Health Canada Santé Canada

Health Impacts of Air Pollution in Canada

Estimates of morbidity and premature mortality outcomes

2019 Report

Canada

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Summary

A large body of scientific evidence has accumulated over the last 25 years attributing a wide range of significant adverse health effects to exposure to air pollution, ranging from respiratory symptoms to development of disease and premature mortality. Advances in understanding over this period in both the health and atmospheric science have provided the ability to quantitatively estimate the population health impacts of major air pollutants. In general, these estimates depend on an evaluation of population exposure (both short-term and long-term) to specific pollutants and evidence from scientific studies of the relationship between exposure and increased risk of adverse health effects. In Canada and internationally, health impact estimates identify air pollution as one of the most important risk factors for premature mortality and several non-fatal outcomes.

The approach for quantitatively estimating the population health impacts of air pollution is well recognized by health science organizations internationally. The approach relies on three main components: 1) estimates of the 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. The objective of this analysis is to update and enhance an earlier Health Canada publication (Health Canada 2017), based on recent data and scientific advancements. Compared with previous estimates, the current report updates the analysis of premature deaths associated with exposure to air pollution, provides analyses for morbidity outcomes, and gives an estimate of the economic valuation of the health impacts. In addition, results are presented at the provincial and territorial level using updated Canadian population data and geographic units based on the 2011 Census of Population. These modifications improve upon previous efforts by Health Canada to quantify air pollution health impacts.

Canadian population exposure to anthropogenic air pollution was estimated by comparing current ambient concentrations to background concentrations. Background concentrations represent ambient air pollution levels if all North American anthropogenic sources of emissions are removed. Air quality analyses show that the national average ambient concentrations (population-weighted) are 6.1 $\mu g/m^3$ for fine particulate matter ($PM_{2.5}$), 39.0 ppb for annual ozone (O_3), 42.8 ppb for summer O_3 (May-September), and 5.6 ppb for nitrogen dioxide (NO_2). The estimated average background concentrations for Canada are 1.8 $\mu g/m^3$ for $PM_{2.5}$, 0.15 parts per billion by volume (ppb) for NO_2, 26 ppb for annual O_3 and 28 ppb for summer O_3. These result in national average air pollution exposure values (population-weighted) of 4.3 $\mu g/m^3$ for $PM_{2.5}$, 5.4 ppb for NO_2, 13.0 ppb for annual O_3 and 14.8 ppb for summer O_3.

Health Canada estimates the number of annual mortalities in Canada that can be attributed to air pollution from human sources in North America to be 14,600 deaths per year based on 2015 population counts. This estimate is based on selected mortality endpoints that are considered the most appropriate in generating an estimate of air pollution-related premature mortality for Canada. The endpoints include all internal (i.e. non-accidental) causes of mortality associated with chronic exposure to fine particulate matter ($PM_{2.5}$) and acute exposure to nitrogen dioxide (NO_2) and annual ozone (O_3), as well as respiratory mortality associated with chronic exposure to warm season O_3.

Compared with the previous estimate published in 2017, the updated estimate represents an increase of 200 premature deaths or 1.4%. The increase in the number of premature deaths should be interpreted in light of the components of the approach cited above. The population-weighted average exposures to air pollution in the current analysis decreased compared to the 2017 report by 0.4 $\mu g/m^3$ for $PM_{2.5}$, 2.9 ppb for NO_2 and 0.8 ppb for summer O_3, whereas it increased by 0.2 ppb for annual O_3. The same risk factors as in the previous analysis were used and no change is expected from this component. In terms of demographics, the Canadian population in 2015 was greater by approximately 1.5 million people compared to 2011 (the reference year for the previous analysis). Thus, while Canadians are exposed to lower air pollution levels on average, the higher number of exposed individuals leads to an overall increase in mortality attributable to air pollution. An alternative measure of the air pollution health impacts is presented in terms of the number of mortalities per 100,000 people, thereby limiting the influence of changes in population. The current analysis for 2015 shows

that premature death associated with air pollution affected 41 per 100,000 Canadians, compared with 42 per 100,000 Canadians for the previous analysis based on 2011 populations.

In terms of annual morbidity outcomes, the number of asthma symptom days reaches 2.7 million, while the count of acute respiratory symptom days amounts to 35 million. The total economic cost of the all health impacts attributable to air pollution is $114B per year (2015 currency).

On a provincial and territorial basis, health impact estimates are generally proportional to population counts. For example, the premature mortality count is 6,700 in Ontario, 3,800 in Québec, 1,600 in British Columbia and 1,200 in Alberta.

The mortality and morbidity outcomes associated with air pollution in the current analysis were estimated based on ambient concentration projections for $PM_{2.5}$, NO_2 and O_3 exclusively. Other air contaminants also contribute to air pollution health impacts. Further, not all health endpoints that have been associated with $PM_{2.5}$, NO_2 and O_3 in the scientific literature can be currently quantified. Additional pollutants and health endpoints could not be evaluated owing to data limitations and knowledge gaps. Hence, the quantitative estimates of population health outcomes provided in this analysis are assumed to represent an underestimate of the full impacts of air pollution in Canada.

Overall, this analysis indicates that despite improvements in air quality and the relatively low levels of air contaminants in Canada compared to other regions of the world, air pollution continues to have impacts on population health in Canada.

Rationale

Air pollution is recognized globally as a major health risk. Exposure to ambient air pollution, for example, increases the risk of premature mortality from heart disease, stroke and lung cancer. Air pollution represents the largest environmental risk to health (WHO 2016). It is currently considered the fifth-leading mortality health risk in the world and was responsible 8.7% of deaths globally in 2017 (or 4.9 million premature mortalities worldwide) (IHME and HEI 2019). Similar estimates from the Global Burden of Disease (GBD) collaborators show that air pollution from ambient fine particulate matter (particulate matter with an aerodynamic diameter of less than 2.5 micrometres or $PM_{2.5}$) and ozone (O_3) was responsible for 4.5 million deaths from all non-accidental causes in 2015 (GBD 2015 Risk Factors Collaborators 2016), whereas estimates using the Global Exposure Mortality Model indicate that 8.9 million deaths were attributable to outdoor air exposure to $PM_{2.5}$ alone in 2015 (Burnett et al. 2018). The latter analysis by Burnett et al. (2018) represents a considerable increase compared to previous estimates owing to the inclusion of more causes of deaths than the ones considered for the GBD and Institute for Health Metrics and Evaluation (IHME) analyses (i.e. lung cancer, ischemic heart disease, chronic obstructive pulmonary disease, stroke, and lower respiratory infections), and a more advanced understanding of the relationship between exposure and mortality risk.

Estimates of air pollution attributable mortality have been developed globally and for many countries in the world (e.g. Cohen et al. 2017; IHME and HEI 2018; WHO 2016). Internationally, Canada is among the ten countries with the lowest national $PM_{2.5}$ exposure levels (IHME and HEI 2019). In Canada, estimates of air pollution attributable mortality have been developed by Health Canada in 2017 (Health Canada 2017), by Stieb and co-authors in 2015 (Stieb et al. 2015), and by the Canadian Medical Association in 2008 (CMA 2008). In general, all such estimates depend on an evaluation of population exposure (both short-term and long-term) to specific pollutants and evidence from scientific studies of the quantitative relationship between exposure and increased risk of mortality (referred to as concentration-response functions or CRFs).

This analysis is intended to provide a comprehensive estimate of morbidity and mortality outcomes in Canada related to ambient levels of $PM_{2.5}$, O_3 and nitrogen dioxide (NO_2). These pollutants were selected based on robust epidemiological evidence of adverse health impacts as well as our ability to accurately estimate their ambient levels and spatial distribution across Canada. This analysis is an update and an

enhancement of Health Canada's earlier efforts to quantitatively estimate the population health impacts of air pollution in Canada (e.g. Health Canada 2017).

Estimates of air pollution-attributable mortality and morbidity outcomes are expected to change over time owing to continuous improvements regarding data availability and methodology, both in terms of the understanding of the relationship of exposure and risk and with respect to the spatial representations of air pollution exposure, as well as changes in population health and demographics. For example, new scientific information enhances specific analyses of causes of death associated with exposure to air pollution. In addition, new air pollution data and modelling tools provide better and updated estimates of air pollution levels for all regions of Canada and thus a more precise understanding of population exposure. As a result, quantitative health impact estimates now apply to the entire Canadian population instead of being limited to the larger urban centres.

The objective of this analysis is to update the estimate of mortality outcomes attributable to air pollution in Canada based on the best available science and data. Estimates of morbidity outcomes associated with air pollution are also provided, with all estimates presented at both national and provincial and territorial levels. In addition, an economic valuation of health impacts is presented. The method described here is considered to be comprehensive and appropriate for the Canadian context.

Methods

Pollutants included in the estimate

This analysis of air pollution health impacts in Canada focuses on $PM_{2.5}$, NO_2, and O_3. These three pollutants have been most consistently associated with mortality in epidemiological studies and account for the majority of population health impacts from air pollution. Further, high resolution ambient concentration maps covering all of Canada are available for $PM_{2.5}$, NO_2 and O_3, providing exposure estimates for the whole population.

For each of these pollutants ($PM_{2.5}$, NO_2 and O_3), there is robust scientific evidence of health effects at very low concentrations and no evidence of an exposure threshold: that is, any incremental increase in air pollutant concentration is associated with an increased risk of adverse health outcomes. The current analysis estimates the mortality and morbidity outcomes that are associated with the incremental ambient air pollutant levels resulting from human source (anthropogenic) emissions in North America only. Health impacts associated with "background" pollutant concentrations (which include emissions from natural and non-North American sources) are not included.[1] This approach is taken given that anthropogenic emissions are generally targeted for the purposes of air quality management.

Calculating the background concentrations of air pollution

Background concentrations for $PM_{2.5}$, NO_2 and O_3 were estimated in collaboration with Environment and Climate Change Canada (Judek et al. 2004). This complex initiative involves a combination of qualitative (i.e. expert judgment) and quantitative (i.e. data-driven) approaches based on concentration measurements at rural and remote monitoring sites. This results in a set of *monthly-average* background concentrations for O_3, which has a strong seasonal cycle in its ambient concentrations, and *annual-average* background concentrations for NO_2 and $PM_{2.5}$. Regional differences likely exist in the background concentrations, but for the purposes of this analysis, a single background concentration is applied across Canada. Very low concentrations at rural and remote sites are used to establish the background concentrations based on one of the two following methods:

[1] 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 in the current report to represent all contributions other than from North American anthropogenic sources.

1. The data from rural and remote measurement sites are separated into sectors of different air mass origin and the background concentrations are selected as the monthly or annual average concentrations associated with the sectors containing no major anthropogenic sources; or
2. Many years of rural and remote measurement data are plotted in a time series that allows a qualitative selection of the lowest values that are, in turn, considered to be most representative of background air masses.

The estimated average background concentrations for Canada using the above methods are 1.8 micrograms per cubic metre (µg/m^3) for $PM_{2.5}$ (annual), 0.15 parts per billion by volume (ppb) for NO_2 (annual), and 26 ppb (annual) and 28 ppb (May-September) for O_3.

Calculating the contribution from anthropogenic sources to air pollution

Quantifying the health impacts of air pollution for Canada requires measurements of air pollution representative of all areas. Routine ground-level air pollution monitoring in Canada, however, only occurs at a number of discrete monitoring stations across the country. Therefore, spatially resolved estimates of ambient air pollution levels (including both anthropogenic and natural and non-North American contributions) are produced for $PM_{2.5}$, NO_2 and O_3 by combining a number of data sources as described below. Ambient concentrations are averaged over two to three years of available data to ensure that results are not influenced by inter-annual variations in concentrations.

- Estimates of median annual $PM_{2.5}$ concentrations for 2014–2016 were derived from remote sensing observations 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). These are then combined with information obtained from the Goddard Earth Observing System chemical transport model (GEOS-Chem) and the Canadian National Air Pollution Surveillance (NAPS) network (i.e. ground-based air pollutant monitoring) to provide final national estimates of $PM_{2.5}$ levels (van Donkelaar et al. 2015b). The median annual $PM_{2.5}$ concentration estimates are available on a grid with a spatial resolution of approximately 1 km × 1 km.
- O_3 estimates of both (1) the annual average and (2) the average of the daily 8-hour maximum in the warm season for 2014 and 2015 were derived from an interpolation technique that weighs and combines modelled O_3 forecasts with measurements (observations) of O_3 (Robichaud and Ménard 2014; Kalnay 2003). The technique is termed objective analysis. The modelled O_3 forecast was provided by the Global Environmental Multiscale - Modelling Air quality and CHemistry (GEM-MACH), the operational regional air quality forecast model (e.g. Makar et al. 2018; Moran et al. 2010; Whaley et al. 2018), while measurements came from the Canadian Air and Precipitation Monitoring Network (CAPMon) and the Canadian NAPS network. 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 O_3 concentrations over areas lacking monitoring data compared to standard interpolation techniques (such as spatial kriging). Estimates for Canada are available for 2014 and 2015, at a horizontal resolution of 10 km x 10 km.
- NO_2 concentrations were estimated using a national land use regression model developed using data from the NAPS network, the Aura satellite (NASA 2011), GEOS-Chem 2011, and geographic data (e.g. land use, population density, road network, point sources) (Hystad et al. 2011; Lamsal et al. 2008). Estimates were based on data for the period 2014–2016. The NO_2 estimates were developed on a fine scale (30 m) grid in order to more appropriately estimate concentrations for the highly variable nature of this pollutant within urban environments.

Air pollution concentration estimates were provided as a surface of grid cells, which was then mapped to the Canadian population (using the 2011 census, with population projections to 2015) by area weighting based

on 478,780 dissemination blocks. From the area-weighted values for each dissemination block, population-weighted air pollution concentrations were estimated for up to 293 census divisions (CDs) of Canada.[2] Ambient air pollutant maps for $PM_{2.5}$, annual O_3, summer O_3 and NO_2 are shown in figures 1 to 4.

Figure 1. Annual average $PM_{2.5}$ concentrations across Canadian census divisions for the period 2014–2016

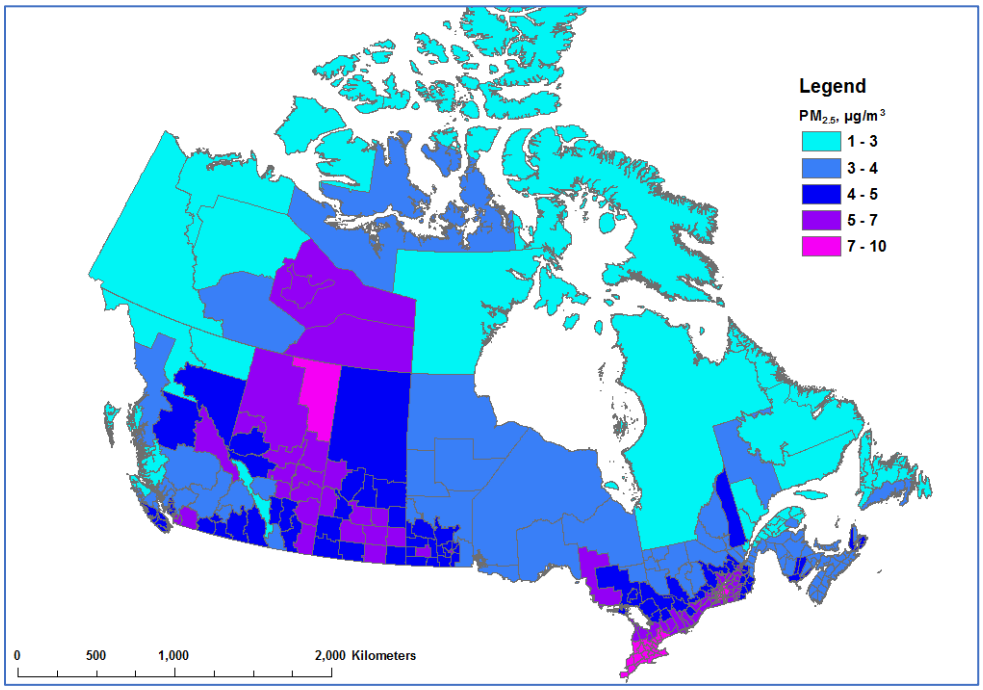
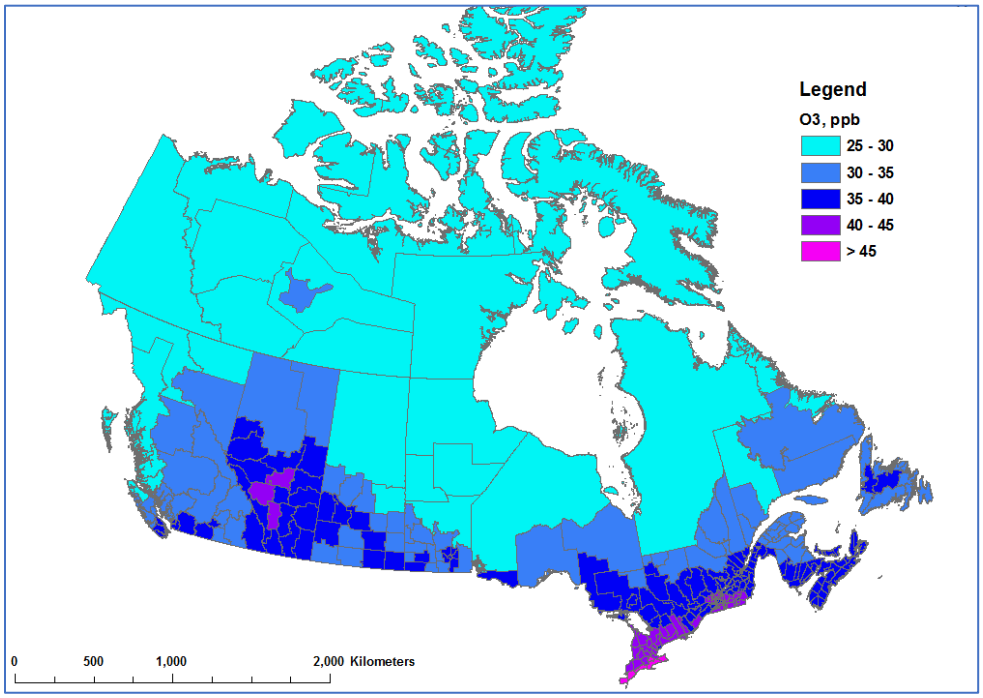


Figure 2. Annual daily maximum average O_3 concentrations across Canadian census divisions for the period 2014–2015



[2] Missing data are associated with a few census divisions in remote and low population northern areas.

Figure 3. Summer daily maximum average O_3 concentrations across Canadian census divisions for the period 2014–2015

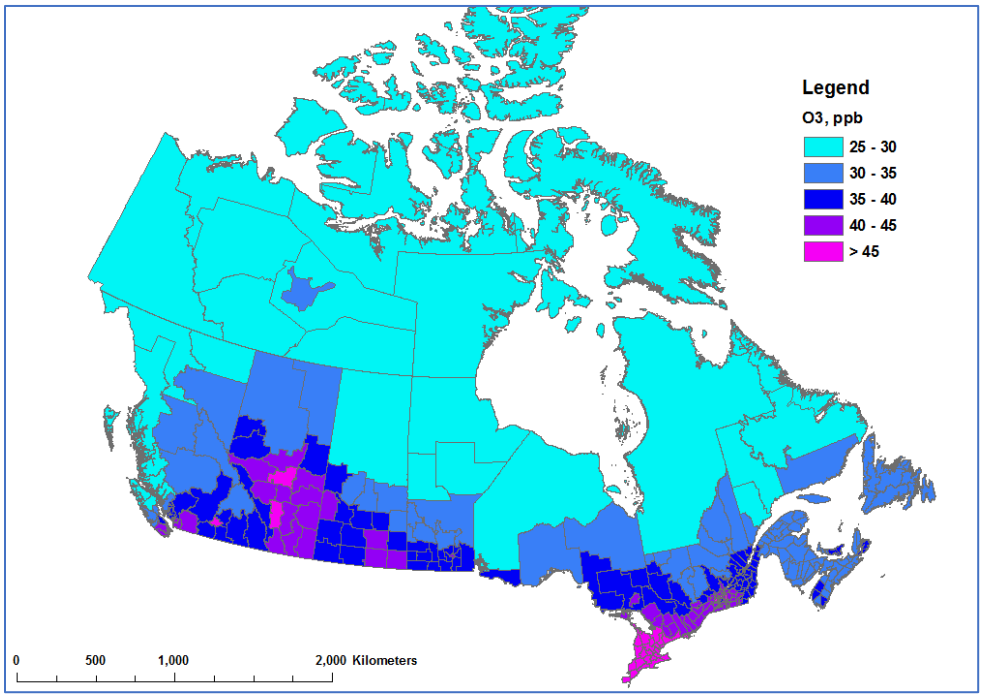
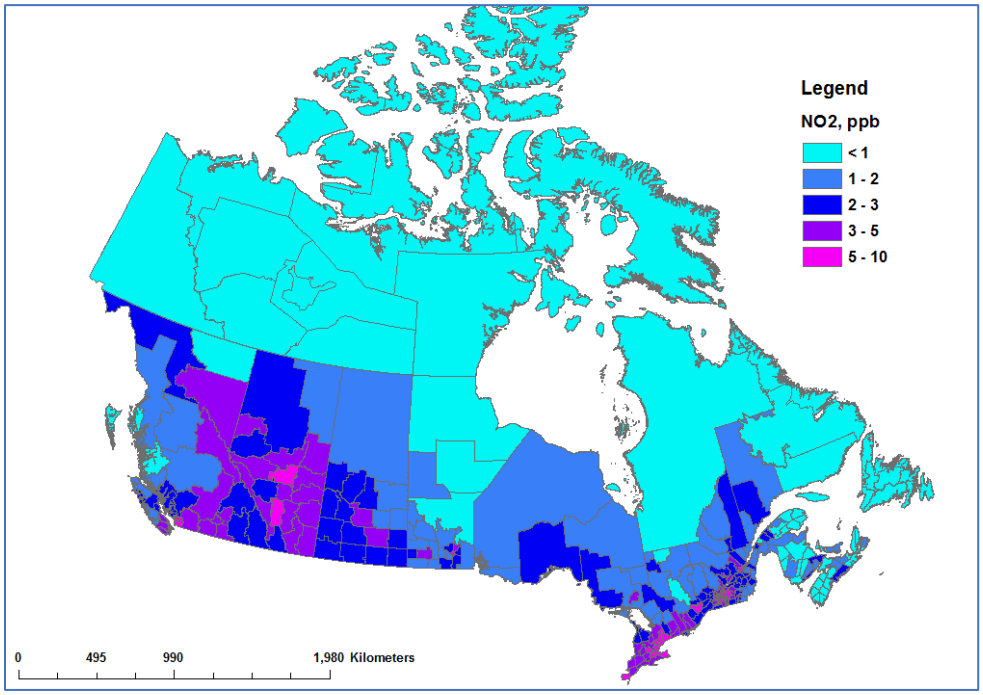


Figure 4. Annual average NO_2 concentrations across Canadian census divisions for the period 2014–2016



The maps represent the estimated ambient air concentrations in 2015 and reflect the contribution from all natural and anthropogenic sources. As expected, higher $PM_{2.5}$, O_3, and NO_2 concentrations are observed in many of the higher population CDs. Figure 1 also indicates that areas of western Canada have higher $PM_{2.5}$ concentrations, probably due to the influence of industrial and resource development and possibly from wildfires. For O_3, higher concentrations are observed in summer (Figure 3) as environmental and meteorological conditions in warmer months promote the formation of O_3. The national population-weighted

average ambient concentrations are 6.1 µg/m^3 for $PM_{2.5}$, 39.0 ppb for annual O_3 , 42.8 ppb for summer O_3 (May-September), and 5.6 ppb for NO_2.

Calculating the morbidity and mortality outcomes due to air pollution

The health outcomes attributable to air pollution were estimated for ambient concentrations of $PM_{2.5}$, O_3 and NO_2 above background concentrations. The analysis was conducted using Health Canada's Air Quality Benefits Assessment Tool (AQBAT) (Judek et al. 2012), version 3.0.[3] The AQBAT model produces an estimate of the number of premature deaths and other health outcomes in Canada associated with a specified change in air pollution concentration. Health effect information for the three air pollutants is included in the form of concentration response functions (CRFs). A CRF represents the percentage of excess health risk for a given endpoint (e.g. asthma symptoms, chronic bronchitis, and acute exposure mortality) associated with a unit increase in ambient pollutant concentration. For example, the risk of premature mortality from all internal causes increases by 10% for an increase in $PM_{2.5}$ exposure of 10 µg/m^3. A CRF is a statistically derived estimate, from a single study or a meta-analysis of multiple studies.

The health endpoints (related to acute or chronic exposure), the associated CRFs and the applicable population group(s) (e.g. age-specific groups) are pre-defined within the AQBAT and represent Health Canada-endorsed values drawn from the health science literature. The pollutants and their associated health effects considered in this analysis are provided in Table 1. Background information regarding 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 the AQBAT have been published previously (Crouse et al. 2012; Judek et al. 2012; Shin et al. 2013; Stieb et al. 2015). This information is also summarized in Appendix A. Health outcomes were considered to have no threshold for effect (i.e. effects were assumed to occur at all levels of exposure).

CRFs were calculated for all internal cause mortality for $PM_{2.5}$ from a Canadian cohort (Crouse et al. 2012), for NO_2 and annual O_3 from Canadian data (Burnett et al. 2004), and summer O_3 from an American cohort (Jerrett et al. 2009).[4] CRF values for the three pollutants are reported in Table 3. 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 mean of the output distribution, as well as low- and high-end estimates equal to the 2.5th and 97.5th percentiles of the output distribution.

Baseline health endpoint incidence rates are a key factor in estimating the count of health outcomes for a pollutant concentration change. They are estimated from detection, observation and reporting through formal means (e.g. death certificates, hospital admission records) based on data provided by Statistics Canada or from 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). 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 (of the health endpoints in question) for the target population were included to estimate the number of excess health outcomes associated with the increased risk due to a change in air pollutant concentration. Annual baseline health endpoint rates of events are assigned to specified populations. For example, the Restricted Activity Days endpoint is assigned to 94% of people 20 years and

[3] Guoliang Xu and Dave Stieb, personal communications, Health Canada, 2019

[4] CRF values for $PM_{2.5}$ mortality from ischemic heart disease (IHD), cerebrovascular disease (CVD), lung cancer (LC) and chronic obstructive pulmonary disease (COPD), for adults 25 years and over, are also included in AQBAT (Shin et al. 2013). This mirrors the approach employed in the Global Burden of Disease analyses (Cohen et al. 2017; Lim et al. 2010; www.healthdata.org/gbd), led by the Institute for Health Metrics and Evaluation, and the World Health Organisation, which include estimates on air quality and health impacts across the world. IHD, CVD, LC and COPD are a subset of all internal cause (non-accidental) chronic exposure mortality. Chronic exposure mortality (i.e. all internal cause) estimates are reported herein. They are considered the main premature mortality metric associated with chronic exposure to $PM_{2.5}$ for the current report. This endpoint generally leads to higher mortality estimates than the sum of specific causes of death.

older (i.e. non-asthmatics). Exposure to pollutants typically has a minor influence on the baseline incidence rates.

Table 1. Averaging periods and associated health endpoints considered for CRFs associated with NO_2, O_3 and $PM_{2.5}$ in the AQBAT

Pollutant[a]	Averaging period	Health endpoint
NO_2	24 h	Acute exposure mortality[b,c]
O_3	1 h maximum	Acute exposure mortality[b]
O_3 summer (May–September only)	1 h maximum	Acute respiratory symptom days
		Asthma symptom days
		Chronic exposure respiratory mortality
		Minor restricted activity days
		Respiratory emergency room visits
		Respiratory hospital admissions
$PM_{2.5}$	24 h	Acute respiratory symptom days
		Adult chronic bronchitis cases
		Chronic exposure mortality
		Asthma symptom days
		Cardiac emergency room visits
		Cardiac hospital admissions
		Child acute bronchitis episodes
		Respiratory emergency room visits
		Respiratory hospital admissions
		Restricted activity days

AQBAT: Air Quality Benefits Assessment Tool; CRF: concentration–response function; NO_2: nitrogen dioxide; O_3: 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] The CRF between acute exposure mortality and gaseous pollutants is from a model including CO, NO_2, O_3 and SO_2 and may not precisely reflect the true attribution of risk to individual pollutants.

[c] It is recognized that the CRF for acute exposure mortality associated with NO_2 exposure may reflect a 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.

Baseline cause- and age-specific mortality rates were derived from counts of mortality obtained for each census division (CD) with the exception of Québec where these data were not available. Québec mortality counts for each CD were derived by applying national age- and cause-specific rates to the population age distribution of individual CDs. Rates are averaged over the three most recent years of available data to improve stability (Stieb et al. 2015). For each morbidity and mortality health endpoint in the AQBAT, a data file contains estimated annual events per million specified population, for every geographic area, age group, scenario year and population projection. Additional details and references on the derivation of baseline rates have been published previously (Judek et al. 2012; Stieb et al. 2015).

In the context of this analysis, CRFs pertaining to acute exposure were derived from studies examining effects of air pollutants in the days before health outcomes, while CRFs pertaining to chronic exposure were derived from studies of air pollutants averaged over the years prior to health outcomes.

Calculating the economic valuation of health outcomes due to air pollution

Economic valuation estimates consider the potential social, economic and public welfare consequences of the health outcomes, including medical costs, reduced workplace productivity, pain and suffering, and the effects of increased mortality risk. Expressing impacts using monetary valuation provides a common metric across health endpoints to estimate an overall monetary valuation of benefits or damages. The sum provides an indication of the relative social benefits or damages resulting from reduced or increased *risks* to health.

Each health endpoint is assigned a valuation estimate, typically derived from survey, accounting, economic or actuarial data. The endpoint valuation has an uncertainty reflected by a distribution of possible values with corresponding parameters (i.e. economic valuation estimates are entered as a distribution in the AQBAT). The valuation estimates used in the model and references to the studies from which they are derived are provided in Table 2. Endpoint valuation estimates are expressed in Canadian dollars. Values were adjusted based on the Consumer Price Index as defined by Statistics Canada for estimating costs in 2015 currency (Judek et al. 2012; Statistics Canada, annual).

Table 2. Economic valuation of health endpoints used in Health Canada's AQBAT model

Endpoint [reference]	Currency year	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%)
Chronic exposure lung cancer 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 & 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. (2012)

COPD: chronic obstructive pulmonary disease; prob.: probability of value being selected in 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 based on the proportion of emergency room visits that result in admission to hospital. Hospital admissions are assigned a value of zero. This avoids double counting of costs.

The valuation estimate for mortality endpoints is considerably higher than other health endpoint valuations. The recommended central estimate of an avoided premature death for policy analysis is $6.5 million (in 2007 currency), based on a review of Canadian studies by Chestnut and De Civita (2009). It is based on analyses indicating 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 $65 over 100,000 Canadians (for which one death is avoided) equals the value of one avoided death. The recommended low value is $3.5 million and the recommended high value is $9.5 million. These values represent a reasonable range for a primary analysis and they should not be considered as lower and upper bounds (Chestnut and De Civita 2009). The values above 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.[5] Following adjustments based on the Consumer Price Index, the value of an avoided premature death in 2015 is $7.4 million.

Results

Reducing ambient $PM_{2.5}$ concentrations in Canada from current to background levels would result in a 3.9% reduction in all internal causes of mortality from chronic exposure to $PM_{2.5}$, representing 9,700 deaths per year.

It is estimated that 0.4% of all internal causes of death in Canada can be attributed to acute exposure of the Canadian population to above-background concentrations of NO_2 (i.e. mortality that occurs a few days after an elevation in ambient NO_2). This represents 940 mortalities a year.

Acute exposure to above-background annual O_3 is associated with 1.1% of all internal cause mortality (2,700 deaths per year), whereas chronic exposure to above-background summer O_3 is linked with 5.4% of respiratory-related mortalities, equivalent to 1,300 respiratory mortalities per year.

Overall, the total mortality attributable to anthropogenic air pollution in Canada is estimated to be 14,600 deaths per year, based on population estimates for 2015 and air pollutant concentrations from 2014 to 2016.[6] Specifically, this represents the estimated population health impacts of $PM_{2.5}$, O_3 and NO_2. The results are summarized in Table 3 on a count basis for each air pollutant, as well as on a per 100,000 population basis. The latter metric provides a population-based normalized value allowing, notably, appropriate comparisons of health impact estimates among geographic or political entities of different population size. The Canadian values presented herein have not been published previously. The provincial and territorial results, for each pollutant evaluated, are shown in Table 4.

Table 3. Estimate of premature mortality and morbidity outcomes attributable to above-background levels of air pollution: results, analysis parameters, exposure, and sources

Pollutant	**$PM_{2.5}$**	**NO_2**	**O_3[a]**
Mortality outcomes			
Number of mortalities (% of baseline mortality by cause of death)	**9,700** (3.9%) – chronic exposure	**940** (0.4%) – acute exposure	**2,700** (1.1%) – acute exposure **1,300** (5.4%) – chronic exposure
Number of mortalities per 100,000 Canadians	27	2.6	7.4 – acute exposure 3.6 – chronic exposure
Causes of death	All [b]	All [b]	All [b] – acute Respiratory – chronic
CRF – % increase per Δx [Source of CRF]	10% per 10 µg/m^3 [Crouse et al. 2012]	1.5% per 20 ppb [Burnett et al. 2004[c]]	Acute: 1.7% per 20 ppb [Burnett et al. 2004[c]] Chronic: 8.2% per 20 ppb [Jerrett et al. 2009]

[5] Empirical studies of willingness to pay (WTP) for mortality risk reductions estimate average monetary amounts that individuals are willing to pay for small reductions in premature mortality. The valuation context or an individual's circumstances influence his 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 to reduce their own risk of death. Therefore, it can exceed the value of the financial impact to an individual associated with the change in risk.

[6] $PM_{2.5}$: 2014–2016; O_3: 2014–2015; NO_2: 2014–2016

Pollutant	$PM_{2.5}$	NO_2	O_3 [a]
Morbidity outcomes			
Morbidity effects estimated	Acute Respiratory Symptom Days; Adult Chronic Bronchitis Cases; Asthma Symptom Days; Cardiac Emergency Room Visits; Cardiac Hospital Admissions; Child Acute Bronchitis Episodes; Respiratory Emergency Room Visits; Respiratory Hospital Admissions; Restricted Activity Days	None	Chronic: Acute Respiratory Symptom Days; Asthma Symptom Days; Minor Restricted Activity Days; Respiratory Emergency Room Visits; Respiratory Hospital Admissions
Exposure estimates			
Years of exposure data	2014–2016	2014–2016	2014–2015
Source of exposure data	Satellite observations, observations from NAPS monitors and chemical transport models; van Donkelaar et al. 2015a,b	Model incorporating observations from NAPS monitors, satellite observations, land use patterns and distance from highways and major roads; Hystad et al. 2011	Objective analysis: combination of values from the GEM-MACH air quality forecast model and observations from NAPS monitors; Robichaud and Ménard 2014
National population weighted average ambient concentration	**6.1 µg/m^3**	**5.6 ppb**	**39.0 ppb (annual)** **42.8 ppb (May-Sept)**
Estimated natural background concentration	**1.8 µg/m^3**	**0.15 ppb**	**26 ppb (annual)** **28 ppb (May-Sept)**
National population weighted average exposure to anthropogenic air pollution	**4.3 µg/m^3**	**5.4 ppb**	**13.0 ppb (annual)** **14.8 ppb (May-Sept)**
Economic valuation – All health outcomes			
Canadian currency, 2015 ($1,000,000)	**77,000**	**7,000**	**19,700 (annual)** **9,800 (May-Sept)**

CRF: concentration-response function; GEM-MACH: Global Environmental Multiscale - Modelling Air quality and CHemistry; µg/m^3: micrograms per cubic metre; NAPS: National Air Pollutant Surveillance network; ppb: parts per billion by volume

[a] O_3 acute exposure associated with annual O_3; O_3 chronic exposure associated with summer O_3 (May-September)

[b] All – excluding external causes, e.g. accidents, suicide

[c] Supplementary analysis model including CO, NO_2, O_3, and SO_2

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

Region—population	Premature mortality counts[a]					Valuation ($1,000,000)[a,d]
	per pollutant				per 100,000 population	
	NO_2	$PM_{2.5}$	O_3[b]	All[c]	All[c]	All[c]
Canada—35,851,774	940	9,700	4,000	14,600	41	108,000
Alberta—4,196,457	90	740	400	1,200	29	9,100
British Columbia—4,683,139	140	980	440	1,600	33	11,500
Manitoba—1,293,378	30	260	110	400	31	3,000
New Brunswick—753,871	6	110	64	180	24	1,400
Newfoundland and Labrador—527,756	1	36	41	79	15	580
Northwest Territories—44,088	–	5	0	6	13	41
Nova Scotia—943,002	8	160	93	260	27	1,900
Nunavut—36,919	–	0	0	0	1	4
Ontario—13,792,052	400	4,500	1,800	6,700	49	49,700
Prince Edward Island—146,447	1	19	17	37	25	270
Québec—8,263,600	260	2,600	910	3,800	46	28,000
Saskatchewan—1,133,637	16	270	87	380	33	2,800
Yukon—37,428	–	0	1	1	2	5

[a] Values represent mean 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 for values of 10,000 or more.

[b] Acute and chronic exposure premature mortalities combined.

[c] NO_2, O_3, and $PM_{2.5}$; totals may not match because of rounding.

[d] Endpoint valuation estimates expressed in Canadian dollars and based on 2015 currency.

Large variations are observed across geographic regions. Higher mortality outcomes are estimated in the provinces of Ontario, Québec, British Columbia and Alberta, which correspond with the most populated provinces and the regions with the highest projected air pollution levels (see figures 1 to 4). The deaths per 100,000 values highlight that air pollution mortality risks are highest in Ontario and Québec, reflecting the confluence of high population density and air pollution levels, and lowest in Nunavut and the Yukon. There are also regional variations associated with the influence of population densities, meteorological conditions and geographic features. For example, while the provincial populations for British Columbia and Alberta are comparable, the elevated population density and vehicle activity in Vancouver likely contribute to distinctly higher impacts associated with NO_2 and $PM_{2.5}$ air pollution in British Columbia compared to Alberta, which has two smaller and less densely populated urban centres.

Anthropogenic air pollution is also associated with a considerable number of morbidity outcomes. National count estimates by health endpoint are provided in Table 5. The highest counts are predicted for acute respiratory symptoms days, restricted activity days and asthma symptom days.

The total annual economic value of health outcomes associated with air pollution is approximately $114 billion. This amount primarily reflects premature mortalities valued at $108 billion. Although the monetary value associated with morbidity endpoints is low ($5.5B) compared with that associated with mortalities, the morbidity impacts represent a health burden for the Canadian population.

Because people are exposed concurrently to multiple air pollutants in ambient air, rather than to individual pollutants in isolation, it can be difficult in epidemiological studies to separate the true independent effects of individual pollutants. Where possible, the CRFs employed in this analysis were derived from statistical models that included the other pollutants, providing a measure of adjustment for possible overlapping effects among pollutants. However, it is still possible that there is double counting of effects among pollutants, or that effects attributed to one pollutant are not fully disentangled from those attributed to other pollutants.

Table 5. National count estimates by health endpoint

Health endpoint	Pollutant	Count[a]	Valuation ($1,000,000) [a,d]
Mortality[a]			
Acute exposure mortality	NO_2	940	7,000
	O_3	2,700	19,700
Chronic exposure respiratory mortality	O_3 summer[c]	1,300	9,500
Chronic exposure mortality	$PM_{2.5}$	9,700	72,000
Total mortality[b]	All pollutants	14,600	108,000
Morbidity[a]			
Acute respiratory symptom days	O_3 summer, $PM_{2.5}$	35,000,000	360
Adult chronic bronchitis cases	$PM_{2.5}$	9,100	3,900
Asthma symptom days	O_3 summer, $PM_{2.5}$	2,690,000	190
Cardiac emergency room visits	$PM_{2.5}$	1,000	6
Cardiac hospital admissions	$PM_{2.5}$	790	-[e]
Child acute bronchitis episodes	$PM_{2.5}$	42,500	19
Minor restricted activity days	O_3 summer	2,340,000	72
Respiratory emergency room visits	O_3 summer, $PM_{2.5}$	7,000	20
Respiratory hospital admissions	O_3 summer, $PM_{2.5}$	1,400	-[e]
Restricted activity days	$PM_{2.5}$	13,000,000	870
Total morbidity[b]	All pollutants	n.a.	5,500

n.a.: not applicable; NO_2: nitrogen dioxide; O_3: ozone; $PM_{2.5}$: fine particulate matter or particulate matter with an aerodynamic diameter of 2.5 µm or less

[a] Values represent mean 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 for values of 10,000 or more.

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

[c] May–September only.

[d] Endpoint valuation estimates expressed in Canadian dollars and based on 2015 currency.

[e] No economic valuation associated with hospital admissions. It is assumed that air pollution-related hospital admissions involve an initial emergency room visit of the same type (cardiac or respiratory) that includes a cost.

Discussion

This estimate of 14,600 deaths per year represents Health Canada's current estimate of the number of annual mortalities in Canada that can be attributed to air pollution from human sources in North America. This represents a slight increase over the previous estimate of 14,400 produced in 2017 (Health Canada 2017). Table 6 compares the current estimates with the 2017 report values.

The increase in the number of premature deaths should be interpreted in light 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. The population-weighted average exposures to air pollution (above background) in the current analysis decreased compared to the 2017 report by 0.4 µg/m^3 for $PM_{2.5}$, 2.9 ppb for NO_2 and 0.8 ppb for summer O_3, whereas it increased by 0.2 ppb for annual O_3. The population-weighted exposure values for the 2014–2016 period are generally lower for all pollutants, but especially so for NO_2. The air pollution data used in the current analysis spanned from 2014 to 2016 for NO_2 and $PM_{2.5}$, and from 2014 to 2015 for O_3. By contrast, data used in the previous analysis represented 2009–2011 for NO_2, 2007–2009 for O_3 and 2010–2012 for $PM_{2.5}$. Although the national average estimates are comparable, small regional discrepancies can influence the results. Air quality monitoring data in Canada, as presented for the Canadian Environmental Sustainability Indicators, suggest

comparable trends overtime.[7] However, the air pollution exposure maps developed for the current analysis are not based solely on monitoring data. As such, discrepancies between monitoring data and exposure maps using a combination of approaches and data sources should not be interpreted as diverging; rather, they reflect conditions across different geographical spaces.

Table 6. Comparison of the 2017 and 2019 reports – population-weighted exposure to anthropogenic air pollution (above background) and premature mortality estimates

Pollutant	Background levels	2017 Report Exposure surface: 2007–2012 Population (2011): 34,342,780		2019 Report Exposure surface: 2014–2016 Population (2015): 35,851,774	
		Population-weighted exposure	Mortality count	Population-weighted exposure	Mortality count
NO_2	0.15 ppb	8.3 ppb	1,300	5.4 ppb	940
O_3 annual	26 ppb	12.8 ppb	2,400	13.0 ppb	2,700
O_3 summer	28 ppb	15.6 ppb	1,200	14.8 ppb	1,300
$PM_{2.5}$	1.8 µg/m^3	4.7 µg/m^3	9,500	4.3 µg/m^3	9,700
Total			**14,400**		**14,600**
per 100,000 Canadians			**42**		**41**

The risk estimates for the different health effects, represented herein by CRFs, were not modified since the 2017 report and they are not considered a source of variation. However, the baseline rates for mortality and morbidity outcomes were recently updated for AQBAT version 3.0 and these can influence the results.

In terms of demographics, the current estimates used the 2011 population census instead of the 2006 census, with projections to the appropriate years. The Canadian population in 2015 was greater by approximately 1.5 million people compared to 2011 (the reference year for the previous analysis). The age profile also evolved, indicating a slightly older and potentially more vulnerable population. Higher population counts translate into a higher number of potentially exposed individuals, and as a result, higher health outcome estimates can be expected for the same air pollutant concentrations. Although population weighted air pollution exposure has decreased, population health impacts reflect changes in population demographics and health status (e.g. baseline incidence rates). An alternative measure of the air pollution health impacts is to express the number of health outcomes per 100,000 people, thereby limiting the influence of changing populations. The results normalized by population show that premature death associated with air pollution affected 41 per 100,000 Canadians in 2015, compared with 42 per 100,000 Canadians in 2011.

The provincial results indicate that the highest health impacts associated with air pollution are occurring in Ontario and Québec, both on a mortality count basis and based on premature deaths per 100,000 individuals. This is not unexpected as approximately 63% of the Canadian population resides in these two provinces. Further, some of the highest air pollution levels in Canada are projected in the southern parts of Ontario and Québec that include the highly populated and industrialized region of the Windsor-Québec corridor.

In this analysis, an estimated North American background concentration was applied as the reference for the calculation. This approach was taken given that anthropogenic emissions are generally targeted for the purposes of air quality management. Although air pollution levels are low in Canada compared with those in other developed nations,[8] recent Canadian studies show air pollution-attributable mortality even at quite low ambient concentrations (Crouse et al. 2015; Pinault et. al. 2017).

This updated analysis uses the same CRFs as in the 2017 report since for most endpoints, newer information did not indicate risk associations had markedly changed. However, much effort is made to characterize the relationship of air pollution to mortality and recently, an enhanced CRF for all cause non-accidental mortality associated with chronic exposure to $PM_{2.5}$ has been developed (Pinault et al. 2017). This new CRF is based on

[7] Government of Canada. Air quality. www.canada.ca/en/environment-climate-change/services/environmental-indicators/air-quality.html

[8] www.who.int/phe/health_topics/outdoorair/databases/cities/en/

the Canadian Census Health and Environment Cohort (CanCHEC) as is the AQBAT CRF from Crouse et al. (2015). However, it was derived using a newer statistical method, the Shape Constrained Health Impact Function (SCHIF). This method fits several different shapes of association, and describes the relative risk between $PM_{2.5}$ concentrations and mortality by a set of functions. The SCHIF method was recently employed to derive the Global Exposure Mortality Model (GEMM) that includes 41 international cohorts 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 (i.e. more than double) in global mortality attributable to outdoor $PM_{2.5}$ air pollution compared to previous estimates.

While the current version of AQBAT does not include a CRF derived using the SCHIF method, new health data and analyses may support its inclusion in the future. To assess the potential influence of a revised CRF on estimates of health impacts attributable to air pollution in Canada, the new non-linear CRF for $PM_{2.5}$ from Pinault et al. (2017) was employed in the current evaluation as a sensitivity analysis. It results in an additional 1200 premature mortalities from chronic exposure to ambient $PM_{2.5}$. The health impact estimates for NO_2 and O_3 are unchanged. Overall, premature mortalities attributable to air pollution in Canada would total 15,800 using the SCHIF approach.

Other attempts have been made to estimate the impact of air pollution on Canadians, most notably on the basis of the approaches developed under the Institute for Health Metrics and Evaluation's (IHME) Global Burden of Disease (GBD) project. The GBD-based analysis produced lower estimates of premature mortality for Canada (7,630), though it is based on similar principles to those used here. The differences lie in some of the details of the approaches taken including the basic CRF for outcomes, which in the current analysis used the Canadian-specific study of Crouse et al. (2015), while the GBD analysis used an amalgam of several international studies and somewhat different approach to classification of mortality effects, both of which influence the calculation of impacts. Additionally, while both approaches incorporate mortality effects for $PM_{2.5}$ and O_3, the GBD approach does not include NO_2. In Canadian analyses of the health effects of air pollution, NO_2 is a significant participant in the mortality estimates for air pollution. As well, the assignment of exposures can have important impacts on estimate calculations. The GBD analysis was based on global considerations and approaches. By contrast, for this analysis, we have developed detailed and Canadian-specific approaches to the estimation of concentration and population exposure. Finally, the analysis provided here utilizes Canadian analyses of background concentrations that results in some additional differences versus other approaches.

Mortality estimates also depend on detailed demographic data, such as population count and distribution, as well as baseline rates of mortality in Canada. The data and method used in the current analysis are considered to be more comprehensive, recent and appropriate for the Canadian context than previous estimates as they integrate new science and knowledge on the health effects of air pollution in Canada.

In this analysis, three different methods were used to calculate exposure of the Canadian population to each of the three pollutants: Satellite observations and ground observations coupled to a chemical transport model for $PM_{2.5}$; objective analysis (OA) that considers ground observations coupled to a chemical transport model for O_3; and land use regression (LUR) modelling informed by various sources of data for NO_2. The data periods are not exactly the same; while data from 2014 to 2016 informed the NO_2 and $PM_{2.5}$ concentrations, O_3 was based on data from 2014 and 2015 because the 2016 data was not available at this time. These three estimation methods currently provide the best available national exposure estimates for each pollutant. Health Canada and Environment and Climate Change Canada continue to collaborate on the evaluation of approaches to developing more integrated exposure estimates that can be updated annually or biannually.

It should be noted that variations in health impact estimates are to be expected in future updates by Health Canada. If annual trends are generated from the analyses, the health impact estimates will be recalculated for all years included in the analysis to ensure that trends are internally consistent. Variations or discrepancies between estimates may occur owing to, for example: changes in data or methods to assess population exposure to air pollutants; changes in exposure–response or concentration-response relationships; changes in the baseline rates of adverse outcomes in Canada; or changes in population

demographics. Mortality estimates can be especially influenced by values used for exposure–response relationships, which are regularly updated as new data are produced, become available, and are integrated in the AQBAT.

The mortality and morbidity outcomes associated with air pollution in the current analysis were estimated based on ambient concentration projections for $PM_{2.5}$, NO_2 and O_3 exclusively. Other air pollutants also contribute to air pollution health impacts. Further, not all health endpoints that have been associated with $PM_{2.5}$, NO_2 and O_3 in the scientific literature can be currently quantified. Additional pollutants and health endpoints could not be evaluated owing to data limitations and knowledge gaps. Hence, the quantitative estimates of population health outcomes provided in this analysis are assumed to represent an underestimate of the full impacts of air pollution in Canada.

Conclusions

Air pollution is recognized globally as a leading risk factor for premature mortality, based on the weight of evidence available in a robust database of international epidemiological studies and supporting evidence from toxicological investigations. As a result of extensive research and assessment, including comprehensive reviews by Health Canada, $PM_{2.5}$, NO_2, and O_3 have been found to exert the largest population health impacts.

The current analysis estimates the Canadian mortality, morbidity and economic costs associated with the incremental ambient air pollutant levels resulting from human source emissions – the air pollution that is targeted by air quality management measures. Health Canada estimates that 14,600 premature deaths per year in Canada can be attributed to human sources of $PM_{2.5}$, NO_2, and O_3. When combined with non-fatal health outcomes, including 35 million acute respiratory symptoms days, 2.69 million asthma symptom days and 8,000 emergency room visits, the total economic valuation of the air pollution health impacts reach $114 billion per year.

This estimate of 14,600 deaths per year indicates a slight increase over the previous estimate of 14,400 produced in 2017 (Health Canada 2017). Whereas the population-weighted average exposures to air pollution in the current analysis decreased compared to the 2017 report, the Canadian demographic and health profile has changed. For example, the population has increased by 1.5 million people. Consequently, although Canadians are exposed to lower air pollution levels on average, the higher number of exposed individuals leads to an overall increase in mortality attributable to air pollution. An alternative measure of the air pollution health impacts expressing the number of health outcomes per 100,000 people shows that premature death associated to air pollution affected 41 per 100,000 Canadians in 2015, compared with 42 per 100,000 Canadians in 2011.

The data and methods used in the current analysis (e.g. background concentrations, CRFs) integrate the best available science, data and knowledge on the health effects of air pollution in Canada compared with previous Canadian estimates. In addition, this is the most comprehensive analysis available. Nonetheless, evidence exists suggesting that air pollution may be associated with additional adverse health outcomes that were not considered in the current analysis. Further, air pollutants other than NO_2, $PM_{2.5}$ and O_3 are responsible for adverse health effects. As a result, the quantitative estimates of population health outcomes in this analysis are assumed to represent an underestimate of the full impacts of air pollution in Canada

While all three pollutants considered here exert impacts, exposure to $PM_{2.5}$ is associated with the majority of the observed premature mortality (66%) while O_3 and NO_2 account for 27% and 6%, respectively. For morbidity outcomes, both O_3 and $PM_{2.5}$ are associated with health impacts. NO_2 is absent because, while considered to be causally associated with several important respiratory effects, there are currently no CRFs in the AQBAT for NO_2 and morbidity outcomes.

Although impacts of air pollution are felt in all regions of the country, the largest effects are seen in relation to the largest populations, and are greatest for Ontario, Québec, British Columbia and Alberta. Overall, this

analysis indicates that despite improvements in air quality, air pollution continues to have impacts on population health in Canada.

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Appendix A. NO_2, O_3 and $PM_{2.5}$ concentration-response functions in AQBAT v3.0

Appendix A. NO_2, O_3 and $PM_{2.5}$ concentration response functions in AQBAT v3.0

Pollutant	Endpoint	Source(s)	Details	Averaging period	Regression type	Form	Mean beta	SE beta
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, O_3 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
O_3				1 h			8.39E-04	1.36E-04
O_3 (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 O_3 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	Averaging period	Regression type	Form	Mean beta	SE beta
O_3 (May–Sept.)	Acute respiratory symptom days	Krupnick et al. (1990)	The authors reported on the association between O_3 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 CoH, 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 O_3 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 non-asthmatic (85.7%) children aged 5–19 years.	1 h	Linear	Normal	7.86E-04	3.86E-04

Pollutant	Endpoint	Source(s)	Details	Averaging period	Regression type	Form	Mean beta	SE beta
O_3 (May–Sept.)	Asthma symptom days	Mortimer et al. (2002) Schildcrout et al. (2006)	Numerous panel studies have been conducted on the association between O_3 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 O_3 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 O_3 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 O_3. 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 O_3 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 O_3 (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	Averaging period	Regression type	Form	Mean beta	SE beta
O_3 (May–Sept.)	Minor restricted activity days	Ostro and Rothschild (1989)	Ostro and Rothschild (1989) reported an association between O_3 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 O_3 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 O_3 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 non-asthmatic (85.7%) children aged 5–19 years.	1 h	Log(RR) or Log(OR)	Normal	5.30E-04	2.91E-03
O_3 (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
O_3 (May–Sept.)	Respiratory hospital admissions	Burnett et al. (1997)	Burnett et al. (1997) reported the results of a study on O_3 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 O_3. 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	Averaging period	Regression type	Form	Mean beta	SE beta
$PM_{2.5}$	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 O_3, 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 µg/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 µg/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 non-asthmatic (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 µg/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 µg/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	Averaging period	Regression type	Form	Mean beta	SE beta
$PM_{2.5}$	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	Averaging period	Regression type	Form	Mean beta	SE beta
$PM_{2.5}$	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 O_3 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	Averaging period	Regression type	Form	Mean beta	SE beta
$PM_{2.5}$	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[b]	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 cerebro-vascular mortality			24 h	Log(RR) or Log(OR)	Gamma	4.884E+0	3.375E-03

Pollutant	Endpoint	Source(s)	Details	Averaging period	Regression type	Form	Mean beta	SE beta
	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
$PM_{2.5}$	Chronic Exposure Internal Cause Mortality	Crouse et al. 2012	Crouse et al. (2012) examined the association between PM2.5 derived from satellite observations and mortality during ten years of follow-up of a cohort of 2.1 million Canadians 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 µg/m3 PM2.5. 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 non-linear	Normal	Θ =0.143	SE(θ) = 0.01807
							Additional parameters: α = 1.6 µ = 15.5 τ * r = 36.8 threshold concentration = 2.4	

Pollutant	Endpoint	Source(s)	Details	Averaging period	Regression type	Form	Mean beta	SE beta
$PM_{2.5}$	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 O_3 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
$PM_{2.5}$	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 $\mu g/m^3$ $PM_{2.5}$. 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 non-asthmatic (85.7%) children aged 5–19 years.	24 h	Log(RR) or Log(OR)	Normal	4.81E-03	1.01E-03

Appendix A. NO_2, O_3 and $PM_{2.5}$ concentration response functions in AQBAT v3.0

AQBAT: Air Quality Benefits Assessment Tool; CI: confidence interval; CO: carbon monoxide; CoH: coefficient of haze; CRF: concentration–response function; NO_2: nitrogen dioxide; O_3: ozone; PM: particulate matter; PM_{xx}: particulate matter with an aerodynamic diameter of xx µm or less; ppb: parts per billion; pphm: parts per hundred million; ppm: parts per million; SE: standard error; SO_2: sulphur dioxide; TSP: total suspended particulate; WHO: World Health Organization

[a] Tables and figures referred to in lowercase are referring to items in the source paper.

[b] CO = 1 ppm; NO_2 = 20 ppb; O_3 = 20 ppb; $PM_{2.5}$ = 10 µg/m^3; SO_2 = 5 ppb

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