Impact of a 10-year shift in ambient air quality on mortality in Canada: a causal analysis of multiple pollutants

Hong Chen, Matthew Quick, Jay S Kaufman, Chen Chen, Jeffrey C Kwong, Aaron van Donkelaar, Randall V Martin, Michael Tjepkema, Tarik Benmarhnia, Richard T Burnett

Summary

Background The impact of past air quality improvements on health and equity at low pollution levels near the revised WHO air quality guidelines remains largely unknown. Less is known about the influence of simultaneous reductions in multiple major pollutants. Leveraging real-world improvements in air quality across Canada, we sought to directly evaluate their health benefits by quantifying the impact of a joint shift in three criteria pollutants on mortality in a national cohort.

Methods In this population-based cohort study, we assembled a cohort of 2·7 million adults living in Canada in 2007 who were followed up through 2016. Annual mean concentrations of fine particulate matter ($PM_{2·5}$), nitrogen dioxide (NO_2), and ozone (O_3) were assigned to participants' residential locations. For each pollutant individually and combined, we conducted a causal analysis of the impact of the decadal shift in annual exposure from the pre-baseline level (2004–06) on the risk of non-accidental mortality using the parametric g-formula, a structural causal model. To check the robustness of our results, we conducted multiple sensitivity analyses, including exploring alternative exposure scenarios. We also evaluated differential benefits across regions and socio-demographic subgroups.

Findings Between 2007 and 2016, annual mean exposures to $PM_{2·5}$ and NO_2 decreased (from 7·1 μg/m³ [SD 2·3] to 5·5 μg/m³ [1·9] for $PM_{2·5}$ and from 11·1 ppb [SD 6·6] to 8·0 ppb [4·9] for NO_2), whereas O_3 declined initially and then rebounded (from 38·6 [SD 8·3] ppb to 36·0 [6·0] ppb and then 38·1 [5·4] ppb). Compared to pre-baseline (2004–06) levels, the joint change in the pollution exposures beginning in 2007 resulted in, per million population, 70 (95% CI 29–111) fewer deaths by 2009, 416 (283–549) fewer deaths by 2012, and 609 (276–941) fewer deaths by 2016, corresponding to a –0·7% change in mortality risk over the decade. Stratified analyses showed greater beneficial impacts in men, adults aged 50 years and older, low income-earners, and residents in regions undergoing substantial air quality improvements. Had all regions experienced pollution reductions similar to the most improved region, approximately three times as many deaths would have been averted (2191 fewer deaths per million). Conversely, if the observed air quality improvements had been delayed in all regions by 3 years, there would have been 429 more deaths per million by 2016.

Interpretation In Canada, substantial health gains were associated with air quality improvements at levels near the revised WHO guidelines between 2007 and 2016, with notable heterogeneity observed across socio-demographic subgroups and regions. These findings indicate that modest declines in air pollution can considerably improve health and equity, even in low-exposure environments.

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Introduction

Air pollution, comprising particulate matters and gases, is one of the greatest environmental risks to human health.[1] Globally, the population-weighted concentration of fine particulate matter ($PM_{2·5}$) in 2019 was unchanged from that in 2000.[2] However, in some regions, significant reductions in $PM_{2·5}$ have been achieved due to air quality regulations and technological advancements.[3] Between 1990 and 2019, $PM_{2·5}$ concentrations declined by 42% in North America and by 39% in western Europe.[4] Similarly large reductions in nitrogen dioxide (NO_2) were also observed,[5] although reductions in ozone (O_3) were less pronounced.[6] These declines, particularly in $PM_{2·5}$ since the 1990s, have been associated with detectable health benefits, including improved respiratory health,[7–9] slower cognitive decline,[10,11] and increased life expectancy.[12–15] However, the potential health gains from further reductions in air pollution to lower concentrations remain less well understood.

Understanding this potential health benefit is crucial, as progress in further reducing air pollution has stalled in many high-income regions[4] and even reversed in other regions (eg, south Asia).[2,5,6] Although many

Research in context

Evidence before this study

We searched the MEDLINE and Google Scholar databases for epidemiological studies estimating health benefits from past reductions in long-term exposure to air pollution. Studies published in the peer-reviewed literature from Jan 1, 1980, up to May 1, 2024, were included, regardless of the language of publication. We perused the bibliographies of these articles and of previously published reviews. We searched the bibliographic databases using the following search string: ("outdoor" OR "ambient") AND ("air pollution" OR "$PM_{2·5}$" OR "fine particulate matter" OR "O_3" OR "ozone" OR "NO_2" OR "nitrogen dioxide") AND ("long-term exposure" or "chronic exposure") AND ("improvement" OR "reduction" OR "decline") AND ("health" OR "impact" OR "benefit"). A few studies reported improvements in lung function and reductions in bronchitis and asthma incidence in children in response to decreasing air pollution since the 1990s, a period marked by high levels of air pollution. Several other studies linked these reductions in air pollution to slower cognitive decline and lowered dementia incidence in adults, as well as increased survival in the general population. There is also growing evidence showing positive health effects from abrupt reductions in $PM_{2·5}$ due to some changes in economic activity or policy over the past three decades. The converging lines of evidence show that substantial declines in high levels of air pollution are beneficial. However, no study has so far evaluated the survival benefits of past reductions in air pollution at low levels. Additionally, previous studies have typically focused on a single exposure, even though humans are simultaneously exposed to multiple pollutants, which are likely to have a greater effect. Furthermore, few studies have assessed the potential differential benefits across various socio-demographic subpopulations.

Added value of this study

Our study expanded on existing research by quantifying the combined effects of long-term changes in three major pollutants on mortality in Canada, where air pollution levels are considerably lower than in most other countries. We used the g-formula, a structural causal model uniquely suited for estimating the marginal causal effect of joint exposures using observational data with time-varying exposures, confounders, and health outcomes. By analysing data from a national cohort of 2·7 million adults from 2007 to 2016, we found that within just 3 years, a joint shift in annual exposures to fine particulate matter ($PM_{2·5}$), nitrogen dioxide (NO_2), and ozone (O_3) was associated with tangible benefits in terms of avoided deaths and increased life-years, with greater benefits observed over the longer term. We also estimated that if all regions in Canada had reductions in air pollution similar to the most improved region, three times as many deaths would have been averted. Conversely, if the observed air quality improvements had been delayed in all regions by 3 years, there would have been 429 more deaths per million by 2016. This study provides important insights into the public health benefits of real-world air pollution reductions at levels near the 2021 revision of the WHO air quality guidelines.

Implications of all the available evidence

Over the past few decades, air quality has improved substantially at diverse locations around the world, especially in high-income regions. However, recent progress in reducing air pollution has stagnated in many high-income regions and even reversed in others. This study adds weight to previous findings that reductions in air pollution improve survival. More importantly, this study provides direct evidence that modest declines in air pollution can yield large health benefits, even in low-exposure environments. Given that most of the global population is exposed to air pollution surpassing the revised WHO guidelines, this study substantially advances the case for continuing efforts to lower ambient air pollution. Our findings also reveal greater benefits among men, older adults, low-income earners, and residents in regions undergoing substantial air quality improvements, highlighting the role of air pollution reductions in improving health equity. In light of the growing need to address health disparities worldwide, these findings can have important implications for policies aimed at protecting vulnerable populations.

studies[16–18] and the 2021 revision of WHO air quality guidelines[1] suggest that chronic exposure to pollution at low levels (eg, at or >5 μg/m³ for $PM_{2·5}$) remains a considerable health threat, inferring the actual benefits from exposure reductions at these low concentrations requires making several strong assumptions. These include assuming the same concentration–response relationships across populations, reliance on uniform (often simplistic) thresholds for exposure reduction regimens, and assuming time-invariant relationships between air pollution and health, despite the dynamic nature of populations and changes in the mixtures of air pollutants they are exposed to. Additionally, past studies have predominantly focused on $PM_{2·5}$, despite the fact that individuals are simultaneously exposed to multiple pollutants, leaving the overall health benefits from changes in air pollution as a mixture largely unexplored.

Given mounting interest in the benefits of further tightening current air quality regulations,[3] several recent studies have simulated $PM_{2·5}$ abatement at lower concentrations and linked the hypothetical reductions to improved respiratory health[19] and decreased mortality.[19–21] To more directly evaluate the health benefits of air quality improvements at low levels near the revised WHO air quality guidelines, we aimed to quantify the impact of a joint shift in annual $PM_{2·5}$, NO_2, and O_3, three criteria pollutants, on mortality in a national cohort of 2·7 million Canadians by leveraging real-world changes

in concentrations of these pollutants from 2007 to 2016. Canada represents an ideal setting for this study given the existence of a well characterised national cohort and the fact that air pollution levels in Canada are among the lowest globally.[4]

Methods

Study population

For this population-based cohort study, we created a national cohort using the 2006 wave of the Canadian Census Health and Environment Cohort (CanCHEC). Constructed by Statistics Canada through linking quinquennial census surveys of the Canadian population with health administrative databases, vital statistics, and income tax files, CanCHEC provides detailed information on ethnocultural and socioeconomic characteristics, health status, and personal residential history.[18] This cohort has been used to assess the relationship between $PM_{2·5}$ and mortality.[18]

Individuals were eligible for inclusion in this study if they were aged 30–79 years and had lived in Canada for more than 5 years on Jan 1, 2007 (baseline). Those with incomplete exposure and covariate data, as well as those who died before baseline, were excluded. Our outcome of interest was non-accidental death, obtained from the national vital statistics database (appendix p 3). We followed up the cohort through to Dec 31, 2016.

This study was approved by the Research Ethics Board of Health Canada and the Public Health Agency of Canada.

Air pollution exposures

Annual mean exposures to $PM_{2·5}$, NO_2, and O_3 were derived for each participant on the basis of their residential postal code addresses between 1997 (10 years before baseline) and 2016. We used version V4.NA.02.MAPLE of $PM_{2·5}$ estimates, developed with satellite observations in combination with outputs from a global atmospheric chemistry transport model (GEOS-Chem).[22] The $PM_{2·5}$ estimates were further calibrated with a geographically weighted regression of land cover, elevation, and aerosol composition, producing annual mean concentrations of $PM_{2·5}$ (1×1 km) across North America between 1998 and 2016.[22] These estimates closely agreed with measurements at fixed-site monitors (R^2=0·73, n=2312).[22]

For NO_2, we used a national land-use regression model that included monitoring data from Environment and Climate Change Canada's National Air Pollution Surveillance Network, satellite estimates of NO_2, and data measuring industrial land use, road length, and mean summer rainfall.[23] To incorporate local-scale variations of NO_2 due to vehicle emissions, the NO_2 estimates were calibrated by applying spatially varying multipliers that represented distance-decay gradients.[23] The final land-use regression model explained 73% of annual 2006 measurements of NO_2 (100×100 m).[23]

For O_3, we used a long-term average O_3 surface derived using an optimal interpolation technique.[24] This approach combines measured O_3 with physically based air quality prediction models that account for meteorological and chemical patterns of O_3. Using this approach, Environment and Climate Change Canada produced annual mean warm-season exposure surfaces of O_3 across Canada yearly between 2002 and 2015.[24] From 2002 to 2009, the O_3 estimates had a spatial resolution of 21×21 km, which was subsequently refined to 10×10 km.[24]

Because our exposure surfaces were derived at certain time periods (eg, $PM_{2·5}$ exposure surface was derived in 1998–2016), we conducted yearly calibration of these surfaces using spatiotemporal adjustment factors derived from ground-level observations at fixed-site stations across Canada.[18] This approach allowed us to create annual mean exposures to $PM_{2·5}$, NO_2, and O_3 for each year between 1997 and 2016 (appendix p 3).[18]

Confounding variables

Based on existing literature (appendix p 4),[16–18,25–28] we obtained the following time-fixed individual-level variables from the census questionnaire: age, sex, racialised population (visible minority status [defined by Statistics Canada as "persons, other than Aboriginal peoples, who are non-Caucasian in race or non-White in colour"] and Indigenous identity), immigration status, marital status, educational attainment, occupational class, and employment status. From income tax files, we created time-varying annual household income (in deciles; appendix p 4). We also derived four time-varying neighbourhood-level deprivation measures: residential instability, material deprivation, dependency, and ethnic concentration.[29] To characterise active commuting and transit use, we created a time-varying urban form variable[18] and, to account for regional differences in mortality that might be caused by factors other than pollution, we created time-varying variables representing community population size and the six Canadian regions (British Columbia, Prairies, Ontario, Quebec, Atlantic, and Territories). Finally, we derived four time-varying variables to represent meteorological conditions: annual mean and SD of air temperature and relative humidity. All area-level variables were assigned to annual residential postal codes and the nearest census year.

See Online for appendix

Statistical analysis

We used the parametric g-formula to estimate the average causal effect of the joint shift in annual exposures to $PM_{2·5}$, NO_2, and O_3 on mortality. The parametric g-formula provides unbiased estimates of marginal causal effect of treatments under standard identifiability assumptions (appendix p 5).[30,31] This approach has been described in detail elsewhere.[30,31] It has been increasingly applied to evaluate sustained and dynamic interventions, including air quality policies in Canada.[7,20,30,31]

In this study, we compared mortality risk from observed exposures to $PM_{2·5}$, NO_2, and O_3 between 2007 and 2016 (natural course exposure) with counterfactual mortality risk had exposures been held constant at pre-baseline levels. Pre-baseline exposure for each participant was defined as the average from 2004 to 2006 to allow for a stable reference for previous exposure. To capture cumulative effects, we considered a 3-year history of annual $PM_{2·5}$, NO_2, and O_3 exposures.[18,20]

As done previously,[30] we first fitted separate parametric regression models for all time-varying covariates and the outcome at each annual time interval, conditional on covariate histories and baseline covariates (see appendix p 5). Then, we conducted Monte Carlo simulation based on these parametric models to estimate the predicted probability of non-accidental mortality using random draws with replacement (n=10 000) from the original study population under each exposure scenario.

In each time-varying covariate model, we included baseline age, sex, race or ethnicity, immigration status, marital status, education, occupation, employment, categorical years since baseline, and all other time-varying covariates that came before it, based on a directed acyclic graph (appendix p 4). In the outcome model, we a-priori introduced three 2-way interactions among the three pollutants,[18] as well as three 2-way interactions between each pollutant and categorical years since baseline to allow for time-varying concentration–response relationships. We used the Shape Constrained Health Impact Function approach[32] to characterise the shape of concentration–response relationships for the outcome model (appendix p 6).

We computed the absolute difference in average mortality risk between the natural course exposure and the exposure scenario where $PM_{2·5}$, NO_2, and O_3 were maintained at pre-baseline levels, expressed as a mortality risk difference in mortality (per million population). As a complementary effect measure, we also calculated the percentage change in mortality as [(1–risk ratio)×100%], where the risk ratio contrasted the average mortality risk under the natural course exposure to that under pre-baseline exposure on a multiplicative scale. 95% CIs were constructed with 200 non-parametric bootstrap resamples. To contextualise our findings, we calculated the number of deaths averted (per million) throughout the 10-year period. Using restricted mean survival time estimated for the two exposure scenarios, we calculated years of life gained (per million).

Secondary analysis

To evaluate the robustness of the estimates, we conducted additional analyses, including different causal ordering of covariate models; alternative scenarios had exposures been held at the 2002–06, 2000–06, and 1997–2006 levels; restricted cubic spline to characterise the concentration–response relationships for $PM_{2·5}$, O_3, and NO_2 in the outcome model (appendix p 6); quantised exposures for $PM_{2·5}$, O_3, and NO_2 (in deciles) in the outcome model; and a 5-year history of exposures (appendix p 6). To illustrate the importance of evaluating multiple exposures simultaneously, we further conducted the g-formula analysis by treating each pollutant as the sole exposure for air pollution by omitting other pollutants in the outcome model and covariate models.

To evaluate possible effect heterogeneity, we performed stratified analyses by age, sex, income (lowest *vs* highest tertile), urbanity, and three larger regions (western [British Columbia and Prairies], central [Ontario and Quebec], and Atlantic), all based on the additive scale.[33] These analyses were done by calculating the average causal effect within specific strata of the study population (eg, younger *vs* older adults). To inform future air pollution policies, we further assessed the effect on mortality had all regions experienced the same reduction in pollution as the most improved region relative to the pre-baseline exposure. Furthermore, we evaluated another hypothetical scenario had the observed air quality improvements been delayed in all regions by 3 years.

All analyses were conducted with SAS Enterprise Guide (version 7.11) and R software (version 4.0.5) with the gfoRmula package (Syntax of R code for implementation of the g-formula approach provided in the appendix p 10).

Role of the funding source

The funder of the study had no role in study design, data collection, data analysis, data interpretation, the writing of the report, or the decision to submit the paper for publication.

Results

Of 3 343 370 potential study participants, exclusions were made for the following reasons: 432 580 (12·9%) did not meet the age criteria; 83 630 (2·5%) were resident in Canada for 5 years or less; 31 835 (1·0%) had missing air pollution exposure; 126 805 (3·8%) had missing covariates; and 4880 (0·1%) died before baseline, yielding a cohort of 2 663 645 with 25·7 million person-years of observations. The mean age at baseline was 50·9 (SD 12·8) years, 1 293 890 (48·6%) were men and 1 369 755 (51·4%) were women, 279 855 (10·5%) belonged to a visible minority, 542 060 (20·4%) had less than a high school education, and 1 744 340 (65·5%) were employed (appendix p 34). The average follow-up was 9·6 (SD 1·4) years. The annual mortality rate was 0·8% (or 213 882 deaths in total).

Between 2007 and 2016, annual $PM_{2·5}$ mean concentrations decreased from 7·1 μg/m³ (SD 2·3) to 5·5 μg/m³ (SD 1·9) and NO_2 decreased from 11·1 ppb (SD 6·6) to 8·0 ppb (SD 4·9; figure 1). Conversely, annual O_3 mean concentrations decreased from 38·6 (SD 8·3) ppb to 36·0 (6·0) ppb during the first 5 years and rebounded in subsequent years to 38·1 (5·4) ppb in 2016. Relative to

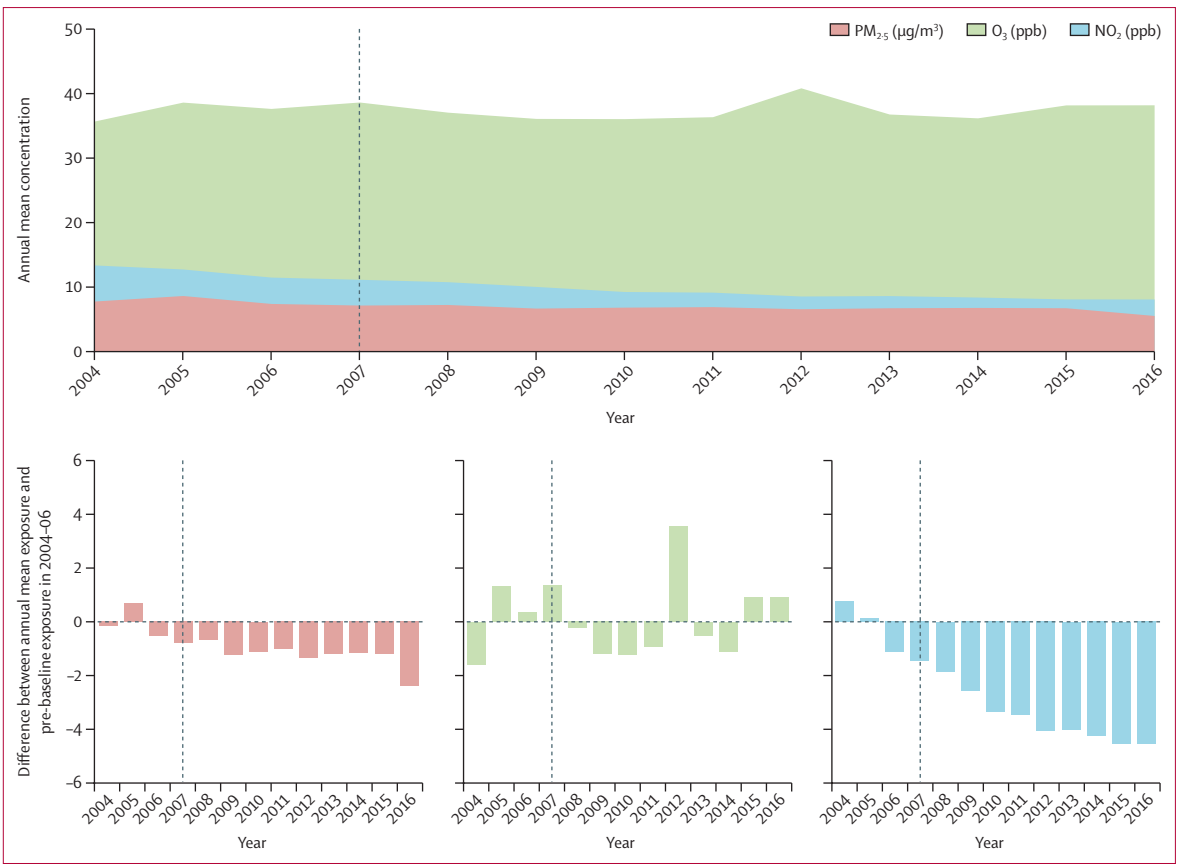

Figure 1: **Observed changes in annual mean exposures to three critera pollutants in the 2006 CanCHEC from 2004 to 2016**
Annual mean exposures to fine particulate matter ($PM_{2·5}$), ozone (O_3), and nitrogen dioxide (NO_2; top) and difference between the annual mean exposures and pre-baseline exposures in 2004–06 (bottom) in the 2006 Canadian Census Health and Environment Cohort (CanCHEC) between 2004 (3 years before cohort onset) and 2016 (end of follow-up). In the top panel, the vertical axis represents the annual mean exposure to each of the three pollutants (ie, superimposed on each other). The vertical dashed line in both panels denotes the year of cohort onset.

3-year mean exposure during the pre-baseline period (2004–06), similar trends were observed, with an average decrease of 1·2 µg/m³ (SD 1·5) in $PM_{2·5}$ and 3·4 ppb (SD 3·7) in NO_2, and a slight increase of 0·2 ppb (SD 4·8) in O_3 (figure 1). Annual $PM_{2·5}$ mean concentrations were correlated with NO_2 (Pearson correlation coefficient, r=0·60) and O_3 (r=0·42) whereas O_3 was weakly correlated with NO_2 (r=0·10).

The joint shift in $PM_{2·5}$, O_3, and NO_2 from pre-baseline was associated with substantial declines in mortality: per million population, 70 (95% CI 29–111) fewer deaths by 2009; 416 (283–549) fewer deaths by 2012; and 609 (276–941) fewer deaths by 2016 (figure 2). This corresponds to an approximate –0·7% change in 10-year mortality risk (appendix p 37). A similar improvement was observed in terms of life-years: per million population, 43·6 (95% CI 4·5–82·7) life-years were gained by 2009; 761·3 (505·6–1017·1) life-years were gained by 2012; and 2652·2 (1562·0–3742·4) life-years were gained by 2016.

Considering each pollutant as the sole exposure, the 10-year shift in annual mean $PM_{2·5}$ concentration was

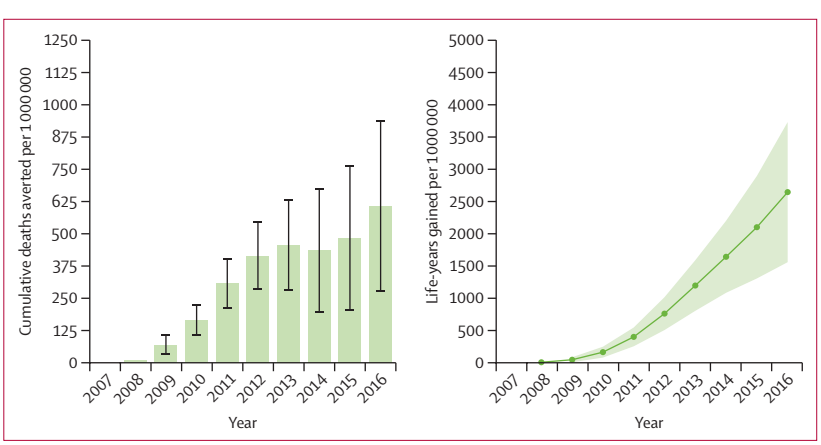

Figure 2: **Impact of a joint shift in three criteria pollutants on mortality risk and life-years in the 2006 CanCHEC**
Cumulative number of deaths averted (left) and life-years saved (right) per million people in the 2006 Canadian Census Health and Environment Cohort (CanCHEC) following a decadal change in the ambient levels of fine particulate matter ($PM_{2·5}$), nitrogen dioxide (NO_2), and ozone (O_3), compared to pre-baseline (2004–06) levels, estimated with the g-formula adjusted for all available covariates. The shading in the right panel and error bars in the left panel represent 95% CIs.

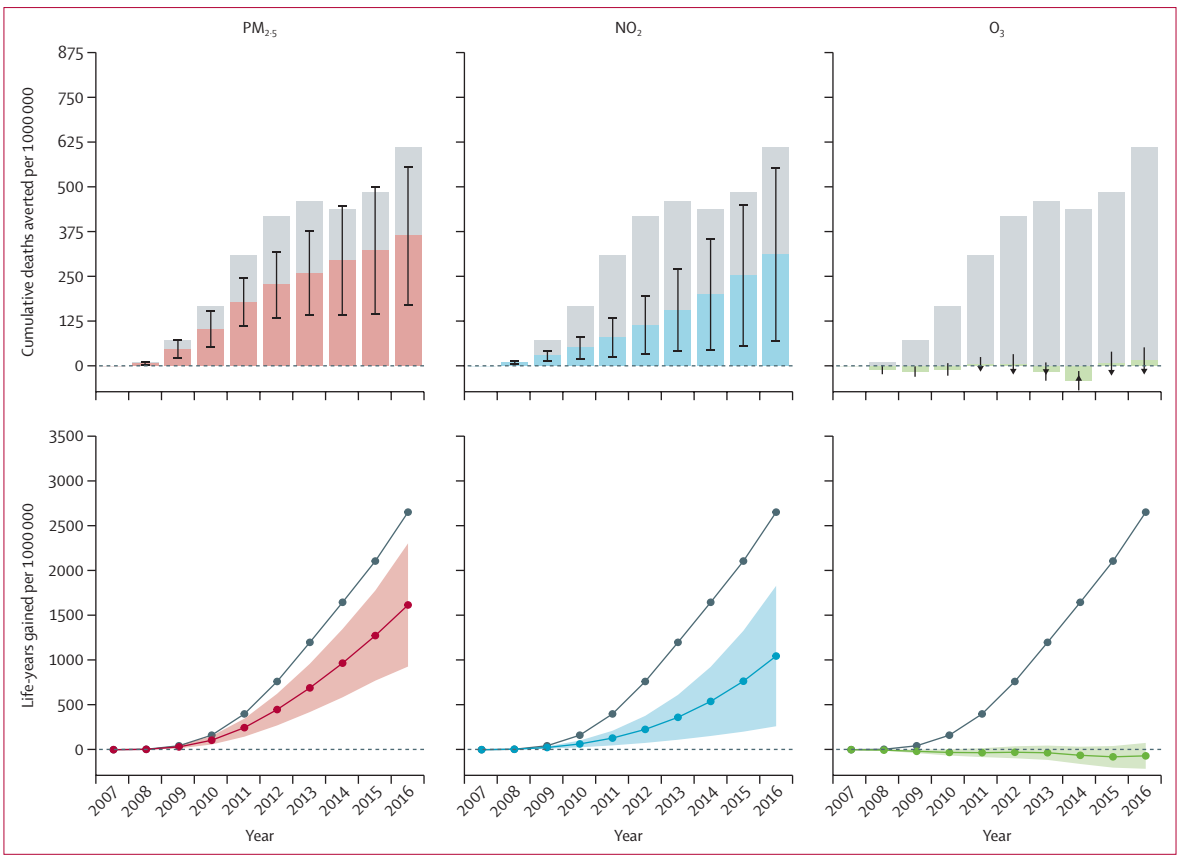

Figure 3: **Impact of a 10-year shift in each criteria pollutant on mortality risk and life-years in the 2006 CanCHEC, analysed by a single-pollutant approach**
Cumulative number of deaths averted (top) and life-years saved (bottom) per million people in the 2006 Canadian Census Health and Environment Cohort (CanCHEC) following a decadal change in fine particulate matter ($PM_{2·5}$), nitrogen dioxide (NO_2), and ozone (O_3), compared to pre-baseline (2004–06) levels. The grey bars (top) and lines (bottom) indicate the cumulative number of deaths averted and life-years saved from the decadal changes in the three pollutants as a mixture, estimated with the g-formula adjusted for all available covariates. Error bars and shading represent 95% CIs.

associated with 363 (95% CI 169 to 556) fewer deaths and 1615·7 (95% CI 928·1 to 2303·4) life-years gained per million by 2016 (figure 3). The shift in NO_2 was associated with 310 (95% CI 67 to 553) fewer deaths and 1045·5 (262 to 1829) life-years saved per million by 2016. By contrast, the shift in O_3 had minimal impact on survival, resulting 15 fewer deaths (95% CI –22 to 55) and 79 (95% CI –76 to 213) life-years lost per million by 2016.

When considering different pre-baseline periods, the health benefits were maintained (figure 4). For example, the joint shift of $PM_{2·5}$, O_3, and NO_2 from the 10-year pre-baseline (1997–2006) resulted in, per million population, 178 (95% CI 92 to 265) fewer deaths and 176·6 (70·8 to 282·4) life-years gained by 2009, 594 (365 to 822) fewer deaths and 1305·8 (774·4 to 1837·2) life-years gained by 2012, and 826 (351 to 1301) fewer deaths and 3883·5 (2058·3 to 5708·7) life-years gained by 2016.

Our findings remained consistent when we varied the ordering of covariate models and when we characterised the concentration–response relationships using spline-based and quantisation-based approaches (appendix p 41). We also obtained similar results using 5-year history of exposures. Furthermore, all models showed excellent performance in estimating mortality risk and the distributions of risk factors under the natural course (appendix p 42).

Additionally, the beneficial impact of the joint shift in $PM_{2·5}$, O_3, and NO_2 was more pronounced in adults aged 50 years and older ($p_{interaction}<0·0001$; figure 5). Larger health impacts were also evident in low-income compared to high-income earners (eg, per million, 5964·5 *vs* 2172·6 life-years gained by 2016; $p_{interaction}=0·02$) and in men compared to women (3878·9 *vs* 1696·7 life-years gained; $p_{interaction}= 0·08$). Furthermore, the joint shift of these pollutants resulted in 5068·3 life-years gained among participants in central Canada versus 2634·3 life-years gained among those in western Canada by 2016 ($p_{interaction}=0·28$). Air quality improvements were most pronounced in central Canada and in urban areas, yet were similar across other subgroups defined by age, sex, and income (appendix pp 43–47). Had all regions experienced the same pollution reductions as central Canada, there would have been an estimated 8116·1 life-years gained and 2191 deaths averted per million by 2016

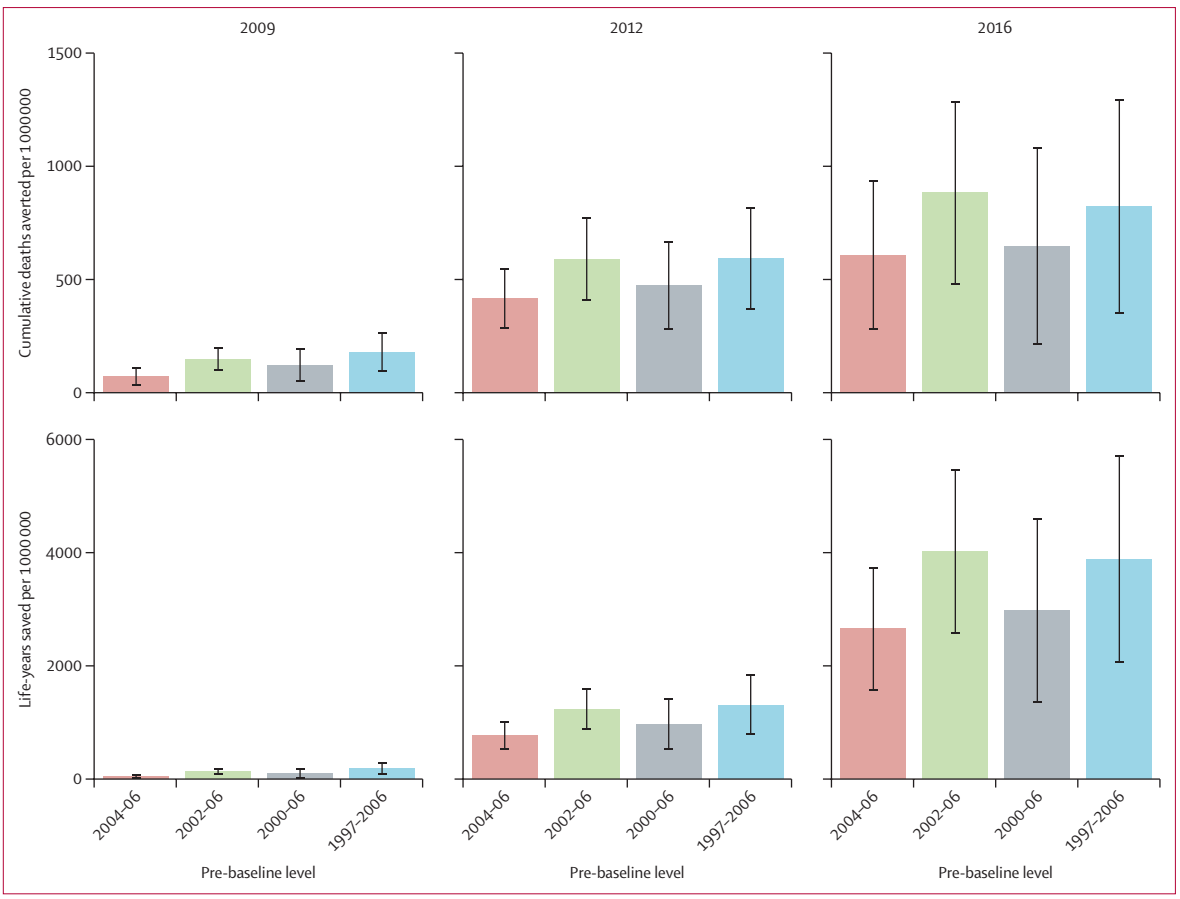

Figure 4: **Impact of a joint shift in three criteria pollutants on mortality risk and life-years in the 2006 CanCHEC, by counterfactual exposure and timepoint**
Cumulative number of deaths averted (top) and life-years saved (bottom) per million people in the 2006 Canadian Census Health and Environment Cohort (CanCHEC) following a decadal change in fine particulate matter ($PM_{2·5}$), nitrogen dioxide (NO_2), and ozone (O_3), in comparison with four pre-baseline levels (2004–06, 2002–06, 2000–06, and 1997–2006) and by three timepoints (2009, 2012, and 2016), estimated with the g-formula adjusted for all available covariates. Error bars represent 95% CIs.

(appendix p 38). Conversely, if the observed shift in $PM_{2·5}$, O_3, and NO_2 had been delayed in all regions by 3 years, 429 more deaths per million would have occurred by 2016 (appendix p 39).

Discussion

Between 2007 and 2016, air pollution in Canada declined towards the 2021 revision of WHO guideline levels,[1] particularly for $PM_{2·5}$ and NO_2. Compared with pre-baseline levels, the combined shift in annual mean concentrations of $PM_{2·5}$, NO_2, and O_3 was associated with 609 fewer deaths per million in 2016, reflecting a –0·7% change in 10-year mortality risk. Substantial health gains were also observed in terms of life-years, with 2652·2 additional life-years gained per million. These benefits were more pronounced in men, older adults, low-income earners, and residents of regions undergoing substantial air quality improvements, underscoring the importance of air pollution declines in reducing health disparities. Furthermore, we found that had all regions experienced pollution reductions similar to the most improved region, the health benefits would have more than tripled. Conversely, if the observed air quality improvements had been delayed in all regions by 3 years, there would have been 429 more deaths per million by 2016.

Globally, air quality trends have varied substantially by region, with notable declines in high-income regions and increases in others.[2,5,6] Additionally, there are considerable pollutant-specific differences in temporal trends.[2,5,6] To the best of our knowledge, this is the first study to quantify the survival benefits associated with real-world air pollution reductions near levels recommended by the revised WHO air quality guidelines.[1] To date, a small number of studies have assessed the health impacts of past air quality improvements.[7,10–15] For example, Pope and colleagues[12] reported a 0·61-year gain in life expectancy per 10 μg/m³ decrease in $PM_{2·5}$ in 211 US counties over two time periods: the late 1970s to early 1980s (mean $PM_{2·5}$ concentration 20·6 μg/m³) and the late 1990s to early 2000s (14·1 μg/m³). Similarly, Correia and colleagues[13] observed a 0·35-year gain in life

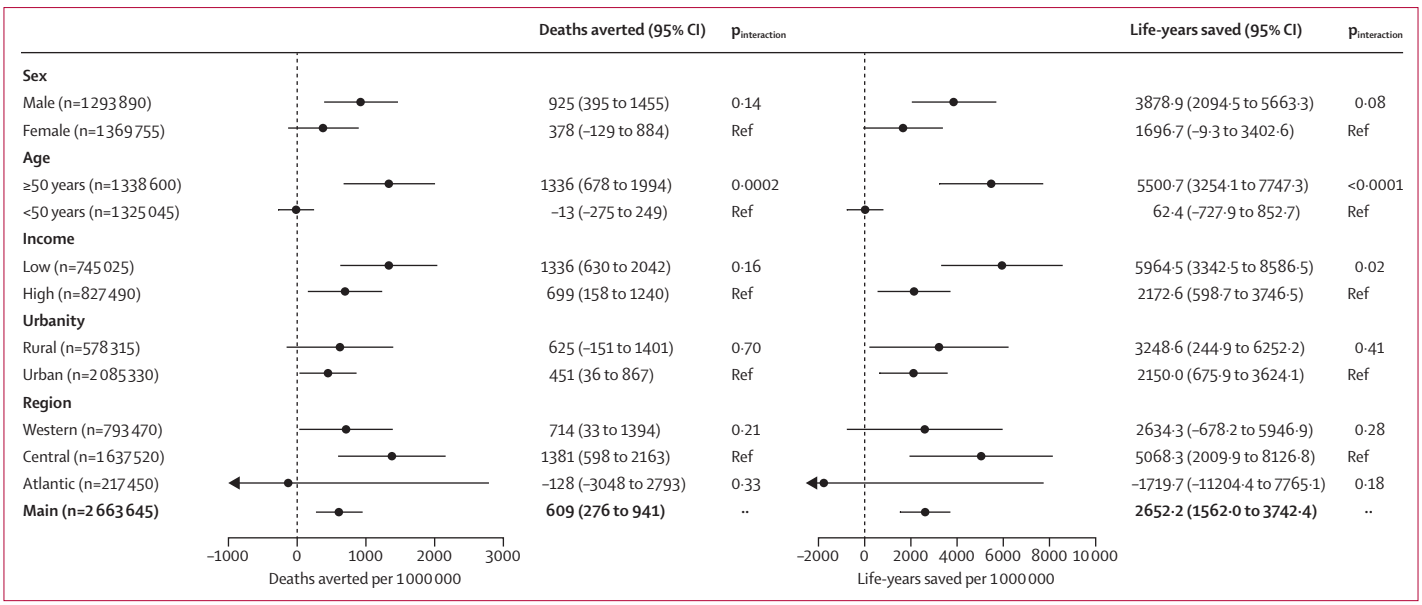

Figure 5: **Impact of a joint shift in three criteria pollutants on mortality risk and life-years in the 2006 CanCHEC, by selected characteristics**
Cumulative number of deaths averted (left) and life-years saved (right) per million people in the 2006 Canadian Census Health and Environment Cohort (CanCHEC) following a decadal change in the ambient levels of fine particulate matter ($PM_{2·5}$), ozone (O_3), and nitrogen dioxide (NO_2), compared with pre-baseline (2004–06) levels, by selected characteristics, estimated with the g-formula adjusted for all available covariates. Error bars represent 95% CIs. *All counts were rounded up to the nearest five in compliance with privacy requirements by Statistics Canada.

expectancy per 10 μg/m³ decrease in 545 US counties between 2000 and 2007. These results were supported by additional studies linking reduced $PM_{2·5}$ concentrations to increased county-level life expectancy in the USA since the 1980s.[14,15,34] More recently, in a study of 4140 school-age children in southern California from 1993 to 2014, Garcia and colleagues[7] observed a 16% reduction in childhood asthma incidence following a decrease in ambient NO_2 and, to a lesser degree, $PM_{2·5}$. These substantial improvements in air quality since the 1990s have also been linked to slower cognitive decline[10] and decreased dementia incidence.[11] Our findings reinforce previous evidence that air pollution reductions enhanced survival and show that even modest air quality improvements in low-exposure environments could yield substantial public health benefits. In our cohort, annual mean exposure decreased to 5·5 μg/m³ for $PM_{2·5}$ and 8·1 ppb for NO_2, with O_3 exposure at 38·1 ppb. These findings are timely given the recent slowdown in air quality improvements in many regions globally.[4] Supporting our results, a recent study in Stockholm, Sweden, reported significant associations between reduced air pollution and increased lung function growth in 1104 children, with median $PM_{2·5}$ concentrations declining from 8·2 μg/m³ to 5·2 μg/m³ between 2002 and 2019.[35]

Our study also highlights the importance of considering multiple exposures when evaluating the benefits of air quality improvements. To date, most studies on the health benefits or risks associated with changes in air pollution have focused on individual pollutants, especially $PM_{2·5}$,[7,10–15,34] despite the fact that humans are exposed to air pollution as a mixture. The single-pollutant approach might not adequately capture the health impacts of ongoing changes in atmospheric mixture, particularly as regulatory actions often target multiple pollutants simultaneously.[36] In this study, our analyses focusing solely on $PM_{2·5}$ estimated approximately 40% fewer health benefits in terms of deaths averted and life-years gained compared with analyses examining the three pollutants jointly. This finding aligns with the recognition that air pollution is a mixture of particulates and gases; the known effect of annual mean $PM_{2·5}$ on mortality;[25] and growing evidence linking mortality to annual mean NO_2 and O_3.[37,38] NO_2 is also a known marker for traffic-related air pollution, and thus might capture exposures to other pollutants not fully represented by $PM_{2·5}$.[38]

Focusing on Canadian population subgroups, we found that some of the largest health gains attributed to air pollution were accrued in adults aged 50 years and older. This finding contrasts with past research reporting stronger associations between long-term $PM_{2·5}$ exposure and mortality in younger adults.[25,39] One explanation for this difference is that this study quantified health impacts on the additive scale whereas past studies assessed health impacts on the multiplicative scale. Because older adults face significantly higher baseline mortality risks than younger adults, even if air pollution exposure produced a nearly constant risk ratio across age groups on the multiplicative scale, the absolute increase in risk for older adults would be notably larger. Indeed, evaluating effect modification on the additive scale is increasingly known to be more informative for identifying subpopulations that could experience the largest benefits.[33]

We also found larger health gains among men and low-income earners. This finding supports past research.[40,41] Importantly, however, because there were similar changes in exposures between men and women, and between low-income and high-income groups in this study, the larger health gains for these subgroups were likely to be attributed to differences in their underlying mortality risks rather than differences in exposures. This observation suggests that simultaneously addressing disparities in exposures and underlying mortality risks will offer larger public health benefits than reducing exposures alone. Finally, residents in central Canada had greater health gains than those in other regions. Between 2007 and 2016, this region underwent the most substantial improvements in air quality, largely due to national and bilateral regulations in Canada and the USA, the phase-out of coal burning electricity generating utilities, reduced industrial emissions, and other actions (appendix p 9). Collectively, these actions led to an average reduction of 1·8 μg/m³ for $PM_{2·5}$, 1·0 ppb for O_3, and 4·0 ppb for NO_2, compared with the pre-baseline levels in this region (appendix p 47).

Our causal analysis incorporated several assumptions. First, we assumed that the covariates were adequate to control for confounding.[42] Although we selected numerous individual-level and neighbourhood-level covariates that represented key demographic characteristics, socioeconomic status, and meteorological conditions based on our subject-matter knowledge appendix p 4),[16–18] data on other individual-level factors such as smoking and physical activity were unavailable. However, socioeconomic characteristics are strongly associated with these lifestyle variables,[43] and several previous studies have shown that directly adjusting for smoking and physical activity did not alter the association between $PM_{2·5}$ and mortality.[18,44] Additionally, we did not have repeated measurements for occupation and employment. To address their potential impact, we included time-varying annual income and neighbourhood socioeconomic status, which are likely to be part of the causal pathways linking these factors to air pollution exposure. Despite our best efforts, we recognise that the possibility of residual confounding cannot be ruled out, given the nature of observational analysis.[42]

Second, we assumed positivity, which posits that exposure scenarios were observed in all population subgroups defined by a combination of covariates.[42] Positivity violations can introduce bias into effect estimates due to smoothing over gaps in the data.[45,46] To address this concern, we used a large national cohort (2·7 million) and simulated the intervention regimen within the range of observed (natural course) exposures. By using cumulative exposure metrics, such as a 3-year history of exposure from 2007 to 2016, we further improved the overlap between natural course exposures and the simulated intervention regimen. We also used flexible models that incorporated non-linear dose–response functions and accounted for temporal changes in the conditional probability of mortality in the estimation, as supported by previous research. Additionally, we explored a complementary shifted intervention that would delay the observed air quality improvements by 3 years, which was less prone to positivity violations, and we found similar evidence in favour of air quality improvements in reducing mortality risk. Although our measures helped mitigate potential positivity violations, we acknowledge that they did not fully eliminate them, resulting in some uncertainty in our effect estimates.

Third, we implicitly assumed that the exposure scenarios being compared corresponded to well defined interventions (hence, consistency).[42] The declines in air pollution in many high-income countries, including Canada, have primarily been driven by regulations and technological developments. Although different interventions (eg, improving engine technology or adopting clean fuels) might result in varied reductions in pollutants, the effects of these interventions on mortality are expected, at a minimum, to occur in the same direction. However, the presence of potential effect modification by the mechanism through which a given amount of air pollution abatement is achieved might hinder transportability of the results to other populations and time periods. Indeed, $PM_{2·5}$ is a mixture that arises from multiple sources, and if certain $PM_{2·5}$ components or sources are more toxic than others, this assumption could be violated. In such cases, the average effect of a $PM_{2·5}$ reduction on mortality would depend on the proportion of cohort members under each version of the intervention. Given the absence of clear consensus on the relative toxicity of $PM_{2·5}$ components or sources,[47] we assumed that the effects of all intervention specifications on mortality were similar, as long as the changes in air pollution exposure occurred. Nonetheless, the term "air quality improvements" does not correspond to a unique intervention, which could complicate the interpretation of our results.

Fourth, we relied on the spatially derived exposure measures and we did not have information on daily activity, which could either overestimate or underestimate personal exposure. Additionally, our use of different traditional exposure methods inevitably involved varying measurement errors between exposures, which could introduce additional uncertainty to our results. Future air quality intervention research can benefit from using more advanced exposure assessment techniques, such as machine learning.[48] Fifth, data constraints prevented us from identifying participants who might have been lost to follow-up due to emigration from Canada. However, less than 0·2% of the Canadian population emigrates annually,[49] so this was unlikely to have a major impact on this study. Finally, we cannot completely rule out potential model misspecification. Nonetheless, because the modelled natural course values resembled the

observed values and because the results were consistent across many covariate model specifications, gross model misspecification under the natural course is highly unlikely.

This study has several strengths, including its large size and national representation of adults throughout Canada. Additionally, we gathered extensive individual-level information (eg, income, education, and race or ethnicity), allowing us to control for various known risk factors. Another novel aspect of our study is the application of the g-formula, a powerful technique for causal inference that is uniquely suited to evaluate sustained and dynamic interventions, in the context of air pollution mixtures (appendix p 8). Unlike previous health impact assessments that typically focused on one pollutant at a time, this method is particularly valuable for assessing the combined impacts of multiple exposures that can fluctuate in different magnitudes and directions over time. This method also offers insights into the trajectory of health benefits gained from sustained interventions, such as progressive reductions in pollution. Moreover, the g-formula, operating under less restrictive conditions than traditional regression-based methods,[30,31] allows for derivation of marginal causal effects on both relative and additive scales, further enhancing our policy relevance. Finally, the available high-resolution data measuring $PM_{2·5}$, NO_2, and O_3, along with personal residential histories, offered a unique opportunity to construct comprehensive exposures for the cohort over a decade.

Our causal analysis of multiple common pollutants in Canada shows that real-world air pollution reductions at levels near the revised WHO guidelines led to significant public health gains, with notable differences across socio-demographic groups and regions. This study adds to the growing evidence supporting the benefits of long-term reductions in air pollution and strengthens the case for global air quality improvement. Given the changing nature of air pollution, further research is needed to better understand the interactions and combined effects of major pollutants.

Contributors

HC developed the research idea and study design for the study. TB, JSK, CC, and RTB contributed to study design. MQ prepared and cleaned the data. HC, MQ, JSK, CC, and TB contributed to the statistical methodology. HC, MQ, RTB, and MT contributed to data curation. AvD and RVM contributed to exposure assessment. MQ and HC contributed to data analyses and visualisation. HC took the lead in drafting the manuscript. HC, MQ, JSK, CC, JCK, AvD, RVM, MT, TB, and RTB contributed to the interpretation of data, provided critical revisions to the manuscript, and approved the final draft. HC and MQ accessed and verified the underlying data in the study. All authors had full access to all the data in the study and had final responsibility for the decision to submit the manuscript for publication.

Declaration of interests

We declare no competing interests.

Data sharing

Statistics Canada's policy on data privacy and confidentiality prohibits the analytical cohort data used to be freely available in the manuscript or in a public repository. However, access can be granted through Statistics Canada's Research Data Centre programme. Environmental exposures are available upon request to the original authors of the data. The analytical code used was all standard R and SAS code (eg, gfoRmula, data steps).

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