Health Canada Santé Canada

Health Impacts of Air Pollution in Canada

Estimates of premature deaths and nonfatal outcomes

2021 Report

Canada

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Summary

A large body of scientific evidence has accumulated over the past 25 years attributing a wide range of adverse health effects to ambient (outdoor) air pollution exposure. These effects range in severity from respiratory symptoms to the development of disease and premature death. Significant advances in the health and atmospheric sciences over the last two decades have also made it possible to estimate the number of deaths and illnesses associated with air pollution. In Canada and internationally, health impact assessments identify air pollution as one of the largest risk factors for premature death and disability.

In this report, air pollution is defined as pollutants that scientific studies have associated with wide-ranging health effects and to which the population is ubiquitously exposed in the outdoor environment. These pollutants include fine particulate matter ($PM_{2.5}$), ground-level ozone, and nitrogen dioxide (NO_2). This is an update to previous health impacts of air pollution reports published by Health Canada. It relies on data and scientific knowledge, including ambient air pollution exposure estimates and demographic data from 2014 to 2017. The approach for quantitatively estimating the population health impacts of air pollution is well established by international health science organizations. In alignment with established approaches, Health Canada estimated 1) exposures to ambient air pollution across Canada, 2) the associated adverse health impacts in the population and 3) the corresponding economic costs of these health impacts. This analysis accounts for national demographics, including population counts, age profiles and baseline health status. Health impacts are presented nationally, as well as by province and territory (using 2016 population data).

This report considers Canadians' exposure to above-background levels. Above-background levels correspond to the difference between ambient concentrations and background concentrations. Background concentrations are equivalent to minimum ambient air pollution levels, such as those present in remote areas uninfluenced by human activity. The above-background air pollution is comprised mostly of human-source (anthropogenic) emissions, but it also includes emissions from natural events such as forest fires. Exposure to air pollutants in indoor environments was not considered. The focus on above-background air pollution is relevant to air quality management in Canada because policies and regulations to improve air quality generally target anthropogenic emissions. The national average exposure to above-background air pollution estimates were 4.3 $\mu g/m^3$ for $PM_{2.5}$, 7.2 ppb for NO_2, 13.2 ppb for annual ozone and 14.4 ppb for summer ozone. These averages are population-weighted to account for the geographic distribution of the Canadian population.

Health Canada estimates that above-background air pollution, including air pollution from human sources in North America, contributes to 15,300 premature deaths per year in Canada.[1] This includes an estimated 6,600 premature deaths in Ontario, 4,000 in Quebec, 1,900 in British Columbia and 1,400 in Alberta. National morbidity or nonfatal health outcomes include 2.7 million asthma symptom days and 35 million acute respiratory symptom days per year, with the total economic cost of all health impacts attributable to air pollution for the year being $120 billion (2016 CAD). This is equivalent to approximately 6% of Canada's 2016 real gross domestic product.

The mortality estimates reported in this assessment are based on risk information from epidemiological studies deemed to be the most relevant to Canada. The mortality endpoints include all-cause mortality[2] associated with long-term exposure to ambient $PM_{2.5}$, and short-term exposure to NO_2 and annual ozone, as well as respiratory mortality associated with long-term exposure to warm-season ozone. All risk information contributing to Health Canada's mortality estimates was derived from Canadian cohort and time-series studies, with the exception of chronic exposure respiratory mortality associated with ozone that was derived from an American cohort. In the context of this analysis, short-term exposure is related to effects that occur a few days after an elevation in ambient air pollution (i.e. acute health effects), while long-term exposure refers to exposures averaged over the years preceding the development of disease or death (i.e. chronic health effects).

[1] The reference year for the health impact estimates is 2016 and corresponds with the population data considered for analysis.

[2] All-cause nonaccidental mortality

In addition to the effects of changes in air pollution exposure over time, population growth also influences the overall health burden of air pollution, leading to a greater number of exposed individuals and resulting in an overall net increase in premature deaths attributable to air pollution. These variations in population can be standardized by presenting the death rates per 100,000 population. The current estimate of 15,300 premature deaths is equivalent to 42 premature deaths per 100,000 Canadians. Health Canada estimated, in 2017, 14,400 premature deaths per year, and, in 2019, 14,600 premature deaths per year, which were equivalent to 41–42 deaths per 100,000 Canadians.[3]

In this analysis, the air pollution-associated premature death and nonfatal effect counts are based on exposure to ambient concentrations of $PM_{2.5}$, NO_2 and ozone exclusively. These pollutants are included because there is robust epidemiological evidence of their adverse health impacts as well as the ability to accurately estimate the spatial distribution of their ambient concentrations across Canada. However, owing to data limitations and knowledge gaps, not all health effects that have been associated with exposure to $PM_{2.5}$, NO_2 and ozone in the scientific literature can currently be quantified. Further, there are other air contaminants that contribute to air pollution health impacts, but they are beyond the scope of this work. The quantitative estimates of population health effects provided in this report are therefore assumed to underestimate the full impact of exposure to air pollution in Canada. Overall, this analysis indicates that despite the relatively low levels of air pollutants in Canada compared to other regions of the world, air pollution continues to impact population health.

[3] The population reference year was 2011 for the Health Canada report published in 2017, 2015 for the report published in 2019, and 2016 for the current analysis.

1. Introduction

Air pollution is recognized globally as a major contributor to the development of disease and premature death and represents the largest environmental risk factor to human health (WHO 2016). Exposure to air pollution increases the risk of premature mortality from heart disease, stroke and lung cancer.[4] The health and atmospheric sciences have advanced significantly in the past two decades, making it possible to estimate the number of deaths and illnesses associated with air pollution. These values are estimated using information from the peer-reviewed scientific literature, which relates population-level pollution exposure (both short-term and long-term) to the risk of adverse health outcomes, including premature death and hospital visits. The quantitative relationship between exposure and increased risk of adverse health outcomes is referred to as the concentration-response function (CRF). Estimates of air pollution-attributable deaths and other adverse health outcomes have been developed globally and for many individual countries, including by Cohen et al. (2017), the Institute for Health Metrics and Evaluation (IHME) and the Health Effects Institute (HEI) (2018), and the World Health Organization (WHO) (2016).

According to the Global Burden of Disease (GBD) project, air pollution is the fifth leading mortality risk in the world and was responsible for 8.7% of deaths globally in 2017 (or 4.9 million premature deaths worldwide) (IHME and HEI 2019). Internationally, Canada is among the top 10 countries with the lowest national $PM_{2.5}$ exposure levels (IHME and HEI 2019). According to the GBD analyses, air pollution ranks as the 11th largest risk factor overall for premature death and disability in Canada, and is the top environmental risk.[5]

Estimates of air pollution-attributable mortality in Canada have previously been developed by Health Canada (2017, 2019), Stieb et al. (2015), the Canadian Medical Association (2008), and as part of the GBD project.[6] The previous edition of this report (Health Canada 2019) estimated that 14,600 premature deaths were associated with ambient air pollution exposure in 2015. In this context, air pollution is defined as pollutants that scientific studies have associated with wide-ranging health effects and to which the population is ubiquitously exposed. These pollutants include $PM_{2.5}$, ground-level ozone, and NO_2. While both sulphur dioxide and carbon monoxide are also ubiquitous in Canada and have also been associated with such effects in some studies, they appear to have far less important impact than the three pollutants listed above.

Estimates of air pollution-attributable fatal and nonfatal outcomes are expected to change over time as a result of our improving understanding of the relationship between exposure and risk and the spatial representation of air pollution exposure. For example, new scientific information may support or confirm the inclusion of additional causes of death associated with air pollution. In addition, new air pollution exposure data and modelling tools provide more accurate air pollution level estimates with improved spatial and temporal resolution for all regions of Canada. Changes in population health and demographics, including the aging population, will influence the number of health outcomes attributable to air pollution exposure.

The objective of this analysis is to draw on the most recent data and scientific knowledge to provide comprehensive and up-to-date estimates of morbidity and mortality outcomes in Canada related to ambient levels of $PM_{2.5}$, ozone and NO_2. These pollutants were included because there is robust epidemiological evidence of their adverse health impacts as well as the ability to accurately estimate the spatial distribution of their ambient concentrations across Canada. Estimates are provided at the national, provincial and territorial levels. In addition, an economic valuation of health impacts is presented. The methods described here are considered to be comprehensive and appropriate for the Canadian context. Exposure to air pollutants in indoor environments was not considered.

[4] It must be noted that multiple risk factors are involved in the development or worsening of adverse health effects. While air pollution can contribute to increased risk of population health impacts, this does not necessarily imply that air pollution is the sole cause. Exposure to air pollution is a contributing risk factor to the development of adverse health effects.
[5] Institute for Health Metrics and Evaluation (IHME). GBD Country profiles – 2017. http://www.healthdata.org/canada (accessed December 12, 2019)
[6] Ibid.

2. Methods

2.1 Pollutants included in the estimate

This analysis of air pollution health impacts in Canada focuses on $PM_{2.5}$, NO_2, and ozone. Emissions from local, regional, national and international sources directly (primary emissions) and indirectly (secondary formation) contribute to the presence of these pollutants in the country's ambient air. Fuel combustion, including from mobile (e.g. on-road vehicles and off-road equipment) and power generation (e.g. coal or natural gas) sources, directly releases particles and nitrogen oxides (NOx) into the air. In addition, combustion emits a suite of organic and inorganic compounds that contribute to secondary $PM_{2.5}$ and ozone. Ozone is not emitted directly, but formed from precursors such as NOx and volatile organic compounds (VOCs) via secondary reactions in the atmosphere and reactions with sunlight. Health Canada and other international agencies have concluded that $PM_{2.5}$, NO_2 and ozone cause or are likely to cause premature mortality based on extensive evidence from epidemiological studies (e.g. Health Canada 2013, 2016; US EPA 2019). These three pollutants also account for the majority of population health impacts from air pollution. There is robust scientific evidence of health effects at very low concentrations of these pollutants, and no evidence of an exposure threshold in the population. In other words, any incremental increase in air pollutant concentration is associated with an increased risk of adverse health outcomes. General information on emissions and ambient concentrations of NO_2, ozone, and $PM_{2.5}$ in Canada as well as the associated adverse health effects are presented in Appendix A.

2.2 Estimating population exposures to above-background air pollution

The current analysis estimates the mortality and morbidity outcomes associated with ambient air pollution corresponding to above-background levels. While most of the above-background increment is linked to human source (anthropogenic) emissions originating from North America, natural emissions are also included, notably from wildfires. Health impacts associated with "background" pollutant concentrations (which include emissions from other natural sources and sources beyond North America) were not included.[7] This measurement of above-background air pollution is relevant to air quality management in Canada, as policies and regulations generally target anthropogenic emissions to improve air quality. High-resolution estimates of ambient concentrations of $PM_{2.5}$, NO_2, and ozone were used to estimate population-level exposures across Canada. These estimates, which are presented graphically in Figures 1–3, were generated using a combination of ground-level measurements, satellite data, geographic and land-use information, as well as computer model simulations. Background concentrations were then subtracted to obtain the exposure data included in this analysis.

2.2.1 Background concentrations of air pollution

Background concentrations of $PM_{2.5}$, NO_2 and ozone were estimated in collaboration with Environment and Climate Change Canada (Judek et al. 2004). This complex initiative involved a combination of qualitative (i.e. expert judgment) and quantitative (i.e. data-driven) approaches to evaluate concentration measurements at rural and remote monitoring sites. Background concentrations were estimated using either one of the following methods:

1. The data from rural and remote monitoring sites were separated into sectors of different air mass origin, and the background concentrations were selected as the monthly or annual average concentrations for the sectors containing no major anthropogenic sources; or

[7] Although some authors make a distinction between baseline (natural and long-range air pollution contributions) and background (natural contributions only) conditions (TFHTAP 2010), the term "background" is used herein to represent all contributions other than those from North American anthropogenic sources or large natural events such as forest fires. This is comparable to the term "North American Background" (formerly "policy relevant background") used by the United States Environmental Protection Agency (CRS 2019).

2. Many years of rural and remote measurement data were plotted in a time series allowing a qualitative selection of the lowest values that are considered to be the most representative of background air masses.

This resulted in annual average background concentrations for NO_2 and $PM_{2.5}$. A set of monthly-average background concentrations were derived for ozone, for which the ambient concentrations have a strongly seasonal cycle. These monthly averages were then combined into summer and annual average concentrations to be consistent with those used to quantify health risks. Regional differences in background concentrations are likely, but for the purposes of this analysis, a single background concentration was applied across Canada for each pollutant.[8] The estimated background concentrations for Canada are as follows:

- 1.8 micrograms per cubic metre ($\mu g/m^3$) for $PM_{2.5}$ (annual average).
- 0.15 parts per billion by volume (ppb) for NO_2 (annual average).
- 26 ppb for annual ozone (annual average of daily 1-h maximum) and 28 ppb for summer ozone (May–September average of daily 1-h maximum).

2.2.2 Above-background air pollution

To estimate the population health impacts attributable to above background air pollution, it is necessary to calculate the above-background air pollution increment. Air pollution levels are known to vary geographically and can be estimated using a combination of observed and simulated concentrations. Routine ground-level air pollution monitoring in Canada occurs at discrete monitoring stations across the country, which limits the geographic coverage of air pollution exposure estimates that rely solely on direct measurements. For this assessment, we relied on spatially resolved estimates of ambient air pollution levels (including both anthropogenic and natural sources, and non–North American contributions) for $PM_{2.5}$, NO_2 and ozone, produced through a combination of data sources, including ambient monitoring, as described below. In contrast, a single background concentration for each pollutant was developed and was assumed to apply across Canada (as described in the previous section).

2.2.3 Assignment of concentrations to populations

Air pollution concentration estimates for NO_2, $PM_{2.5}$ and ozone were generated and mapped to the Canadian population (using the 2011 census, with population counts for 2016). Ambient concentrations were averaged over three years of available data (between 2014 and 2017) to ensure that results were not influenced by any interannual variations in concentrations. Abnormal weather patterns and air pollution events, including wildfires and stay-at-home orders, are possible causes of interannual variations (Griffin et al. 2020; Matz et al. 2020; Zangari et al. 2020). Air pollution concentrations were estimated for up to 293 census divisions (CDs).[9] Figures 1 to 3 present maps of population-weighted ambient air pollutant concentrations for annual average $PM_{2.5}$, annual 1-h daily maximum ozone, summer 1-h daily maximum ozone (i.e. May–September), and annual average NO_2. The data displayed in these maps represent the estimated distribution of ambient air concentrations from all natural and anthropogenic sources. Canadian background concentrations were then subtracted to estimate exposures to above-background ambient air pollution concentrations. The methods used to estimate air pollutant levels are detailed in the following subsections.

2.2.3.1 Fine particulate matter

Annual average $PM_{2.5}$ concentrations for 2015–2017 were derived from optimal estimation methods combining remote-sensing observations, chemical transport modelling and ground-based observations (van Donkelaar et al. 2015a). Aerosol optical depth (AOD) data were obtained from three satellite instruments: Multi-angle Imaging SpectroRadiometer (MISR), Moderate Resolution Imaging Spectroradiometer (MODIS), and Sea-viewing Wide Field-of-view Sensor (SeaWiFS) (Boys et al. 2014; Crouse et al. 2015; Stieb et al. 2015; van Donkelaar et al. 2010, 2013, 2015a). AOD is a vertically integrated measurement of light extinction in the

[8] Estimates of background concentrations are revisited periodically by Environment and Climate Change Canada scientists. An assessment conducted in 2018 determined that the original estimates provided by Judek et al. (2004) were still representative of Canadian background concentrations (Bob Vet, Environment and Climate Change Canada; personal communications).

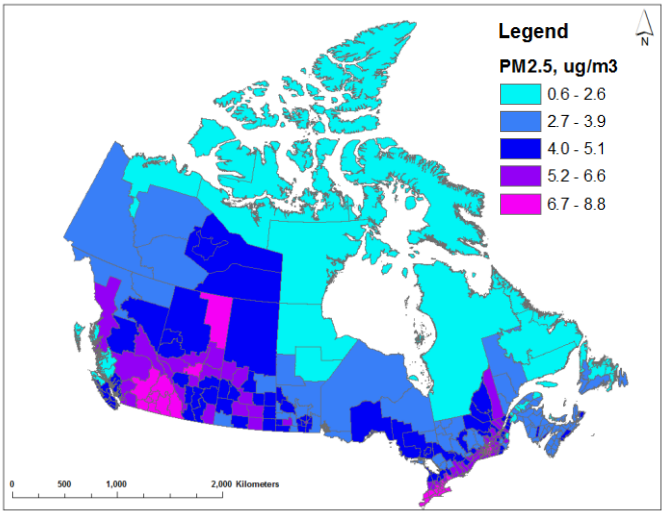
[9] Missing data are associated with a small number of census divisions in remote and low-population areas in the North.

atmosphere, which is associated with aerosols. Factors such as the vertical distribution and composition of aerosols, as well as humidity and other meteorological conditions, can influence estimates of ground-level $PM_{2.5}$ concentrations based on AOD measurements. To account for these factors, AOD values can be normalized or adjusted using output from chemical transport models and ground-based observations. For the current assessment, AOD data were combined with information obtained from the Goddard Earth Observing System chemical transport model (GEOS-Chem) and Canada's National Air Pollution Surveillance (NAPS) network (ground-based air pollutant monitoring) to provide final national estimates of $PM_{2.5}$ levels (van Donkelaar et al. 2015b).

The annual average $PM_{2.5}$ concentration estimates were generated as a gridded surface with a spatial resolution of approximately 1 km × 1 km. The grid cell values were then converted to a point dataset and merged with a dataset representing postal code areas. The nearest point was assigned to each postal code. The postal code results were then combined with dissemination area (DA) population data to calculate population-weighted concentrations for each CD.

Figure 1 shows the distribution of annual average $PM_{2.5}$ concentrations for the years 2015 to 2017. The national population-weighted average ambient $PM_{2.5}$ concentration is 6.1 µg/m^3 during the period of interest. As expected, higher $PM_{2.5}$ concentrations are observed in many of the more populous CDs, such as those in the Lower Fraser Valley of British Columbia, the Calgary–Edmonton Corridor in Alberta, and along the Windsor–Quebec City Corridor in Ontario and Quebec (Figure 1).

Figure 1. Three-year population-weighted average of daily $PM_{2.5}$ concentrations across Canadian census divisions – 2015–2017 (includes air pollution from all sources)



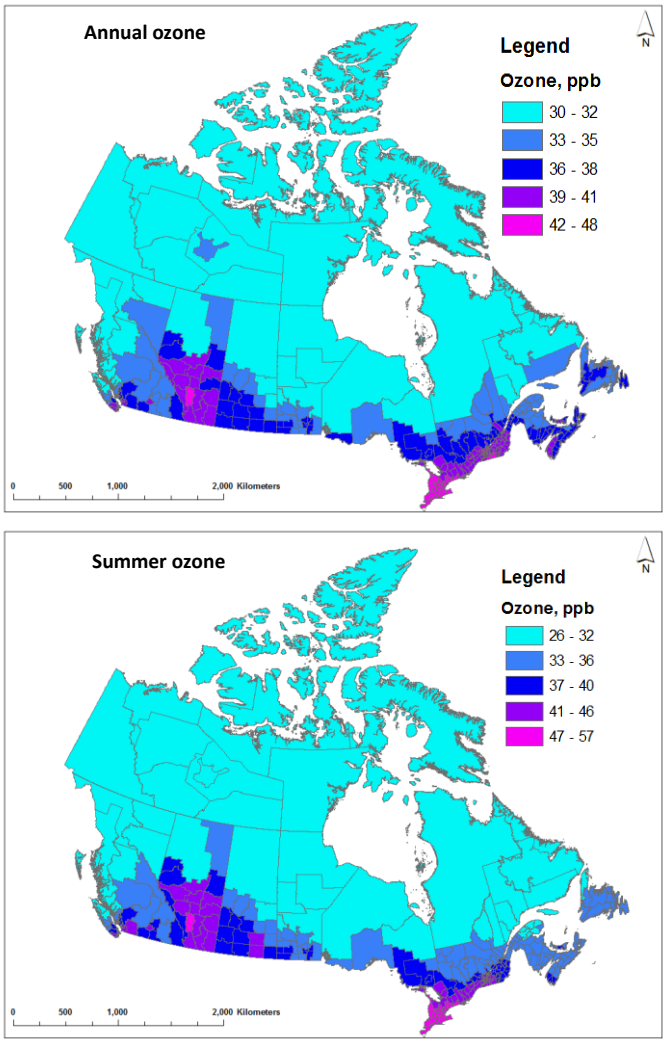
2.2.3.2 Ozone

Estimates of both the (1) annual ozone average and (2) summer ozone average (May–September) were derived from daily 1-h maximum concentrations for 2014, 2015 and 2017. Data for 2016 were not available owing to operational considerations for the underlying model. These estimates were produced by Environment and Climate Change Canada using objective analysis, an interpolation technique that weighs and combines modelled ozone forecasts with observations of ozone (Robichaud and Ménard 2014; Kalnay 2003). The model led ozone forecast was provided by the Global Environmental Multiscale - Modelling Air quality and Chemistry (GEM-MACH) system, Environment and Climate Change Canada's operational regional air quality forecast model (e.g. Makar et al. 2018; Moran et al. 2010; Whaley et al. 2018). Ozone measurements were obtained from the Canadian Air and Precipitation Monitoring Network (CAPMoN) and the Canadian NAPS network. In objective analysis, the optimal combination of modelled and observed values improves the coverage and accuracy of air pollution patterns (Robichaud et al. 2016). Objective analysis leads to better estimates of ambient ozone concentrations in areas lacking monitoring data compared to standard interpolation techniques (such as spatial kriging). Estimates for Canada are available for 2014, 2015 and 2017,

on a grid point surface with a horizontal resolution of 10 km x 10 km. The grid point estimates were then interpolated to CD polygons (using a normalized conservative approach). All grid points within and bordering CD polygons were included, wholly or partially, to estimate the average ozone concentration values by CD.

Figure 2 (top panel) shows the distribution of the annual average of daily 1-h maximum ozone concentrations for the years 2014, 2015 and 2017. The distribution of summer-average daily 1-h maximum ozone is similar (Figure 2–bottom panel). Higher ozone concentrations are observed in the Lower Fraser Valley of British Columbia, in southern Alberta and along the Windsor–Quebec City Corridor, including many of the more populous CDs in Canada. As environmental and meteorological conditions in warmer months promote the formation of ozone, higher concentrations are also observed in the summer: The national population-weighted average ambient concentrations are 39.2 ppb for annual ozone and 42.4 ppb for summer ozone.

Figure 2. Three-year population-weighted annual (top panel) and summer (bottom panel) average of the daily 1-h maximum ozone concentrations across Canadian census divisions – 2014, 2015 and 2017 (includes air pollution from all sources)



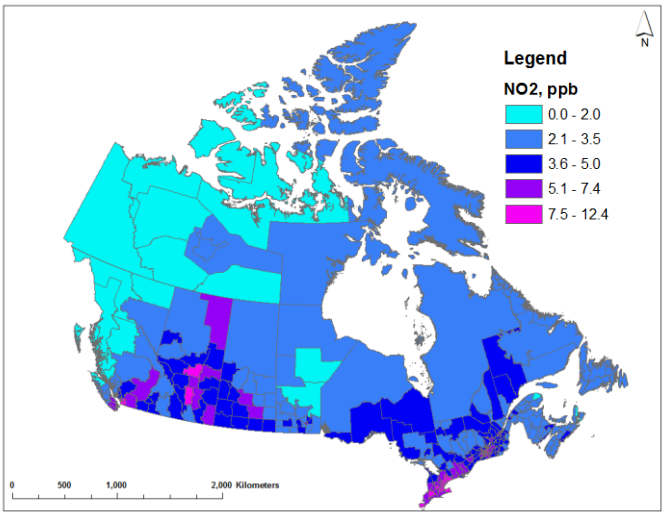
2.2.3.3 Nitrogen dioxide

Annual average NO_2 concentrations were estimated using a national land-use regression (LUR) model for 2015–2017 (Larkin and Hystad 2020). The LUR model predictors included three-year annual average NO_2 concentrations for 2015, 2016, and 2017 using NO_2 vertical column densities (NASA Earth Observations

database[10]) from the Ozone Monitoring Instrument (OMI), as well as land use and meteorological descriptors (e.g. Boersma et al. 2011; Hystad et al. 2011; Lamsal et al. 2008). In addition to the OMI data, the model predictors were population density, railways, temperature, industrial use, highways and expressways, and the normalized difference vegetation index (NDVI). The NO_2 estimates were developed on a high-resolution grid (30 m) in order to best capture the fine spatial gradients in NO_2 concentrations. The LUR model performance was assessed by comparing predicted and observed NO_2 concentrations. Observations corresponded with the three-year annual average NO_2 data from the NAPS network for 2015, 2016, and 2017 (180 monitoring stations). A coefficient of determination (R^2) of 0.68 was reported between the NO_2 model results and the corresponding NAPS data (Larkin and Hystad 2020).

In this analysis, the 2015–2017 annual average NO_2 estimates were derived for dissemination block (DB) centroids (or nearest valid location). Estimates were available for 486,676 DBs (2016 Census). DB estimates ranged from 0 to 20 ppb, with a mean of 5.4 ppb. The DB results were used to calculate population-weighted concentrations for each CD. Figure 3 shows the distribution of annual average NO_2 concentrations, averaged over 2015 to 2017. The national population-weighted average ambient concentration is 7.4 ppb for NO_2. As is the case for $PM_{2.5}$ and ozone, higher NO_2 concentrations were observed in southwestern British Columbia, around the Calgary–Edmonton Corridor in Alberta, in southern Saskatchewan, and along the Windsor–Quebec City Corridor in Ontario and Quebec.

Figure 3. Three-year population-weighted average of daily NO_2 concentrations across Canadian census divisions – 2015–2017 (includes air pollution from all sources)



2.3 Estimating premature deaths and nonfatal outcomes from air pollution

This analysis used Health Canada's Air Quality Benefits Assessment Tool (AQBAT) version 3.0 (Judek et al. 2019; Xu and Stieb[11]) to link population-level above-background air pollution exposure to health outcomes. The AQBAT model estimates the number of premature deaths and other health outcomes associated with specified changes in air pollution concentrations across geographic units (i.e. CDs) in Canada. Outcomes can then be aggregated to provincial, territorial and national health impact estimates, as was done here. Health effect information for the three air pollutants is included in the form of CRFs. A CRF represents the excess health risk for a given endpoint, such as asthma symptoms, chronic bronchitis, and mortality, that follows a unit increase in ambient pollutant concentration. For example, an increase in $PM_{2.5}$ chronic exposure of 10 $\mu g/m^3$ leads to a corresponding 10% increase in the risk of premature mortality from nonaccidental causes. A CRF is a statistically derived estimate, from a single study or a meta-analysis of multiple studies.

[10] [NASA] National Aeronautics and Space Administration. Aura Satellite.
[11] Guoliang Xu and Dave Stieb; personal communications, Health Canada, 2019

Health endpoints (related to acute or chronic exposure), the associated CRFs and the applicable population group(s) (e.g. age-specific groups) are predefined in AQBAT. In the context of this analysis, short-term exposure contributes to effects that occur within a few days of an increase in ambient air pollution (acute health effects), while long-term exposure refers to exposures averaged over the years preceding the development of disease or death (chronic health effects). Pollutant-specific CRFs for individual adverse health outcomes are drawn from the health science literature and are the consensus selection of a panel of Health Canada experts. They are therefore Health Canada-endorsed values. Table 1 presents the pollutants and their associated health effects considered by this analysis. Previous studies (e.g. Crouse et al. 2012; Judek et al. 2019; Shin et al. 2013; Stieb et al. 2015) contain background information on the CRF estimates used in this analysis (i.e. references to the scientific literature upon which the risk estimates are based) and the analysis undertaken to produce the estimates within AQBAT. This information is also summarized in Appendix B. Health outcomes were considered to have no threshold for effect (i.e. effects were assumed to occur at all levels of exposure), which is consistent with Health Canada's conclusions upon evaluation of the overall literature on each of these pollutants (Health Canada 2013, 2016).

Table 1. Averaging periods and associated acute and chronic health endpoints for NO_2, ozone and $PM_{2.5}$ CRFs in AQBAT version 3.0

Pollutant[a]	Averaging period	Health endpoint	Causality determination[d]	Reference
NO_2	24-h	Acute exposure mortality[b,c]	Likely causal	Health Canada 2016
Ozone	1-h maximum	Acute exposure mortality[b]	Likely causal	Health Canada 2013
Summer ozone	1-h maximum	Acute respiratory symptom days	Causal	Health Canada 2013
		Asthma symptom days	Causal	Health Canada 2013
		Chronic exposure respiratory mortality	Suggestive to be causal	Health Canada 2013
		Minor restricted activity days	Causal	Health Canada 2013
		Respiratory emergency room visits	Causal	Health Canada 2013
		Respiratory hospital admissions	Causal	Health Canada 2013
$PM_{2.5}$	24-h	Acute respiratory symptom days	Causal	Health Canada 2013
		Adult chronic bronchitis cases	Suggestive to be causal	Health Canada 2013
		Asthma symptom days	Causal	Health Canada 2013
		Cardiac emergency room visits	Causal	Health Canada 2013
		Cardiac hospital admissions	Causal	Health Canada 2013
		Child acute bronchitis episodes	Causal	Health Canada 2013
		Chronic exposure mortality	Causal	Health Canada 2013
		Respiratory emergency room visits	Causal	Health Canada 2013
		Respiratory hospital admissions	Causal	Health Canada 2013
		Restricted activity days	Causal	Health Canada 2013

AQBAT: Air Quality Benefits Assessment Tool; CRF: concentration–response function; NO_2: nitrogen dioxide; ozone: ozone; $PM_{2.5}$: fine particulate matter or particulate matter with a diameter of 2.5 µm or less

[a] Unless otherwise specified, CRFs reflect an exposure to the pollutant at any time during the year.

[b] CRFs relating acute exposure mortality and gaseous pollutants are from a copollutant model including CO, NO_2, ozone and SO_2 and may not precisely reflect the true attribution of risk to individual pollutants.

[c] It is recognized that the CRF for NO_2-related acute exposure mortality may reflect a likely causal relationship with NO_2, or NO_2 may be acting as a surrogate for a specific component of the air pollution mixture, such as vehicle exhaust emissions.

[d] **Causal:** Evidence is sufficient to conclude that there is a causal relationship with relevant pollutant exposures; **Likely to be causal:** Evidence is sufficient to conclude that a causal relationship is likely to exist with relevant pollutant exposures, but important uncertainties remain; **Suggestive to be causal:** Evidence is suggestive of a causal relationship with relevant pollutant exposures, but is limited.

Population-level premature deaths were estimated using the following CRFs and health endpoints:

- Nonaccidental causes of death[12] associated with long-term exposure to $PM_{2.5}$ from a Canadian cohort (Crouse et al. 2012).
- Nonaccidental causes of deaths associated with short-term exposure to NO_2 and ozone from a Canadian time-series analysis (Burnett et al. 2004).
- Death from respiratory causes associated with long-term exposure to summer ozone from an American cohort (Jerrett et al. 2009).

CRF values for premature deaths are shown in Table 2 and for all health endpoints in Appendix B. Table 2 also summarizes methodological considerations for the current health impact assessment, including a list of fatal and nonfatal health effects associated with each air pollutant, data sources for estimating exposures to air pollution, and national population-weighted average exposure estimates.

CRFs can be input as a distribution function in the calculations, accounting for inherent uncertainty in the CRF estimates. Monte Carlo simulations employing 10,000 iterations were used to propagate this uncertainty in the CRFs. The model generates a central estimate of the most likely health impacts equal to the median of the output distribution, as well as low- and high-end estimates equal to the 2.5 and 97.5 percentiles of the output distribution.

CRF values for $PM_{2.5}$ mortality from ischemic heart disease (IHD), cerebrovascular disease (CVD), lung cancer and chronic obstructive pulmonary disease (COPD) for adults 25 years and over, are also included in AQBAT (Shin et al. 2013).[13] These four individual causes correspond to the approach employed by the IHME and the WHO in the GBD analyses (Cohen et al. 2017; Lim et al. 2012; www.healthdata.org/gbd), which include estimates of air quality and health impacts across the world. IHD, CVD, lung cancer and COPD are a subset of nonaccidental chronic exposure mortality (i.e. nonaccidental deaths), which are reported herein. For the purposes of this report, nonaccidental causes of death is considered the most appropriate premature mortality metric associated with chronic exposure to $PM_{2.5}$; it generally leads to higher mortality estimates than the sum of specific causes of death.

[12] Nonaccidental causes of death are referred to as (all) internal causes of death in previous Health Canada publications on the health impacts of air pollution in Canada (2017, 2019).

[13] The user has the option of using the "all-cause" nonaccidental mortality endpoint or the four individual causes.

Table 2. Summary of health effects and exposure estimates considered in the health impacts analysis

Health effects	**$PM_{2.5}$**	**NO_2**	**Annual ozone**	**Summer ozone**
Cause of death and exposure type **% increase per change** **[key reference]**	All[a] – chronic 10% per 10 µg/m^3 [Crouse et al. 2012]	All[a] – acute 1.5% per 20 ppb [Burnett et al. 2004[c]]	All[a] – acute 1.7% per 20 ppb [Burnett et al. 2004]	Respiratory – chronic 8.2% per 20 ppb [Jerrett et al. 2009]
Morbidity outcomes	Acute Respiratory Symptom Days; Adult Chronic Bronchitis Cases; Asthma Symptom Days; Cardiac ER Visits; Cardiac HA; Child Acute Bronchitis Episodes; Respiratory ER Visits; Respiratory HA; Restricted Activity Days	None	None	Acute Respiratory Symptom Days; Asthma Symptom Days; Minor Restricted Activity Days; Respiratory ER Visits; Respiratory HA
National exposure estimates	**$PM_{2.5}$**	**NO_2**	**Annual ozone**	**Summer ozone**
Years of exposure data	2015–2017	2015–2017	2014, 2015, 2017	
Type of exposure data **[key reference]**	Satellite observations, ground observations (NAPS), and chemical transport models (GEOS-Chem) [van Donkelaar et al. 2015a,b]	Satellite observations, ground observations (NAPS), geographic data (e.g. land use, distance to roadways) [Larkin and Hystad 2020]	Objective analysis of ground observations (NAPS) and chemical transport model (GEM-MACH) [Robichaud and Ménard 2014]. Annual and summer (May–September) averages	
Average ambient concentration[b]	**6.1 µg/m^3**	**7.4 ppb**	**39.2 ppb**	**42.4 ppb**
Natural background concentration	**1.8 µg/m^3**	**0.15 ppb**	**26 ppb**	**28 ppb**
Average air pollution concentration[b,c]	**4.3 µg/m^3**	**7.2 ppb**	**13.2 ppb**	**14.4 ppb**

CRF: concentration-response function; ER: emergency room; GEM-MACH: Global Environmental Multiscale - Modelling Air quality and CHemistry; GEOS-Chem: Goddard Earth Observing System chemical transport model; HA: hospital admissions; µg/m^3: micrograms per cubic metre; NAPS: National Air Pollutant Surveillance network; ppb: parts per billion by volume

[a] All – nonaccidental causes
[b] National population-weighted concentrations
[c] Above-background air pollution

2.3.1 Baseline incidence rates

Baseline incidence rates of the health endpoints considered are a key factor in estimating the count of health outcomes associated with a change in pollutant concentration. Baseline incidence rates are pre-defined in AQBAT as a model parameter. These rates are estimated through detection, observation and formal means of reporting (e.g. death certificates, hospital admission records) based on data provided by Statistics Canada or epidemiological studies (e.g. Abbey et al. 1995; Hoek et al. 2012; Krupnick et al. 1990; Ostro 1987; Osto and Rothschild 1989; Weinmayr et al. 2010). They are expressed in annual events per million people. Details regarding the source data, formulas, and algorithms used to estimate annual baseline health endpoint incidence rates are provided in the AQBAT user guide (Judek et al. 2019).

Baseline cause- and age-specific mortality rates were derived from counts of mortality obtained for each CD outside of Québec, where these data were not available. Rates are averaged over the three most recent years of available data to improve stability (Stieb et al. 2015). For Quebec, mortality counts were derived for each CD by applying national age- and cause-specific rates to the CDs' individual population age distribution. For each morbidity and mortality health endpoint in AQBAT, a data file contains estimated annual events per million specified people for every geographic area, age group, scenario year and population projection. The national, provincial and territorial baseline rates for health endpoints associated with NO_2, ozone and $PM_{2.5}$ in the current version of AQBAT are summarized in Appendix C.[14]

Incidence rates are generally associated with many factors, such as age, gender, race, education, income, environmental factors and lifestyle habits. Age-specific baseline incidence rates (for the health endpoints in question) for the target population were included to estimate the number of excess health outcomes associated with the increased risk following a change in air pollutant concentration. Annual baseline health endpoint rates of events are assigned to specific populations that correspond to those examined in the underlying epidemiologic studies. For example, the Restricted Activity Days endpoint is assigned to 94% of people aged 20 years and older (i.e. nonasthmatics). Exposure to pollutants typically has a minor influence on the baseline incidence rates. Additional details and references on the process of deriving baseline rates have previously been published (Judek et al. 2019; Stieb et al. 2015).

2.4 Estimating the economic value of health outcomes from air pollution

Estimating the economic value (or valuation) of air pollution-related health impacts monetizes health outcomes, allowing impacts to be expressed in monetary units. In doing so, the potential social, economic and public welfare consequences of a health endpoint are considered, including medical costs, reduced workplace productivity, pain and suffering, and the other effects of increased health risks. Expressing impacts in monetary terms provides a common metric across health endpoints to estimate the overall benefits or damages in order to inform air quality management strategies. The sum provides an indication of the relative benefits or damages to society resulting from reduced or increased risks to health.

In AQBAT, each health endpoint is assigned a monetary value, typically derived from surveys, accounting or economic or actuarial data. The valuation estimates used in the model, along with references to the studies from which they are derived, are provided in Table 3. Endpoint valuations have inherent uncertainties, which are captured by a distribution of possible values within the model and defined by a distribution form and a set of parameters (Table 3). Endpoint valuations are expressed in Canadian dollars and can be temporally adjusted from the source years of the underlying studies based on the consumer price index, as defined by Statistics Canada (Judek et al. 2019; Statistics Canada, annual). In the current analysis, the currency year 2016 (2016 CAD) was used.

[14] The full list of baseline rates is provided with the AQBAT model package or can be obtained upon request from Health Canada at hc.air.sc@canada.ca.

Table 3. Economic value of health endpoints used in Health Canada's AQBAT model

Endpoint [reference]	Currency year of original study	Source type	Form[a]	Parameter 1 (prob.)	Parameter 2 (prob.)	Parameter 3 (prob.)
Mortality [Chestnut and De Civita 2009]	2007	WTP/ WR	Discrete	$3,500,000 (25%)	$6,500,000 (50%)	$9,500,000 (25%)
Acute respiratory symptom days [Stieb et al. 2002]	1997	WTP	Normal	$13	$7	–
Adult chronic bronchitis cases [Krupnick and Cropper 1992; Viscusi et al. 1991]	1996	WTP	Discrete	$175,000 (33%)	$266,000 (34%)	$465,000 (33%)
Asthma symptom days [Stieb et al. 2002]	1997	WTP	Triangular	$7	$28	$120
Cardiac emergency room visits[b] [Stieb et al. 2002]	1997	WTP	Normal	$4,400	$590	–
Child acute bronchitis episodes [Krupnick and Cropper 1989]	1996	WTP	Discrete	$150 (33%)	$310 (34%)	$460 (33%)
Elderly cardiac hospital admissions [Stieb et al. 2002]	1997	WTP	Normal	$5,200	$610	–
Minor restricted activity days [Stieb et al. 2002]	1997	WTP	Normal	$22	$9	–
Respiratory emergency room visits[b] [Stieb et al. 2002]	1997	WTP	Normal	$2,000	$210	–
Restricted activity days [Stieb et al. 2002]	1997	WTP	Normal	$48	$18	–

Adapted from Judek et al. (2019)

prob.: probability of value being selected in the analysis; WR: wage risk; WTP: willingness to pay

[a] For valuations represented by discrete values, parameters 1, 2 and 3 represent low, medium and high estimates respectively. For valuations represented by normal distributions, parameters 1 and 2 represent the mean and standard error of the estimates respectively. For valuations represented by triangular distributions, parameters 1, 2 and 3 represent minimum, most likely and maximum values respectively.

[b] Respiratory and cardiac emergency room visits include the costs of subsequent hospital admissions, which are calculated based on the proportion of emergency room visits that result in admission to hospital. Hospital admissions are assigned a value of zero to avoid double-counting of costs.

As evident in Table 3, the monetary value of mortality is considerably higher than that of any other health endpoint. For the purposes of policy analysis, the recommended central estimate of an avoided premature death is $6.5 million (CAD 2007) based on a review of Canadian studies by Chestnut and De Civita (2009). The underlying data indicate that an average Canadian would be willing to pay approximately $65 in order to reduce the risk of premature death by 1 out of 100,000. The aggregate willingness to pay (WTP) of 100,000 Canadians ($65 each) equals the value of the one avoided death. The uncertainty in this estimate is addressed by a recommended low value of $3.5 million and a high value of $9.5 million (Table 3). These values provide a reasonable range of WTP but should not be considered as lower and upper bounds (Chestnut and De Civita 2009). The above values are not equivalent to the economic worth of an identified person's life, but rather an aggregation of individual values people are willing to pay for small changes in risk.[15] Following adjustments based on the consumer price index, the value of an avoided premature death in 2016 CAD is $7.5 million.

[15] Empirical studies of willingness to pay (WTP) for mortality risk reductions provide estimates of the average monetary amounts that individuals are willing to pay for small reductions in premature mortality. The valuation context or individual circumstances influence WTP values; that is, they may vary for the same amount of risk reduction in different contexts and for different individuals. WTP reflects all the reasons individuals put a value on the reduction of their own risk of death. Therefore, it can exceed the value of the financial impact on an individual associated with the change in risk.

3. Results

Table 4 presents the health impact and economic value results for mortality endpoints associated with $PM_{2.5}$, ozone and NO_2 air pollution for national, provincial and territorial geographies. Metrics in Table 4 include the count of incidences and normalized values per 100,000 population. The latter metric allows for comparisons of health impact estimates among geographic regions of different population sizes. All results represent the health impacts attributable to above-background concentrations, as outlined in the Methods section. The Canadian values presented herein have not previously been published.

Overall, the total mortality attributable to above-background air pollution in Canada was estimated to be 15,300 premature deaths per year, based on population data for 2016 and air pollutant concentrations from 2014 to 2017.[16] More specifically, the following population health impacts of $PM_{2.5}$, ozone and NO_2 were estimated:[17]

- Chronic exposure to $PM_{2.5}$ air pollution contributed to 8.0% of all-cause nonaccidental mortality among Canadians over 25 years of age, equivalent to 10,000 deaths per year or 27 deaths per 100,000 population.
- Acute exposure to NO_2 air pollution contributed to 0.9% of all-cause nonaccidental mortality among Canadians of all ages, equivalent to 1,300 deaths per year or 4 deaths per 100,000 population.
- Acute exposure to ozone was associated with 2.7% of all-cause nonaccidental mortality among Canadians of all ages, equivalent to 2,800 deaths per year or 8 deaths per 100,000 population. This estimate was derived using the annual average of daily 1-h maximum ozone concentrations.
- Chronic exposure to ozone was associated with 10% of respiratory-related mortality among Canadians over 30 years of age, equivalent to 1,300 deaths per year or 4 deaths per 100,000 population. This estimate was derived using the summer average of daily 1-h maximum ozone concentrations.

The economic cost of the 15,300 premature deaths associated with air pollution is estimated at $114 billion per year (2016 CAD). Health Canada recognizes the possibility of overlap or double counting of endpoints; this is addressed in the Uncertainties and limitations section.

Large variations in premature death counts and normalized risks are observed across geographic regions (Table 4). Higher mortality counts are estimated for the provinces of Ontario, Quebec, British Columbia and Alberta, which are the most populous provinces and those with the highest projected air pollution levels (Figures 1 to 3). Reported premature deaths per 100,000 population indicate that air pollution mortality risks are highest in Ontario and Quebec, reflecting the confluence of high population density and air pollution levels, and lowest in Nunavut and the Northwest Territories. There are also variations between regions with populations of comparable size. For example, the death rate per 100,000 population in British Columbia (40) is somewhat higher than in Alberta (33). Results at the CD level, discussed below, underline potential reasons for these differences.

[16] $PM_{2.5}$: 2015–2017;ozone: 2014, 2015 and 2017; NO_2: 2015–2017

[17] Values for individual pollutants may not match total because of rounding.

Table 4. Annual premature deaths attributable to air pollution by province and territory in 2016

Region—population	Counts of premature deaths[a]						Valuation (2016 CAD) × $1,000,000[a]
	Pollutant					per 100,000	
	NO_2	$PM_{2.5}$	Ozone[b]	Ozone[c]	All[d]	All[d]	All[d]
Canada—36,229,449	1,300	10,000	2,800	1,300	15,300	**42**	114,000
Alberta—4,286,341	120	850	290	140	1,400	**33**	10,400
British Columbia—4,689,131	170	1,200	310	160	1,900	**40**	13,900
Manitoba—1,307,764	35	300	72	28	430	**33**	3,300
New Brunswick—760,167	14	110	52	14	190	**25**	1,400
Newfoundland and Labrador—521,209	10	35	37	9	92	**18**	690
Northwest Territories—44,648	0	3	1	0	4	**9**	32
Nova Scotia—946,936	20	150	74	24	270	**29**	2,000
Nunavut—37,382	0	0	1	0	1	**3**	6
Ontario—13,979,393	570	4,200	1,200	580	6,600	**47**	49,200
Prince Edward Island—149,904	3	10	12	4	30	**20**	220
Québec—8,348,780	330	2,800	640	280	4,000	**48**	30,200
Saskatchewan—1,120,939	29	280	67	30	410	**36**	3,000
Yukon—36,855	0	3	1	0	4	**11**	31

[a] Values represent median estimates of health outcome counts and economic valuation. Values are rounded to the nearest integer and given to a maximum of two significant figures for values below 10,000, and three significant figures if equal to or above 10,000.

[b] Acute exposure premature deaths; annual ozone.

[c] Chronic exposure premature deaths; summer ozone.

[d] NO_2, ozone, and $PM_{2.5}$; totals may not match because of rounding.

National estimates for all health endpoints, both fatal and nonfatal, are provided in Table 5. Air pollution is associated with a considerable number of morbidity or nonfatal outcomes; the highest counts are predicted for the following outcomes: acute respiratory symptom days, restricted activity days, and asthma symptom days. However, the annual costs associated with morbidity outcomes, which total $5.6 billion (2016 CAD), are much less than those for mortality, owing to the large monetary value assigned to increased risk of death (Table 3).

The 2.5 and 97.5 percentiles reported in Table 5 represent the low- and high-range estimates. They are generally within a factor of two to three of the central estimate, except for minor restricted activity days. The estimates for this health endpoint have a broader range owing to the associated CRF. Specifically, the regression coefficient used to derive the CRF is not statistically significant, and the distribution overlaps 0 and is truncated at 0 (Judek et al. 2019). It may also have wide confidence intervals. Nil values are also estimated as the lower range for adult chronic bronchitis cases linked to exposure to $PM_{2.5}$. The CRF parameters explain this range in values (see Appendix B).

Table 5. National premature death and morbidity counts and valuations attributable to air pollution in 2016

Health endpoint	Pollutant	Count[a] [2.5 / 97.5 percentile]	Valuation (2016 CAD) × $1,000,000 [a] [2.5 / 97.5 percentile]
Mortality			
Acute exposure	NO_2	1,300 [450 / 2,100]	9,700 [2,600 / 20,200]
	Ozone	2,800 [1,900 / 3,600]	20,600 [8,800 / 36,400]
Chronic exposure – respiratory	Summer ozone[b]	1,300 [440 / 3,100]	9,500 [2,600 / 19,600]
Chronic exposure	$PM_{2.5}$	10,000 [5,300 / 14,500]	74,600 [27,200 / 142,000]
Total mortality[c]	All pollutants	15,300 [8,000 / 22,400]	114,000 [41,000 / 218,000]
Morbidity			
Acute respiratory symptom days	Summer ozone, $PM_{2.5}$	35,000,000 [377,000 / 69,900,000]	360 [0 / 1,400]
Adult chronic bronchitis cases	$PM_{2.5}$	9,200 [0 / 17,900]	4,000 [0 / 10,500]
Asthma symptom days	Summer ozone, $PM_{2.5}$	2,660,000 [362,000 / 5,420,000]	200 [17 / 560]
Cardiac emergency room visits	$PM_{2.5}$	1,100 [570 / 1,600]	7 [3 / 10]
Cardiac hospital admissions[d]	$PM_{2.5}$	810 [430 / 1,200]	N/A
Child acute bronchitis episodes	$PM_{2.5}$	42,300 [0 / 91,700]	19 [0 / 53]
Minor restricted activity days	Ozone summer	2,300,000 [0 / 9,540,000]	72 [0 / 350]
Respiratory emergency room visits	Ozone summer, $PM_{2.5}$	7,000 [2,400 / 11,600]	20 [0 / 350]
Respiratory hospital admissions	Ozone summer, $PM_{2.5}$	1,400 [480 / 2,300]	N/A
Restricted activity days	$PM_{2.5}$	13,100,000 [7,750,000 / 18,400,000]	890 [220 / 1,800]
Total morbidity[c]	All pollutants	N/A	5,600 [2,400 / 14,700]

N/A: not applicable

[a] Values represent median estimates of health outcome counts and economic valuation. Values are rounded to the nearest integer and given to a maximum of two significant figures for values below 10,000, and three significant figures if equal to or above 10,000.

[b] May–September only.

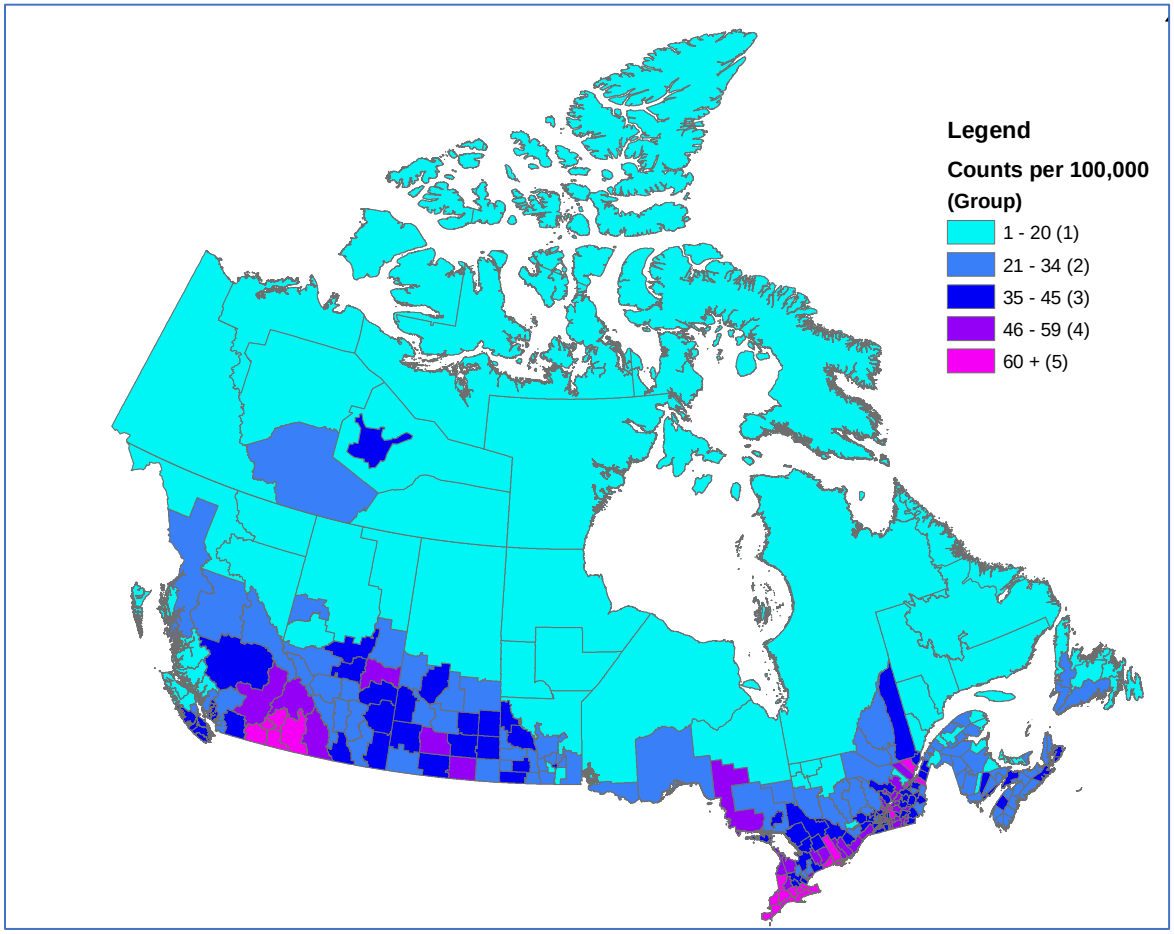
[c] Total or difference may not calculate as expected because of rounding.

[d] No economic valuation associated with hospital admissions (HAs). It is assumed that air pollution-related HAs involve an initial emergency room (ER) visit of the same type (cardiac or respiratory) that includes a cost. The valuation for ERs represent a mixture of costs related to ERs and HAs and consequently avoids under-valuation of these health outcomes.

Figure 4 shows variations in premature death rates per 100,000 population for CDs across Canada. Five groups were created to categorize normalized mortality rates attributable to air pollution exposure. Groups 1 and 2 are associated with rates that are lower than the national average of 42 premature deaths per 100,000 (group 3), and groups 4 and 5 are associated with higher-than-average rates. The geographic distribution of normalized mortality rates generally reflects the distribution of air pollution (see Figures 1 to 3), with CDs in groups 4 and 5 corresponding with regions of higher air pollution. The results illustrate that population size alone does not determine the rate of premature deaths: While higher population areas are generally characterized by higher air pollution levels, it does not necessarily translate to higher death rates per 100,000 population. Appendix D lists the results for the most populous CDs in Canada (Table D1) and the CDs with the highest rates (Table D2) and the lowest rates (Table D3) of premature deaths per 100,000 population. For high-population CDs (i.e. above 500,000), the rates range from 26 in Calgary (Division No. 6; 1,577,299 population) to 67 in Hamilton (564,030). The CDs with the highest rates could be divided into two distinct groups: southern British Columbia and southern Ontario. These CDs are clearly visible in Figure 5 and correspond with regions of higher air pollution levels. The British Columbia CDs in particular align with the

distribution of $PM_{2.5}$ concentrations associated with forest fire pollution in the province (Figure 1). These CDs are also generally characterized by relatively large populations (approximately 50,000–400,000 per CD). Lastly, the CDs with the lowest rate of deaths (Table D3) correspond with rural, northern or remote areas, including sparsely populated CDs in Nunavut, the Northwest Territories and Labrador.

Figure 4. Premature deaths per year, per 100,000 population associated with exposure to NO_2, ozone and $PM_{2.5}$ air pollution for census divisions in Canada for 2016



Notes: National average is 42 premature deaths per 100,000 population. Group 1 = 57 CDs; Group 2 = 93 CDs; Group 3 = 78 CDs; Group 4 = 37 CDs; Group 5 = 28 CDs

The total annual economic value of health outcomes associated with air pollution in Canada is approximately $120 billion (2016 CAD). This is largely driven by premature deaths, which represent $114 billion (2016 CAD) in annual damages, or 95% of the overall monetized health burden. Although nonfatal endpoints have a lower monetary value ($5.6 billion per year [2016 CAD]) than premature deaths, air pollution-related morbidity impacts remain a health burden for the Canadian population.

4. Discussion

Health Canada estimates that 15,300 deaths per year are attributable to ambient air pollution in Canada, corresponding to 42 deaths per 100,000 population in 2016. The total monetary value of health outcomes associated with air pollution is approximately $120 billion per year (2016 CAD), a figure equivalent to 6% of Canada's total real gross domestic product in 2016.[18] These estimates reflect the contribution from human sources of emissions in North America to Canada's ambient concentrations of NO_2, ozone, and $PM_{2.5}$, as well

[18] Annual Economic Indicators, Global Affairs Canada, Government of Canada

as contributions from irregular natural events such as forest fires. In this analysis, air pollution data from 2015 to 2017 were used for NO_2 and $PM_{2.5}$, and from 2014, 2015 and 2017 for ozone. Uniform Canadian background concentrations were subtracted from these three-year average exposure surfaces to estimate the above-background component of ambient air pollution. This approach was taken because this component, which includes anthropogenic emissions, is generally the subject of air quality management measures. Although Canada's air pollution levels are low compared to those of other developed nations,[19] recent Canadian studies indicate that air pollution increases the risk of mortality even at low ambient concentrations (Crouse et al. 2015; Pinault et. al. 2017; Pappin et al. 2019).

The provincial results (Table 4) indicate that Ontario and Quebec see the heaviest health impacts from air pollution, both in terms of mortality count and premature deaths per 100,000 population. This is not unexpected, as approximately 63% of the total Canadian population resides in these two provinces. Further, some of the highest air pollution levels in Canada are found in the southern regions of Ontario and Quebec, which include the highly populated and industrialized Windsor-Quebec City Corridor (encompassing the Greater Toronto and Hamilton Area and Greater Montreal). On the CD level (Tables D1 to D3, Appendix D), results show that higher rates of premature deaths are not correlated solely with higher population; they reflect a combination of environmental factors, age distribution and demographic characteristics, including higher pollution levels and baseline incidence rates. For example, an analysis of health impacts associated with $PM_{2.5}$ from wildfire smoke in Canada (Matz et al. 2020) indicated that between 2013 and 2018, the 10 CDs with the greatest average wildfire-$PM_{2.5}$ exposures (45–70% of the total exposure to $PM_{2.5}$) were all located in British Columbia. Five of those CDs were identified in Table D2 (CDs with the highest rates of premature death per 100,000). Wildfire activity was also common in Alberta, Saskatchewan and Manitoba in 2014, 2015 and 2017 (Matz et al. 2020). In addition, baseline incidence rates integrate various health and demographic variables, notably age distribution. Generally, CDs with older populations will have higher baseline incidence rates and consequently will be associated with higher rates of health outcomes for a given air pollution increment. Notwithstanding the exact causes used for calculating baseline incidence rates across all Canadian CDs, it was observed that several of the CDs with the highest premature death rates are associated with relatively elevated baseline incidence rates (Table D2, Appendix D). Thus, in a given CD, higher rates of health outcomes per 100,000 population represents the integration of several factors that influence the risks associated with exposure to air pollution.

Compared to previous analyses by Health Canada (see Table 6), the mortality health burden of air pollution in Canada for 2016 represents a slight increase in absolute terms: 14,400 in 2011 and 14,600 in 2015 (Health Canada 2017, 2019). The change in the number of premature deaths between analyses should be interpreted in consideration of: 1) estimates of exposure to air pollution across Canada; 2) estimates of the risk of health effects from exposure to air pollutants; and 3) demographic data, including population counts, age profiles and baseline health status. As presented in Table 6, the population-weighted average exposures to air pollution (above-background) in this analysis generally decreased from the previous reports for $PM_{2.5}$ and summer ozone, whereas a slight increase was estimated for annual ozone. Although the national average estimates are comparable, regional variability can also influence the results. There was an estimated 1.8 ppb-increase in NO_2 exposure since the last analysis.

The variation in NO_2 exposure estimates is possibly related in part to modifications in the LUR modelling since the 2014–2016 analysis (Health Canada 2019). Overall, the predictability of the 2014-2016 model reached an R^2 of 0.73, compared to 0.68 for the 2015–2017 version. Two important changes include an update to the remote-sensing data[20] and the use of monitoring data from a near-road monitoring site. Notably, the new model integrates data from a new NAPS station located in downtown Vancouver (station 100141) that were characterized by higher NO_2 concentrations than those measured at other monitoring stations. A mean

[19] WHO Global Urban Ambient Air Pollution Database, World Health Organization.

[20] NO_2 near-surface estimates were previously based on remote-sensing data from the Scanning Imaging Absorption Spectrometer for Atmospheric Chartography (SCIAMACHY) and Global Ozone Monitoring Experiment-2 (GOME-2) instruments as well as calculations by Geddes et al. (2016). The near-surface estimates were only available up to 2012 with this approach, and were substituted with 2015–2017 OMI vertical column density data.

annual average of 22.0 ppb was recorded at this station, over 3 ppb higher than the station with the next highest average (18.7 ppb), also located in downtown Vancouver, and more than 6 ppb higher than the stations with the highest concentrations in other provinces. The inclusion of locations with higher levels of air pollution increased model error.[21] However, this also increased model generalizability by capturing a unique combination of dense emission sources that are sparsely represented in the NAPS air monitoring network. Thus, while variations in air pollution exposure estimates across analyses can effectively reflect increases or decreases in ambient concentrations, methodological modifications also contribute to variability over time. The recent changes in LUR modelling for NO_2 are considered incremental and necessary improvements.

Table 6. Comparison of Health Canada analyses – population-weighted exposure to air pollution (above-background) and premature mortality estimates

Exposure Period[a]	Population (Year)	NO_2		Annual ozone		Summer ozone		$PM_{2.5}$		Total Deaths	
		pwe (ppb)	count	pwe (ppb)	count	pwe (ppb)	count	pwe (µg/m^3)	count	count	per 100,000
2007–2012[b]	34,342,780 (2011)	8.3	1,300	12.8	2,400	15.6	1,200	4.7	9,500	14,400	42
2014–2016[c]	35,851,774 (2015)	5.4	940	13.0	2,700	14.8	1,300	4.3	9,700	14,600	41
2014–2017[d]	36,229,449 (2016)	7.2	1,300	13.2	2,800	14.4	1,300	4.3	10,000	15,300	42

pwe: population-weighted exposure

[a] Background levels for all analyses: 0.15 ppb NO_2, 26 ppb annual ozone, 28 ppb summer ozone, 1.8 µg/m^3 $PM_{2.5}$

[b] Health Canada, 2017

[c] Health Canada, 2019

[d] Current analysis: 2015–2017 for NO_2 and $PM_{2.5}$; 2014, 2015 and 2017 for ozone

In terms of demographics, the current estimates used population data for the year 2016. Previous analyses used the 2011 population (Health Canada 2017) and the 2015 population (Health Canada 2019). The Canadian population count for 2016 represented an approximate increase of 378,000 people over 2015 (1.1% increase) and 1.9 million people over 2011 (5.5% increase). On the CD level, variations in population counts between the 2016 and 2015 data ranged from approximately -2.3 to 2.7%. An alternative means of measuring air pollution health impacts is to express the number of health outcomes per 100,000 population, thereby removing the influence of population changes. The results normalized by population show that estimates of premature death associated with air pollution are consistent across analyses: 42 per 100,000 Canadians in 2016, compared with 41 per 100,000 Canadians in 2015 and 42 per 100,000 in 2011. The air pollution estimates also suggest that spatial characteristics of air pollution in Canada have not changed substantially in recent years. In the current analysis, the air pollution estimates (Figure 1) are comparable to those of the previous analysis (reproduced in Figures D1 to D4, Appendix D). The highest air pollution estimates are reported in the same CDs, generally those associated with the southern regions of British Columbia and Alberta, as well as areas along the Windsor–Quebec City Corridor.

The risk estimates for the various health outcomes, represented herein by CRFs, were consistent between this analysis and previous reports, and therefore are not considered a source of variation across the estimates. The baseline incidence rates for mortality and morbidity outcomes were updated for AQBAT version 3.0 and as such are consistent with the previous analysis (Health Canada 2019), but differ from analyses based on earlier versions of AQBAT (e.g. Health Canada 2017).

Other studies have estimated the impact of air pollution on Canadians, most notably using the approaches developed under the IHME's Global Burden of Disease (GBD) project. The GBD analysis estimated 7,136 premature deaths from exposure to ozone and $PM_{2.5}$ in Canada for the year 2016, equivalent to 20 premature deaths per 100,000 population.[22] Overall, Canada is characterized by low levels of air pollution and relatively low incidence of air pollution-related deaths compared to other countries. The GBD estimates of

[21] It was observed that removing air monitor 100141 from the dataset reduced model error and increased R^2 by 2.5%.

[22] IHME. GBD Compare – 2016 (accessed June 23, 2020).

premature mortality in Canada in 2016 (7,136) is lower than Health Canada's estimate for the same year (15,300). The differences lie in the details of the approaches, including the basic CRFs and exposure surfaces for pollutants considered in the analyses. For example, in the current analysis, the $PM_{2.5}$ CRF was for all-cause mortality and used the Canada-specific study of Crouse et al. (2012), while the GBD analysis used an amalgam of several international studies, five specific causes of mortality, and a somewhat different approach to classification of mortality effects. In addition, while both the Health Canada and GBD approaches incorporate mortality effects for ozone, the specific causes of death or CRFs are different and lead to dissimilar estimates. Moreover, in Canadian analyses of the health effects of air pollution, NO_2 is a significant predictor of premature mortality. Exposure to NO_2 in 2016 is associated with 1,300 premature deaths in our current analysis; the GBD approach does not include NO_2. This demonstrates the influence of the CRF and underlines the importance of selecting the most scientifically robust value for the Canadian population.

Other differences arise from the relatively coarser resolution exposure surfaces for $PM_{2.5}$ and ozone used in the GBD analysis (e.g. Cohen et al. 2017). For this analysis, high-resolution surfaces were developed for $PM_{2.5}$ (1-km resolution), ozone (10-km resolution) and NO_2 (30-m resolution) in order to ascertain population exposure. Finally, the analysis provided here utilizes Canadian analyses of background concentrations, representing a lower counterfactual scenario than the GBD analysis (i.e. theoretical minimum risk exposure level [Cohen et al. 2017]), which result in some additional differences versus other approaches.

4.1 Sensitivity analysis

As a sensitivity analysis, an alternative and nonlinear CRF was applied for nonaccidental mortality associated with long-term exposure to $PM_{2.5}$ (i.e. chronic exposure mortality; Pinault et al. 2017). This CRF was derived from an analysis of the 2001 Canadian Census Health and Environment Cohort (CanCHEC). By contrast, the AQBAT CRF from Crouse et al. (2012, 2015) was based on the 1991 CanCHEC. Pinault et al. (2017) derived the CRF using a statistical curve-fitting method: the Shape Constrained Health Impact Function (SCHIF). This method fits several mathematical functions, including linear and nonlinear curves, to describe the shape of association. The SCHIF method was recently employed to derive the Global Exposure Mortality Model (GEMM) for 41 cohorts internationally, across the global range of $PM_{2.5}$ exposures, to assess global estimates of mortality associated with long-term exposure to ambient $PM_{2.5}$ (Burnett et al. 2018). The results from GEMM show a considerable increase in global mortality attributable to outdoor $PM_{2.5}$ air pollution (8.9 million deaths) compared to previous GBD estimates (4.1 million deaths) (Cohen et al. 2017).

To assess the potential influence of a nonlinear CRF for $PM_{2.5}$, the CRF for $PM_{2.5}$ of Pinault et al. (2017) was employed in the current evaluation as a sensitivity analysis. The selection of this CRF is relevant since it was developed using a large Canadian cohort (CanCHEC) of 2.4 million people. A supralinear shape of association was reported, which indicates higher incremental risks across the lowest range of $PM_{2.5}$ air pollution (Pinault et al. 2017). This supralinearity concurs with the GEMM and findings from Burnett et al. (2018). A large fraction of the Canadian population is expected to be exposed to air pollution levels in the lower range of exposures (i.e. less than 5 $\mu g/m^3$). Use of the alternative nonlinear CRF (nonaccidental deaths) for chronic exposure to ambient $PM_{2.5}$ resulted in an additional 3,000 premature deaths, valued at $22.4 billion (the health impact estimates for NO_2 and ozone are unchanged).

4.2 Uncertainties and limitations

The air quality and health impacts modelling conducted in the current assessment made use of the best available tools and data for Canadian scenarios. As with other health impact assessments, the health impact estimates are subject to uncertainty introduced by assumptions implicit in the assignment of population exposure, representation of health risks via CRFs, and the valuation of health endpoints. This section addresses these categories of uncertainty, which are associated specifically with the analysis of health impacts, by means of qualitative discussions and quantitative sensitivity analyses. When possible, uncertainties were evaluated according to their potential influence on the direction and magnitude of estimated health impacts, as well as the amount of evidence available to support the approach or

assumptions selected for the current assessment. This approach aims to follow the WHO uncertainty framework (2008) and the United States Environmental Protection Agency's approach to the qualitative assessment of uncertainty outlined in the assessment of the National Ambient Air Quality Standard (NAAQS) for PM (2012). Overall, it is expected that uncertainties in the current assessment likely lead to an underestimation of health impacts.

4.2.1 Exposure uncertainty

In this analysis, three different methods were used to calculate Canadians' exposure to each of the three pollutants. These included satellite observations and ground observations fused with a chemical transport model ($PM_{2.5}$); objective analysis (OA) that considered ground observations fused with a chemical transport model (ozone); and LUR modelling (NO_2). These three estimation methods currently provide the best available national exposure estimates for each pollutant. Ongoing work on model development and testing could eventually lead to a uniform approach for all pollutants that is consistent in its assumptions and spatial representativeness; for example, an OA approach for all pollutants. However, there is currently no evidence indicating that the use of a single modelling approach would generate better or more consistent predictions. The three approaches utilize methods that are optimal (at time of writing) for each pollutant and may represent the best analysis, since each pollutant is governed by different processes and parameters. It is unclear how the use of distinct methods impacts the magnitude and direction (i.e. increase or decrease) of air pollution estimates.

The ambient concentrations that informed this analysis were averaged over three years of available data to ensure that results were not influenced by interannual variability. The data periods for each pollutant differ slightly: While data from 2015 to 2017 informed the NO_2 and $PM_{2.5}$ concentrations, ozone concentrations were based on data for 2014, 2015 and 2017 because the 2016 data were not useable at the time of this assessment owing to operational considerations for the underlying model.[23] Health Canada and Environment and Climate Change Canada continue to collaborate on the evaluation of approaches for developing more integrated exposure estimates that can be updated annually or biannually. The uncertainty associated with excluding ozone data for the year 2016 is expected to have a minor impact on the magnitude and direction of health impact estimates.

The use of uniform background concentrations to estimate population exposures is a source of uncertainty. Regional differences in background concentrations likely exist across Canada for each pollutant. Spatial variations in background concentrations can lead to under- or overestimates of population exposures to air pollution. Continuing analyses of air quality in Canada could provide new, spatially-resolved, background air pollution concentrations for use in future analyses. It is uncertain how the use of new background concentrations would impact the magnitude and direction of air pollution exposure estimates across Canada.

Another potential source of uncertainty is the variation in spatial resolution among air pollution exposure and epidemiologic data. First, the spatial resolution of the modelled exposure surfaces applied in this analysis differ from those used in the epidemiological source studies. Specifically, in this analysis, the exposure estimates were based on air pollution estimates for CD geographies, with population exposure estimates in particular being based on residential locations and a presumption that individuals remained within their CDs to carry out their daily activities. The CD estimates were weighted or interpolated from relatively high-resolution national-scale exposure surfaces for NO_2 (30-m grid), ozone (10-km grid) and $PM_{2.5}$ (1-km grid). The CRFs in AQBAT for acute exposure mortality were derived from epidemiological studies that assessed population health effects based on monitoring data collected at centrally located sites. In addition, the mortality CRFs for chronic exposure to $PM_{2.5}$ applied in AQBAT relied upon less spatially refined satellite-

[23] The 2016 OA data could not be used owing to multiple changes to the size and orientation of the GEM-MACH grid over a short period in 2016. The 10-km horizontal resolution of the grid remained unchanged, but the size of the grid varied from 347,116 grid cells (506 × 686) to 366,444 grid cells (522 × 702), and finally 487,176 grid cells (766 × 636). Moreover, the grid orientation was rotated during the last expansion and thus excluded colocalized grid cells among grid versions. Since annual or seasonal metrics for ozone require the interpolation of all data on the same grid, these changes influenced the results, and introduced error and uncertainty in the ozone concentration estimates.

derived data (10-km grid resolution). These epidemiological studies used exposure data that were not as highly resolved as that of the current analysis. Meanwhile, population health effect studies based on high-resolution exposure estimates were not readily available. The magnitude and direction of the bias owing to this exposure misclassification are difficult to assess without a thorough sensitivity analysis, which was beyond the scope of the current assessment. An analysis of a population-based Canadian environmental cohort, the 2001 CanCHEC, examined the sensitivity of $PM_{2.5}$ mortality associations to exposure surface resolution. Brauer et al. (2019) found improved model fit and higher risk estimates for the finest resolution surface analyzed (1 km) compared to 5 km and 10 km. It is likely that the CRF for chronic exposure mortality and $PM_{2.5}$ exposure, which was derived using a 10-km exposure surface (Crouse et al. 2012), underestimates risk at the 1-km resolution applied in this assessment. The magnitude of this effect likely varies according to the pollutant examined in light of differences in the atmospheric lifetimes and spatial gradients in concentration as well as the resolutions employed for each species (30 m for NO_2, 1 km for $PM_{2.5}$, and 10 km for ozone).

Populations are exposed concurrently to multiple air pollutants in the ambient air mixture, and not to individual pollutants in isolation. It can be difficult for epidemiological studies to statistically separate the true, independent effects of individual pollutants. Where possible, the CRFs employed in this analysis were derived from models that statistically adjusted for the effects of other pollutants in multipollutant models. However, it remains possible that some pollutant effects have been double-counted, or that effects attributed to one pollutant were not fully disentangled from those attributed to other pollutants. One example is the association between NO_2 concentrations and daily mortality rates (i.e. acute exposure mortality). Health Canada (2016) has concluded that acute exposure to NO_2 is likely to cause an increased risk of mortality; however, NO_2 originates from combustion sources and it is possible that NO_2 exposure reflects, in part, the influence of other combustion products, including gases and particulates (Brook et al. 2007). A growing number of studies have also linked NO_2 to chronic exposure mortality, but this was not included in AQBAT because Health Canada (2016) concluded that the evidence was merely suggestive of a causal relationship, primarily in light of the uncertainty in the potential mode of action and the possibility that it is acting as a surrogate for specific sources or other pollutants. If there is a causal relationship between chronic NO_2 exposure and mortality, this would represent an underestimate in the current analysis.

There is also the potential for overlap between or double counting of acute and chronic health effects (WHO 2013). Double counting from this perspective is believed to be controlled in the selection of air pollutant and health endpoint CRFs in AQBAT. The uncertainty associated with double-counting premature deaths linked to acute and chronic exposure to air pollution is expected to have a minor impact on the magnitude and direction of the overall health impact estimates. Additional epidemiological studies addressing acute and chronic health outcomes in Canada and internationally are needed to further clarify this uncertainty.

4.2.2 Exposure-response uncertainty

The selection of a specific CRF for use in AQBAT is non-trivial. As the tool is used in part for regulatory purposes, the consistency and stability of model parameters are important considerations. While new studies may report alternative or updated CRFs, these must be evaluated by Health Canada, and the AQBAT model is updated periodically rather than continuously. Recent analyses, including those applying nonlinear curve-fitting techniques (e.g. Burnett et al. 2018; Pinault et al. 2017) suggest a supralinear association between long-term exposure to $PM_{2.5}$ and mortality from various causes. This supralinear shape of the relationship is especially relevant for conditions in Canada, which are typically characterized by low ambient air pollution concentrations. A supralinear CRF implies higher incremental effects per unit of exposure across the lowest range of $PM_{2.5}$ air pollution levels and, when used in AQBAT, leads to additional premature deaths (health impacts for NO_2 and ozone are unchanged). This new CRF for long-term exposure to $PM_{2.5}$ has not been officially included in AQBAT; rather, it is used here for sensitivity analyses. These analyses indicate that current AQBAT results may underestimate $PM_{2.5}$-associated premature deaths by approximately 30% (and total premature deaths by 20%). Chen et al. (2020) have also investigated the use of the SCHIF to characterize the joint impact of $PM_{2.5}$ concentration and composition on cardiovascular disease outcomes across regions in Ontario. The results obtained by using the $PM_{2.5}$-component-adjusted approach indicated improved

prediction of the health impacts of $PM_{2.5,}$ especially when considering nonlinear air pollutant–outcome relationships. This new approach improves understanding of the health effects of $PM_{2.5}$ and the quantification of population health impacts in future analyses.

Regarding CRFs in general, AQBAT includes a set of values that are endorsed by Health Canada based on the robustness of the database. In light of new research findings, CRFs may be added, modified or removed from AQBAT. Similar to the GBD analyses, the inclusion of new CRFs or new risk-outcome pairs in AQBAT depends on the strength of evidence for a causal association between the risk (in this case exposure to an air pollutant) and the health outcome, as well as the feasibility of developing an exposure surface for the air pollutant of interest (Shaffer et al. 2019). The decision to remove, add or modify CRFs requires the availability of robust data and a clear consensus among Health Canada experts that the proposed values are more relevant for the current Canadian population.

4.2.3 Uncertainty in endpoints and valuation

In the current analysis, the mortality and morbidity outcomes associated with air pollution are based on ambient concentration estimates of $PM_{2.5}$, NO_2 and ozone exclusively. AQBAT includes health endpoints associated with these air pollutants, for which there is a strong weight of evidence for effect and robust information quantifying the relationship between air pollution exposure and adverse health outcome. In addition, many effects that have been linked to air pollution are not included in AQBAT but may be in the future. For example, air pollution-related health endpoints of interest include neurological outcomes (e.g. dementia, intellectual disability) and reproductive outcomes (e.g. low birth weight) (Shaffer et al. 2019). Overall, it is expected that AQBAT results are underestimating fatal and nonfatal outcomes associated with air pollution. The magnitude of the bias is uncertain.

The inclusion of a health outcome in AQBAT requires the quantification of estimates of the associated baseline incidence rates and of target population characteristics, such as population count and age distributions. For some health endpoints, data reflecting regional differences in incidence rates are available, while for others, uniform incidence rates are applied across Canada in the absence of more detailed geographic data (e.g. cause-specific mortality related to long-term $PM_{2.5}$ exposure). The assumption of geospatially constant baseline incidence rates is simplistic and a source of uncertainty that may lead to over- or underestimation of impacts depending on the characteristics of populations across regions of Canada.

Costs are assigned to the health outcomes for economic valuation purposes. The current version of AQBAT includes willingness to pay values that were temporally adjusted using the consumer price index from the source years of the underlying studies. However, these studies were published more than a decade ago (see Table 3) and may not reflect the most recent data and approaches. While the probabilistic approach in AQBAT accounts for valuation uncertainties, updating the underlying data could potentially improve estimates of the economic costs of air pollution. For air pollutants and health endpoint CRFs included in AQBAT version 3.0, the magnitude and direction of this bias are uncertain.

5. Conclusions

Air pollution is recognized globally as a leading risk factor for premature mortality based on an established database of international epidemiological studies and toxicological investigations. Comprehensive risk assessments performed by Health Canada (2013, 2016) have concluded that, based on extensive research and assessment, exposures to $PM_{2.5}$, NO_2, and ozone have been found to exert the largest population health impacts in Canada.

The current analysis provides estimates of mortality, morbidity and economic costs associated with the above-background component of ambient air pollutant levels in Canada, which corresponds to air pollution that is targeted by air quality management measures. Health Canada estimates that in 2016, 15,300 premature deaths in Canada could be attributed to air pollution from $PM_{2.5}$, NO_2, and ozone. Nonfatal health outcomes attributable to air pollution include 35 million acute respiratory symptoms days, 2.7 million asthma symptom days and 8,100 emergency room visits. The total economic value of adverse air pollution health

impacts is estimated to be $120 billion per year (2016 CAD), equivalent to 6% of total real gross domestic product in 2016. Although air pollution affects the health of Canadians in all regions of the country, the largest impacts are seen in the most populous provinces and those with the largest sources of emissions: Ontario, Quebec, British Columbia and Alberta.

While all three pollutants considered here exert impacts, exposure to $PM_{2.5}$ represents the majority of the estimated mortality burden (65%), with ozone and NO_2 accounting for 26% and 8%, respectively. Regarding nonfatal outcomes, both ozone and $PM_{2.5}$ are associated with health impacts. This is not the case for NO_2 because, while considered to be causally associated with several important respiratory effects, there are currently no CRFs in AQBAT for NO_2 and morbidity outcomes.

This estimate of 15,300 deaths per year is equivalent to 42 per 100,000 Canadians and is consistent with previous analyses. The higher number of exposed individuals in 2016 compared to previous years of analysis (owing to population increase) leads to an overall higher mortality count. It is estimated that Canadians are currently exposed, on average, to lower air pollution levels for $PM_{2.5}$ and summer ozone compared to exposure periods that informed previous analyses. In contrast, this assessment uses overall higher concentrations for annual ozone, while NO_2 exposure estimates do not suggest any clear directional change. The normalized value per 100,000 population provides a more objective picture of the health burden and suggests that per capita air pollution-related health risks in Canada have remained consistent over the last decade. While Canadians benefit from relatively good air quality, air pollution continues to have impacts on population health.

The data and methods (e.g. background concentrations, CRFs) used in the current analysis, the most comprehensive analysis available, incorporate the most up-to-date science, data and knowledge on the health effects of air pollution in Canada compared with previous Canadian estimates. Nonetheless, evidence suggests that air pollution may be associated with additional adverse health outcomes that were not considered here. Further, there are air pollutants other than NO_2, $PM_{2.5}$ and ozone that are responsible for adverse health effects. As a result, the quantitative estimates of population health outcomes in this analysis are assumed to underestimate the adverse health impacts of air pollution in Canada.

Changes in health impact estimates are to be expected following each update by Health Canada. Variations or discrepancies between estimates may occur owing to changes in data or methods to assess population exposure to air pollutants, changes in concentration-response relationships, changes in the baseline rates of adverse outcomes in Canada, or changes in population demographics. AQBAT is periodically updated as new evidence is evaluated for inclusion in the model. For example, recent studies have reported a supralinear CRF between exposure to ambient $PM_{2.5}$ and premature mortality, which would increase count estimates for Canada if it were included in AQBAT for health burden analyses. Evaluation of the relative risks and CRFs at low levels of air pollution, which is particularly relevant for Canada, is also an active area of research (Shaffer et al. 2019). Sensitivity analyses, such as the one discussed above, were conducted to explore the influence of different factors. Finally, there is the possibility of re-estimating the health impacts for years included in Health Canada analyses to ensure that estimates are based on internally consistent methods and data.

6. References

Abbey DE, Lebowitz MD, Mills PK, Petersen FF, Beeson L, Burchette RJ. 1995. Long term ambient concentrations of particulates and oxidants and development of chronic disease in a cohort of nonsmoking California residents. Inhal Toxicol 7(1): 19–34.

Boersma KF, Eskes HJ, Dirksen RJ, van der A RJ, Veefkind JP, Stammes P, Huijnen V, Kleipool QL, Sneep M, Claas J, Leitão J, Richter A, Brunner D. 2011. An improved retrieval of tropospheric NO_2 columns from the Ozone Monitoring Instrument. Atmos Meas Tech 4: 1905–1928.

Boys B, Martin RV, Van Donkelaar A, MacDonell R, Hsu NC, Cooper MJ, Yantosca RM, Lu Z, Streets DG, Zhang Q, Wang SW. 2014. Fifteen-year global time series of satellite-derived fine particulate matter. Environ Sci Technol 48: 11109–11118.

Brauer M, Brook JR, Christidis T, Chu Y, Crouse D, Erickson A, Hystad P, Li C, Martin RV, Meng J, Pappin AJ, Pinault LL, Tjepkema M, van Donkelaar A, Weichenthal S, Burnett RT. 2019. Mortality–Air Pollution Associations in Low-Exposure Environments (MAPLE): Phase 1. Research Report 203

Brook JR, Burnett RT, Dann TF, Cakmak S, Goldberg MS, Fan X, Wheeler AJ. 2007. Further interpretation of the acute effect of nitrogen dioxide observed in Canadian time-series studies. J Exp Sci Environ Epidem 17: S36–S44.

Burnett RT, Stieb D, Brook JR, Cakmak S, Dales R, Raizenne M, Vincent R, Dann T. 2004. Associations between short-term changes in nitrogen dioxide and mortality in Canadian cities. Arch Environ Health 59(5): 228–36.

Burnett R, Chen H, Szyszkowicz M et al. 2018. Global estimates of mortality associated with long-term exposure to outdoor fine particulate matter. Proceedings of the National Academy of Sciences 115 (38): 9592–9597.

Canadian Medical Association. 2008. No Breathing Room: National Illness Cost of Air Pollution Summary Report. Ottawa: CMA. www.healthyenvironmentforkids.ca/resources/no-breathing-room-costs-of-air-pollution

Chen H, Zhang Z, van Donkelaar A, Bai L, Martin RV, Lavigne E, Kwong JC, Burnett RT. 2020. Understanding the joint impacts of fine particulate matter concentration and composition on the incidence and mortality of cardiovascular disease: a component-adjusted approach. Environ Sci Technol doi 10.1021

Chestnut LG, De Civita P. 2009. Economic valuation of mortality risk reduction: review and recommendations for policy and regulatory analysis. Prepared for the Government of Canada Policy Research Initiative. PRI Project – Regulatory strategy.

Cohen AJ, Brauer M, Burnett R, Anderson HR, Frostad J, Estep K, Feigin V et al. 2017. Estimates and 25-year trends of the global burden of disease attributable to ambient air pollution: an analysis of data from the Global Burden of Diseases Study 2015. The Lancet 389(10082): 1907–1918.

Crouse DL, Peters PA, van Donkelaar A, Goldberg MS, Villeneuve PJ, Brion O, Khan S, Atari DO, Jerrett M, Pope CA, Brauer M, Brook JR, Martin RV, Stieb D, Burnett RT. 2012. Risk of nonaccidental and cardiovascular mortality in relation to long-term exposure to low concentrations of fine particulate matter: a Canadian national-level cohort study. Environ Health Perspect 120(5): 708–714.

Crouse DL, Peters PA, Hystad P, Brook JR, van Donkelaar A, Martin RV, Villeneuve PJ, Jerrett M, Goldberg MS, Pope CA, Brauer M, Brook RD, Robichaud A, Menard R, Burnett RT. 2015. Ambient $PM_{2.5}$, ozone, and NO_2 exposures and associations with mortality over 16 years of follow-up in the Canadian Census Health and Environment Cohort (CanCHEC). Environ Health Perspect 123(11): 1180–1186.

[CRS] Congressional Research Service. 2019. Background ozone: challenges in science and policy. Prepared for Members and Committees of Congress. R45482. 18 pp. https://fas.org/sgp/crs/misc/R45482.pdf

Geddes JA, Martin RV. Boys BL, van Donkelaar A. 2016. Long-term trends worldwide in ambient NO_2 concentrations inferred from satellite observations. Environ Health Perspect 124(3): 281–289.

Griffin D, McLinden C, Racine J, Moran M, Fioletov V, Pavlovic R, Eskes H. 2020. Assessing the impact of Corona-virus-19 on nitrogen dioxide levels over southern Ontario, Canada. Geophys Res Letters [submitted] https://doi.org/10.1002/essoar.10503538.2

Health Canada. 2013. Canadian smog science assessment – Volume 2: Health Effects. Air Health Sciences Division. Cat. En88-5/2-2013E-PDF. 533 pp.

Health Canada. 2016. Human health risk assessment for ambient nitrogen dioxide. Water and air quality Bureau, Safe Environments Directorate. Cat. H144-31/2016E-PDF. 288 pp.

Health Canada. 2017. Health impacts of air pollution in Canada – An estimate or premature mortalities. Government of Canada. ISBN 978-0-660-23740-4. 13 pp. http://publications.gc.ca/site/eng/9.846412/publication.html

Health Canada. 2019. Health impacts of air pollution in Canada – Estimate of morbidity and premature mortality outcomes – 2019 report. Government of Canada. ISBN 978-0-660-31165-4. 38 pp. http://publications.gc.ca/site/eng/9.874080/publication.html

Hoek G, Pattenden S, Willers S, Antova T, Fabianova E, Braun-Fahrländer C, et al. 2012. PM_{10} and children's respiratory symptoms and lung function in the PATY study. Eur Respir J 40(3): 538–47.

Hystad P, Setton E, Cervantes A, Poplawski K, Deschenes S, Brauer M, van Donkelaar A, Lamsal L, Martin R, Jerrett M, Demers P. 2011. Creating national air pollution models for population exposure assessment in Canada. Environ Health Perspect 119: 1123–1129.

[IHME and HEI] Institute for Health Metrics and Evaluation and Health Effects Institute. 2018. State of Global Air/2018. Institute for Health Metrics and Evaluation, and Health Effects Institute. www.stateofglobalair.org/

IHME and HEI. 2019. State of Global Air/2019. Institute for Health Metrics and Evaluation, and Health Effects Institute. www.stateofglobalair.org/

Jerrett M, Burnett RT, Pope CA III, Ito K, Thurston G, Krewski D, Shi Y, Calle E, Thun M. 2009. Long-term ozone exposure and mortality. N Engl J Med 360(11): 1085–1095.

Judek S, Stieb D, Xi G, Jovic B, Edwards B. 2019. Air Quality Benefits Assessment Tool (AQBAT) – User Guide – Version 3. Healthy Environments and Consumer Safety Branch, Health Canada. 205 pp.

Judek S, Jessiman B, Stieb D, Vet R. 2004. Estimated number of excess deaths in Canada due to air pollution. Ottawa: Health Canada.

Kalnay E. 2003. Atmospheric Modeling, Data Assimilation and Predictability: New York: Cambridge University Press.

Krupnick AJ, Cropper ML. 1989. Valuing chronic morbidity damages: medical costs, labor market effects and individual valuations. Final report to Office of Policy Analysis, US Environmental Protection Agency. 269 pp.

Krupnick AJ, Harrington W, Ostro B. 1990. Ambient ozone and acute health effects: Evidence from daily data. J Environ Econ Manage 18(1): 1–18.

Krupnick AJ, Cropper ML. 1992. The effect of information on health risk valuations. J Risk Uncertain 5: 29–48.

Lamsal LN, Martin RV, van Donkelaar A, Steinbacher M, Celarier EA, Bucsela E, et al. 2008. Ground- level nitrogen dioxide concentrations inferred from the satellite-borne Ozone Monitoring Instrument. J Geophys Res Atmos 113: D16308.

Larkin A, Hystad P. 2020. Update of the Canadian NO_2 land use regression model for years 2015–2017, and prediction of Canadian NO_2 concentrations. Produced for Health Canada, contract 4500401021. January 31, 2020. 13 pp.

Lim SS, Vos T, Flaxman AD, et al. 2012. A comparative risk assessment of burden of disease and injury attributable to 67 risk factors and risk factor clusters in 21 regions, 1990–2010: A systematic analysis for the Global Burden of Disease Study 2010. Lancet 380: 2224–2260.

Makar P, Akingunola A, Aherne J, Cole A, Aklilu Y, Zhang J, Wong I, Hayden K, Li S, Kirk J, Scott K, Moran M, Robichaud A, Cathcart H, Baratzedah P, Pabla B, Cheung P, Zheng Q, Jeffries DS. 2018. Estimates of exceedances of critical loads for acidifying deposition in Alberta and Saskatchewan. Atmos Chem Phys 18: 9897–9927.

Matz CJ, Egyed M, Guoliang X, Racine J, Pavlovic R, Rittmaster R, Henderson SB, Stieb DM. 2020. Health impact analysis of $PM_{2.5}$ from wildfire smoke in Canada (2013–2015, 2017–2018). Sci Total Environ 725: 138506.

Moran MD, Ménard S, Talbot D, Huang P, Makar PA, Gong W, Landry H, Gravel S, Gong S, Crevier L-P, Kallaur A, Sassi M. 2010. Particulate-matter forecasting with GEM-MACH15, a new Canadian air-quality forecast model. Air pollution modelling and its application XX, edited by Steyn DG and Rao, ST, Springer, Dordrecht. pp. 289–292.

Ostro BD. 1987. Air pollution and morbidity revisited: A specification test. J Environ Econ Manage 14(1): 87–98.

Ostro BD, Rothschild S. 1989. Air pollution and acute respiratory morbidity: An observational study of multiple pollutants. Environ Res 50(2): 238–47.

Pappin AJ, Christidis T, Pinault LL, Crouse DL, Brook JR, Erickson A, Hystad P, Li C, Martin RV, Meng J, Weichenthal S, van Donkelaar A, Tjepkema M, Brauer M, Burnett RT. 2019. Examining the shape of the association between low levels of fine particulate matter and mortality across three cycles of the Canadian Census Health and Environment Cohort. Environ Health Perspect 127(10): 107008.

Pinault LL, Weichenthal S, Crouse DL, Brauer M, Erickson A, van Donkelaar A, Martin RV, Hystad P, Chen H, Finès P, Brook JR, Tjepkema M, Burnett RT. 2017. Associations between fine particulate matter and mortality in the 2001 Canadian Census Health and Environment Cohort. Environmental Research 159: 406–415.

Robichaud A, Ménard R, Zaïtseva Y, Anselmo D. 2016. Multi-pollutant surface objective analyses and mapping of air quality health index over North America. Air Qual Atmos Health 9:743–759.

Robichaud A, Ménard R. 2014. Multi-year objective analyses of warm season ground-level ozone and $PM_{2.5}$ over North America using real-time observations and the Canadian operational air quality models. Atmos Chem Phys 14(4): 1769–1800.

Shaffer RM, Sellers SP, Baker MG, de Buen Kalman R, Frostad J, Suter MK, Anenberg SC, Balbus J, Basu N, Bellinger DC, Birnbaum L, Brauer M, Coehn A, Ebi KL, Fuller R, Grandjean P, Hess JJ, Kogevinas M, Kumar P, Landrigan PJ, Lanphear B, London SJ, Rooney AA, Stanaway JD, Trasande L, Walker K, Hu H. 2019. Improving and expanding estimates of the Global Burden of Disease due to environmental health risk factors. Environ Health Perspect 127(10): 105001-1–16.

Shin HH, Cohen A, Pope CA III, Ezzati M, Lim SS, Hubbell B, et al. 2013. Critical issues in combining disparate sources of information to estimate the Global Burden of Disease attributable to ambient fine particulate matter exposure. Working paper prepared for: Methods for Research Synthesis: A Cross-Disciplinary Workshop. Cambridge (MA): Harvard Center for Risk Analysis. www.hsph.harvard.edu/wp-content/uploads/sites/1273/2013/09/Shin-et-al.-Sept-2013.pdf

Statistics Canada. Annual. Table 18-10-0005-01 – Consumer Price Index, annual average, not seasonally adjusted. www150.statcan.gc.ca/t1/tbl1/en/tv.action?pid=1810000501

Stieb DM, De Civita P, Johnson FR, Manary M, Anis A, Beveridge RC, Judek S. 2002. Economic evaluation of the benefits of reducing acute cardiorespiratory morbidity associated with air pollution. Environ Health 1: 1–13.

Stieb DM, Judek S, van Donkelaar A, Martin RV, Brand K, Shin HH, Burnett RT, Smith-Doiron M. 2015. Estimated public health impact of changes in fine particle air pollution in Canada, 2000–2011. Can J Public Health 106(6): 362–368.

[TFHTAP] Task Force on Hemispheric Transport of Air Pollution. 2010. Hemispheric transport of air pollution 2010 – Part A: Ozone and particulate matter. Air Pollution Studies No. 17. Editors: Dentener F, Keating T & Akimoto H. Economic Commission for Europe. 304 pp. http://www.htap.org

[US EPA] United States Environmental Protection Agency. 2012. Regulatory impact analysis for the proposed revision to the National Ambient Air Quality Standards for particulate matter. Office of Air Quality Planning and Standards, US Environmental Protection Agency. Research Triangle Park, NC. EPA-452/R-12-003. 522 pp.

US EPA. 2013. Integrated science assessment for ozone and related photochemical oxidants. (EPA/600/R-10/076F). Research Triangle Park, NC: US Environmental Protection Agency, National Center for Environmental Assessment. http://cfpub.epa.gov/ncea/isa/recordisplay.cfm?deid=247492

US EPA. 2019. Integrated science assessment for particulate matter. Office of Research and Development. 1967 pp. Available online at: https://cfpub.epa.gov/ncea/isa/recordisplay.cfm?deid=347534#tab-3

van Donkelaar A, Martin RV, Brauer M, Kahn R, Levy R, Verduzco C, Villeneuve PJ. 2010. Global estimates of ambient fine particulate matter concentrations from satellite-based aerosol optical depth: development and application. Environ Health Perspect 118: 847–855.

van Donkelaar A, Martin RV, Spurr RJD, Drury E, Remer LA, Levy RC, Wang J. 2013. Optimal estimation for global ground-level fine particulate matter concentrations. J Geophys Res 118: 5621–5636.

van Donkelaar A, Martin RV, Brauer M, Boys BL. 2015a. Use of satellite observations for long-term exposure assessment of global concentrations of fine particulate matter. Environ Health Perspect 123(2): 135–143.

van Donkelaar A, Martin RV, Spurr RJ, Burnett RT. 2015b. High-resolution satellite-derived $PM_{2.5}$ from optimal estimation and geographically weighted regression over North America. Environ Sci Technol 49: 10482–10491.

Viscusi WK, Magat WA, Huber J. 1991. Pricing environmental health risks: survey assessments of risk–risk and risk–dollar trade-offs for chronic bronchitis. J Environ Econ Manage 21(1): 32–51.

Weinmayr G, Romeo E, De Sario M, Weiland SK, Forastiere F. 2010. Short-term effects of PM_{10} and NO_2 on respiratory health among children with asthma or asthma-like symptoms: a systematic review and meta-analysis. Environ Health Perspect 118(4): 449–57.

Whaley C, Makar PA, Shephard MW, Zhang L, Zhang J, Zheng Q, Akingunola A, Wentworth GR, Murphy JG, Kharol SK, Cady-Pereira KE. 2018. Contributions of natural and anthropogenic sources to ambient ammonia in the Athabasca Oil Sands and north-western Canada. Atmos Chem Phys 18: 2011–2034.

[WHO] World Health Organization. 2008. Part 1: Guidance document on characterizing and communicating uncertainty in exposure assessment – harmonization project document No. 6. Published under joint sponsorship of the World Health Organization, the International Labour Organization and the United Nations Environment Programme. WHO Press: Geneva, Switzerland. Available online at: www.who.int/ipcs/methods/harmonization/areas/uncertainty%20.pdf

WHO. 2013. Health risks of air pollution in Europe – HRAPIE project. Recommendations for concentration–response functions for cost–benefit analysis of particulate matter, ozone and nitrogen dioxide. HWO Regional Office for Europe: Copenhagen, Denmark. 60 pp.

WHO. 2016. Ambient air pollution: a global assessment of exposure and burden of disease. Geneva www.who.int/phe/publications/air-pollution-global-assessment/en/

Zangari S, Hill DT, Charette AT, Mirowsky JE. 2020. Air quality changes in New York City during the COVID-19 pandemic. Sci Total Environ 742: 140496.

Appendix A. Information on air emissions levels and sources associated with ambient concentrations of $PM_{2.5}$, ozone and NO_2 in Canada

Extensive scientific studies indicate that there are significant health and environmental effects associated with exposure to the air pollutants considered in the current assessment, namely fine particulate matter ($PM_{2.5}$), ground-level ozone, and nitrogen dioxide (NO_2). These pollutants are notably responsible for the formation of smog. This appendix provides general information on Canadian sources of emissions for these pollutants, as well as their associated health effects. In addition, emissions data are provided for nitrogen oxides (NOx) and volatile organic compounds (VOCs), precursors for the formation of ozone and $PM_{2.5}$.

> Note: This appendix summarizes findings published in Canada's Air Pollutant Emissions Inventory Report, a report released annually by Environmental and Climate Change Canada (ECCC 2020). Additional information and data were extracted from the State of the Air Report, available on the website of the Canadian Council of Ministers of the Environment. For additional details, please refer to the original publications.

Particulate matter

Emissions in Canada

Particulate matter consists of aerosols (i.e. airborne particles) in solid or liquid form. It exists in various sizes that are often classified as coarse (aerodynamic diameter between 2.5 and 10 µm), fine (2.5 µm or less) and ultrafine (0.1 µm or less).

PM may be defined as primary or secondary:

- Primary PM is emitted by a source directly into the atmosphere. Examples include combustion emissions from smokestacks and vehicle exhaust or noncombustion emissions from wind-entrained dust produced by soil, as well as paved and unpaved roads.
- Secondary PM is not emitted directly, but rather formed in the atmosphere through a series of chemical and physical reactions involving gases such as sulphur oxides, NOx, and VOCs.

In 2015–2017, approximately 1.6 million tonnes (Mt) of $PM_{2.5}$ were emitted in Canada (Table A1). The largest contributions were associated with noncombustion emissions. Dust sources accounted for 58% of total $PM_{2.5}$ emissions, mostly from construction activities (55% of dust emissions) and unpaved roads (43% of dust emissions). Agriculture was the second largest contributing sector to $PM_{2.5}$ emissions, accounting for 24% of total $PM_{2.5}$ emissions.

In terms of combustion-related emissions, the largest contribution originated from commercial, residential, and institutional sources (11% of total $PM_{2.5}$ emissions), mainly associated with residential wood burning (10% of total $PM_{2.5}$ emissions). The ore and mineral industry sector and transportation and mobile equipment sources each contributed 2% of total $PM_{2.5}$ emissions, while other source sectors accounted for less than 1%.

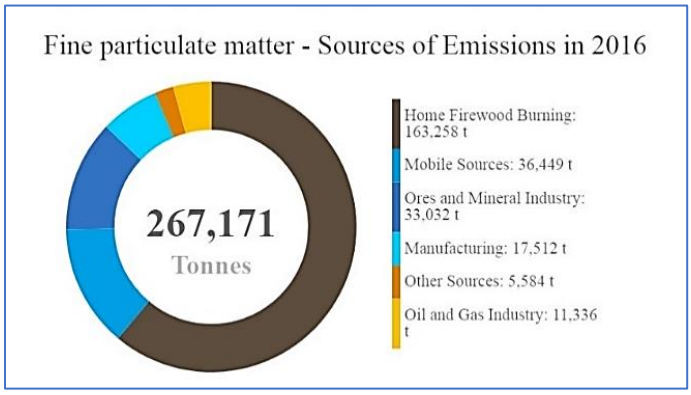
Figure A1 presents the distribution of $PM_{2.5}$ emissions in 2016, not including dust emissions and natural sources (e.g. forest fires). As indicated above, residential wood burning is responsible for the largest share of $PM_{2.5}$ emissions in Canada. Figure A2 shows trends in $PM_{2.5}$ emissions from 1990 to 2018. Dust emissions have steadily increased in absolute and relative terms over this period and accounted for more than 50% of total $PM_{2.5}$ emissions. Particulate emission from other sectors have generally decreased owing to new technologies and practices.

Table A1. National summary of annual $PM_{2.5}$ emissions (in tonnes) between 2015 and 2017

Sectors	2015	2016	2017
Ore and Mineral Industry	31,000	32,000	35,000
Oil and Gas Industry	12,000	11,000	13,000
Electric Power Generation (Utilities)	3,500	3,400	3,300
Manufacturing	18,000	17,000	17,000
Transportation and Mobile Equipment	37,000	33,000	35,000
Agriculture	370,000	380,000	380,000
Commercial/Residential/Institutional	180,000	180,000	180,000
Incineration and Waste	2,700	2,700	2,800
Paints and Solvents	15	16	23
Dust	920,000	930,000	930,000
Fires	10,000	9,100	4,800
Total	**1,600,000**	**1,600,000**	**1,600,000**

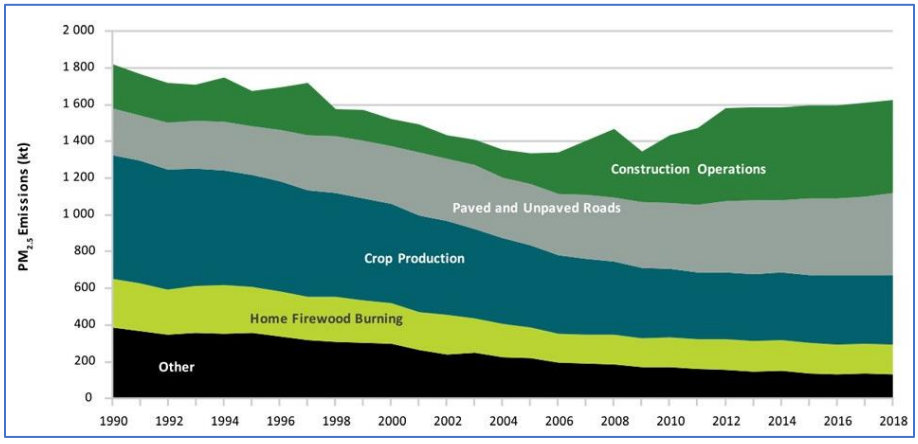
Source: Canada's Air Pollutant Emissions Inventory Report 2020: chapter 2.1, Environment and Climate Change Canada.

Figure A1. Human sources of $PM_{2.5}$ emissions (excluding dust emissions) in Canada in 2016



Source: Canada's Air, Canadian Council of Ministers of the Environment.

Figure A2. National trends in annual $PM_{2.5}$ emissions in kilotonnes (kt) from 1990 to 2018



Source: Canada's Air Pollutant Emissions Inventory Report 2020: chapter 2.1, Environment and Climate Change Canada.

Health effects

Exposures to $PM_{2.5}$ can negatively impact the heart and lungs and can lead to health issues such as asthma symptoms, chronic bronchitis, and heart attacks. Exposure to $PM_{2.5}$ is also linked to increased emergency room visits and hospitalization for respiratory and cardiovascular problems, as well as increased risk of

premature death. Vulnerable population groups, such as children and those with pre-existing cardiovascular and respiratory disease, are more sensitive to these effects (Health Canada 2013).

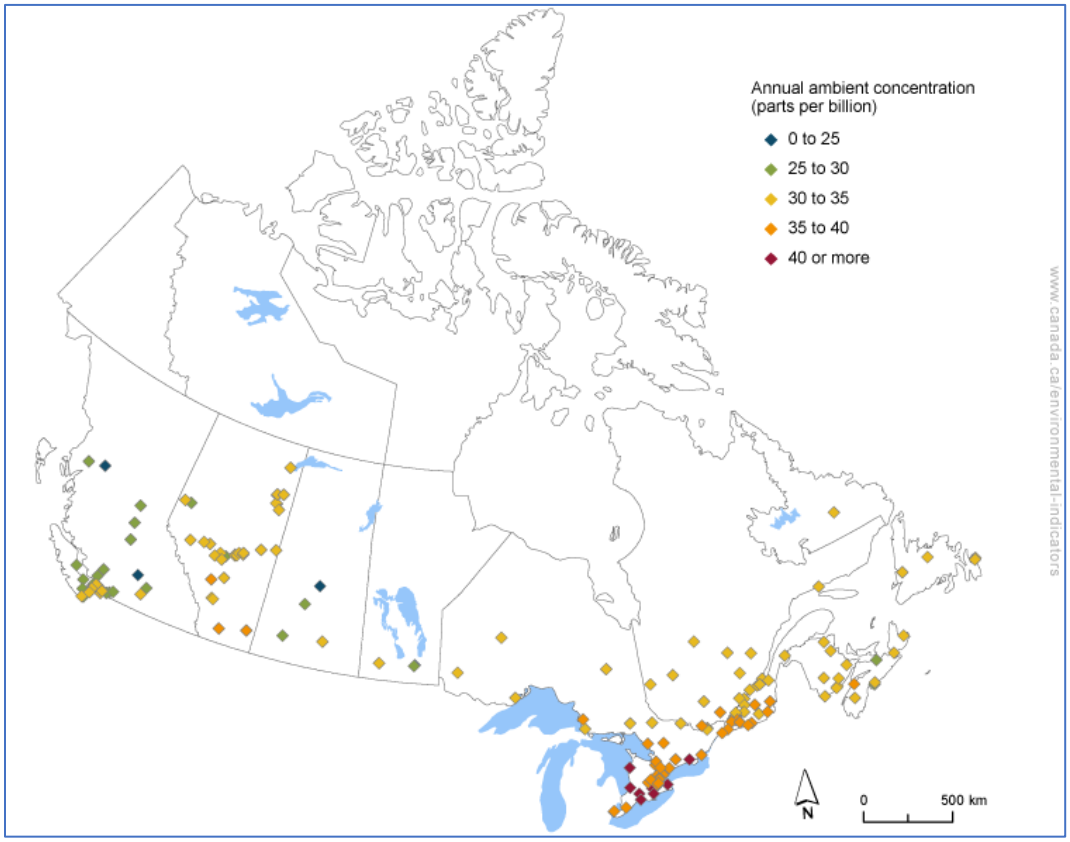
Ground-level ozone

Precursor emissions and ambient levels in Canada

Ozone is an colourless, odourless and highly irritating gas and a major component of smog. Populations are exposed to ozone that forms at ground level, which is also referred to as tropospheric ozone. Ozone is not emitted directly by sources: It is considered a secondary pollutant because it is formed through photochemical reactions between NOx and VOCs in the presence of sunlight. Ozone levels are generally higher in summer than in winter owing to favourable environmental conditions that increase ozone formation.

Figure A3 presents the distribution of ozone across Canada in 2016, as measured at National Air Pollution Surveillance (NAPS) stations. Annual average ambient concentrations generally vary between 30 and 40 parts per billion (ppb), reaching values above 40 ppb at stations located in southern Ontario. Average ozone concentrations over the last decade do not indicate any trend. By contrast, peak ozone concentrations, as indicated by the 4th highest daily maximum 8-hour average ozone concentration, shows a slight downward trend (from 68 ppb in 2002 to 57 ppb in 2016, a 0.75% annual decrease).

Figure A3. Average ozone concentrations measured at National Air Pollution Surveillance stations in 2016



Source: ECCC 2018

Health effects

Exposure to ozone is associated with respiratory symptoms (e.g. throat irritation, coughing, shortness of breath and reduced lung function) and can aggravate existing conditions like asthma or other chronic lung diseases. Exposure to ozone also increases the risk of premature death. Sensitive populations (e.g. children and people with respiratory or cardiovascular conditions) are at higher risk (Health Canada 2013).

Nitrogen dioxide

Emissions in Canada

NO_2 and other NOx compounds are formed primarily through the burning of fossil fuels. Although a large fraction of NOx is released as NO, the latter reacts quickly in the atmosphere and converts to NO_2. At higher concentrations, NO_2 has a strong, harsh odour and can form a brownish haze in polluted urban air.

In 2015–2017, approximately 1.7 Mt of NOx were emitted in Canada (Table A2). The largest contributions were associated with transportation and mobile equipment, which accounted for 51% of total NOx emissions on average, followed by emissions from the oil and gas industry (28%) and electric power generation (9%).

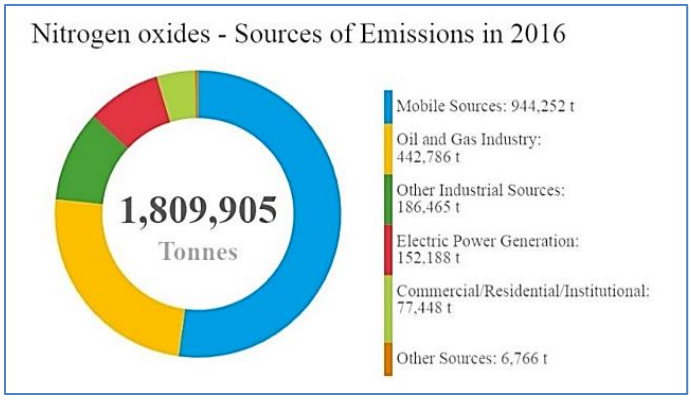
Table A2. National summary of annual NOx emissions (tonnes) between 2015 and 2017

Sectors	2015	2016	2017
Ore and Mineral Industries	81,000	78,000	86,000
Oil and Gas Industry	480,000	470,000	480,000
Electric Power Generation (Utilities)	150,000	150,000	150,000
Manufacturing	69,000	69,000	70,000
Transportation and Mobile Equipment	890,000	840,000	880,000
Agriculture	4,100	4,200	4,200
Commercial/Residential/Institutional	80,000	77,000	79,000
Incineration and Waste	4,200	4,200	5,200
Paints and Solvents	23	23	17
Dust	-	-	-
Fires	2,000	1,700	990
Total	**1,800,000**	**1,700,000**	**1,700,000**

Source: Canada's Air Pollutant Emissions Inventory Report 2020: chapter 2.3, Environment and Climate Change Canada.

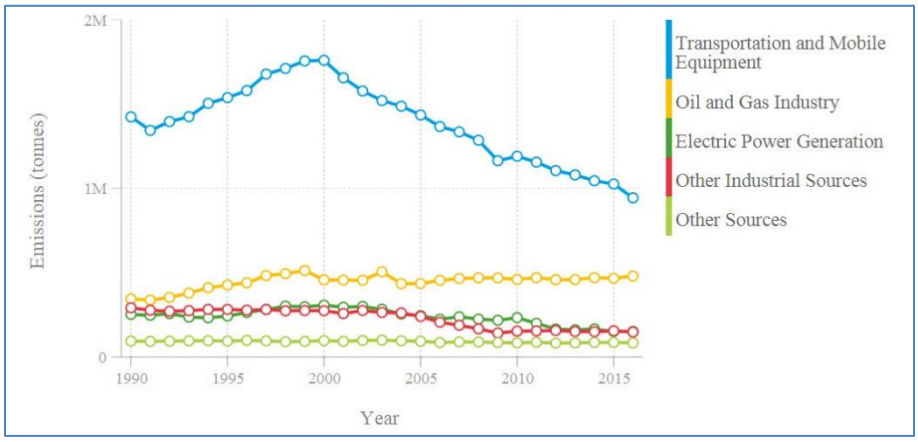
Figure A4 presents the distribution of NOx emission sources, excluding natural source emissions. Figure A5 presents the trends in NOx emission for major source sectors from 1990 to 2016. A considerable decrease in mobile source emissions has been observed since 2000, driven by decreases in emissions from light-duty gasoline trucks and vehicles following the adoption of increasingly stringent vehicle regulations. Upstream oil and gas industry and marine transportation are the sectors where NOx emissions have increased in recent decades, which can be attributed to expansion and growth in activity (ECCC 2018).

Figure A4. Human sources of NOx emissions in Canada in 2016



Source: Canada's Air, Canadian Council of Ministers of the Environment.

Figure A5. National NOx emissions by source type from 1990 to 2016



Source: Canada's Air, Canadian Council of Ministers of the Environment.

Health effects

Short-term exposure to NO_2 can provoke a range of adverse respiratory effects, including decreased lung function, increased respiratory symptoms, and airway inflammation, and can aggravate respiratory diseases, particularly asthma and chronic obstructive pulmonary disease. Short-term exposure to NO_2 also increases the risk of premature death. Long-term exposure to NO_2 may contribute to allergic responses, asthma development and may increase susceptibility to respiratory infections. Further investigations are needed to characterize and confirm relationships between exposure to NO_2 and health outcomes (Health Canada 2016).

Volatile organic compounds

Emissions in Canada

VOCs are organic chemicals that vaporize under normal atmospheric conditions. VOCs exposed to sunlight react photochemically with NOx, producing ozone and organic aerosols, two key components of smog. VOCs include a range of chemicals that can be found in natural emissions (biogenic emissions), combustion products (e.g. engine exhaust) and evaporative emissions from consumer products (e.g. oil-based paint, cleaning products, solvents).

Between 2015 and 2017, approximately 1.8 Mt of VOCs were released in Canada (Table A3). The largest human sources are related to oil and gas extraction (37% of total VOC emissions), the use of paints and solvents (21% of total VOC emissions), and transportation and home firewood burning (each 16% of total VOC emissions) (Table A3 and Figure 6).[24]

From 1990 to 2018, VOC emissions decreased by 37% (1.1 Mt) (ECCC 2018). This decrease was driven by increasingly stringent regulations targeting off-road spark ignition engines (i.e. those powered by gasoline, liquefied petroleum gas or compressed natural gas), as demonstrated in Figure A7 (covering 1990–2016). Reductions in VOC emissions from on-road light-duty gasoline vehicles and trucks also contributed to the downward trend. By contrast, emissions from the oil and gas industry have increased slightly over time.

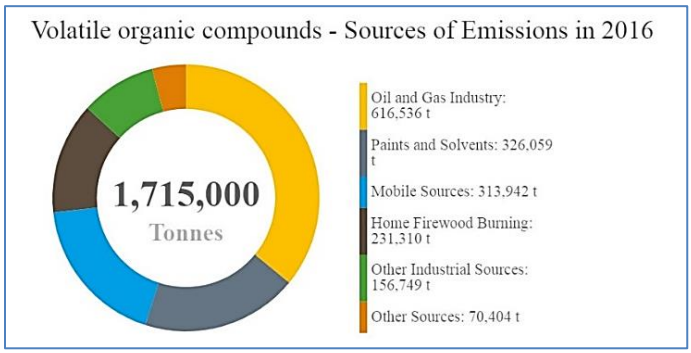
[24] Values in Table A3 and Figure A6 were obtained from different datasets; slight discrepancies are possible. For example, differences may vary based on rounding and sector combination.

Table A3. National summary of annual VOC emissions (tonnes) between 2015 and 2017

Sectors	2015	2016	2017
Ore and Mineral Industries	11,000	11,000	11,000
Oil and Gas Industry	730,000	660,000	660,000
Electric Power Generation (Utilities)	1,600	1,600	1,300
Manufacturing	110,000	100,000	100,000
Transportation and Mobile Equipment	300,000	280,000	290,000
Agriculture	110,000	110,000	120,000
Commercial/Residential/Institutional	290,000	290,000	290,000
Incineration and Waste	9,900	10,000	10,000
Paints and Solvents	360,000	360,000	370,000
Dust	-	-	-
Fires	5,900	4,900	2,900
Grand Total	**1,900,000**	**1,800,000**	**1,800,000**

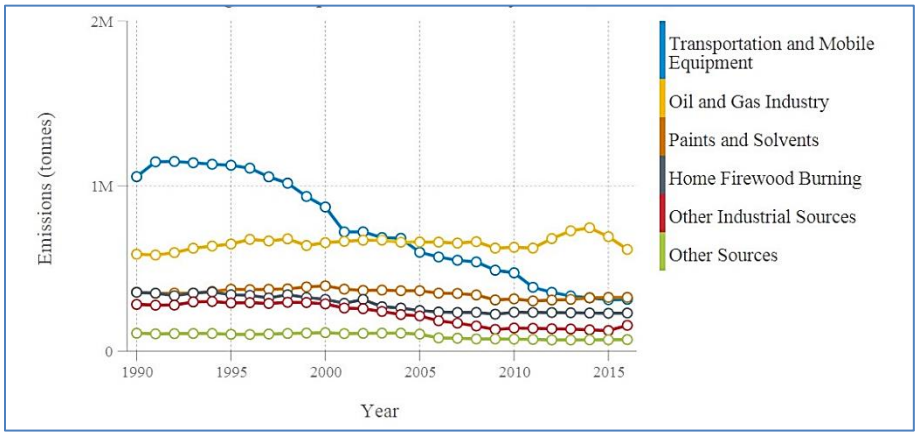
Source: Canada's Air Pollutant Emissions Inventory Report 2020: chapter 2.4, Environment and Climate Change Canada.

Figure A6. Source of VOC emissions from human sources in Canada in 2016



Source: Canada's Air, Canadian Council of Ministers of the Environment.

Figure A7. National VOC emissions by source type from 1990 to 2016



Source: Canada's Air, Canadian Council of Ministers of the Environment.

References

[ECCC] Environment and Climate Change Canada. 2018. Canadian Environmental Sustainability Indicators – Air quality. Cat. EN4-144/57-2018E-PDF. Available at: www.canada.ca/en/environment-climate-change/services/environmental-indicators/air-quality.html

ECCC. 2020. Canada's air pollutant emissions inventory report. Cat. En81-30E-PDF. Available online at www.canada.ca/en/environment-climate-change/services/air-pollution/publications/emissions-inventory-report-2020.html

Health Canada. 2013. Canadian smog science assessment – Volume 2: Health Effects. Air Health Sciences Division. Cat. En88-5/2-2013E-PDF. 533 pp.

Health Canada. 2016. Human health risk assessment for ambient nitrogen dioxide. Water and air quality Bureau, Safe Environments Directorate. Cat. H144-31/2016E-PDF. 288 pp.

Appendix B. NO_2, ozone and $PM_{2.5}$ concentration-response functions in AQBAT version 3.0

Pollutant	Endpoint	Source(s)	Details[a]	Averaging period	Regression type	Form[a]	Mean beta[b]	SE beta[c]
NO_2	Acute exposure mortality	Burnett et al. (2004) Results from model with four gases provided by R.T. Burnett, in addition to published results	Analysis of air pollution and mortality in 12 Canadian cities. The lead author provided results from additional multi-pollutant models not reported in the paper; the four-gas model was selected based on the overall t-value among the candidate models. Percent excess mortality (associated with the mean pollutant concentration) from Poisson regression models for CO, NO_2, ozone and SO_2, respectively, was 0.19% (t = 0.73, 1.0 ppm), 1.69% (t = 3.00, 22.4 ppb), 2.60% (t = 6.16, 30.6 ppb) and 0.23% (t = 2.09, 5.0 ppb). These results translate into regression coefficients (SE) of 0.00190 (0.00260), 0.000748 (0.000249), 0.000839 (0.000136) and 0.000459 (0.000220) for the same four pollutants, respectively. Although this multi-pollutant model excluded PM, it was selected as the model that best reflected the impact of the overall air pollution mix. Because of multi-co-linearity among pollutants, this model should nonetheless still reflect impacts of PM. In any case, the effects of PM in this study were reduced substantially when it was modelled together with NO_2, the effect of which predominated in this analysis. The AQBAT CRF is applied to all members of all age groups.	24 h	Log(RR) or Log(OR)	Normal	7.48E-04	2.49E-04
Ozone				1 h			8.39E-04	1.36E-04
Ozone (May–Sept.)	Respiratory mortality	Jerrett et al. (2009)	Jerrett et al. (2009) analyzed data from the American Cancer Society cohort study. The relative risk of death from respiratory causes was 1.040 (95% CI 1.010–1.067) per 10 ppb ozone in a model with $PM_{2.5}$; exposure was based on average of quarterly averages with ≥ 75% of daily values. This translates into a coefficient of 0.00392 with SE 0.00132. The AQBAT CRF is applied to the Canadian population ≥ 25 years of age.	1 h	Log(RR) or Log(OR)	Normal	3.92E-03	1.32E-03

Pollutant	Endpoint	Source(s)	Details[a]	Averaging period	Regression type	Form[a]	Mean beta[b]	SE beta[c]
Ozone (May–Sept.)	Acute respiratory symptom days	Krupnick et al. (1990)	The authors reported on the association between ozone and the occurrence of acute respiratory symptoms in a panel of California families. They employed a Markov model that accounted for the occurrence of symptoms on the previous day and adjusted for Coefficient of Haze (CoH)[d], NO_2 and SO_2 as co-pollutants. The incremental change in frequency of symptoms was calculated by substituting the coefficient from table V, column 3, divided by 10 to convert from pphm to ppb, together with the transitional probabilities, $p_1 = 0.775$ and $p_2 = 0.0468$ (provided by the authors), into equation 3 on page 12 of the paper. The baseline frequency of symptoms was calculated by substituting p_1 and p_0 into equation 2. Thus, the proportional change per 1 ppb ozone is the output from equation 3 divided by that of equation 2, 0.000786 (SE 0.000386). The AQBAT CRF is applied to adults and nonasthmatic (85.7%) children aged 5–19 years.	1 h	Linear	Normal	7.86E-04	3.86E-04

Pollutant	Endpoint	Source(s)	Details[a]	Averaging period	Regression type	Form[a]	Mean beta[b]	SE beta[c]
Ozone (May–Sept.)	Asthma symptom days	Mortimer et al. (2002) Schildcrout et al. (2006)	Numerous panel studies have been conducted on the association between ozone and asthma exacerbations in children. Several of these were carried out in summer camps, which may not reflect typical exposure conditions, in that campers would be expected to spend more time outdoors compared with non-campers. Others have been conducted in locations such as Mexico City and Los Angeles, which experience very high ozone concentrations not representative of conditions in Canada. We therefore selected two large multicentre North American panel studies as the source of the CRF. Mortimer et al. (2002) analyzed data collected in summer 1993 for 846 inner-city children aged 4–9 years from eight American cities. The average 8 h maximum ozone concentration among all cities was 48 ppb. The odds ratio for morning asthma symptoms was 1.16 (95% CI 1.02–1.30) in relation to a 15 ppb increment in average of lag 1–5 day ozone. This was reduced to 1.07 (0.92–1.26) in a joint model with NO_2 in seven cities and to 1.04 (0.70–1.55) in a joint model with PM_{10} based on three cities (table 4). Schildcrout et al. (2006) analyzed data collected from 1993 to 1995 for 990 children aged 5–13 years, also from eight cities and including Toronto, and only with Baltimore in common with the Mortimer et al. (2002) analysis. Median 1 h maximum ozone concentrations ranged from 43 to 65.8 ppb. The odds ratio for asthma symptoms was 1.06 (95% CI 0.92–1.23) in relation to a 30 ppb increment in lag 0 ozone (the largest effect among lags considered; figure 1). Joint models with other pollutants were not run. The log odds ratio from Mortimer et al. (2002) based on the 8 h maximum (joint model with NO_2) was multiplied by 1.13 (the ratio of 1 h maximum to 8 h maximum in Canadian cities) and pooled with the Schildcrout et al. (2006) result to obtain an odds ratio of 1.05 (95% CI 0.96–1.14) per 20 ppb. The same baseline frequency of asthma symptoms and prevalence of current wheeze as for $PM_{2.5}$ was applied to 14.3% of children aged 5–19 years.	1 h	Log(RR) or Log(OR)	Normal	2.38E-03	2.19E-04

Pollutant	Endpoint	Source(s)	Details[a]	Averaging period	Regression type	Form[a]	Mean beta[b]	SE beta[c]
Ozone (May–Sept.)	Minor restricted activity days	Ostro and Rothschild (1989)	Ostro and Rothschild (1989) reported an association between ozone and minor reduced activity days based on an analysis of data from the US Health Interview Survey. They reported results by year for 1976–1981 based on a Poisson regression model including both ozone and $PM_{2.5}$ (table 4, column 2). Coefficients were pooled using a random effect model; a pooled estimate of 0.000530 (SE 0.00291) per 1 ppb daily 1 h maximum ozone was obtained. The baseline daily rate of minor reduced activity days per person was 7.8/365 = 0.0214. The AQBAT CRF is applied to adults and nonasthmatic (85.7%) children aged 5–19 years.	1 h	Log(RR) or Log(OR)	Normal	5.30E-04	2.91E-03
Ozone (May–Sept.)	Respiratory emergency room visits	Burnett et al. (1997) Stieb et al. (2000)	Substantially more data are available pertaining to air pollution and hospital admissions in Canada relative to emergency department visits. We therefore elected to represent the effects of air pollution on respiratory emergency department visits using the results for hospital admissions scaled up in number based on the relative frequency of hospital admissions and emergency visits for these conditions. Thus, the coefficient per unit air pollution was the same as for hospital admissions based on Burnett et al. (1997), i.e. 0.000791 (SE 0.000355) per 1 ppb. The baseline rate of emergency visits is equal to the baseline rate of hospital admissions divided by 0.198, the proportion of visits resulting in hospital admission as reported by Stieb et al. (2000). The AQBAT CRF is applied to all members of all age groups.	1 h	Log(RR) or Log(OR)	Normal	7.91E-04	3.55E-04
Ozone (May–Sept.)	Respiratory hospital admissions	Burnett et al. (1997)	Burnett et al. (1997) reported the results of a study on ozone and respiratory hospital admissions in 16 Canadian cities. Based on results from a Poisson regression model, which simultaneously adjusted for dew point temperature, CO and CoH, they reported a relative risk of 1.024 (p = 0.0258) per 30 ppb daily 1 h maximum ozone. Taking the natural logarithm of the relative risk and dividing by 30 yields a coefficient of 0.000791 (SE 0.000355) per 1 ppb. The AQBAT CRF is applied to all members of all age groups.	1 h	Log(RR) or Log(OR)	Normal	7.91E-04	3.55E-04

Pollutant	Endpoint	Source(s)	Details[a]	Averaging period	Regression type	Form[a]	Mean beta[b]	SE beta[c]
$PM_{2.5}$	Acute respiratory symptom days	Krupnick et al. (1990)	The authors reported on the association between CoH and the occurrence of acute respiratory symptoms in a panel of California families. They employed a Markov model that accounted for the occurrence of symptoms on the previous day and adjusted for ozone, NO_2 and SO_2 as co-pollutants. The incremental change in frequency of symptoms was calculated by substituting the coefficient from table V, column 3, multiplied by 0.211 to convert from CoH to $PM_{2.5}$, together with the transitional probabilities, $p_1 = 0.775$ and $p_2 = 0.0468$ (provided by the authors), into equation 3 on page 12 of the paper. The conversion from CoH to $PM_{2.5}$ was calculated by dividing the ratio of CoH to TSP (0.116) provided by the authors by the ratio of PM_{10} to TSP (0.55) provided by Environment Canada. This assumes that the toxicity of $PM_{2.5}$ per 1 $\mu g/m^3$ is the same as that of PM_{10}. The baseline frequency of symptoms was calculated by substituting p_1 and p_0 into equation 2. Thus, the proportional change per 1 $\mu g/m^3$ $PM_{2.5}$ is the output from equation 3 divided by that of equation 2, 0.00266 (SE 0.00139). The AQBAT CRF is applied to adults and nonasthmatic (85.7%) children aged 5–19 years.	24 h	Linear	Normal	2.66E-03	1.39E-03
$PM_{2.5}$	Adult chronic bronchitis cases	Abbey et al. (1995)	Abbey et al. (1995) reported the results of a cohort study of air pollution and the development of chronic lung disease among non-smoking Seventh Day Adventists living in California. Based on a logistic regression model, which also included personal characteristics, they reported an odds ratio of 1.81 (95% CI 0.98–3.25) for the development of chronic bronchitis per 45 $\mu g/m^3$ $PM_{2.5}$ (table 2, row 2). Taking the natural log of the odds ratio and dividing by 45 yields a coefficient of 0.0132 (SE 0.006 80) per 1 $\mu g/m^3$ $PM_{2.5}$. They reported that the 10-year incidence of chronic bronchitis was 6.26% (117 new cases occurred among 1868 subjects for whom $PM_{2.5}$ exposures could be estimated). We calculate the annual incidence, p_1, from the expression: $0.0626 = 1 - (1 - p_1)^{10}$, so that $p_1 = 0.006\ 44$. The AQBAT CRF is applied to the Canadian population ≥ 25 years of age.	24 h	Log(RR) or Log(OR)	Normal	1.32E-02	6.80E-03

Pollutant	Endpoint	Source(s)	Details[a]	Averaging period	Regression type	Form[a]	Mean beta[b]	SE beta[c]
$PM_{2.5}$	Asthma symptom days	Weinmayr et al. (2010) Ward and Ayres (2004) Dell et al. (2010)	These parameters are derived using the same approach as described in the Health Risk of Air Pollution in Europe project of the WHO European Centre for Environment and Health. Weinmayr et al. (2010) conducted a systematic review and meta-analysis based on 36 studies of the association between air pollution and asthma symptoms in children. The pooled odds ratio was 1.028 (95% CI 1.006–1.051) per 10 µg/m^3 PM_{10} (table 2) based on a random effect model including all studies. This is based on single pollutant models, as results from multi-pollutant models were not consistently available. However, the derived effect size is nonetheless much smaller than that observed in a multi-pollutant model for North American cities in Mortimer et al. (2002). In order to derive an odds ratio for $PM_{2.5}$, we multiplied the log odds ratio for PM_{10} by 2.37, which is the average of the ratio of log pooled odds ratios for $PM_{2.5}$ vs. PM_{10} for cough and other respiratory symptoms reported by Ward and Ayres (2004; tables 3 and 4) in their earlier meta-analysis. The result is an odds ratio for $PM_{2.5}$ of 1.07 (95% CI 1.01–1.12). The baseline daily frequency of asthma symptoms in asthmatic children varies widely in panel studies. We have conservatively estimated it at 20%. The population to which this is applicable is based on the prevalence of current wheeze in Canada from the National Longitudinal Survey of Children and Youth (14.3%; Dell et al. 2010). This is applied to asthmatic children (14.3%) aged 5–19 years.	24 h	Log(RR) or Log(OR)	Normal	6.545E-03	2.646E-03

Pollutant	Endpoint	Source(s)	Details[a]	Averaging period	Regression type	Form[a]	Mean beta[b]	SE beta[c]
$PM_{2.5}$	Cardiac emergency room visits	Burnett et al. (1995) Stieb et al. (2000)	Substantially more data are available pertaining to air pollution and hospital admissions in Canada relative to emergency department visits. We therefore elected to represent the effects of air pollution on cardiac emergency department visits using the results for hospital admissions scaled up in number based on the relative frequency of hospital admissions and emergency visits for these conditions. Thus, the change in frequency per unit air pollution was the same as for hospital admissions based on Burnett et al. (1995) – i.e. 0.0711% (SE 0.0170) increase per 1 $\mu g/m^3$. The baseline rate of emergency visits is equal to the baseline rate of hospital admissions divided by 0.760, the proportion of visits resulting in hospital admission as reported by Stieb et al. (2000). The AQBAT CRF is applied to all members of all age groups.	24 h	Linear	Normal	7.11E-04	1.70E-04
$PM_{2.5}$	Cardiac hospital admissions	Burnett et al. (1995)	Burnett et al. (1995) reported a 3.3% (95% CI 1.7–4.8) increase in cardiac hospital admissions per 13 $\mu g/m^3$ sulphate based on a linear regression model that also included ozone and temperature (table 5, row 2). Multiplying by the average ratio of sulphate to $PM_{2.5}$ of 0.28 (Environment Canada), this equates to a 0.0711% (SE 0.0170) increase per 1 $\mu g/m^3$ $PM_{2.5}$. The AQBAT CRF is applied to all members of all age groups.	24 h	Linear	Normal	7.11E-04	1.70E-04

Pollutant	Endpoint	Source(s)	Details[a]	Averaging period	Regression type	Form[a]	Mean beta[b]	SE beta[c]
$PM_{2.5}$	Child acute bronchitis episodes	Hoek et al. (2012) Dockery et al. (1996)	These parameters are derived using the same approach as described in the Health Risk of Air Pollution in Europe project of the WHO European Centre for Environment and Health. Hoek et al. (2012) conducted a meta-analysis of eight cross-sectional studies from Europe and North America, including the 24 cities study, which included data from several Canadian communities. The random effect pooled estimate of the odds ratio was 1.08 (95% CI 0.98–1.19) per 10 µg/m^3 PM_{10} (table 3), adjusted for age, sex, maternal education, paternal education, household crowding, current parental smoking, mother smoking during pregnancy, gas cooking, unvented gas/oil/kerosene heater, mould, nationality, birth order and "ever had a pet." The effect size was reduced based on joint models with SO_2, but this was based on only three studies (table 4). The average prevalence of bronchitis among the studies was 18.6% (table 2). In the 24 cities study, the odds ratio for bronchitis for $PM_{2.5}$ was identical to that for PM_{10} across the exposure difference between highest- and lowest-exposure communities, 17.3 and 14.9 µg/m^3 for PM_{10} and $PM_{2.5}$, respectively (tables 1 and 4). We therefore multiply the log of the pooled odds ratios for PM_{10} by this ratio (1.16) in order to derive a log odds ratio per 10 µg/m^3 $PM_{2.5}$, resulting in an odds ratio of 1.09 (95% CI 0.98–1.22). This is applied to the population of children 5–19 years of age.	24 h	Log(RR) or Log(OR)	Normal	8.927E-03	5.745E-03
$PM_{2.5}$	Chronic obstructive pulmonary disease mortality	H. Shin, personal communication, Health Canada, 2013	Parameters were derived from a meta-analysis of cohort studies of air pollution and cause-specific mortality. The central estimate was set at the result of the American Cancer Society cohort study, and the CIs were based on a gamma distribution reflecting the distribution of results from other studies. The values specified here for beta and SE are actually the alpha and beta parameters of the gamma distribution. The AQBAT CRFs are applied to all Canadians ≥ 25 years of age.	24 h	Log(RR) or Log(OR)	Gamma	1.457E+01	6.010E-04
	Chronic exposure cerebrovascular mortality			24 h	Log(RR) or Log(OR)	Gamma	4.884E+0	3.375E-03
	Chronic exposure ischemic heart disease mortality			24 h	Log(RR) or Log(OR)	Gamma	1.156E+0	2.117E-03
	Chronic exposure lung cancer mortality			24 h	Log(RR) or Log(OR)	Gamma	4.930E+0	3.168E-03

Pollutant	Endpoint	Source(s)	Details[a]	Averaging period	Regression type	Form[a]	Mean beta[b]	SE beta[c]
$PM_{2.5}$	Chronic Exposure Internal Cause Mortality	Crouse et al. 2012	Crouse et al. (2012) examined the association between $PM_{2.5}$ derived from satellite observations and mortality during ten years of follow-up of a cohort of 2.1 million Canadian adults (≥ 25 years of age) based on the 1991 long form census. Using a spatial random-effects Cox model including individual and ecological covariates and an urban/rural indicator, and accounting for spatial autocorrelation among cohort members, they reported a hazard ratio of 1.10 (95% CI 1.05-1.15) per 10 $\mu g/m^3$ $PM_{2.5}$. This translates to a β of 0.00953 with standard error 0.00232. [note: choose either 4 specific causes or internal causes, not both]	24 h	Log(RR) or Log(OR)	Normal	9.53E-3	2.32E-03
$PM_{2.5}$	Chronic Exposure Internal Cause Mortality	Burnett et al. 2018	Burnett et al. modelled the association between $PM_{2.5}$ and mortality in 41 cohorts from 16 countries. Data were analyzed using log linear models employing transformations, T(z), of concentration, which permit a variety of shapes (linear, log linear, supralinear, sublinear, S-shaped) of the concentration response function. The model form is $R(z) = \exp\{\theta T(z)\}$, where $T(z) = \log(1 + z/\alpha)\omega(z)$. $\omega(z) = 1/(1 + \exp\{-(z - \mu)/(\tau r)\})$ is a logistic weighting function of z, μ and τ where r represents the range in the pollutant concentrations, τ controls the curvature and μ controls the shape of the concentration response. The CRF is applied to all Canadians ≥ 25 years of age. [note: choose either 4 specific causes or internal causes, not both]	24 h	Burnett nonlinear	Normal	Θ =0.143 Additional parameters: α = 1.6 μ = 15.5 τ * r = 36.8 threshold concentration = 2.4	SE(θ) = 0.01807

Pollutant	Endpoint	Source(s)	Details[a]	Averaging period	Regression type	Form[a]	Mean beta[b]	SE beta[c]
$PM_{2.5}$	Respiratory emergency room visits	Burnett et al. (1995) Stieb et al. (2000)	Substantially more data are available pertaining to air pollution and hospital admissions in Canada relative to emergency department visits. We therefore elected to represent the effects of air pollution on respiratory emergency department visits using the results for respiratory hospital admissions scaled up in number based on the relative frequency of hospital admissions and emergency visits for these conditions. Thus, the change in frequency per unit air pollution was the same as for hospital admissions based on Burnett et al. (1995) – i.e. 0.0754% (SE 0.0132) increase per 1 $\mu g/m^3$. The baseline rate of respiratory emergency visits is equal to the baseline rate of hospital admissions divided by 0.198, the proportion of visits resulting in hospital admission as reported by Stieb et al. (2000). The AQBAT CRF is applied to all members of all age groups.	24 h	Linear	Normal	7.54E-04	1.32E-04
$PM_{2.5}$	Respiratory hospital admissions	Burnett et al. (1995)	Burnett et al. (1995) reported a 3.5% (95% CI 2.3–4.7) increase in respiratory hospital admissions per 13 $\mu g/m^3$ sulphate based on a linear regression model that also included ozone and temperature (table 4, row 2). Multiplying by the average ratio of sulphate to $PM_{2.5}$ of 0.28 (Environment Canada), this equates to a 0.0754% (SE 0.0132) increase per 1 $\mu g/m^3$ $PM_{2.5}$. The AQBAT CRF is applied to all members of all age groups.	24 h	Linear	Normal	7.54E-04	1.32E-04

Pollutant	Endpoint	Source(s)	Details[a]	Averaging period	Regression type	Form[a]	Mean beta[b]	SE beta[c]
$PM_{2.5}$	Restricted activity days	Ostro (1987) Ostro and Rothschild (1989) Chestnut et al. (1999)	Ostro (1987) reported an association between $PM_{2.5}$ and reduced activity days based on an analysis of data from the US Health Interview Survey. They reported results by year for 1976–1981 based on a Poisson regression model (table III, column 2). We pooled these coefficients using a random effect model and obtained a pooled estimate of 0.00481 (SE 0.00101) per 1 μg/m^3 $PM_{2.5}$. The baseline daily rate of reduced activity days per person was 0.052 (Chestnut et al. 1999). Ostro and Rothschild (1989) also reported an analysis of $PM_{2.5}$ and respiratory reduced activity days, in which they adjusted for the simultaneous effects of ozone. The effects of $PM_{2.5}$ were unaffected by this adjustment; thus, we opted to use the results from their earlier analysis on the grounds that reduced activity days are a more global outcome than the more narrowly defined respiratory reduced activity days. The AQBAT CRF is applied to adults and nonasthmatic (85.7%) children aged 5–19 years.	24 h	Log(RR) or Log(OR)	Normal	4.81E-03	1.01E-03

[a] Distribution forms: Normal, Gamma, Discrete, and Triangular
[b] Mean beta: mean of the pollutant coefficient (regression parameter)
[c] SE beta: standard error of the pollutant coefficient
[d] Coefficient of haze is a measure of the atmospheric impedance of light caused by suspended atmospheric particles or aerosols.

References

Abbey DE, Lebowitz MD, Mills PK, Petersen FF, Beeson WL, Burchette RJ. 1995. Long term ambient concentrations of particulates and oxidants and development of chronic disease in a cohort of nonsmoking California residents. Inhal Toxicol 7: 19–34.

Burnett RT, Dales R, Krewski D, Vincent R, Dann T, Brook JR. 1995. Associations between ambient particulate sulphate and admissions to Ontario hospitals for cardiac and respiratory diseases. Am J Epidemiol 142(1): 15–22.

Burnett RT, Brook JR, Yung WT, Dales RE, Krewski D. 1997. Association between ozone and hospitalization for respiratory diseases in 16 Canadian cities. Environ Res 72: 24–31.

Burnett RT, Stieb D, Brook JR, Cakmak S, Dales R, Raizenne M, Vincent R, Dann T. 2004. Associations between short-term changes in nitrogen dioxide and mortality in Canadian cities. Arch Environ Health 59(5): 228–236.

Burnett R, Chen H, Szyszkowicz M et al. 2018. Global estimates of mortality associated with long-term exposure to outdoor fine particulate matter. Proceedings of the National Academy of Sciences 115 (38): 9592–9597.

Chestnut LG, Mills D, Ragland S, Rowe RD. 1999. Air Quality Valuation Model (AQVM 3.0) report 2: Methodology. Final report prepared for Environment Canada and Health Canada by Stratus Consulting Inc.

Crouse DL, Peters PA, van Donkelaar A, Goldberg MS, Villeneuve PJ, Brion O, Khan S, Atari DO, Jerrett M, Pope CA, Brauer M, Brook JR, Martin RV, Stieb D, Burnett RT. 2012. Risk of nonaccidental and cardiovascular mortality in relation to long-term exposure to low concentrations of fine particulate matter: a Canadian national-level cohort study. Environ Health Perspect 120(5): 708–714.

Dell SD, Foty RG, Gilbert NL, Jerret M, To T, Walter SD, Stieb DM. 2010. Asthma and allergic disease prevalence in a diverse sample of Toronto school children: results from the Toronto Child Health Evaluation Questionnaire (T-CHEQ) Study. Can Respir J 17(1): e1–6.

Dockery DW, Cunningham J, Damokosh AI, Neas LM, Spengler JD, Koutrakis P, Ware JH, Raizenne M, Speitzer FE. 1996. Health effects of acid aerosols on North American children: respiratory symptoms. Environ Health Perspect 104: 500–505.

Hoek G, Pattenden S, Willers S, Antova T, Fabianova E, Braun-Fahrländer C, Forastiere F, Gehring U, Luttmann-Gibson H, Grize L, Heinrich J, Houthuijs D, Janssen N, Katsnelson B, Kosheleva A, Moshammer H, Neuberger M, Privalova L, Rudnai P, Speizer F, Slachtova H, Tomaskova H, Zlotkowska R, Fletcher T. 2012. PM_{10}, and children's respiratory symptoms and lung function in the PATY study. Eur Respir J 40(3): 538–547.

Jerrett M, Burnett RT, Pope CA III, Ito K, Thurston G, Krewski D, Shi Y, Calle E, Thun M. 2009. Long-term ozone exposure and mortality. N Engl J Med 360(11): 1085–1095.

Krupnick AJ, Harrington W, Ostro B. 1990. Ambient ozone and acute health effects: evidence from daily data. J Environ Econ Manage 18(1): 1–18.

Mortimer KM, Neas LM, Dockery DW, Redline S, Tager IB. 2002. The effect of air pollution on inner-city children with asthma. Eur Respir J 19(4): 699–705.

Ostro BD. 1987. Air pollution and morbidity revisited: a specification test. J Environ Econ Manage 14: 87–98.

Ostro BD, Rothschild S. 1989. Air pollution and acute respiratory morbidity: an observational study of multiple pollutants. Environ Res 50: 238–247.

Schildcrout JS, Sheppard L, Lumley T, Slaughter JC, Koenig JQ, Shapiro GG. 2006. Ambient air pollution and asthma exacerbations in children: an eight-city analysis. Am J Epidemiol 164(6): 505–517.

Shin HH, Cohen A, Pope CA III, Ezzati M, Lim SS, Hubbell B, Burnett RT. 2013. Critical issues in combining disparate sources of information to estimate the global burden of disease attributable to ambient fine particulate matter exposure. Presented at Methods for Research Synthesis: A Cross-Disciplinary Workshop, October 2, 2013, Harvard Center for Risk Analysis, Cambridge, MA. 24 pp.

Stieb DM, Beveridge RC, Brook JR, Smith-Doiron M, Burnett RT, Dales RE, Beaulieu S, Judek S, Mamedov A. 2000. Air pollution, aeroallergens and cardiorespiratory emergency department visits in Saint John, Canada. J Expo Anal Environ Epidemiol 10(5): 461–477.

Ward DJ, Ayres JG. 2004. Particulate air pollution and panel studies in children: a systematic review. Occup Environ Med 61(4): e13 (doi:10.1136/oem.2003.007088).

Weinmayr G, Romeo E, De Sario M, Weiland SK, Forastiere F. 2010. Short-term effects of PM_{10} and NO_2 on respiratory health among children with asthma or asthma-like symptoms: a systematic review and meta-analysis. Environ Health Perspect 118(4): 449–457.

Appendix C. Baseline incidence rates for key health endpoints by geographic area, in annual events per million population – 2011 estimates included in AQBAT version 3.0

Endpoint	Acute exposure mortality[a]	Acute respiratory symptom days	Asthma symptom days	Cardiac emergency room visits	Child acute bronchitis episodes	Chronic exposure mortality[b]	Chronic exposure respiratory mortality[c]	Minor restricted activity days	Respiratory emergency room visits	Restricted activity days
Population age group	All	NA 5–19; adults	Asthmatic 5–19	All	5–19	25+	30+	NA 5–19; adults	All	NA 5–19; adults
Canada	6,730	64,000,000	73,000,000	9,250	186,000	9,450	1,010	8,000,000	24,500	19,000,000
Newfoundland and Labrador	8,330	64,000,000	73,000,000	12,000	186,000	11,200	1,090	8,000,000	30,500	19,000,000
Prince Edward Island	7,960	64,000,000	73,000,000	13,600	186,000	11,200	1,420	8,000,000	37,100	19,000,000
Nova Scotia	8,390	64,000,000	73,000,000	11,400	186,000	11,500	1,350	8,000,000	26,800	19,000,000
New Brunswick	7,960	64,000,000	73,000,000	14,600	186,000	10,900	1,140	8,000,000	35,700	19,000,000
Quebec	7,210	64,000,000	73,000,000	10,500	186,000	9,830	1,100	8,000,000	29,600	19,000,000
Ontario	6,430	64,000,000	73,000,000	8,790	186,000	9,120	915	8,000,000	20,600	19,000,000
Manitoba	7,610	64,000,000	73,000,000	9,450	186,000	11,200	1,130	8,000,000	25,800	19,000,000
Saskatchewan	8,010	64,000,000	73,000,000	11,100	186,000	11,800	1,280	8,000,000	39,600	19,000,000
Alberta	5,340	64,000,000	73,000,000	6,740	186,000	7,740	891	8,000,000	21,900	19,000,000
British Columbia	6,710	64,000,000	73,000,000	8,360	186,000	9,250	994	8,000,000	21,400	19,000,000
Yukon	5,110	64,000,000	73,000,000	9,140	186,000	7,180	752	8,000,000	25,500	19,000,000
Northwest Territories	3,850	64,000,000	73,000,000	7,430	186,000	5,950	794	8,000,000	40,600	19,000,000
Nunavut	3,580	64,000,000	73,000,000	3,660	186,000	6,210	1,190	8,000,000	63,900	19,000,000

NA: nonasthmatic

[a] all-cause (nonaccidental) mortality among Canadians of all ages

[b] all-cause (nonaccidental) mortality in the Canadian population over 25 years of age

[c] all-cause respiratory mortality in the Canadian population over 30 years of age

Notes for interpreting the values: Baseline rates can vary among provinces or territories or a single value may apply to all Canadians (where more geographically precise values are unavailable). Values exceeding 1 million indicate that the number of incidences for each individual is greater than 1. For example, each asthmatic child aged 5–19 is associated, on average, with 73 asthma symptom days per year. Thus, the annual baseline rate per 1 million individuals for the target population is 73 million asthma days.

Appendix D. Additional air pollution and health impact estimates

Table D1. Premature deaths per 100,000 population – Most populated census divisions in 2016

Province - CD name (identifier)	Population	Deaths per 100,000
ON - Toronto (CD3520)	2,865,131	48
BC - Greater Vancouver (CD5915)	2,504,989	34
QC - Montréal (CD2466)	2,020,217	59
AB - Division No. 6 (CD4806)	1,577,299	26
ON - Peel (CD3521)	1,456,494	32
AB - Division No. 11 (CD4811)	1,434,083	40
ON - York (CD3519)	1,154,377	31
ON - Ottawa (CD3506)	969,510	32
MB - Division No. 11 (CD4611)	729,641	41
ON - Durham (CD3518)	669,507	44
QC - Québec (CD2423)	587,028	60
ON - Halton (CD3524)	566,264	44
ON - Hamilton (CD3525)	564,030	67
ON - Waterloo (CD3530)	549,606	45

AB: Alberta; BC: British Columbia; MB: Manitoba; ON: Ontario; QC: Quebec

Table D2. Premature deaths per 100,000 population – Census divisions with the highest rates in 2016

Province - CD name (identifier)	Population	Deaths per 100,000
BC - Okanagan-Similkameen (CD5907)	81,347	104
ON - Chatham-Kent (CD3536)	107,275	89
ON - Lambton (CD3538)	131,434	87
ON - Niagara (CD3526)	455,683	80
ON - Prince Edward (CD3513)	25,672	78
ON - Haldimand-Norfolk (CD3528)	113,312	74
ON - Essex (CD3537)	409,070	73
BC - Kootenay Boundary (CD5905)	30,466	73
ON - Northumberland (CD3514)	87,550	71
BC - Central Kootenay (CD5903)	59,462	70
ON - Elgin (CD3534)	91,940	69
ON - Brant (CD3529)	146,231	68
QC - Pierre-De Saurel (CD2453)	51,706	68
BC - North Okanagan (CD5937)	85,110	67
BC - Central Okanagan (CD5935)	197,703	67

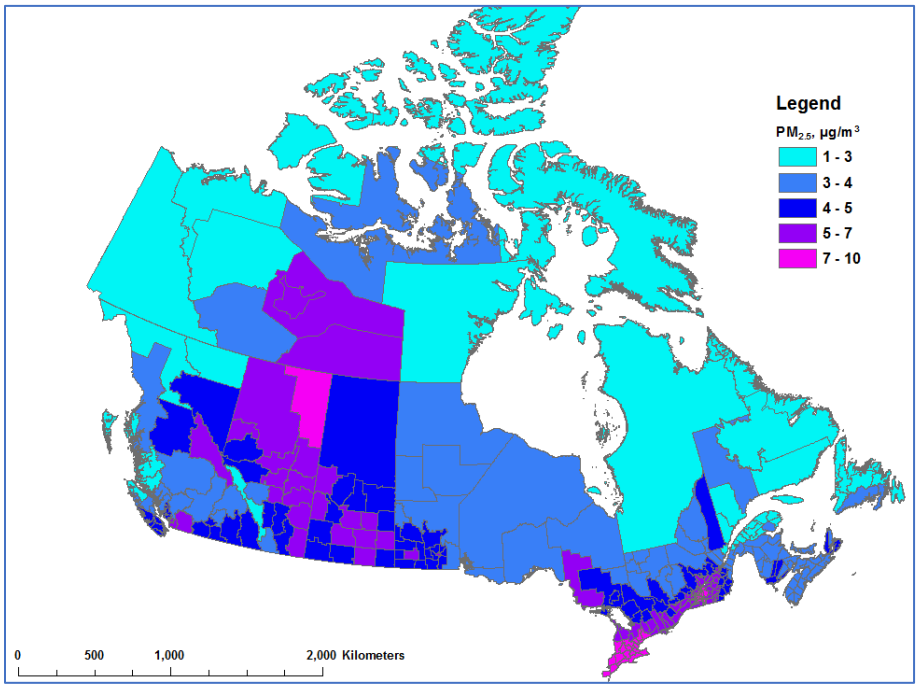
BC: British Columbia; ON: Ontario; QC: Quebec

Table D3. Premature deaths per 100,000 population – Census divisions with the lowest rates in 2016

Province - CD name (identifier)	Population	Deaths per 100,000
NU - Kitikmeot (CD6208)	6,728	1
NT - Region 1 (CD6101)	7,041	1
NU - Keewatin (CD6205)	10,883	2
NU - Baffin (CD6204)	19,771	2
NL - Division No. 11 (CD1011)	2,637	3
QC - Nord-du-Québec (CD2499)	45,162	3
NT - Region 6 (CD6106)	21,438	3
NL - Division No. 10 (CD1010)	24,608	4
BC - Skeena-Queen Charlotte (CD5947)	18,317	4
MB - Division No. 23 (CD4623)	9,599	4
BC - Central Coast (CD5945)	3,234	4
MB - Division No. 22 (CD4622)	44,528	4
QC - Minganie–Le Golfe-du-Saint-Laurent (CD2498)	11,607	6
BC - Mount Waddington (CD5943)	11,280	7
MB - Division No. 19 (CD4619)	18,569	8
BC - Northern Rockies (CD5959)	5,796	8
NT - Region 2 (CD6102)	2,455	10

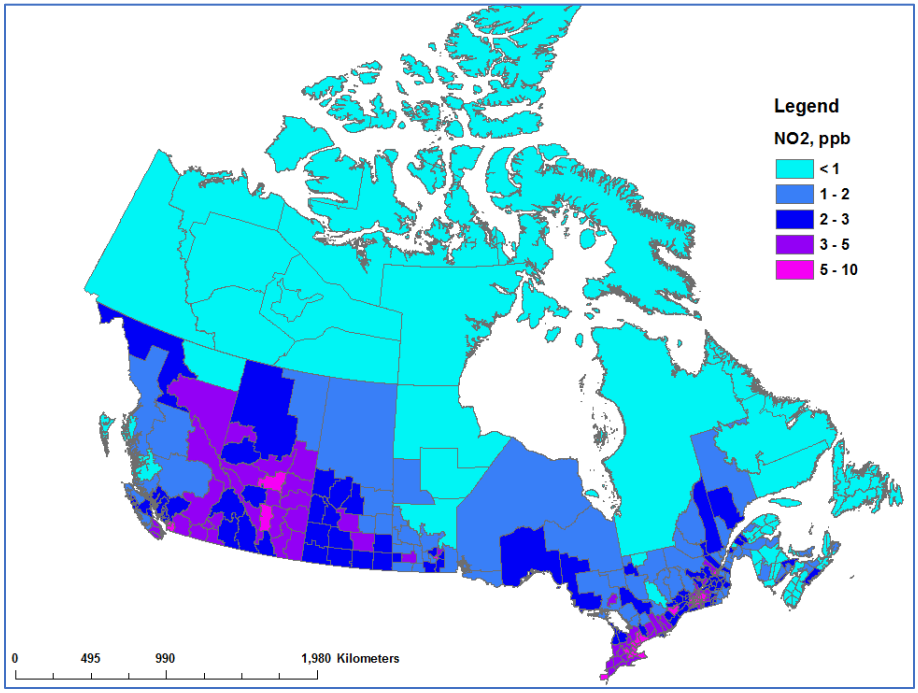
BC: British Columbia; MB: Manitoba; NL: Newfoundland and Labrador; NT: Northwest Territories; NU: Nunavut; ON: Ontario; QC: Quebec

Figure D1. Three-year average of daily $PM_{2.5}$ concentrations across Canadian census divisions – 2014–2016



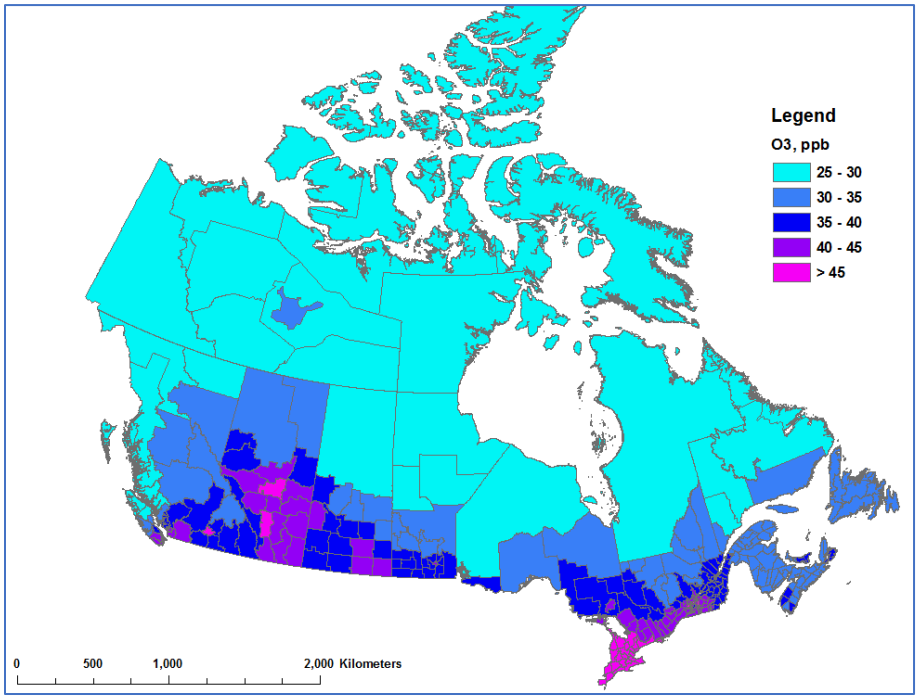
Source: Health impacts of air pollution in Canada: estimates of morbidity and premature mortality outcomes, 2019 report, Health Canada.

Figure D2. Three-year average of daily NO_2 concentrations across Canadian census divisions – 2014–2016



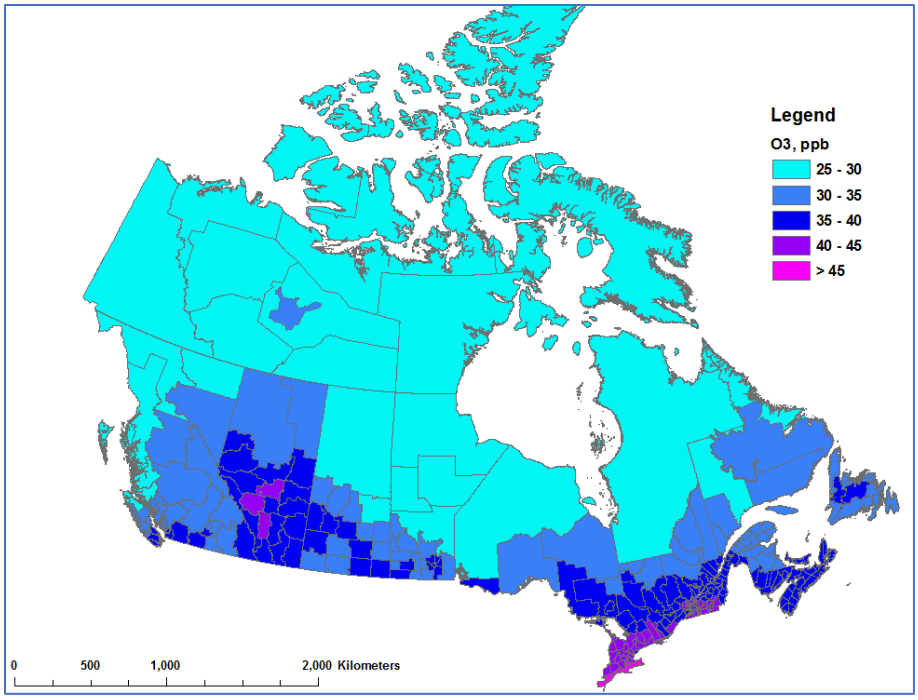
Source: Health impacts of air pollution in Canada: estimates of morbidity and premature mortality outcomes, 2019 report, Health Canada.

Figure D3. Three-year summer average of daily maximum ozone concentrations across Canadian census divisions – 2014 and 2015



Source: Health impacts of air pollution in Canada: estimates of morbidity and premature mortality outcomes, 2019 report, Health Canada.

Figure D4. Three-year annual average of daily maximum ozone concentrations across Canadian census divisions – 2014 and 2015



Source: Health impacts of air pollution in Canada: estimates of morbidity and premature mortality outcomes, 2019 report, Health Canada.

Health Canada Santé Canada

Canada