

**Health Assessment for the Cumulative Air  
Quality Modelling Study – Wards 30 and 32  
including the South Riverdale and The  
Beaches neighbourhoods**

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## Executive Summary

At the request of the Board of Health, the Toronto Environmental Office undertook an air modelling study for Wards 30 and 32 which includes the South Riverdale and The Beaches neighbourhoods of Toronto. The study modelled air concentrations for 30 pollutants which included the 25 priority substances in Toronto's Environmental Reporting and Disclosure Bylaw (ChemTRAC program) and 5 other air pollutants. The report of the air modelling study is located at <http://www.toronto.ca/teo/reports-resources.htm>.

This report presents the results for a air quality health assessment performed for Wards 30 and 32 based on the Toronto Environment Office modelled concentrations of air pollutants.

Air modelling studies typically compare the estimated levels of pollutants against air standards or health benchmarks to identify if the exposure to the pollutants could be at levels of concern in a specific area. For most of the substances modelled, the predicted ambient concentrations were below Ontario's Ambient Air Quality Criteria (AAQCs). The model predicted that levels of benzene, nitrogen dioxides, polyaromatic hydrocarbons (modelled as benzo[a]pyrene) and particulate matter (PM<sub>10</sub>) might exceed air quality objectives in some areas, some of the time. The modelling study showed that transportation is the largest local source of these pollutants.

The air quality modelling enabled identification of the proportion of ambient concentrations in Wards 30 and 32 attributable to sources outside of Toronto. The model estimates that on average, 39% of the air pollution (by weight) comes from the United States and 25% comes from other parts of Ontario. Of the 36% of air pollution that comes from Toronto sources, residential and commercial sources appear to be the most important, contributing about 18% to the total ambient concentrations while mobile sources contribute about 13%. Off-road mobile sources contribute a further 4%, and large industrial sources in Toronto (including those in Wards 30 and 32) also contribute about 4% to the total concentrations to these neighbourhoods.

As people are exposed to a mixture of pollutants it is useful to also consider the combined impacts of these pollutants, even when most are individually below levels of concern. The science for assessing the health impacts of mixtures of chemicals continues to evolve and there is no common approach to assessing combined exposures to the complete range of substances considered in this study. Therefore pollutants were grouped into three categories and the cumulative impact estimated for each group of substances separately. These categories were:

- 1) Toxic substances associated with non-cancer effects, for which there is a health threshold
- 2) Substances associated with cancer, and
- 3) Common air contaminants (CACs, or criteria air contaminants), which are mainly associated with premature death from cardiovascular and respiratory diseases.

The results of these separate cumulative assessments are described below.

### 1) Cumulative risk for non-cancer effects

In general, for non-cancer effects it is assumed that there is a threshold – a level below which exposure to the substance will have no adverse health impacts. The health benchmark is set at this level. Since substances have different levels of toxicity it is not possible to just add the modelled air concentrations to estimate the cumulative impacts; a common measure is needed. One such measure is the *hazard ratio*, which is obtained by dividing the exposure level with the health benchmark for each pollutant. For each substance this tells us what fraction of the health benchmark a person might be exposed to. If the hazard ratio is less than one, then a person or community is being exposed at a level which current knowledge suggests is not a concern. The hazard ratio was calculated for 22 substances with health benchmarks for non-cancer effects

The hazard ratio values for the individual non-carcinogenic substances are all much less than one; acrolein had the largest hazard ratio at 0.1. This confirms that there is little or no risk of adverse health effects from exposures to these substances individually. When the hazard ratios for the 22 pollutants were added together, the cumulative hazard index is 0.31; this is still well below one. This suggests that the combined exposure to these air pollutants do not pose a health risk for non-cancer effects.<sup>1</sup>

### 2) Cumulative risk of cancer

For carcinogens it is assumed that every amount of exposure has a risk of causing cancer. Toronto Public Health uses a concentration level in air that is associated with increasing cancer by one in one million over a lifetime as the health benchmark for a carcinogen. The risk of cancer for a single carcinogen is calculated by multiplying the level of the carcinogen in the air by its associated risk factor. Seven of the 19 carcinogens had modelled annual concentrations above the one in one million excess lifetime cancer risk benchmark in parts or the whole of the two wards. These were benzene, benzo[a]pyrene, 1,3-butadiene, chromium (VI), 1,4-dichlorobenzene, formaldehyde and tetrachloroethylene (perchloroethylene). With the exception of chromium and tetrachloroethylene, on and off-road vehicles are the largest local source of these carcinogens.

The estimated risk for each substance was added to give a total estimate of the risk. If the average annual risk is summed across all 19 carcinogenic substances, the average cumulative cancer risk in these two wards is 83 in one million. While 83 in a million is greater than the benchmark that TPH uses for individual cancer risk, the total risk is still

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<sup>1</sup> There are limitations to this approach. It assumes that the effect of the individual pollutants is in direct proportion to the level of exposure and the effect of each pollutant is additive. In some circumstances, this could overestimate the risk since it does not take into account that different pollutants affect different parts of the body and ignores the natural mechanism of the body to eliminate or detoxify these substances. At the same time, the approach could underestimate the risk since it does not take into account potential interactions between these pollutants that could increase the health impacts.

quite small. This total risk is around two percent of the overall cancer incidence rate in Toronto - about 400 per 100,000 in 2007 (Vital Statistics, 2007).

Chromium (VI), benzene, 1,3-butadiene, and benzo[a]pyrene account for most of this risk. While the cumulative risk was somewhat above the one in a million cancer risk benchmark in all parts of the two wards, only two areas had more elevated risks – one next to the Don Valley Parkway (DVP) and the other around an industrial area close to the Port Lands.

The largest part of the total cancer risk in these two wards comes from chromium (VI). The modelling shows that most of this chromium comes from sources outside Toronto and thus is a health risk that is likely common to other parts of the city. The elevated risk next to the DVP is mostly from 1,3-butadiene, benzene, and benzo[a]pyrene. As indicated above, these substances are mostly released from transportation sources. These are also the substances that contribute a large part of the overall cumulative cancer risk in the other parts of the study area. The Toronto Environment Office and the Ontario Ministry of the Environment have investigated the facility for which the modelling results indicate high exposure. The facility has taken steps to reduce its emissions.

### **3) Cumulative risk from common air contaminants (CACs)**

Our current knowledge of the health effects from the five common air pollutants (carbon monoxide, ozone, nitrogen dioxide, particulate matter (PM) and sulphur dioxide) shows that there is no threshold for these effects. Therefore, Toronto Public Health used an approach similar to the one used for carcinogens to estimate the cumulative risk from this group of pollutants. In this case, instead of excess cancer risk we used the estimate of excess risk of premature death to calculate the cumulative impact.

Annual average values were used for estimating percent excess risk of premature death, as they are most representative of chronic, long-term exposures. The common air pollutants have a cumulative excess risk of 8.9 percent (that is, they increase the overall mortality for respiratory and cardiovascular diseases by this amount). Fine particulate matter (PM<sub>2.5</sub>) and nitrogen dioxide are the pollutants that contribute most to this risk. This level of excess risk is similar to what has previously been calculated in the Burden of Illness in Toronto (Toronto Public Health, 2004).

Similar to the analysis for carcinogens, there is a higher risk in the area close to the DVP and industrial sources near the Port Lands. Nitrogen dioxide is the pollutant that accounts for most of the risk near the DVP, while fine particulate matter (PM<sub>2.5</sub>) was the pollutant associated with the higher risk around the point source. The change in the processes in the facility that have taken place since suggest that the facility has reduced its emissions of particulate matter.

### **4) Conclusions**

This health assessment indicates that many of the thirty air contaminants selected for this study, mainly the non-carcinogenic ones, occur below levels of concern to health in Wards 30 and 32 even when the combined exposure is taken into account. However, it is

possible that some carcinogens are present at levels above the one in one million excess cancer risk benchmark. Other pollutants such as ozone, nitrogen dioxides, and particulate matter are also found at levels that are known to have an adverse impact on health. For many substances of greatest concern, such as benzene, 1,3-butadiene, and nitrogen dioxides, the locally generated emissions are mainly from transportation sources. Therefore, it is important to continue efforts to reduce air pollution from both on and off-road transportation sources.

The study and modelling utilized for cumulative health effects has several limitations. It is difficult to compare the multiple health impacts into a single measure of health risk for the community. As the modelling is based on one year, 2006, the lifetime risk of diseases such as cancer are being estimated based on the air quality situation from one year. This assessment cannot account for past exposures from sources in the community that may contribute to current and future health problems.

This study is a novel way of assessing cumulative health risks from multiple pollutants for a specific neighbourhood within a large urban area. For the first time, the contribution of pollution from different geographic areas and sectors to health risks at the local level was assessed. The results aid in setting priorities and determining effective strategies for pollution prevention to reduce exposures and improve the health of Toronto residents. There is currently limited data available on the small commercial and industrial sources of air pollutants in the area of study. The data collected through the Environmental Reporting and Disclosure Bylaw (ChemTRAC program) will help improve future estimate of the cumulative exposure in these and other Toronto neighbourhoods and direct pollution prevention priorities in these communities.

## Background

Residents in the Toronto neighbourhoods of South Riverdale and the Beaches have long been concerned about the health and environmental impacts of pollutants in their community. Historically, the community was home to a heavy industrial presence. While many of the facilities in the area closed down by the end of the 1990s, the potential for cumulative impacts from current and past exposures to pollutants remains a question for area residents.

In 2001, plans to make major modifications to the Ashbridges Bay Treatment Plant (ABTP) combined with community concerns about the facility's emissions prompted a series of studies including a Community Health Status Study and Air Emissions Study (see [http://www.toronto.ca/health/hphe/abtp\\_emissions.htm](http://www.toronto.ca/health/hphe/abtp_emissions.htm)). The treatment plant is one of the large-scale facilities that still operates in the community. The air emission study was undertaken to provide a comprehensive picture of total air emissions from the treatment plant and to assess its impact on the surrounding South Riverdale and Beaches community. The emission study found that the planned modifications to the treatment plant would both reduce its impact on local emissions, and that all substances studied would meet regulated levels and health-based criteria by 2010.

However, the community and the researchers recognized that although the treatment plant is a large point source of emissions, it is not the only source of pollution in the community and its contribution to local pollution is relatively small when compared with existing levels of pollutants in the City's ambient air. As a result, in July 2005, the Toronto Board of Health requested that the City examine the air quality impact of emissions from all sources of air pollution in South Riverdale and the Beaches.

### *This Study*

In response to this request, the Toronto Environment Office (TEO) recently completed a sophisticated air quality modelling study to estimate and map concentrations of thirty substances for Wards 30 and 32 which include the neighbourhoods of South Riverdale and The Beaches. The goals of the study included characterizing how much of the air pollution comes from different sources such as industrial, residential/commercial, transportation, and biogenic sources. The study also estimated the proportion of pollution that comes from the United States, other parts of Ontario, and from within Toronto. Finally, combining all the sources the resulting concentrations for each substance at all receptor points were estimated and mapped to show how levels of each substance vary across the neighbourhood.

Toronto Public Health (TPH) used the estimates from the modelling to conduct a health assessment of the findings to respond to the community's interest in understanding cumulative impacts from multiple pollutants. A cumulative health assessment approach can help to answer important questions related to the potential health effects of neighbourhood-level air pollution, including:

- Is air pollution present in the neighbourhood at levels that are a health concern?
- What are the patterns of exposure to air pollution?
- Which sources contribute most to potential health impacts?
- Which air pollutants should be priorities for reduction?

Ultimately, the health assessment should facilitate identification of problem chemicals and support development, implementation and measurement of risk reduction strategies.

This document outlines the selection of substances included in the project, and describes the approach taken to assess the health implications of the modelled concentrations. While it provides some background information about air quality modelling, the main focus is on the methods and results from the health assessment. More detailed information about the air quality modelling methods and findings is available in the technical report entitled, *An All Sources Cumulative Air Quality Impact Study of South Riverdale - Leslieville – Beaches*, available from the Toronto Environment Office (<http://www.toronto.ca/teo/reports-resources.htm>).

## Assessing Air Quality

Urban air pollution is made up of a complex mix of hundreds of substances. The potential for health risk from any airborne pollutant depends on (i) people's exposure to the substance, and (ii) its toxicity. Exposure refers to the amount of contact people have with a pollutant, and for air pollution it is often approximated using ambient pollutant concentrations. Toxicity refers to the type and degree of potential harm posed by the pollutant. Exposures to two different substances at the same concentration can result in different types of health effects (for example, impact on the respiratory system or impact on the nervous system). Similarly, exposure to two different substances at the same concentrations could affect the same body system, but at different severity (for example, nasal irritation or extended inflammation of the respiratory system). Pollutants which are of most concern are typically those that are linked to severe health outcomes and those that are linked to health outcomes which affect a large number of people.

The air pollution mix in cities includes common air contaminants such as particulate matter, nitrogen oxides, and ozone that are linked to cardiovascular and respiratory effects (Brunekreef and Holgate, 2002; Dockery et al., 1993; Pope et al., 2002; Samet 2000; Schwartz, 1993). The air pollution mixture also includes low levels of air toxics – substances for which long-term chronic exposure is associated with development of cancer or other serious health effects, such as reproductive effects or birth defects (Caldwell, 1998; Cohen, 2000; Environment Canada, 2009a; US EPA, 2009). Examples of air toxics include benzene, tetrachloroethylene, and lead.

In Toronto, the common air contaminants contribute to approximately 1,700 premature deaths and 6,000 hospitalisations each year (Toronto Public Health, 2004). In children, these pollutants are also linked to illness such as acute bronchitis and asthma. They

contribute to lost work days and diminished quality of life, especially for vulnerable populations including people already suffering from chronic illness.

While the burden of illness from air toxics is unknown, reviews of local data suggest that some air toxics are likely to be present in Toronto's air at levels that pose a risk to health (Toronto Public Health 2002, 2008). At-risk populations include children, seniors, and individuals with pre-existing disease (Schwartz 2004; Samet and Krewski 2007; Krewski and Rainham 2007).

The concentration of air pollutants at any urban location varies from day-to-day, and from one place to the other. Factors that affect neighbourhood pollution levels include the types of local sources such as cars, homes, and businesses, as well as pollution coming from other locations. The weather can affect whether pollution stays in the air for a short or long time, and the time of day can also affect pollution levels, since some pollution is more likely to be emitted at certain times (for example, emissions from cars are greater during the rush hour than in the middle of the night).

There are two tools that are commonly used to evaluate air quality: **monitoring** and **modelling**.

In air quality **monitoring**, specialized equipment is used to measure actual concentrations of pollutants. Depending on the equipment, measurement may be continuous, or may occur at specified time intervals. In Toronto, measurements for some pollutants are taken all the time. For others, the levels are only measured once every six days.

Some benefits of air monitoring are that it allows investigation of trends in air quality over time and it provides information about actual concentrations at a specific location. However, air monitoring equipment is expensive to purchase, and must be maintained regularly. As well, most air quality monitors measure air quality at only one location. In Toronto, there are four monitoring stations that measure the most common air pollutants. They do not provide information about air pollution concentrations at other locations. Finally, most of the time, air monitoring data does not allow to identify the place of origin of the air pollutants.

Air quality **modelling** uses information about known sources, typical emissions rates, the weather, and geography to predict the concentrations of pollutants for a place of interest. The predictions of air quality models are estimated based on complex calculations done by computers. Air quality modelling results can create a continuous "picture" showing expected air quality everywhere in a community, and can provide estimates about what sources contribute to the levels found in a community: for example, how much is from cars, and how much is from industry. As well, models can be used to see what might happen to air quality if a new source is added to the community, or if an existing source is eliminated. Finally, modelling is less expensive than monitoring.

However, modelling requires extensive amounts of detailed data about air pollution sources and weather patterns, and modelling predictions are only as good as the data that

is used as input. If there are problems with the data, or data are not available, this can affect the model's accuracy.

Air quality monitoring and modelling are very different from each other, but taken together, the information from these tools can be very powerful. Air quality monitors in an area being modelled can be used to check air quality model results. If the models are good at predicting what happened in the past and what is happening now at the monitor's location, they should be good at predicting what is happening at other places in the neighbourhood. Air quality models are becoming increasingly reliable and sophisticated, and they facilitate analyses that monitoring data cannot do. For example they can help to identify which sources are responsible for most of the pollution in a community. They also can predict what would happen to local air quality if air pollution emissions changed for some reason.

### ***Modelling Approach***

Air pollution in any neighbourhood comes from a variety of sources, some of which are local. Others may be very far away: emissions from parts of the United States are known to affect air quality in Southern Ontario. In order to ensure that the impact of transboundary sources was fully captured in the model, information about emissions from large areas of the U.S. and Ontario was included in the model.

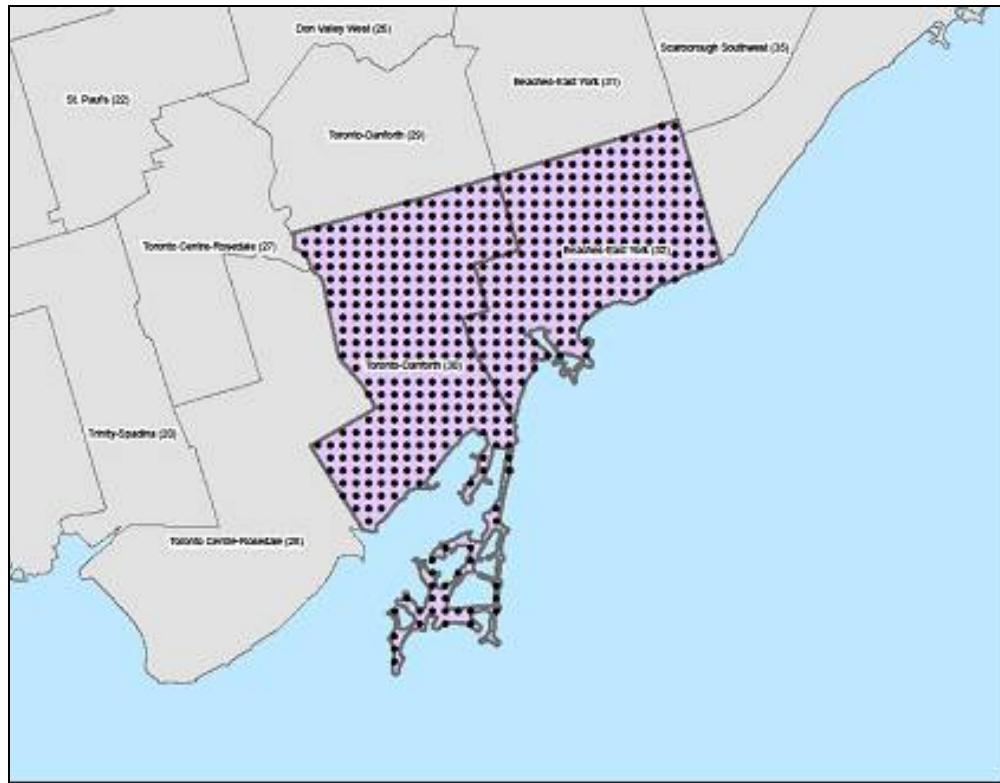
Each substance was modelled for 551 points in the studied area (Figure 1), which created a tight grid of estimated concentrations. Concentrations were estimated for points every 100-200 metres. With such a tight grid, it is possible to see variations in pollution concentrations across the neighbourhood. The model was used to estimate the average annual concentration and the maximum 24-hour concentration of each substance at each of the points.

Under the direction of TEO, consultants designed an air quality modeling approach that will be easily transferable to other neighbourhoods within the City of Toronto. This will reduce significantly the resources that would be required to perform similar modelling for other Toronto neighbourhoods.

Because of resource limitations that prevent modelling of hundreds of substances, this project modelled concentrations for 30 substances. It was important to narrow down the selection to those of greatest health concern. When deciding on which pollutants to model, the population health needs of the City as a whole was considered.

Next section describes how the selection of substances for the modelling was done.

**Figure 1: Illustration of 551 points evenly distributed across Wards 30 and 32. The concentration of each substance was modelled at each point.**



### **Selection of Air Contaminants to be modelled**

The selection of pollutants to be modelled included several considerations:

First, in 2008, Toronto Public Health established a list of 25 substances that are a priority concern for Toronto which were included in the Environmental Reporting and Disclosure bylaw (Toronto Public Health, 2008). This list includes air toxics as well as several criteria air contaminants (CACs). Toronto Public Health had two approaches to identify substances of potential concern in Toronto Air:

- **Prioritize estimated emissions data using a health-based ranking scheme.** Using estimates of emissions, TPH applied a ranking scheme known as Toxicity Equivalence Potential (TEP) (Hertwich et al., 2001). The method uses estimates of the amount of a substance released, the potential for human exposure, and the toxicity of the substance to calculate a relative risk score for each substance.
- **Identifying chemicals in Toronto's air that exceed health-based benchmarks.** Data on air quality were obtained from Environment Canada and the Ontario

Ministry of the Environment. The air quality data were used to identify substances in Toronto's air at levels that may cause adverse health effects. This was determined by comparing levels measured in our air with reference levels from the California Environmental Protection Agency, the Ontario Ministry of the Environment, and the New Jersey Department of Environmental Protection.

The resulting list of 25 substances underlies the "Priority Air Contaminants" included in the cumulative air modelling study. An added advantage of selecting these substances is that the Environmental Reporting and Disclosure bylaw will collect information on smaller sources of these substances in Toronto. This will allow more accurate modelling of emissions of these priority air pollutants in the Toronto.

Second, Toronto Public Health had previously estimated the burden of illness arising from exposure to the common air contaminants (CACs) (Toronto Public Health, 2004, 2007). While PM<sub>2.5</sub> and NO<sub>x</sub> were already among the substances identified as a priority concern for Toronto, not all of the CACs that were included in previous burden of illness studies were on the list. Given the weight of evidence that CACs can cause adverse health effects, and the research indicating that current levels of these substances affect the health of Toronto residents, all CACs are included in the modelling study. This added the following substances to the list of "priority air contaminants" in this study:

1. PM<sub>10</sub> (includes PM<sub>2.5</sub>)
2. O<sub>3</sub>
3. SO<sub>x</sub>
4. CO

Third, the substances included in the 2005 Ashbridges Treatment Plant (ABTP) air emissions study were also considered for inclusion. Some of those substances were already included on the list of Priority Air Contaminants. Those that remained were not associated with a health risk in the original study, and were excluded from the list of Priority Air Contaminants as there was no evidence that the substances would enter the neighbourhood from either local emissions or from "transboundary emissions" sources.

Finally, some minor adjustments were made to the list of "Priority Air Contaminants" before it was finalized:

- Due to uncertainties in the ability to accurately capture modelling results for total volatile organic compounds (VOCs), the substance toluene was added to the list. Toluene is frequently used as a marker for VOCs, and is the most commonly released VOC in Ontario;
- NO<sub>x</sub> and SO<sub>x</sub> were replaced with NO<sub>2</sub> and SO<sub>2</sub>, as these substances are associated with defined health endpoints;
- Benzo[a]pyrene (B[a]P) was used as a marker for the mixture of substances known as polycyclic aromatic hydrocarbons (PAHs). B[a]P is believed to be among the most potent of the PAHs and has been used by the Ontario Ministry of Environment (MOE) to set the air quality standards for PAHs in Ontario. Similarly, the World Health Organization and the UK Expert Panel on Air Quality Standards (EPAQS) have considered B[a]P as a marker of the carcinogenic

potency of the PAHs mixture, when recommending their respective guidelines for PAHs in outdoor air.

This led to the following final list of “Priority Air Contaminants” (PACs) for this study:

1. Acetaldehyde	11. 1,2-Dichloroethane	21. PM <sub>2.5</sub>
2. Acrolein	12. Dichloromethane	22. Tetrachloroethylene
3. Benzene	13. Ethylene dibromide	23. Toluene
4. 1,3-Butadiene	14. Formaldehyde	24. Trichloroethylene
5. Cadmium	15. Lead	25. Vinyl Chloride
6. Carbon tetrachloride	16. Manganese	26. Carbon Monoxide (CO)
7. Chloroform	17. Mercury	27. PM <sub>10</sub>
8. Chloromethane <sup>1</sup>	18. Nickel compounds	28. Sulphur Dioxide
9. Chromium	19. Nitrogen Dioxide	29. VOC <sup>3</sup>
10. 1,4-Dichlorobenzene	20. B[a]P (as marker for PAHs) <sup>2</sup>	30. Ozone

<sup>1</sup> This compound was later removed from the Environmental Reporting and Disclosure Bylaw.

<sup>2</sup> B[a]P is modelled as a marker for the mixture of PAHs (not as PAH-equivalents).

<sup>3</sup> Both Natural and anthropogenic sources of VOCs were included in the modelling.

## Health Assessment

### Overview

This health assessment is intended to characterize the health impacts of the 30 priority air contaminants, listed before, when present at the modelled concentrations in Wards 30 and 32.

The modelled concentrations provide a spatial representation of ambient airborne levels of each substance, and provide spatial proxies for exposure. In reality, any individual resident's exposure to air pollution depends on their individual characteristics and behaviours. This may include how much time they typically spend outside, and where they go during the day for work, errands, and play. Despite these limitations, using ambient concentrations as a proxy for exposure is a well-established methodology in the risk assessment and epidemiological literature, and is viewed as providing a reasonable estimate of the magnitude of exposure likely to be experienced by a local resident.

In order to determine whether a given exposure is associated with a health risk, it can be compared with benchmark concentrations which have been established by reputable health agencies. Such benchmarks identify exposures that are associated with particular levels of risk, or are deemed to represent a “safe” level of exposure. Some health benchmarks are developed for chronic exposure – where exposures to low levels of a substance occur over long periods of time, perhaps a whole lifetime. Benchmarks can

also be established for acute exposures, where exposure occurs briefly, but to relatively high concentrations. Such health benchmarks are substance-specific and are often developed based on information from animal research, occupational studies, or epidemiological studies that used ambient concentrations as a measure of exposure.

The communities located in Wards 30 and 32 are especially interested in a cumulative assessment of the health risks. A cumulative assessment combines information about all of the pollutants modelled into summary estimates of health risk posed by “air pollution” as a whole.

As described above, the model estimated the average annual concentrations and maximum 24-hour average concentrations for each substance at each of 551 points within Wards 30 and 32. The annual average concentrations predicted at each point best reflect chronic exposure levels. The maximum 24-hour average concentrations are more representative of potential acute exposures.

In this study, the maximum 24-hour average concentrations also represent a type of “worst-case scenario”, because the value at each point (e.g., the most polluted day of the entire year at each location) was combined into a single dataset to represent the neighbourhood. Using maximum concentrations in this way is a health-protective approach, since it is unlikely that the maximum concentration for any substance would occur at all locations at the same time. It is also unlikely that the maximum concentration for all substances would occur simultaneously at any of the locations. Instead, the spatial profile of individual and total concentrations would be in constant flux. Therefore, when characterizing the risk associated with 24-hour maximum concentrations for individual substances, the risk should be viewed as the “worst-case scenario” for the community.

While it is tempting to explore cumulative impacts by directly comparing or summing the modelled concentrations for each substance, this is not an appropriate way to estimate cumulative health impacts. Each substance may induce health effects at a different concentration, such that some could be harmful at relatively low exposures, while others may not pose a significant threat unless concentrations are much higher. These differences in toxicity must be accounted for. The following section outlines the approach TPH used to conduct a cumulative health assessment.

### ***Methods for the Health Assessment***

Methods for characterizing the health risk from the carcinogens and non-carcinogens were developed based on work conducted in Portland Oregon, and in California (Department of Environmental Quality, 2006; Morello-Frosch et al., 2000).

## Calculating Cumulative Cancer Risk for Individuals

Cancer risks can be assessed using inhalation unit risk (IUR) values for each carcinogenic compound. The inhalation unit risk is the upper-bound excess lifetime cancer risk estimated to result from continuous exposure to an agent at a concentration of  $1 \mu\text{g}/\text{m}^3$  in air. Estimated cancer risks for each carcinogenic substance at each location can therefore be calculated using the formula

$$R_{ij} = C_{ij} * IUR_j$$

Where  $R_{ij}$  is the estimate of individual lifetime cancer risk from pollutant  $j$  at location  $i$ ,  $C_{ij}$  is the concentration of pollutant  $j$  at location  $i$  in  $\mu\text{g}/\text{m}^3$ , and  $IUR_j$  is the inhalation unit risk for a 70-year lifetime, for pollutant  $j$  in  $(\mu\text{g}/\text{m}^3)^{-1}$ .

The cancer risks of different air toxics are assumed to be additive, and can be summed together at each location to estimate a total individual lifetime cancer risk for that location:

$$\text{Cumulative cancer risk}_i = \sum_j R_{ij}$$

The calculated cumulative risk can then be compared to a benchmark to characterize the level of concern that may be associated with the cumulative risk. The definition of tolerable risk may vary by jurisdiction. Many jurisdictions, including the USEPA, use one in one million ( $10^{-6}$ ) as the maximum lifetime risk benchmark for carcinogen. Health Canada often uses as benchmark from one in one hundred thousand to one in one million. Typically, Toronto Public Health uses one in one million.

A common health-protective approach is to assume that most cancer types develop according to a similar multi-stage biological mechanism. Under this assumption, it makes sense to add the potential risk from different substances (which may be linked to different types of cancer) to estimate a cumulative cancer risk arising from multiple substances.

## Specific Substances and Weights

Table 1 lists the substances treated as carcinogens and provides inhalation unit risk values used for weighting.

The inhalation unit risk values for all substances except chloromethane are drawn from the California Office of Environmental Health Hazard Assessment (Cal OEHHA) database (OEHHA 2009). This database includes values for almost every pollutant listed in Table 1, and is regularly updated. The Cal OEHHA method is respected and viewed as being health-protective. The database does not include an inhalation unit risk value for chloromethane, so for this substance, an inhalation unit risk derived by the state of New Jersey was used (New Jersey Department of Environmental Protection 2008). Links to the sources for the values in Table 1 are provided in Appendix A.

The modelling was done for total chromium. However, the health risks arising from exposure to chromium VI and other forms of chromium are different. For example, while chromium VI is primarily associated with lung cancer, chromium III is associated with impaired lung function and irritation. Therefore, when estimating the risk from the predicted chromium concentrations, a weight should be applied to account for the proportion that is likely to be chromium VI.

While the proportion of chromium VI and chromium III is likely to vary by emission source, measurements of chromium present in ambient air can be used to estimate the typical proportions that might be expected to reach the population. Previously, TPH selected 15% to be a health protective, reasonable estimate<sup>2</sup> for the proportion of chromium that is likely to be chromium VI.

**Table 1: Carcinogens**

Air Pollutant	Cancer Inhalation Unit Risk (IUR) ( $\mu\text{g}/\text{m}^3$ ) <sup>-1</sup>
Acetaldehyde	$2.70 \times 10^{-6}$
Benzene	$2.90 \times 10^{-5}$
Benzo[a]Pyrene	$1.10 \times 10^{-3}$
1,3-Butadiene	$1.70 \times 10^{-4}$
Cadmium	$4.20 \times 10^{-3}$
Carbon tetrachloride	$4.20 \times 10^{-5}$
Chloroform	$5.30 \times 10^{-6}$
Chloromethane	$1.80 \times 10^{-6}$
Chromium VI	$1.50 \times 10^{-1}$
1,4-Dichlorobenzene	$1.10 \times 10^{-5}$
1,2-Dichloroethane (note: also called ethylene dichloride)	$2.10 \times 10^{-5}$
Dichloromethane (note: also known as methylene chloride)	$1.00 \times 10^{-6}$
Ethylene dibromide (note: also known as EDB)	$7.10 \times 10^{-5}$
Formaldehyde	$6.00 \times 10^{-6}$
Lead	$1.20 \times 10^{-5}$
Nickel compounds	$2.60 \times 10^{-4}$
Tetrachloroethylene (also known as perchloroethylene)	$5.90 \times 10^{-6}$
Trichloroethylene	$2.00 \times 10^{-6}$
Vinyl chloride	$7.80 \times 10^{-5}$

### Calculating cumulative non-cancer hazard

The hazard posed by air pollutants that exhibit non-cancer effects can be assessed using a reference concentration (RfC). The RfC is an estimate of a continuous inhalation

<sup>2</sup> CEPA 1994 states that 3 - 8 % of air concentrations of total chromium could be chromium VI (CEPA, 1994). The MMM estimates show that 13% of the total chromium air emissions are chromium VI (Toronto Public Health, 2007). Studies conducted in 1991-1993 concluded that 20% of the routinely monitored chromium in Southwestern Ontario was in the hexavalent form (Bell and Hipfner, 1997).

exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. To assess non-cancer risks, the hazard ratio (HR) for each pollutant is calculated at each location by dividing the modelled concentration by its reference concentration:

$$HR_{ij} = C_{ij}/RfC_j$$

Where  $HR_{ij}$  is the hazard ratio for pollutant  $j$  at location  $i$ ,  $C_{ij}$  is the concentration of pollutant  $j$  at location  $i$  in  $\mu\text{g}/\text{m}^3$ , and  $RfC_j$  is the reference concentration for pollutant  $j$  in  $\mu\text{g}/\text{m}^3$ .

An indicator of total non-cancer hazard can be calculated by summing together the hazard ratios for each non-carcinogen pollutant to derive a total hazard index:

$$HI_i = \sum_j HR_{ij}$$

There are no universal values for tolerable hazard ratios. The value of a tolerable hazard ratio depends upon the jurisdiction using it. Many agencies, including Health Canada and the USEPA, assume that a hazard ratio of less than one means that the concentration is less than the benchmark and so is not expected to be a concern for health. Health Canada also considers hazard ratios of 0.2 or less as not of concern for health for a single exposure pathway or when exposure is compared to the total acceptable daily intake. This reflects the possibility that hazard may accumulate from exposure through multiple exposure pathways.

### Specific Substances and Weights

Table 2 lists the substances treated as non-carcinogens in this study.

RfCs can be developed for various averaging time periods. The values in Table 2 represent chronic values wherever possible. There are five substances where RfCs are based on 24-hour averaging times because RfCs were unavailable for longer averaging times (denoted with “\*\*\*”). All others are based on annual averaging periods.

The chronic reference exposure levels used were drawn mainly from Cal OEHHA's database and existing or proposed ambient air quality criteria set by the Ontario Ministry of the Environment (MOE) (OEHHA 2008; Ontario Ministry of the Environment 2008). Both databases include values for almost every pollutant on our list, and are regularly updated (i.e., new values were adopted for acrolein, manganese, and mercury by Cal OEHHA in 2008, and the MOE adopted new standards for chromium on June 2011). Where an MOE annual ambient air quality criterion value for a non-carcinogen endpoint was lower than a California reference concentration, the MOE value was adopted. Otherwise, California's values were used.

**Table 2: Non-carcinogens (\*\* indicates that the RfC is based on a 24-hour averaging time. All others are based on annual averaging periods.)**

Air Pollutants	Non-cancer RfC <sup>1</sup> µg/m <sup>3</sup>
Acetaldehyde	140
Acrolein	0.35
Benzene	60
1,3-Butadiene	20
Cadmium	0.005
Carbon tetrachloride**	2.4
Chloroform	300
Chloromethane**	320
Chromium III **	0.5
Chromium VI	0.2
1,4-Dichlorobenzene**	95
1,2-Dichloroethane (note: also called ethylene dichloride)	400
Dichloromethane (note: also known as methylene chloride)	400
Ethylene dibromide (note: also known as EDB)	0.8
Formaldehyde	9
Lead **	0.5
Manganese	0.09
Mercury compounds	0.03
Nickel compounds	0.05
Tetrachloroethylene (note: also known as perchloroethylene)	35
Trichloroethylene	600
Toluene	300

<sup>1</sup> Links to the sources for these values are provided in Appendix A.

Some substances are classified as both carcinogens and non-carcinogens. These substances were included in the estimate of cumulative cancer risk as well as the hazard index calculation.

### Calculating Cumulative Risk from Common Air Contaminants (CACs)

Common air contaminants (CACs) are associated with multiple respiratory and cardiovascular outcomes. The risk from CACs was evaluated for an endpoint which is common to all CACs and for which rigorous risk coefficients exist: premature mortality. Using acute premature mortality may be akin to selecting a single most significant endpoint: it is the most severe outcome, and enables the risks associated with each individual CAC to be compared to the others. However, it should be recognized that CACs are associated with a significant burden of illness from respiratory and cardiovascular health conditions in Toronto.

The outcomes associated with CAC exposure are common, and would occur in the population even in the absence of CAC exposure. Thus, to characterize the risk posed by CACs, it is best to assess the additional or excess risk posed above baseline levels. The excess risk of premature mortality due to CAC exposure can be estimated based on the set of concentration response function (CRF) coefficients endorsed by Health Canada for

use in its Air Quality Benefits Assessment Tool (AQBAT). These CRF coefficients represent statistically derived estimates of the percent (%) excess health endpoint associated with a unit increase in the pollutant concentration (Health Canada, 2006). Estimated percent excess per capita risk for each CAC at each location can be calculated using the formula<sup>3</sup>:

$$R_{ijk} = \left( e^{(C_{ij}^{CRF})} - 1 \right) * 100$$

Where  $R_{ijk}$  is the estimate of percent excess per capita risk for a one unit increase in pollutant  $j$  at location  $i$  for outcome  $k$ ,  $C_{ij}$  is the concentration of pollutant  $j$  at location  $i$  in  $\mu\text{g}/\text{m}^3$ , and  $CRF_{ijk}$  is the coefficient representing percent excess per capita risk for outcome  $k$  associated with a unit increase in pollutant  $j$  (in applicable units). Overall, the approach is analogous to the approach used for calculating cumulative risk from carcinogens.

The percent excess per capita risks from four of the individual CACs ( $\text{NO}_2$ ,  $\text{O}_3$ ,  $\text{CO}$ , and  $\text{SO}_2$ ) are assumed to be additive, and can be summed together at each location to estimate a total percent excess individual lifetime risk for that location:

$$\text{Cumulative CAC risk}_{ik} = \sum_i R_{ijk}$$

As Table 3 suggests, the estimates for premature mortality for  $\text{PM}_{2.5}$  are based on chronic exposure, whereas those for the remaining CACs ( $\text{NO}_2$ ,  $\text{O}_3$ ,  $\text{CO}$ , and  $\text{SO}_2$ ) are for acute exposure. They are added together to derive a cumulative percent excess per capita risk under the assumption that over the long-term, the acute risk posed by  $\text{PM}_{2.5}$  each day reaches a steady-state, and can be adequately represented as an annual risk.

The approach described above is consistent with methods previously used by TPH to calculate the burden of illness from CACs (Toronto Public Health 2004, 2007). The above calculations generate percent excess per capita risk values, while the burden of illness reports applied percent excess per capita risks to current population incidence to estimate the number of people affected. For a small neighbourhood such as South Riverdale, the health outcome data is not reliable enough to enable a full burden of illness calculation.

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<sup>3</sup> The formula requires specifying a health outcome. Here, we specify acute premature mortality for  $\text{NO}_2$ ,  $\text{O}_3$ ,  $\text{CO}$ , and  $\text{SO}_2$ , and chronic premature mortality for  $\text{PM}_{2.5}$ . Premature mortality was assessed in epidemiological research that related air pollution levels with the number of people dying from non-traumatic causes. For premature mortality to be linked to air pollution exposure, the number of deaths must be higher than expected after exposure to elevated levels of air pollution, either in the long-term (for chronic exposures) or short-term (acute exposures).

## Specific Substances and Weights

The concentration response function coefficients for the CACs are regression coefficients drawn from Health Canada's Air Quality Benefits Assessment Tool (AQBAT) (Health Canada, 2006).

The substances treated as CACs are shown in Table 3. The CRF values were obtained directly from Health Canada.

**Table 3: Concentration Response Function coefficients for the CACs studied.**

Air Pollutants (CRF units)	CRF coefficient (concentration) <sup>-1</sup> (acute premature mortality) <sup>4</sup>	CRF coefficient (concentration) <sup>-1</sup> (chronic premature mortality)
NO <sub>2</sub> (ppb <sup>-1</sup> )	7.48 x 10 <sup>-4</sup>	
PM <sub>2.5</sub> (µg/m <sup>3</sup> ) <sup>-1</sup>		6.76 x 10 <sup>-3</sup>
Ozone (ppb <sup>-1</sup> )	8.39 x 10 <sup>-4</sup>	
CO (ppm <sup>-1</sup> )	1.90 x 10 <sup>-3</sup>	
SO <sub>2</sub> (ppb <sup>-1</sup> )	4.59 x 10 <sup>-4</sup>	

<sup>4</sup> While the CRFs for NO<sub>2</sub>, CO, PM<sub>2.5</sub>, and SO<sub>2</sub> are based on 24-hour averaging times, the CRF for O<sub>3</sub> is based on a 1-hour averaging time

PM<sub>10</sub> and total VOCs were not included in this analysis. This is to prevent double-counting when estimating cumulative risk. PM<sub>10</sub> includes PM<sub>2.5</sub>, and there is general consensus now that of the two measures for PM, PM<sub>2.5</sub> is the best indicator of risk and the best target for policy interventions (COMEAP, 2009). Several of the individual substances modelled including benzene, 1,3-butadiene, and formaldehyde qualify as VOCs, so including total VOCs would double-count these substances. Additionally, there is no health benchmark available for total VOCs. Such a benchmark would be difficult to identify because the toxicity of any VOC mixture depends on the specific combination of VOCs under consideration.

## Results:

### Air Quality Model Findings

Some key findings from the air quality modelling are presented below to aid in interpreting the health assessment findings. A comprehensive presentation of the methods and findings of the air quality modelling component of this research is available in the technical report entitled, *An All Sources Cumulative Air Quality Impact Study of South Riverdale - Leslieville – Beaches* (Toronto Environment Office 2011).

For most substances, the predicted ambient concentrations met Ontario's ambient air quality criteria (AAQCs). The AAQCs are provincial standards that are developed for a

large number of substances to protect human health and the environment. The province has established 24-hour standards for all of the priority air contaminants except VOCs. The model predicted that these standards would be exceeded for NO<sub>x</sub> (24-hour average and 24-hour max), PM<sub>10</sub>, benzene, and PAH at some points and time. The province has also established annual average air standards for 10 of the priority air contaminants. Of those, the model predicted that annual average concentrations of benzene and PAHs could exceed the standards at some points and times.

The air quality modelling enabled identification of the proportion of ambient concentrations in Wards 30 and 32 attributable to sources outside of Toronto. Of the emissions that originate in Toronto, it is possible to identify the proportion originating from each of five sectors. For each of the substances, Table 4 shows the proportion originating from the U.S., from Ontario, and from within Toronto. The Toronto sources are further broken down by sector, where "industrial" refers to large industrial polluters listed in the national pollutant release inventory (NPRI), "residential/commercial" refers to homes, autobody shops, solvent users, and drycleaners, "mobile" refers to all transportation sources, "mobile non-road" refers mainly to airport, marine, rail, lawn and garden equipment, and "biogenic" refers to emissions arising from living organisms or biological processes.

**Table 4: Contribution of Source Categories to Annual Average Predicted Air Concentrations (2006) in Wards 30 and 32 (Toronto Environment office 2011).**

Substance	Contribution from U.S	Contribution from rest of Ontario	Contribution from within Toronto				
			<i>Industrial</i>	<i>Residential/Commercial</i>	<i>Mobile</i>	<i>Nonroad Mobile</i>	<i>Biogenic / Agri</i>
Nitrogen Oxides <sup>(1)</sup>	22%	21%	5.2%	11.3%	32.6%	7.9%	0%
Carbon Monoxide	22%	19%	1.4%	2.8%	44.3%	10.5%	0%
Sulphur Dioxide	71%	17%	0.64%	0.83%	2.4%	8.1%	0%
PM <sub>2.5</sub>	32%	20%	10.9%	16.0%	16.0%	5.1%	0%
PM <sub>10</sub>	30%	20%	5.1%	6.4%	36.3%	2.2%	0%
VOCs	19%	25%	24.4%	14.8%	12.6%	4.0%	0.24%
Formaldehyde	34%	21%	1.2%	2.1%	22.3%	19.3%	0%
Acetaldehyde	40%	16%	4.8%	0%	21.4%	16.8%	0%
Acrolein	14%	36%	1.2%	5.3%	26.0%	17.5%	0%
1,3-Butadiene	22%	24%	0%	0%	49.8%	4.2%	0%
Benzene	26%	19%	8.7%	0%	39.0%	8.3%	0%
Toluene	23%	15%	6.2%	35.9%	17.3%	2.7%	0%
1,4-Dichlorobenzene	2%	15%	0%	84.0%	0%	0%	0%
PAHs (as B[a]Ps)	68%	8%	0.07%	0%	23.9%	0%	0%
Chloromethane	4%	96%	-	-	-	-	-
Dichloromethane	14%	15%	4.4%	67.6%	0%	0%	0%
Chloroform	44%	8%	0.0%	48.0%	0%	0%	0%
Carbon Tetrachloride	100%	0%	0%	0%	0%	0%	0%
Vinyl Chloride	92%	8%	-	-	-	-	-
1,2-Dichloroethane	42%	50%	0%	8.0%	0%	0%	0%
Trichloroethylene	12%	85%	0.12%	2.9%	0%	0%	0%
Tetrachloroethylene	7%	18%	0%	75.0%	0%	0%	0%
Ethylene Dibromide	100%	0%	-	-	-	-	-
Lead	57%	32%	7.2%	2.2%	0%	1.6%	0.0%
Cadmium	26%	16%	11.4%	46.3%	0%	1.4%	0.0%
Chromium	49%	27%	10.8%	7.8%	1.0%	3.4%	0.0%
Nickel compounds	38%	49%	2.5%	9.8%	0.63%	0%	0%
Mercury	61%	22%	3.4%	13.6%	0%	0%	0%
Manganese	71%	26%	1.6%	1.0%	0.14%	0.35%	0.0%
<b>Average</b>	<b>39%</b>	<b>25%</b>	<b>4%</b>	<b>18%</b>	<b>13%</b>	<b>4%</b>	<b>0%</b>

The model estimates that on average, 39% of the air pollution (by weight) comes from the United States and 25% comes from other parts of Ontario. Of the 36% of air pollution that comes from Toronto sources, residential and commercial sources appear to be the most important, contributing about 18% to the total ambient concentrations while mobile sources contribute about 13%. Off-road mobile sources contribute a further 4%, and large industrial sources in Toronto (including those in Wards 30 and 32) also contribute about 4% to the total concentrations to these neighbourhoods.

The proportion of each individual pollutant that comes from inside or outside Toronto, or from a specific sector varies strongly by substance. For example, while the model estimates that 100% of carbon tetrachloride in Wards 30 and 32 originates in the United States, 83% of 1,4-dichlorobenzene is generated within Toronto.

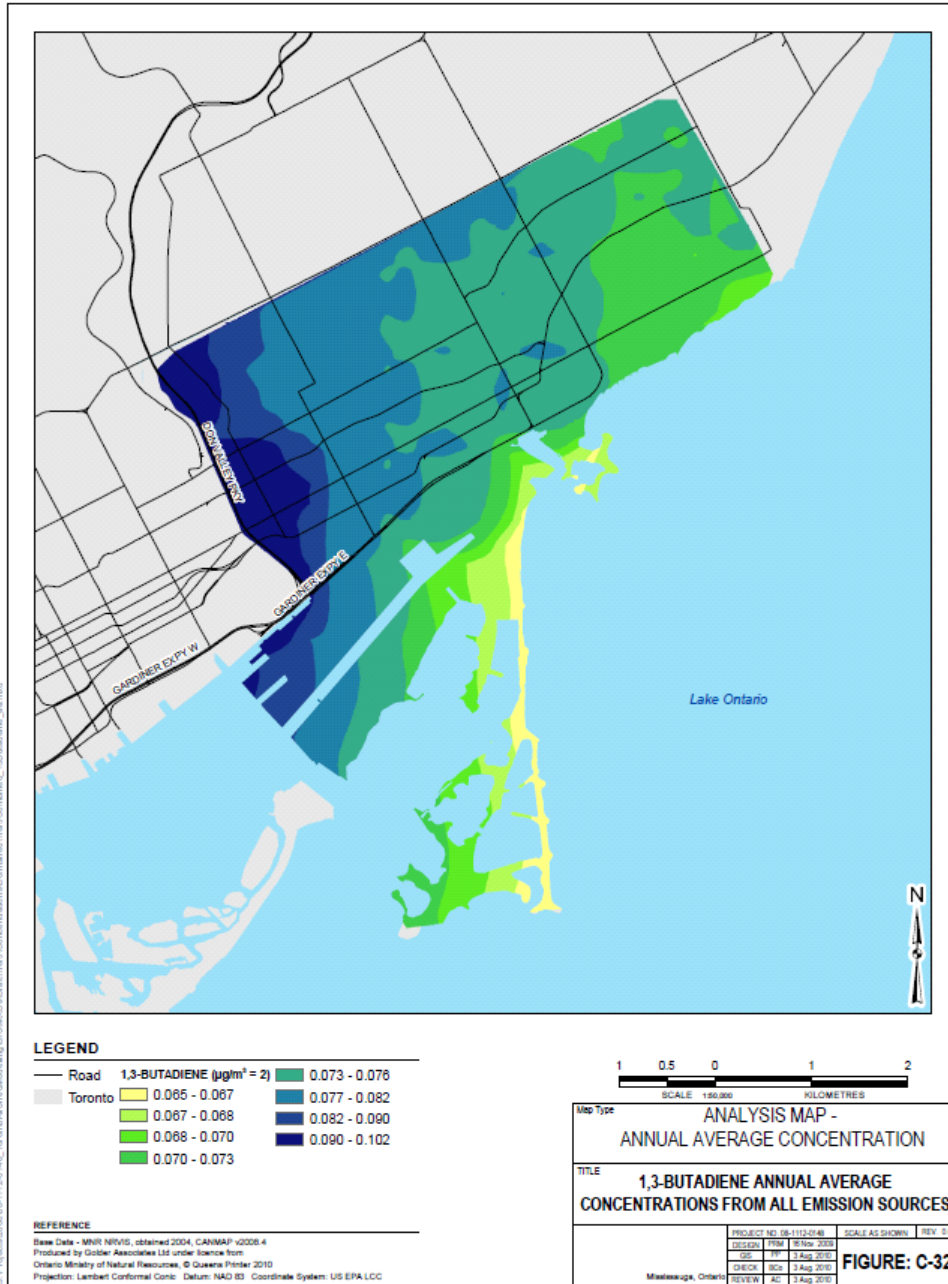
For locally generated pollutants, residential and commercial sources in Toronto appear to be the primary contributors to concentrations of aromatic and halogenated substances, mainly due to emissions from dry cleaners, solvent users and auto-body shops. Residential and commercial sources also release trace metals which come from natural gas consumption and other combustion sources.

When the predicted concentrations were mapped, substances exhibited significant spatial variation. This typically occurred if the substance is emitted mostly by a large industrial facility, where the concentrations are highest close to the facility. Similarly, transportation-related pollutants appear to be most concentrated along major highways and roadways. Within Wards 30 and 32, concentrations are highest close to the Don Valley Parkway (DVP) (see for example, Figure 2).

The results of the model were validated where possible by comparing predicted concentrations with monitored data. Since there are no monitoring stations inside the Wards 30 and 32 area, the comparison was made with data collected at the closest air quality monitor. Monitoring data was available for 11 substances. For most, the match between monitored and modelled concentrations was very close, giving an average monitoring to modelling ratio of 1.03.

However, the modelled concentrations for nitrogen dioxide and sulphur dioxide were higher than the monitored concentrations by more than a factor of 2. This likely occurs because the modelling methodology used is unable to account for the chemical transformations that degrade these substances as they travel long distances from the U.S or parts of Ontario. However, it is important to note that high concentrations of nitrogen dioxide have been recognized as problem for the air quality of Toronto. For dichloromethane, the modelled concentrations were lower than the monitored by more than a factor of 2. This needs to be taken into account when the results of the health assessment are interpreted.

**Figure 2: Spatial distribution of predicted 1,3-butadiene concentrations in Wards 30 and 32. Transportation sources are a key contributor to ambient concentrations of this substance.**



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## Carcinogens

The predicted risk associated with the nineteen carcinogens is summarized in Table 5. Predicted annual average concentrations were used to estimate risk as they are the best representation of chronic exposure, and the cancer risks are estimated based on lifetime exposure. TPH typically uses a risk level of  $10^{-6}$  or "one in a million" to represent a situation without appreciable risk. In Table 5, values that exceed a risk level of  $10^{-6}$  are in bold.

The range from minimum to maximum represents the variation in risk that occurs within the neighbourhood. The minimum values represent the risk at the location in the neighbourhood where the lowest yearly average concentration is predicted, and the maximum values indicate the risk at the location of the highest predicted concentration in the neighbourhood.

**Table 5: Summary of Cancer risks for exposures at average annual levels (Values greater than  $10^{-6}$  are shown in bold)**

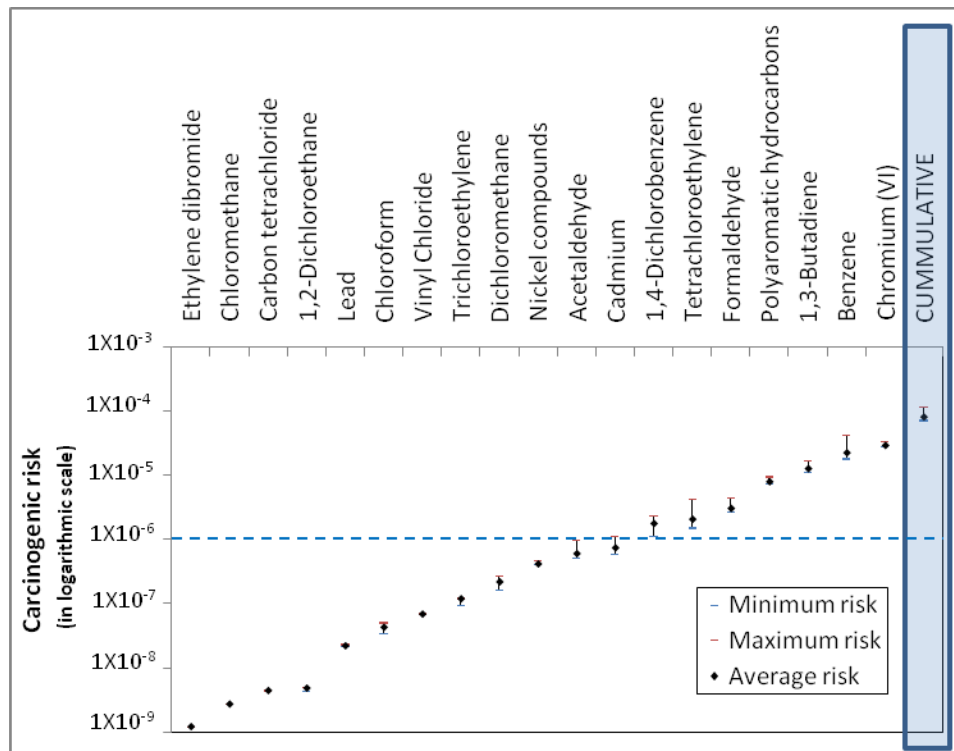
SUBSTANCE	Cancer risk (average annual)		
	minimum	mean	maximum
Acetaldehyde	$5.1 \times 10^{-7}$	$6.1 \times 10^{-7}$	$9.6 \times 10^{-7}$
Benzene	<b><math>1.8 \times 10^{-5}</math></b>	<b><math>2.3 \times 10^{-5}</math></b>	<b><math>4.2 \times 10^{-5}</math></b>
1,3-Butadiene	<b><math>1.1 \times 10^{-5}</math></b>	<b><math>1.3 \times 10^{-5}</math></b>	<b><math>1.7 \times 10^{-5}</math></b>
Cadmium	$5.8 \times 10^{-7}$	$7.5 \times 10^{-7}$	<b><math>1.1 \times 10^{-6}</math></b>
Carbon tetrachloride	$4.4 \times 10^{-9}$	$4.4 \times 10^{-9}$	$4.4 \times 10^{-9}$
Chloroform	$3.4 \times 10^{-8}$	$4.3 \times 10^{-8}$	$5.0 \times 10^{-8}$
Chloromethane	$2.7 \times 10^{-9}$	$2.7 \times 10^{-9}$	$2.7 \times 10^{-9}$
Chromium VI	<b><math>2.8 \times 10^{-5}</math></b>	<b><math>3.0 \times 10^{-5}</math></b>	<b><math>3.4 \times 10^{-5}</math></b>
1,4-Dichlorobenzene	<b><math>1.1 \times 10^{-6}</math></b>	<b><math>1.8 \times 10^{-6}</math></b>	<b><math>2.3 \times 10^{-6}</math></b>
1,2-Dichloroethane	$4.3 \times 10^{-9}$	$4.8 \times 10^{-9}$	$4.9 \times 10^{-9}$
Dichloromethane	$1.6 \times 10^{-7}$	$2.2 \times 10^{-7}$	$2.7 \times 10^{-7}$
Ethylene dibromide	$1.2 \times 10^{-9}$	$1.2 \times 10^{-9}$	$1.2 \times 10^{-9}$
Formaldehyde	<b><math>2.7 \times 10^{-6}</math></b>	<b><math>3.1 \times 10^{-6}</math></b>	<b><math>4.5 \times 10^{-6}</math></b>
Lead	$2.2 \times 10^{-8}$	$2.2 \times 10^{-8}$	$2.3 \times 10^{-8}$
Nickel compounds	$4.0 \times 10^{-7}$	$4.2 \times 10^{-7}$	$4.6 \times 10^{-7}$
Polyaromatic hydrocarbons*	<b><math>7.4 \times 10^{-6}</math></b>	<b><math>8.1 \times 10^{-6}</math></b>	<b><math>9.4 \times 10^{-6}</math></b>
Tetrachloroethylene	<b><math>1.5 \times 10^{-6}</math></b>	<b><math>2.1 \times 10^{-6}</math></b>	<b><math>4.2 \times 10^{-6}</math></b>
Trichloroethylene	$9.4 \times 10^{-8}$	$1.2 \times 10^{-7}$	$1.2 \times 10^{-7}$
Vinyl Chloride	$6.8 \times 10^{-8}$	$6.9 \times 10^{-8}$	$6.9 \times 10^{-8}$

\* As benzo[a]pyrene

The distribution of carcinogen risk values for average exposures across the neighbourhood for each substance is pictured graphically in Figure 3. This figure shows that there are seven substances where the average neighbourhood risks arising from the average annual concentrations were associated with greater than one in a million risk

(benzene, benzo[a]pyrene, 1,3-butadiene, chromium (VI), 1,4-dichlorobenzene, formaldehyde, and tetrachloroethylene). Also, cadmium appears to be associated with a greater than a one in a million risk at locations within the neighbourhood where the concentrations are highest. Figure 3 also shows that individual substances are associated with dramatically different risk (note that the y-axis is a logarithmic scale).

**Figure 3: Average, maximum, and minimum cancer risk values estimated for each substance based on average annual concentrations from the 551 receptor sites.**



### Carcinogens – Cumulative Risk

If the average annual risk is summed across all nineteen carcinogenic substances, the mean cumulative risk is  $8.3 \times 10^{-5}$ . This estimated risk is above the one in a million benchmark. This total risk is around two percent of the overall cancer incidence rate in Toronto - about 400 per 100,000 in 2007 (Vital Statistics, 2007). As Table 6 below shows, these risks are driven mainly by a few substances: chromium VI, benzene, 1,3-butadiene, and benzo[a]pyrene. When the modelling was done, all emissions of PAHs were assumed to be benzo[a]pyrene. This is a very health-protective approach, since

B[a]P is one of the most potent of the carcinogens. As a result, the risks associated with estimated concentrations of B[a]P are likely to be overestimated.

**Table 6: Summary of Percentage Contribution of Individual Air Pollutants to the Cumulative Cancer Risk Estimates risks for exposures at average annual levels ( "---" indicates a contribution of < 0.1%).**

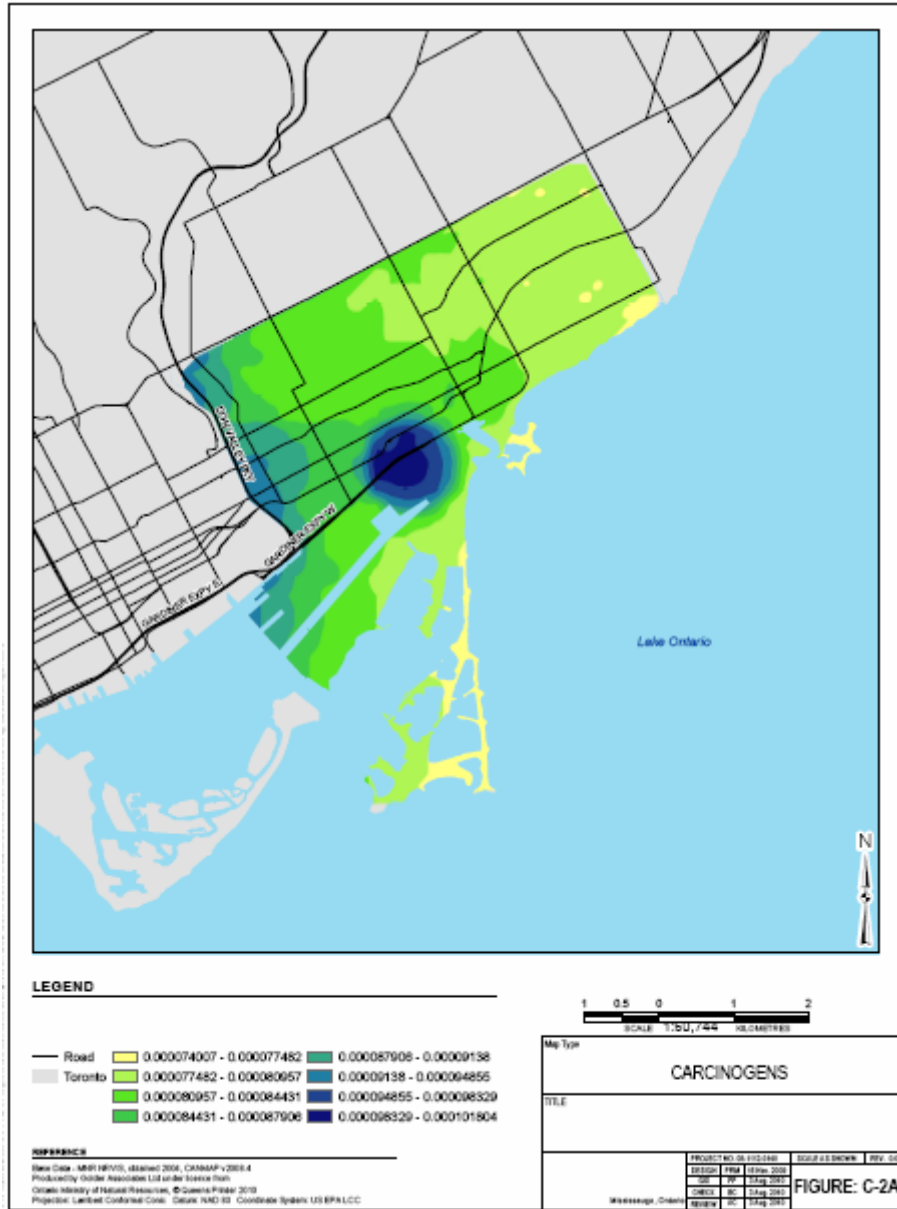
<b>SUBSTANCE</b>	<b>Cancer risk (mean for neighbourhood)</b>
Acetaldehyde	0.7%
Benzene	27.5%
1,3-Butadiene	15.8%
Cadmium	0.9%
Carbon tetrachloride	---
Chloroform	0.1%
Chloromethane	---
Chromium (VI)	35.7%
1,4-Dichlorobenzene	2.2%
1,2-Dichloroethane	---
Dichloromethane	0.3%
Ethylene dibromide	---
Formaldehyde	3.8%
Lead	---
Nickel compounds	0.5%
Polyaromatic hydrocarbons	9.8%
Tetrachloroethylene	2.6%
Trichloroethylene	0.1%
Vinyl Chloride	0.1%

\* As benzo[a]pyrene

Figure 3 shows a map of the cumulative cancer risk across Wards 30 and 32. The figure shows areas of elevated risk centred around a south-central location close to the lake, and a second area of elevated risk close to the Don Valley Parkway, at the left of the map. A review of the risk maps for the substances that drive the cumulative risk estimates suggest that the area of elevated risk close to the lake is due to the emissions of benzene from a single facility located there. Discussions between TEO and MOE revealed that since 2006, (the data year used for the model), emissions of benzene from this facility have significantly decreased, as a result of changes in operations.

Substance-specific data indicate that the areas of elevated risk close to the DVP arise mainly from 1,3-butadiene, benzene, and benzo[a]pyrene. These substances were also significant contributors to the overall cumulative cancer risk in the whole area, and are primarily emitted from transportation sources.

**Figure 3: Spatial distribution of cumulative risk from carcinogens for Wards 30 and 32 neighbourhoods.**



## Non-Carcinogens

The predicted hazard associated with the 22 non-carcinogen substances is summarized in Table 7. For most of the non-carcinogens, it is most appropriate to use the average annual concentrations to predict hazard. This is because the health benchmarks selected represent chronic exposure periods – or lifetime risk. However, there were five substances for which no chronic exposure health benchmark was available: chloromethane, carbon tetrachloride, chromium (III), 1,4-dichlorobenzene, and lead. These substances are identified in the table with the symbol "\*\*\*". For these substances, the available evidence suggests that non-cancer health effects are more likely to arise as a result of acute exposures than chronic exposures. For these substances it is more appropriate to examine the hazards using the maximum 24-hour concentrations.

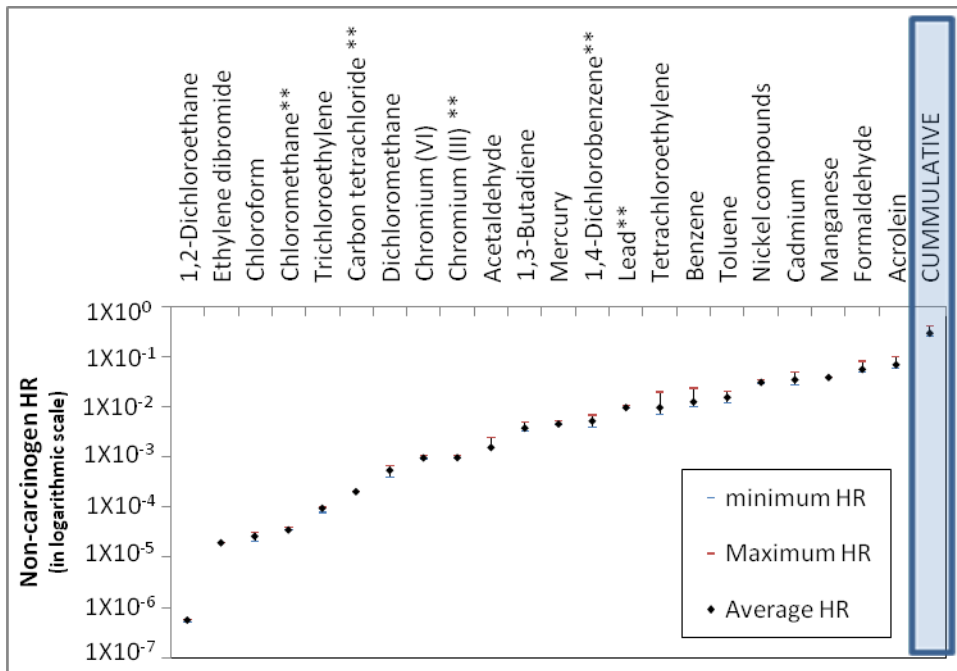
The range from minimum to maximum represents the variation in risk that occurs within the neighbourhood. The minimum values represent the risk at the location in the neighbourhood where the lowest concentration is predicted, and the maximum values indicate the risk at the location of the highest predicted concentration in the neighbourhood.

Health Canada uses a hazard ratio of 0.2 as an indicator for potentially high exposure for a single exposure pathway and individual substance. All values for the hazard ratios, including at the points of maximum exposures, are below 0.2. The substance with the largest hazard ratio is acrolein which a maximum hazard ratio of 0.1. This indicates that exposures to these substances are well below their corresponding health benchmark (see Figure 4 and Table 7).

**Table 7: Summary of Noncancer HRs for exposures at average annual levels, and maximum daily levels.**

SUBSTANCE	Noncancer HR (average annual)			Noncancer HR (max 24-hr)		
	min	mean	max	min	mean	max
Acetaldehyde	$1.4 \times 10^{-3}$	$1.6 \times 10^{-3}$	$2.5 \times 10^{-3}$	--	--	--
Acrolein	$6.0 \times 10^{-2}$	$7.2 \times 10^{-2}$	$1.0 \times 10^{-1}$	--	--	--
Benzene	$1.0 \times 10^{-2}$	$1.3 \times 10^{-2}$	$2.4 \times 10^{-2}$	--	--	--
1,3-Butadiene	$3.3 \times 10^{-3}$	$3.9 \times 10^{-3}$	$5.1 \times 10^{-3}$	--	--	--
Cadmium	$2.8 \times 10^{-2}$	$3.6 \times 10^{-2}$	$5.1 \times 10^{-2}$	--	--	--
Carbon tetrachloride **	--	--	--	$2.1 \times 10^{-4}$	$2.1 \times 10^{-4}$	$2.1 \times 10^{-4}$
Chloroform	$2.1 \times 10^{-5}$	$2.7 \times 10^{-5}$	$3.1 \times 10^{-5}$	--	--	--
Chloromethane **	--	--	--	$3.6 \times 10^{-5}$	$3.6 \times 10^{-5}$	$4.0 \times 10^{-5}$
Chromium (III) **	--	--	--	$9.8 \times 10^{-4}$	$1.0 \times 10^{-3}$	$1.1 \times 10^{-3}$
Chromium (VI)	$9.4 \times 10^{-4}$	$9.9 \times 10^{-4}$	$1.1 \times 10^{-3}$	--	--	--
1,4-Dichlorobenzene **	--	--	--	$3.9 \times 10^{-3}$	$5.4 \times 10^{-3}$	$6.9 \times 10^{-3}$
1,2-Dichloroethane	$5.1 \times 10^{-7}$	$5.7 \times 10^{-7}$	$5.8 \times 10^{-7}$	--	--	--
Dichloromethane	$3.9 \times 10^{-4}$	$5.6 \times 10^{-4}$	$6.8 \times 10^{-4}$	--	--	--
Ethylene dibromide	$2.0 \times 10^{-5}$	$2.0 \times 10^{-5}$	$2.0 \times 10^{-5}$	--	--	--
Formaldehyde	$5.0 \times 10^{-2}$	$5.8 \times 10^{-2}$	$8.3 \times 10^{-2}$	--	--	--
Lead **	--	--	--	$9.7 \times 10^{-3}$	$1.0 \times 10^{-2}$	$1.1 \times 10^{-2}$
Manganese	$3.9 \times 10^{-2}$	$4.0 \times 10^{-2}$	$4.0 \times 10^{-2}$	--	--	--
Mercury	$4.4 \times 10^{-3}$	$4.7 \times 10^{-3}$	$5.3 \times 10^{-3}$	--	--	--
Nickel compounds	$3.0 \times 10^{-2}$	$3.2 \times 10^{-2}$	$3.5 \times 10^{-2}$	--	--	--
Tetrachloroethylene	$7.1 \times 10^{-3}$	$1.0 \times 10^{-2}$	$2.0 \times 10^{-2}$	--	--	--
Toluene	$1.2 \times 10^{-2}$	$1.6 \times 10^{-2}$	$2.1 \times 10^{-2}$	--	--	--
Trichloroethylene	$7.9 \times 10^{-5}$	$9.8 \times 10^{-5}$	$9.9 \times 10^{-5}$	--	--	--

\*\* indicates that the hazard ration was calculated based on an acute health outcome rather than a chronic health outcome.



**Figure 4: Average, maximum and minimum Hazard Ratio (HR) values estimated for non-carcinogens substances based on average annual concentrations at the 551 sites.**

### Non-carcinogens – Cumulative risk

The sum of the average annual hazard indexes of all twenty-two non carcinogenic substances sums to 0.31. As this cumulative hazard ratio is below 1, this suggests that exposure to the combination of substances would not result in adverse health impacts<sup>5</sup> Table 8 shows the contribution of each substance to the cumulative HR. Acrolein is the main contributor (23.7%) followed by formaldehyde (18.9%).

<sup>5</sup> There are limitations to this approach. It assumes that the impacts of exposure are additive. This can over estimate the hazard that substances act through different mechanisms, but at the same time, it does not takes into account potential synergistic effects.

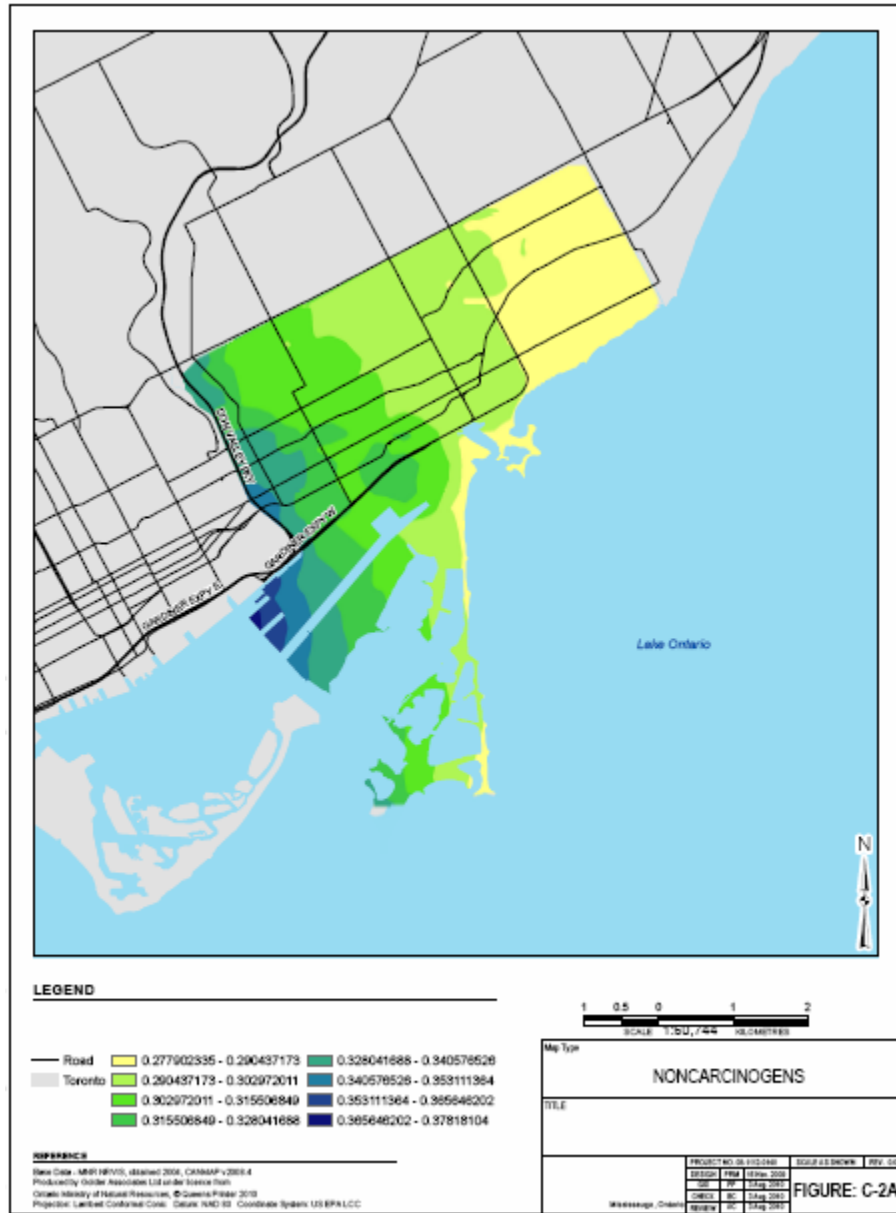
**Table 8: Summary of Percentage Contribution of Individual Air Pollutants to the Cumulative Non-cancer Hazard Index estimates for exposures at average annual levels, and maximum daily levels.**

SUBSTANCE	Non-cancer Hazard (mean for neighbourhood)
Acetaldehyde	0.5%
Acrolein	23.7%
Benzene	4.3%
1,3-Butadiene	1.3%
Cadmium	11.7%
Carbon tetrachloride	0.1%
Chloroform	---
Chloromethane	---
Chromium (III)	0.3%
Chromium (VI)	0.3%
1,4-Dichlorobenzene	1.8%
1,2-Dichloroethane	---
Dichloromethane	0.2%
Ethylene dibromide	---
Formaldehyde	18.9%
Lead	3.3%
Manganese	13.0%
Mercury	1.5%
Nickel compounds	10.5%
Tetrachloroethylene	3.4%
Toluene	5.1%
Trichloroethylene	---

--- indicates a contribution of < 0.1%

Figure 6 shows the distribution of the cumulative non-cancer risk across Wards 30 and 32. The figure shows an area of higher exposure centred on a south-central location near to the Toronto Port, and a second area close to the Don Valley Parkway. This is similar to the results for cumulative cancer risk but the cumulative risk in the centre of the map is not as high as the area around the DVP as was the case of cancer risk.

**Figure 5: Spatial distribution of cumulative hazard from non-carcinogens for Wards 30 and 32.**



## Criteria Air Contaminants

The predicted percent excess per capita risk of premature death associated with the five criteria air contaminants is summarized in Table 9. Annual average values were used for estimating percent excess per capita risk, as they are most representative of chronic, long-term exposures. This is the approach previously used by Toronto Public Health when estimating burden of illness using these coefficients (Toronto Public Health, 2007).

The range from minimum to maximum represents the variation in risk that occurs within the study area. The minimum values represent the risk at the location in the area where the lowest concentration is predicted, and the maximum values indicate the risk at the location of the highest predicted concentration in the area.

Modelling for ozone was conducted separately from all the other substances. Ozone arises from chemical reactions of other pollutants and it is not possible to predict neighbourhood concentrations at a high spatial resolution. The model was only able to predict average concentrations.

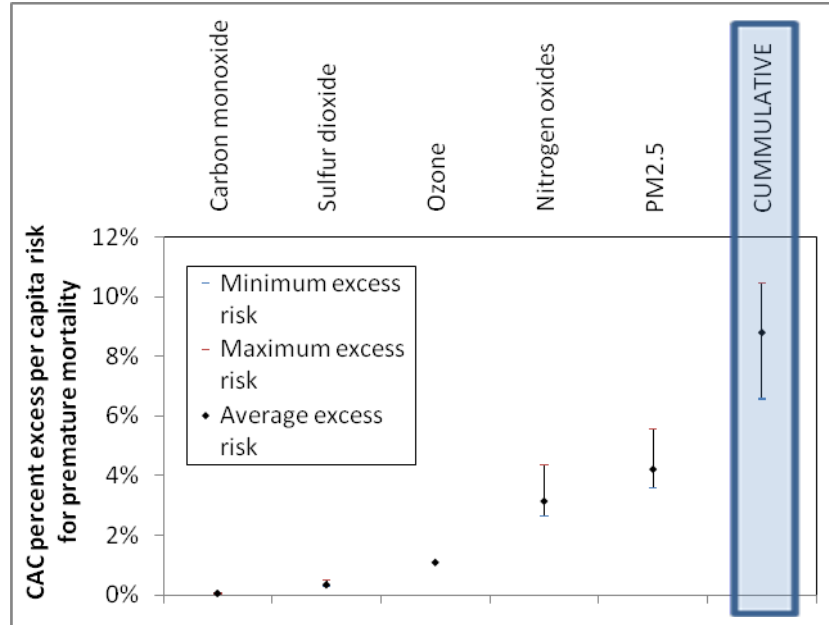
**Table 9: Summary of CAC percent excess per capita risks for exposures at average annual levels<sup>1</sup>.**

SUBSTANCE	CAC risk (average annual)		
	minimum	mean	maximum
CO	<b>0.0003</b>	<b>0.0004</b>	<b>0.0005</b>
SO <sub>2</sub>	<b>0.0030</b>	<b>0.0033</b>	<b>0.0049</b>
Ozone <sup>2</sup>	--	<b>0.0108</b>	--
NO <sub>x</sub> (as NO <sub>2</sub> )	<b>0.0265</b>	<b>0.0313</b>	<b>0.0436</b>
PM <sub>2.5</sub>	<b>0.0359</b>	<b>0.0420</b>	<b>0.0555</b>

<sup>1</sup> All are at levels of concern and therefore in bold.

<sup>2</sup> Because ozone is produced primarily from chemical transformation rather than from emission and dispersion, it was modelled based on annual average NO<sub>x</sub> values. Only an annual average value could be produced to represent the neighbourhood concentration (no maximum or minimum values could be predicted).

The distribution of predicted CAC percent excess per capita risk for average exposures across the neighbourhood for each substance is shown in Figure 7. The figure shows that the excess risk is predominantly associated with PM<sub>2.5</sub> and NO<sub>2</sub>. The cumulative excess per capita risk is estimated at 0.089, or 8.9%. Fine particulate matter (PM<sub>2.5</sub>) and nitrogen dioxide are the pollutants that contribute most to this risk. This level of excess risk is similar to what has previously been calculated in the Burden of Illness in Toronto (Toronto Public Health, 2004).



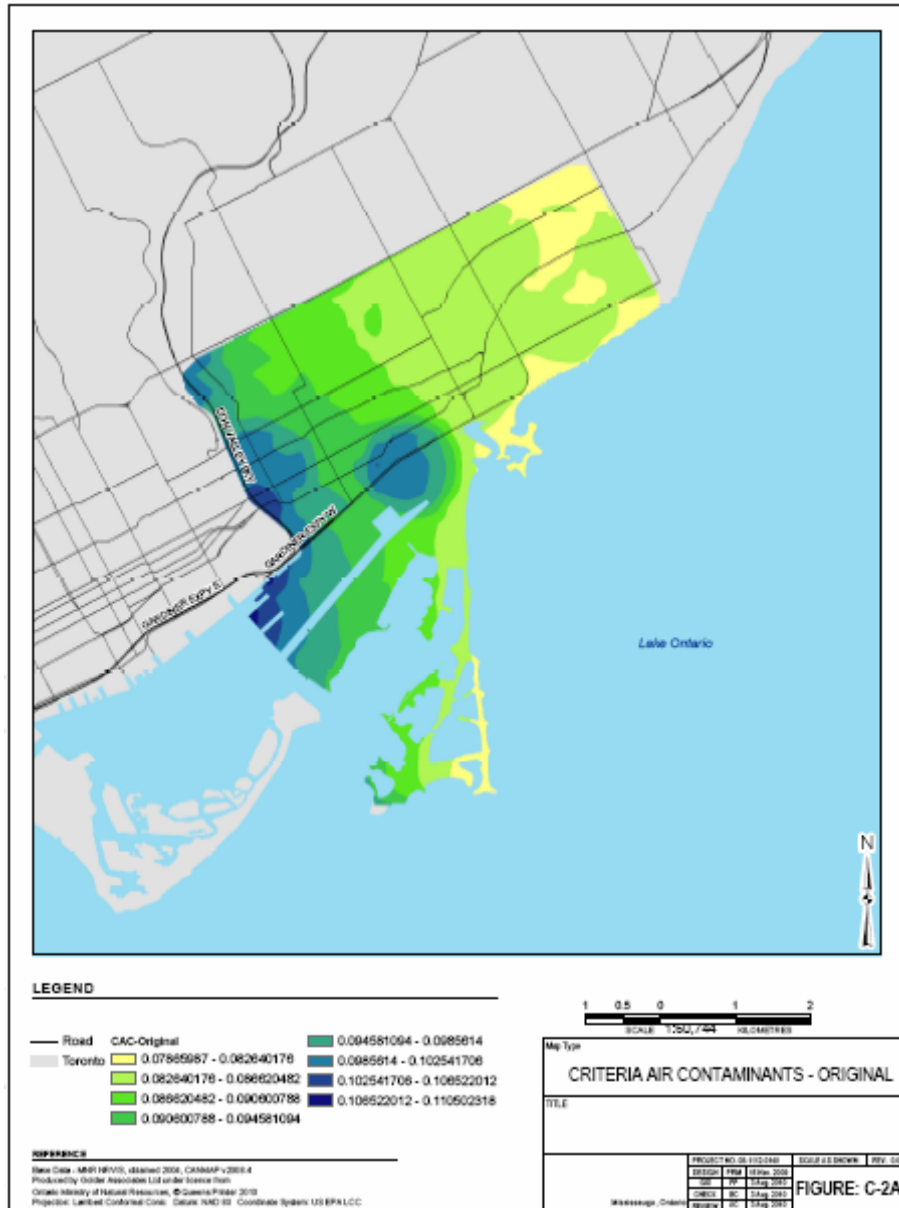
**Figure 7: Average, max and min values for percent excess per capita risk for premature mortality estimated for five CAC based on average annual concentrations at the 551 sites. Any HR < 0.2 is considered tolerable for a single route of exposure.**

It is possible that the model over estimate the levels of SO<sub>2</sub> and NO<sub>2</sub> in the study area. Taking into account possible over estimation the cumulative risk would be lower.<sup>6</sup>

Figure 6 shows the spatial distribution of cumulative percent excess per capita risk from CACs. The map is very similar to the one obtained for cancer risk. Nitrogen dioxide is the pollutant that accounts for most of the risk near the DVP, while fine particulate matter (PM<sub>2.5</sub>) was the pollutant associated with the higher risk around the point source.

<sup>6</sup> The comparison between modelled and monitored data suggests that the concentrations were overestimated by factors of 3.4 and 2, respectively. If the risk estimates were reduced by these factors, the estimated percent excess risk would be 0.165% and 1.65%, respectively.

**Figure 6: Spatial distribution of cumulative percent excess per capita risk from CACs for Wards 30 and 32.**



## Discussion

Some of the thirty priority air contaminants selected for this study may be present in the neighbourhood at levels above recognized health benchmarks in some places in some occasions. The health assessment suggests that many substances that are emitted into Toronto's air are not likely to have an adverse impact on health in the study area. This is valuable information, enabling pollution prevention resources to be focussed on those substances and sources which are priorities from a health perspective.

It is possible to compare these results with those from a similar analysis conducted in Portland, Oregon (Department of Environmental Quality (DEQ) 2006). Although Portland's analysis only included 13 substances, their results are similar in that many of the hazard ratios calculated there were in the  $10^{-4}$  to  $10^{-2}$  range. In their assessment only acrolein and diesel particulate matter were associated with hazard ratios greater than one. For acrolein, this is because they used a health benchmark of  $0.2 \text{ ug/m}^3$  compared to  $0.35 \text{ ug/m}^3$  in this assessment. Using the same newer benchmark as we did in Toronto would result in a hazard ratio less than one for Portland as well. The Toronto analysis did not examine diesel particulate matter specifically, but did look at PAHs and  $\text{PM}_{2.5}$ .

As described earlier in the section on air quality modelling results, the air quality modelling enables identification of the proportion of ambient pollution attributable to specific source types. Table 10 identifies those substances that are at levels above health benchmarks and the proportion originating from the U.S., from Ontario, and from within Toronto. Toronto sources are further broken down by sector. These results are helpful in setting priorities and strategies for pollution prevention that could improve air quality in Wards 30 and 32.

**Table 10: Origins of substances identified as exceeding health benchmarks in Wards 30 and 32.**

Substance	Contribution from U.S	Contribution from rest of Ontario	Contribution from Within Toronto			
			Industrial	Residential/Commercial	Mobile	Mobile Non-road
$\text{NO}_2$	22%	21%	5.2%	11.3%	32.6%	7.9%
CO	22%	19%	1.4%	2.8%	44.3%	10.5%
$\text{SO}_2$	71%	17%	0.64%	0.83%	2.4%	8.1%
$\text{PM}_{2.5}$	32%	20%	10.9%	16.0%	16.0%	5.1%
1,3-Butadiene	22%	24%	0%	0%	49.8%	4.2%
Benzene	26%	19%	8.7%	0%	39.0%	8.3%
Chromium	49%	27%	10.8%	7.8%	1.0%	3.4%
1,4-Dichlorobenzene	2%	15%	0%	84.0%	0%	0%
PAHs (as B[a]Ps)	68%	8%	0.07%	0%	23.9%	0%
Formaldehyde	34%	21%	1.2%	2.1%	22.3%	19.3%
Tetrachloroethylene	7%	18%	0%	75.0%	0%	0%

The results suggest that for some substances that affect these neighbourhoods, most emissions originate outside the City of Toronto. This is the case with SO<sub>2</sub>, 88% of which comes from either the U.S. or Ontario, chromium, of which 76% originates outside Toronto, and PAHs, 76% of which originate outside Toronto. The findings for SO<sub>2</sub> in particular highlight the success of Canadian initiatives to reduce the presence of sulphur in fuels: very little of the substance is emitted in Canada now. The modelling highlights a need for regional strategies and multi-jurisdictional co-operation in addition to local strategies to effectively reduce the presence of these substances in Toronto.

The results also indicate that for many substances, the transportation sector is the main source of the locally-generated emissions. This appears to be especially true for NO<sub>2</sub>, CO, 1,3-butadiene, and benzene. These findings suggest that options to reduce exposure to transportation-related emissions are needed. Strategies for doing this could include new technologies for fuels and vehicle emissions reduction, policies to ensure separation of sensitive land-uses (such as daycares or hospitals) from high-traffic areas, or programs that encourage people to choose transit or active transportation over driving when feasible.

For some substances such as 1,4-dichlorobenzene and tetrachloroethylene, most local emissions fall under the residential/commercial category. These substances are not typically emitted from homes, and so are attributed to commercial sources such as solvent users and drycleaners respectively.

A limitation of the modelling effort was that only three types of commercial emitters were included in the model: drycleaners, solvent users, and autobody shops. As described earlier in this report, in the future, information collected from the Environmental Reporting and Disclosure Bylaw will support more accurate modelling of local concentrations for priority air pollutants in Toronto ([www.toronto.ca/chemtrac](http://www.toronto.ca/chemtrac)). In turn, the air modelling and health assessment results can inform the ChemTRAC program and help to identify which substances should remain priorities for reporting in the long term. As well, a component of the program is to support smaller businesses in finding ways to reduce emissions and adopt safer alternatives where possible.

On average, large industrial sources are minor contributors to overall air pollution and the cumulative risk arising from air pollution in those two Wards. However, industrial emitters are often point sources, so the risk that does exist may be concentrated in a particular area. This pattern occurred with benzene, where there was a clear area of higher risk in these neighbourhoods that was attributable to a single industrial source, even though 70% of benzene emissions in Toronto come from mobile sources.

This project assessed the air quality in Wards 30 and 32, and the conclusions about patterns of exposure and which substances are priorities for reduction apply specifically to this area of Toronto. Other neighbourhoods may be characterized by different source types which could influence the local air quality mix and create different priorities for emissions reduction.

## Limitations of the Health Assessment Methodology

There are some challenges associated with conducting a cumulative assessment of health risk. For example, substances are thought to act through various mechanisms of action. Substances with similar mechanisms of action and affecting similar body systems may exert an additive effect on the health of a population. However, it is more difficult to “sum up” effect on different body systems, particularly as some effects may be deemed more severe than others. For example, the presence of common air contaminants may worsen symptoms among asthmatics in the community, while certain air toxics may increase the lifetime risk of cancer. It is difficult to compare or “add” these two health impacts into a single measure of health risk for the community.

To tackle this difficulty TPH separated substances into three broad categories: those associated with cancer outcomes, those associated with non-cancer outcomes, and CACs, which are associated mainly with cardiovascular and respiratory outcomes, and premature death.

Because cancers are thought to arise according to a given biological process, it is logical to accumulate the potential risk of cancer from individual substances. Similarly, because the risk estimates for CACs are focussed on a single health outcome, accumulating the risks across substances is a logical approach.

Non-cancer health effects attributed to different substances affect different systems/target organs and each may cause harm as a result of different biological mechanism. Some would argue that it may therefore not be appropriate to sum the hazard quotients for the substance-specific non-cancer health endpoints into a single index. However, assessing the hazard separately for each target system/organ ignores the potential for lowered overall resilience from the cumulative impact of multiple environment assaults on all organs/systems at low concentrations. Finally, there is no guarantee that a person may not be subject to more than one health outcome at a time.

If non-cancer hazard indices are calculated separately for each target system/organ, each cumulative (organ-specific) hazard index will be lower than if the hazard index is calculated for all substances as a group. Accumulating all hazard ratios is a way of characterizing the maximum possible non-cancer impact. If a hazard ratio of one is not exceeded from such an accumulation, then the organ-specific and substance-specific non-cancer hazards can also be assumed to be less than one. In this study, the hazard ratio was 0.31 – well below the threshold of 1.00. It appears that there is no need to examine the hazards by body system or target organ, since the hazard ratios for individual target system/organs will all be less than the cumulative hazard, and will therefore all be less than 1.00.

Some pollutants may interact with each other synergistically. This means that their combined effect is greater than what would be expected based on assessing the substances in a simple additive way. Another challenge is that some people and not others may be especially vulnerable to certain pollutants. With no data on these possible

interactions or range of sensitivities, these aspects could not be accounted for in the analysis.

The approach to characterizing risk for CACs examines only one health endpoint: premature mortality. There are other health endpoints associated with exposure to CACs such as cardiovascular and respiratory morbidity – and the risk coefficients (and associated incidence of outcomes) for these health outcomes are higher than those for acute mortality. Thus, the estimated impact of CACs must be viewed as an indicator of potential impact for the neighbourhood and not the full burden of illness. This approach also represents an aggregate level estimate which may not accurately represent the percent excess risk arising for specific age groups.

The predicted concentrations of each substance are based on the year 2006. Thus, lifetime risk of diseases such as cancer are being estimated based on the air quality situation from one year. This assessment cannot account for past exposures from sources which no longer exist in the community. However, past exposures may contribute to current and future health problems.

## **Conclusions**

This study is a novel way of assessing cumulative health risks from multiple pollutants for a specific neighbourhood within a large urban area. For the first time, the contribution of pollution from different geographic areas and sectors to health risks at the local level was assessed. The results aid in setting priorities and determining effective strategies for pollution prevention that will make a difference in improving the health of Toronto residents.

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## Appendix A

### Toronto Air Pollutants: reference levels summary

#### Method for choosing RFCs:

For Air Toxics: Draw from CRELs which are based on annual exposure.

Where a MOE AAQC based on non-carcinogen endpoint is lower than a CREL, adopt AAQC

Priority is to select annual values where possible

#### Method for choosing IUR values:

Adopt CalEPA values which enable use of a complete list from a single source.

#### Method for choosing risk coefficients (representing percent excess risk)

Adopt AQBAT CRFs (Concentration response Functions) for all CACs

Air Pollutants	RFC = reference exposure concentration (eg., CREL) $\mu\text{g}/\text{m}^3$	Reference	Cancer Inhalation Unit Risk $(\mu\text{g}/\text{m}^3)^{-1}$	Reference	unit risk coefficient (premature mortality) units as noted	Reference
Acetaldehyde	140	1	2.70E-06	2	N/A	N/A
Acrolein	0.35	1	N/A	N/A	N/A	N/A
Benzene	60	1	2.90E-05	2	N/A	N/A
Benzo[a]Pyrene	N/A	N/A	1.10E-03	2	N/A	N/A
1,3-Butadiene	20	1	1.70E-04	2	N/A	N/A
Cadmium	0.005	3	4.20E-03	2	N/A	N/A
Carbon tetrachloride	2.4	3	4.20E-05	2	N/A	N/A
Chloroform	300	1	5.30E-06	2	N/A	N/A
Chloromethane	320	3	1.80E-06	4	N/A	N/A
Chromium III	0.5	3	N/A	N/A	N/A	N/A
Chromium VI	0.2	1	1.50E-01	2	N/A	N/A
1,4-Dichlorobenzene	95	3	1.10E-05	2	N/A	N/A
1,2-Dichloroethane (note: also called ethylene dichloride)	400	1	2.10E-05	2	N/A	N/A
Dichloromethane (note: also known as methylene chloride)	400	1	1.00E-06	2	N/A	N/A
Ethylene dibromide (note: also known as EDB or 1,2-Dibromoethane)	0.8	1	7.10E-05	2	N/A	N/A
Formaldehyde	9	1	6.00E-06	2	N/A	N/A
Lead	0.5	3	1.20E-05	2	N/A	N/A
Manganese	0.09	1	N/A	N/A	N/A	N/A
Mercury compounds	0.03	1	N/A	N/A	N/A	N/A
Nickel compounds	0.05	1	2.60E-04	2	N/A	N/A
Perchloroethylene (note: also known as PCE)	35	1	5.90E-06	2	N/A	N/A
Trichloroethylene	600	1	2.00E-06	2	N/A	N/A
Toluene	300	1	N/A	N/A	N/A	N/A
Vinyl chloride	N/A	N/A	7.80E-05	2	N/A	N/A
total VOC	do not include (eliminated since it will cause double-counting for the analysis)					
NO <sub>2</sub>	N/A	N/A	N/A	N/A	0.000748 ppb <sup>-1</sup>	5
PM <sub>2.5</sub>	N/A	N/A	N/A	N/A	0.00676 $\mu\text{g}/\text{m}^3$	5
PM <sub>10</sub>	do not include (eliminated since it will cause double-counting for the analysis)					
ozone	N/A	N/A	N/A	N/A	0.000839 ppb <sup>-1</sup>	5
CO	N/A	N/A	N/A	N/A	0.0019 ppm <sup>-1</sup>	5
SO <sub>2</sub>	N/A	N/A	N/A	N/A	0.000459 ppb <sup>-1</sup>	5

#### References

- 1 CalEPA CRELs (chronic) <http://oehha.ca.gov/air/allrels.html>
- 2 CalEPA Cancer Inhalation Unit Risk <http://www.oehha.ca.gov/risk/pdf/TCDBcas061809.pdf>
- 3 MOE AAQC: <http://www.ene.gov.on.ca/publications/6570e-chem.pdf>
- 4 State of New Jersey <http://www.state.nj.us/dep/standards/pdf/74-87-3-tox.pdf>
- 5 AQBAT CRFs (obtained direct from Health Canada)