MET (WD) deg**	**Ambient Temp.**	**Started (EST)**	**Finished (EST)**	**Elapsed Time (h:min)**	**NOTES**
1	2255 Royal Windsor	EPS 216	-30	-4.5	610288	4817896	2.1	256.5	24.1	14/06/2007 10:15	15/06/2007 10:15	24.00	
2	2509 Royal Windsor	MOE 024	-29	-5.5	609897	4817397	2.1	256.5	24.1	14/06/2007 10:20	15/06/2007 10:20	24.00	
3	2645 Royal Windsor	MOE 009	-28.5	-4	609661	4817076	2.1	256.5	24.1	14/06/2007 10:24	15/06/2007 10:25	24.01	

Notes: No Met Data, only 2 hours during the 24-hour period is available (met data listed above is from 9:00 am)

June 26-27 2007 - Acrolein/Acrylonitrile

					GPS Locations		Wind Parameter			Sampling Time			
Sample #	**Sample Location**	**Canister ID**	**Initial Vacuum (inch Hg)**	**Final Vacuum (inch Hg)**	**X**	**Y**	**MET (WS) Km/h**	**MET (WD) deg**	**Field Notes**	**Started (EST)**	**Finished (EST)**	**Elapsed Time (h:min)**	**NOTES**
1	2255 Royal Windsor	MOE 022	-28	-5.5	610288	4817896	None	None	None	2007/06/26 10.35	27/06/2007 10:35	24.00	
2	2509 Royal Windsor	MOE 001	-30	-7.2	609897	4817397	None	None	None	2007/06/26 10.40	27/06/2007 10:40	24.00	
3	2645 Royal Windsor	MOE 019	-30	-5.5	609661	4817076	None	None	None	2007/06/26 10.45	27/06/2007 10:45	24.00	

Notes: No Met Data

August 28-29 /2007 -
Acrolein/Acrylonitrile

					GPS Locations		Wind Parameter			Sampling Time			
Sample #	**Sample Location**	**Canister ID**	**Initial Vacuum (inch Hg)**	**Final Vacuum (inch Hg)**	**X**	**Y**	**MET (WS) Km/h**	**MET (WD) deg**	**Field Notes**	**Started (EST)**	**Finished (EST)**	**Elapsed Time (h:min)**	**NOTES**
1	2255 Royal Windsor	MOE009	-29	-5	610288	4817896				28/08/2007 16:27	29/08/2007 16:04	23.37	
2	2509 Royal Windsor	MOE016	-29	-7	609897	4817397				28/08/2007 16:44	29/08/2007 16:16	23.32	
3	2645 Royal Windsor	MOE006	-30	-5	609661	4817076				28/08/2007 16:50	29/08/2007 16:25	23.35	
	Notes	No Met Data											

September 20-21/20074-
Acrolein/Acrylonitrile

					GPS Locations		Wind Parameter			Sampling Time			
Sample #	**Sample Location**	**Canister ID**	**Initial Vacuum (inch Hg)**	**Final Vacuum (inch Hg)**	**X**	**Y**	**MET (WS) Km/h**	**MET (WD) deg**	**Field Notes**	**Started (EST)**	**Finished (EST)**	**Elapsed Time (h:min)**	**NOTES**
1	2255 Royal Windsor	MOE013	-29	-3	610288	4817896				20/09/2007 11:16	21/09/2007 11:06	23.50	
2	2509 Royal Windsor	MOE001	-29	-6	609897	4817397				20/09/2007 10:52	21/09/2007 10:45	23.53	
3	2645 Royal Windsor	MOE015	-30	-4	609661	4817076				20/09/2007 11:04	21/09/2007 10:55	23.51	
	Notes	No Met Data											

APPENDIX

K CUMULATIVE HUMAN HEALTH ASSESSMENT

SLATE ASSET MANAGEMENT L.P.

HUMAN HEALTH ASSESSMENT

CLARKSON TRANSIT STATION AREA AIR QUALITY STUDY

DECEMBER 09, 2022

HUMAN HEALTH ASSESSMENT

CLARKSON TRANSIT STATION AREA

SLATE ASSET MANAGEMENT L.P.

PROJECT NO.: 201-06851-00
DATE: DECEMBER 09, 2022

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/M³)						
	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 µg/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 µg/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.

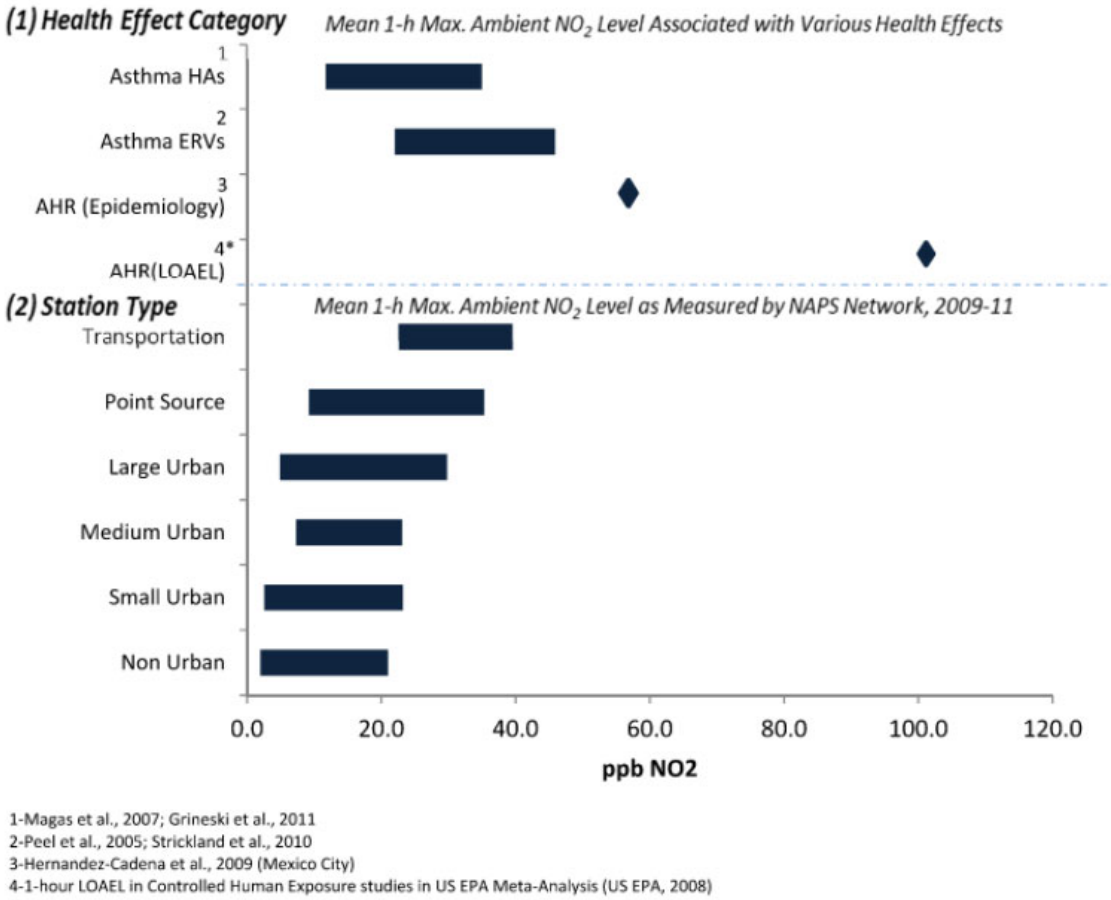
Effects of NO_2 on reproduction in humans are not known. IARC and US EPA have not classified nitrogen oxides for potential carcinogenicity. Nitrogen oxides have caused changes in the genetic material of animal cells, but it is not known if these can cause developmental effects in humans.

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.

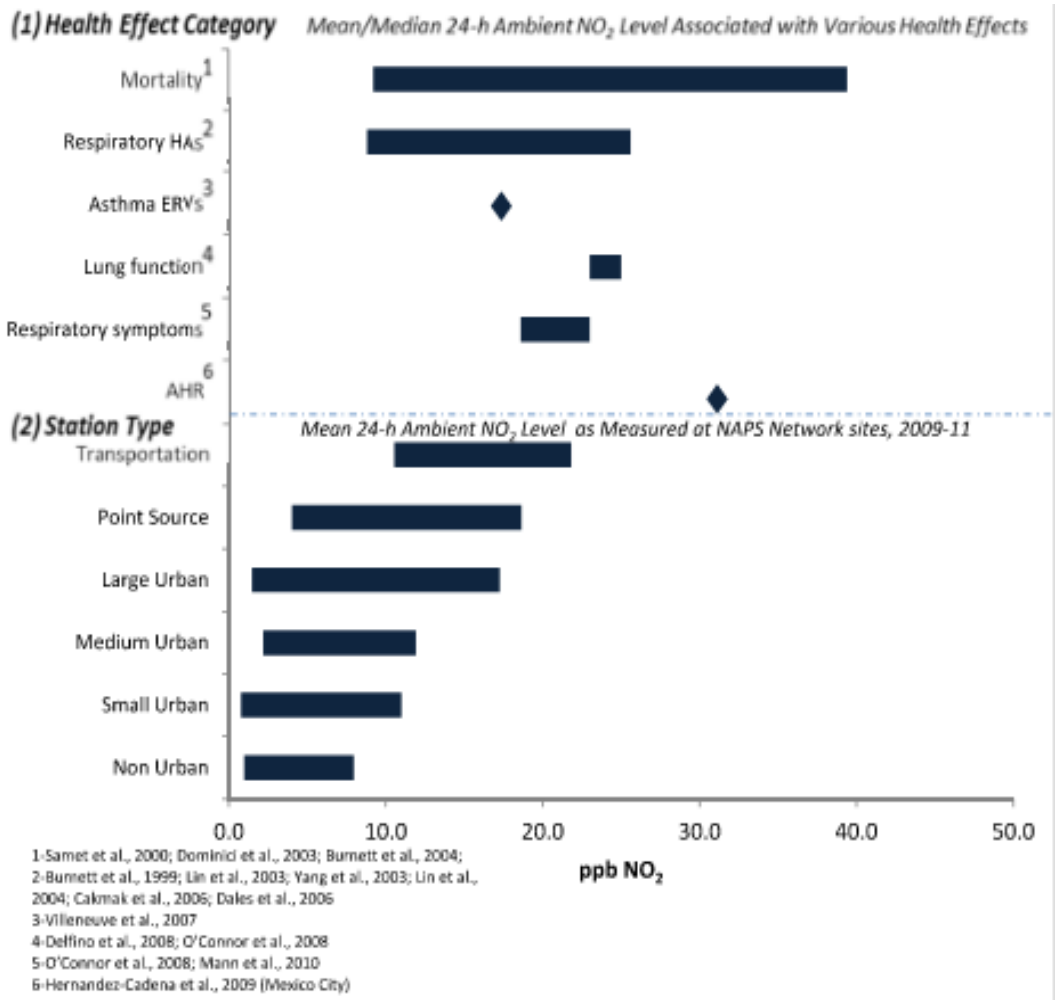
Health Canada (2016) presented the analyses in **Figure 4-1** for the daily 1-hour max NO_2, in **Figure 4-2** for the 24-hour average NO_2, and in **Figure 4-3** for NO_2 as the long-term (annual/multi-year) average. For each figure, the top panel presents the mean or median NO_2 levels associated with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

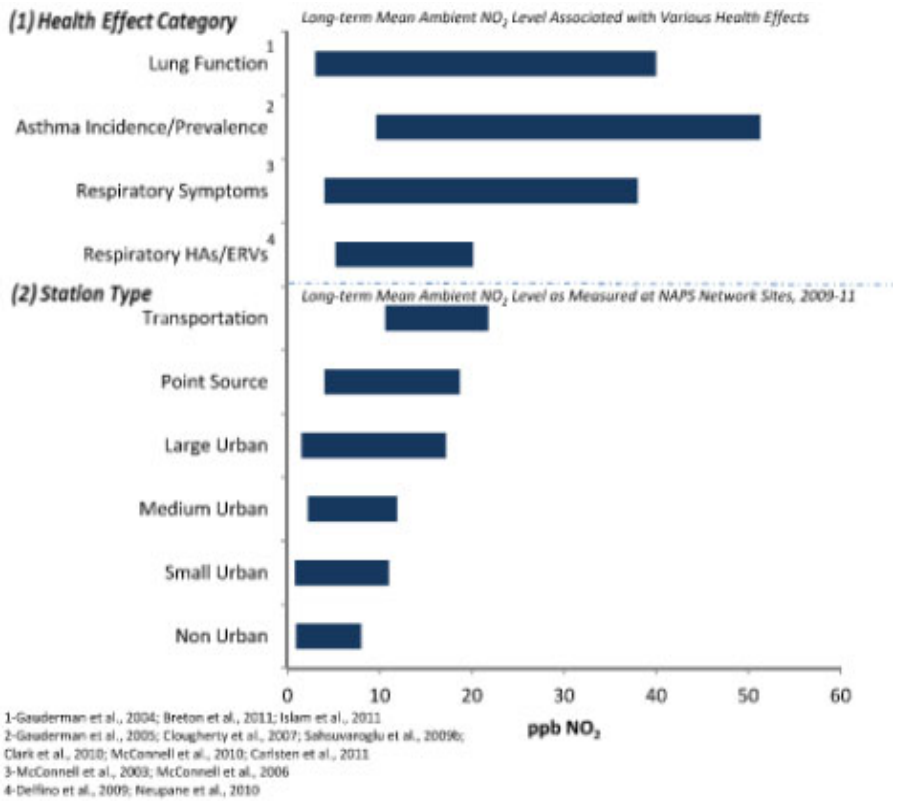
Notes:
HA – hospital admissions
ERV – emergency room visits
AHR – airway hyper-responsiveness

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/m³ (1) (carcinogenic); 30 µg/m³ (non-carcinogenic)	Health Canada (2021), TCEQ (2015) and US EPA (2003)	**Carcinogenic** **Protection against leukemia, mainly acute myelogenous leukemia** This TRV (4.5 µg/m³) 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/m³ 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/m³	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/m³ 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/m³(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/m³ by linear extrapolation from a $BMCL_{10}$ of 0.16 mg/m³ for the occurrence of upper respiratory and upper digestive tract tumors in male hamsters chronically exposed by inhalation.
NO_2	Annual	23 µg/m³	Health Canada (2016)	**Protection of respiratory morbidity** This TRV (23 µg/m³) 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/m³	WHO (2005)	**Protection against excess mortality** This TRV (10 µg/m³) 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/m³ (based on an incremental lifetime cancer risk of 1-in-100,000). This value was converted to 0.45 µg/m³ 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/m³ 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.

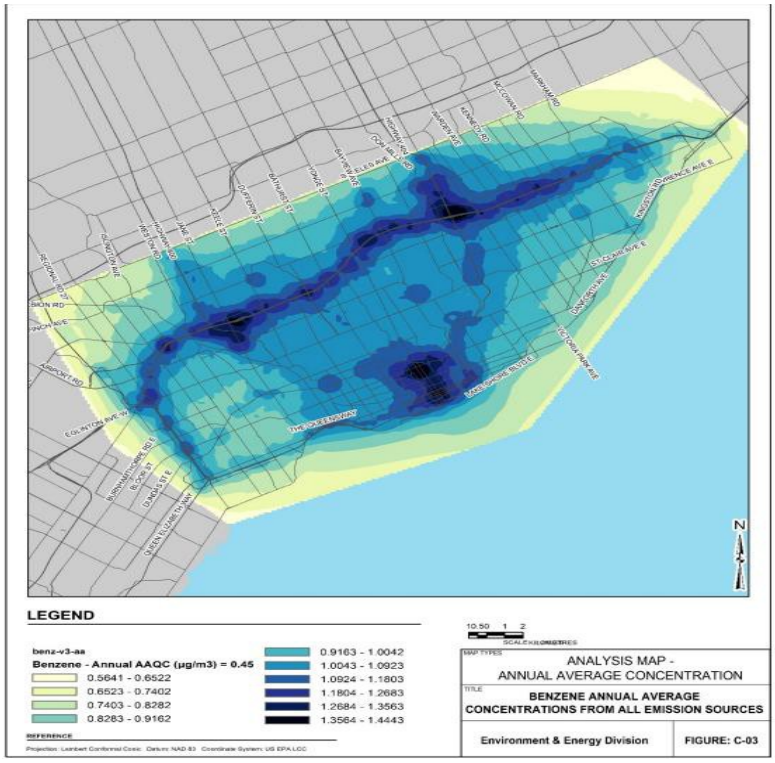


Figure 5-1 **Toronto Modelled Annual Benzene Concentrations (2012), from City of Toronto, 2017**

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.

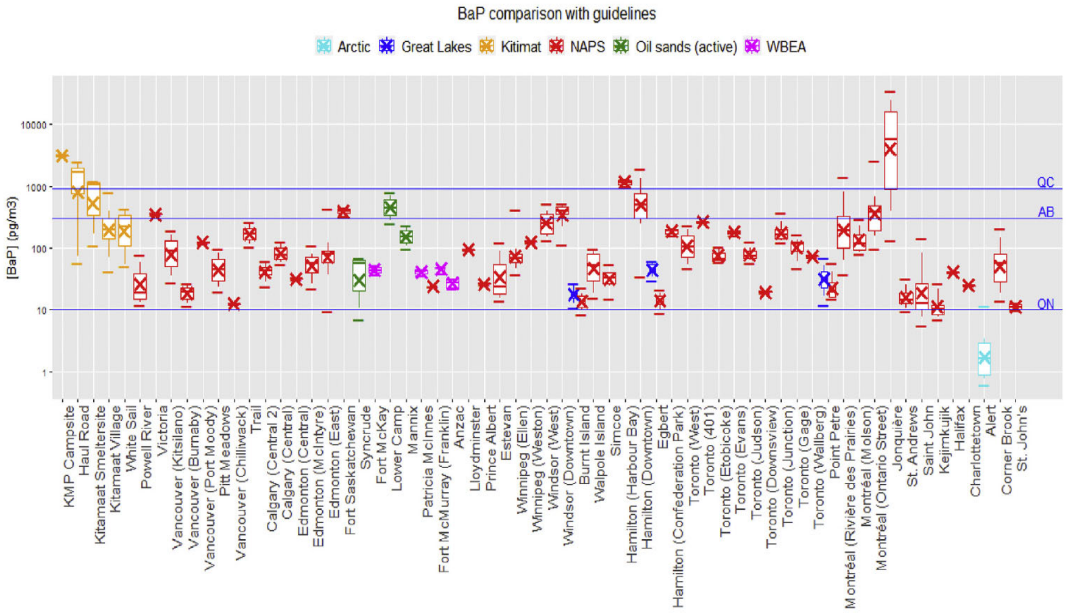


Figure 5-2 **Measured range of annual average benzo(a)pyrene concentrations (pg m^{-3}). Annual average ambient air guidelines from the provinces of Ontario, Alberta, and Quebec are depicted as horizontal blue lines.**

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.

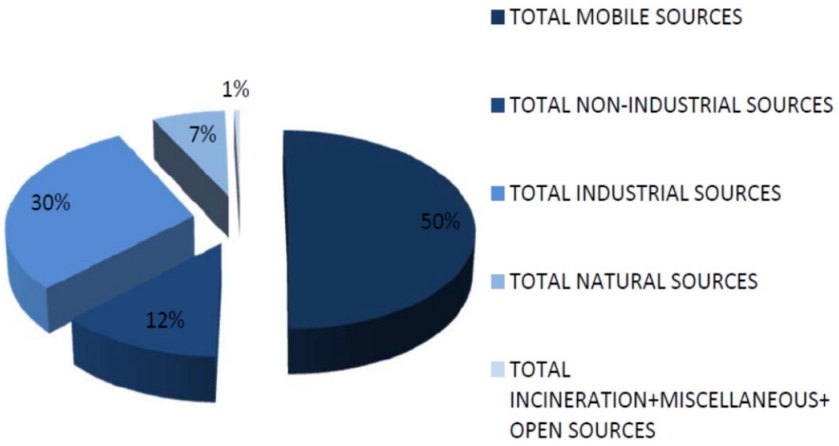


Figure 5-3 2011 NPRI NO_x emissions by broad source category (From NPRI, https://www.ec.gc.ca/inrp-npri/)

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.

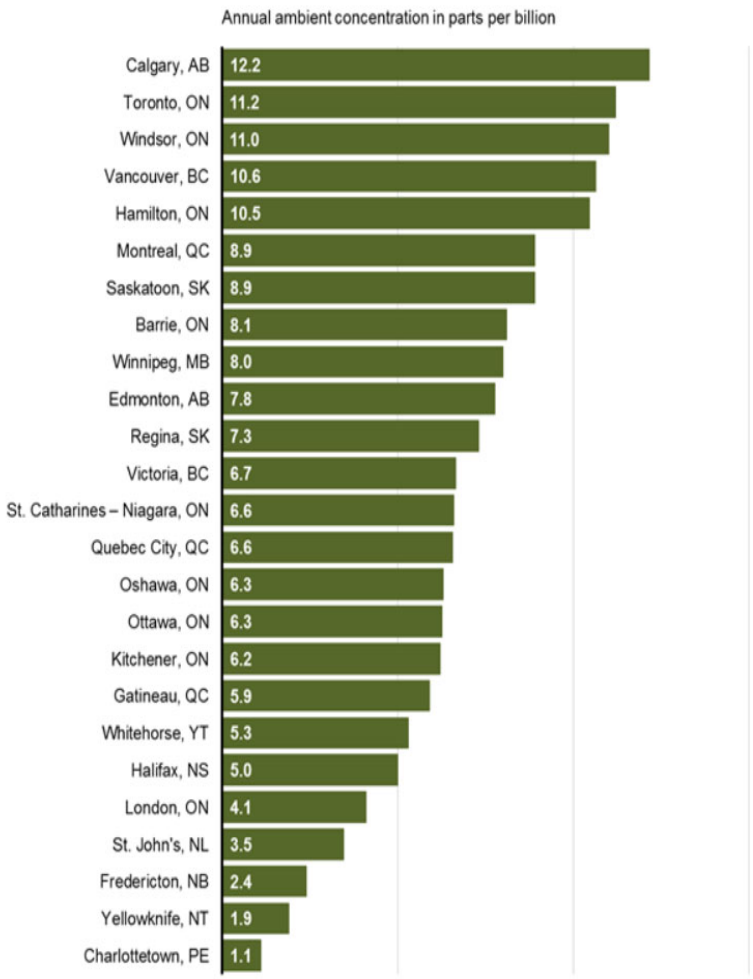


Figure 5-4 **Average NO_2 concentrations in selected Canadian urban areas (2016) (From Environment Canada and Climate Change (ECCC), https://www.canada.ca/en/environment-climate-change/services/environmental-indicators/air-quality.html#NO2-average)**

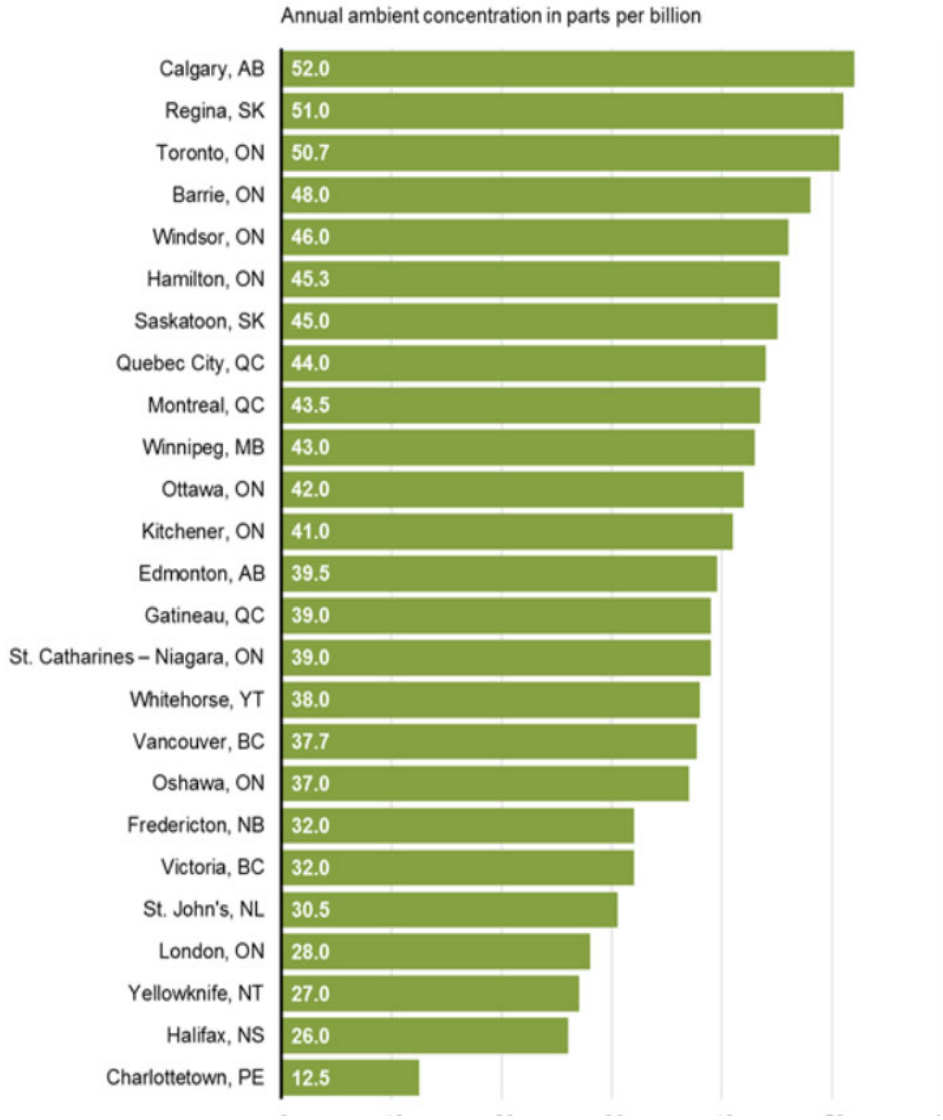


Figure 5-5 **Peak NO_2 concentrations in selected Canadian urban areas (From Environment Canada and Climate Change (ECCC), https://www.canada.ca/en/environment-climate-change/services/environmental-indicators/air-quality.html#NO2-average)**

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.

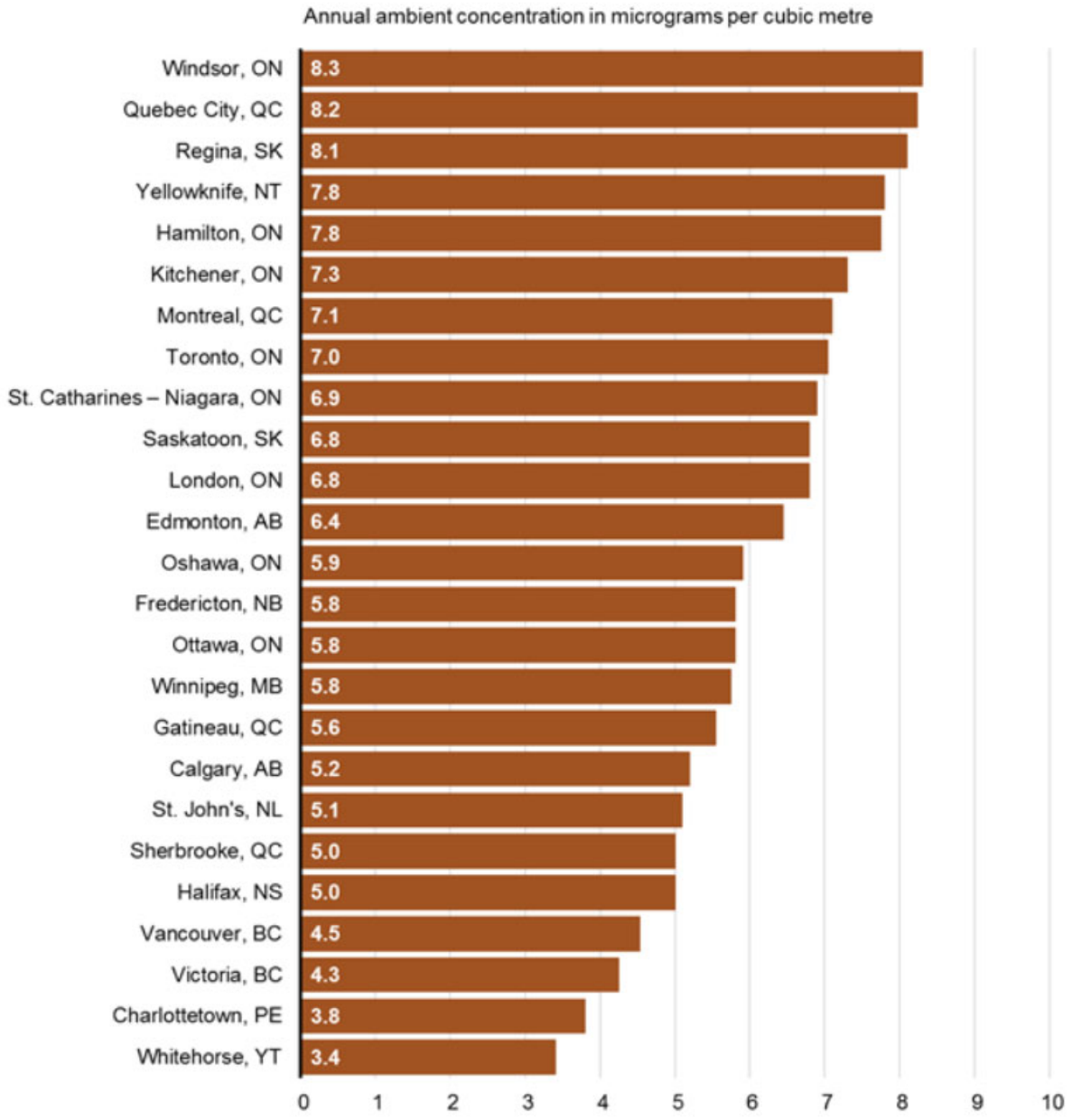


Figure 5-6 **Average $PM_{2.5}$ concentrations in selected Canadian urban areas (From Environment Canada and Climate Change (ECCC), Air quality - Canada.ca)**

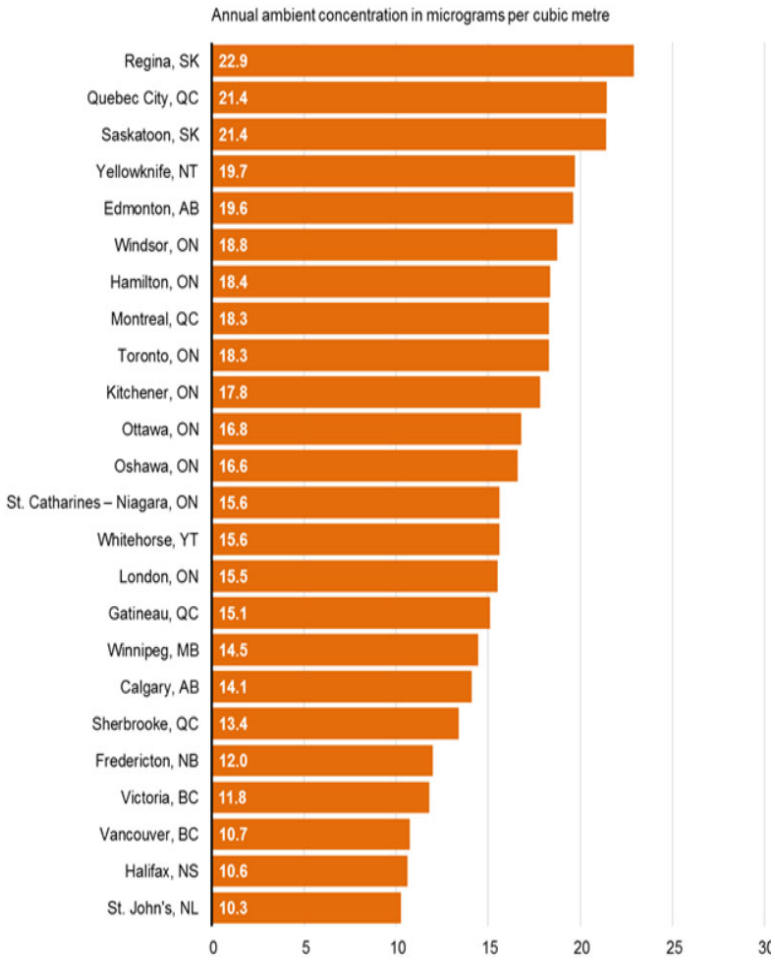


Figure 5-7 **Peak $PM_{2.5}$ concentrations in selected Canadian urban areas (From Environment Canada and Climate Change (ECCC), Air quality - Canada.ca)**

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 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.

Nitrogen Dioxide:

- A predicted cumulative 1-hr NO_2 concentration of 90 µg/m^3 is greater than the TRV of 79 µg/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 µg/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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APPENDIX

L MITIGATION OPTIONS

MEMO

TO: Slate Asset Management L.P.

FROM: WSP Canada Inc.

SUBJECT: **Mitigation Recommendations, Clarkson Transit Station Area**

DATE: **August 26, 2022, revised February 15, 2023**

Based on the WSP Air Study, mitigation is not required at the proposed development; however, mitigation recommendations have been included to improve indoor air quality. This memorandum outlines mitigation recommendations to improve indoor air quality based on the results of two WSP Canada Inc. (WSP) reports:

- Clarkson Transit Station Area Air Quality Study, Monitoring and Dispersion Modelling Report, January 23, 2023 (WSP Air Study); and,

- Human Health Assessment, Clarkson Transit Station Area (TSA) Study, December 9, 2022 (WSP Health Assessment)

The focus of this mitigation memo is to examine the potential for future building construction with appropriate HVAC and air filtration systems to reduce ingress of chemicals of concern into indoor air. Mitigation could be accomplished by adjusting where intake air is drawn into the suites. The modelling completed as part of the WSP Air Quality Study examined concentrations at receptors at various heights at the property boundary. Predicting the concentrations at receptors at the property line at various heights is conservative since the contaminants of concern (COCs) are traffic-related air pollution (TRAP):

- Particulate matter less than 10 µm (PM_{10});
- Particulate matter less than 2.5 µm ($PM_{2.5}$);
- Oxides of Nitrogen (NO_X);
- Acrolein;
- Benzene; and,
- Benzo(a)pyrene [B(a)P].

Table 1 attached displays the model results for the primary contaminants of concern (COCs) listed above and the equivalent time-weighted average Ambient Air Quality Criteria (AAQC). None of the predicted model concentrations result in a value that is elevated compared to the respective AAQC.

Table 2 uses the percentage change of the modelled concentrations in **Table 1** with height and modifies the baseline ambient monitoring concentrations to show their equivalent change with height. This was performed as a direct percentage change since the ambient conditions would change in a similar proportion to the modelled fractions. Correcting the ambient concentrations for

82 Lancaster Street West
Kitchener, ON
Canada N2K 1M3

F: +1 519 743-8778
wsp.com

height was performed assuming that ambient data is collected from an equivalent height as the modelled 4.3 m receptor height, following best practices. **Table 2** demonstrates the background ambient concentration variability with height, and that for all COCs except B(a)P, impacts are not elevated compared to the AAQC at 17.2 m and above.
Table 3 conservatively adds together values from **Table 1** (modelled concentration) and **Table 2** (ambient concentrations). Adding together the modelled results and ambient results is extremely conservative and usually only conducted for Environmental Assessments (EAs) and Transit Project Assessment Process (TPAP) work. In EAs and TPAPs, a future scenario is often examined to show the project; such as highway expansion or rail improvements, has a net positive impact compared to alternatives. By examining cumulative impacts, **Table 3** effectively takes the known sources modelled (**Table 1**) and adds them to all known and unknown sources (**Table 2**). In this case the cumulative impacts show that except for acrolein and B(a)P, there are no concentrations elevated compared to the AAQC at 30.1 m and above. Background concentrations of acrolein and benzo(a)pyrene are elevated compared to the AAQC values; however, B(a)P is elevated anywhere a development were to proceed in an urban area.

Based on the data assessed in this memo, the following recommendations are presented:

- **Local Air Intakes**: If air intakes are designed to be located in each suite, then for any suites below the fourth floor (12.9 m) filters to control particulates ($PM_{2.5}$ and PM_{10}) impregnated with carbon to control benzene could be utilized to improve indoor air quality. Percent reductions required can be calculated from **Table 3**. Filters require ongoing maintenance and monitoring per manufacturer specifications, which generally require replacement after a specified duration of time. It should be noted that mitigation for particulate will also incidentally reduce the concentration of B(a)P since B(a)P binds to particulate and may be partially mitigated through filtration.

- **Monitoring**: Since **Table 3** represents a very conservative approach then it is recommended that a method of ambient monitoring be incorporated to ensure the controls of a local air intake design are working, or even required.

Ducted Air Intakes: An alternative to filtering local air intakes and monitoring could be to have a centralized air intake system ducted from above 30.1 m for any suites located below this level.

- **NO_x**: No additional controls are recommended for NOx given the level of conservatism in the Air Quality Study and as the measured values (baseline) are well below for ambient air quality criteria for NOx as NO_2. The baseline already includes both industry, rail, and roadways emissions. Railway emissions dominated the predicted modelling concentration and are conservative as no reductions have been included for the electrification of the GO Stations. Therefore, baseline combined with modelling is an overpredicting of the concentrations at the Proposed Development and the potential need for mitigation.

- It is recommended that the proponent conduct a detailed design of mitigation as part of the Design Process.

In addition to the recommendations, WSP identifies the following improvements noted for the Clarkson airshed:

- Ongoing MECP compliance for Industry; and,

- Metrolinx Regional Express Rail Lakeshore West line is expected to be electrified in the coming years (some trains will remain diesel, but the majority will be electrified).

Further improvements of air quality are expected based on the City of Mississauga's local initiatives that are ongoing to improve air quality and reduce greenhouse gases.