Estimated public health impacts of changes in concentrations of fine particle air pollution in Canada, 2000 to 2011

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ABSTRACT

OBJECTIVES: To estimate the public health impacts of changes in fine particle air pollution in Canada between 2000 and 2011, employing nationally comprehensive exposure estimates and quantifying the impacts on life expectancy, mortality and morbidity.

METHODS: We employed spatially comprehensive exposure estimates derived from satellite remote sensing to estimate the effects of actual observed changes in concentrations of fine particulate matter (PM), of median aerodynamic diameter <2.5 μm (i.e., $PM_{2.5}$), from 2000 to 2011. We estimated changes in life expectancy using standard life table methods and changes in frequency of health outcomes as the product of population, baseline rate of the health outcome and the proportional change in health outcome per specified change in $PM_{2.5}$ concentration.

RESULTS: A population weighted average decrease in $PM_{2.5}$ of nearly 25% (2.0 μg/m^3) was observed between 2000 and 2011. This was estimated to result in a national population weighted average increase in life expectancy of 0.10 years (95% confidence interval 0.03–0.23; up to 0.34 years in specific census divisions) and reductions in the frequency of mortality and morbidity of up to 3.6%. Increases in $PM_{2.5}$ up to 3.5 μg/m^3 were observed in some census divisions, particularly in the prairies.

CONCLUSION: At the national level, changes in $PM_{2.5}$ concentrations between 2000 and 2011 were associated with an estimated improvement in national population weighted average life expectancy and a net reduction in mortality and morbidity. Areas that failed to improve or that worsened during this period warrant additional scrutiny to identify options for reducing $PM_{2.5}$ concentrations.

KEY WORDS: Air pollution; life expectancy; mortality; morbidity

La traduction du résumé se trouve à la fin de l'article.

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There is a large body of literature linking air pollution exposure with morbidity and mortality.[1,2] A number of estimates have been generated of the public health impacts of air pollution exposure in Canada[3,4] and worldwide.[5] Previous studies have generally involved static estimates comparing current or status quo concentrations with zero, background or counterfactual (a theoretical minimum based on the lowest level at which effects have been observed in epidemiological studies) concentrations. Estimates have also been produced of the projected health impacts of modelled changes in air pollution concentrations resulting from specific proposed policies or programs, using tools such as the United States Environmental Protection Agency's (USEPA) Environmental Benefits Mapping and Analysis Program (BenMAP)[6] or Health Canada's Air Quality Benefits Assessment Tool.[7] While these are informative in positioning air pollution as a public health priority and in weighing the costs and benefits of proposed policies, the background, counterfactual and projected concentrations are nonetheless hypothetical targets. In contrast, examination of the public health impacts of observed temporal changes in pollutant concentrations can be potentially informative with respect to both evaluating the effectiveness of past policies and programs to control emissions, as well as identifying priorities for new control strategies.

Until recently, air pollution exposure estimates in these studies have generally been restricted to areas where ground-based monitoring data are available. A substantial improvement in exposure assignment was realized in the 2010 Global Burden of Disease study, which employed globally comprehensive exposure estimates derived from monitoring data, chemical/meteorological air quality models and remote sensing.[5]

A variety of metrics have been employed to quantify impacts in previous studies, including number of deaths or morbidity outcomes,[3–5] life expectancy,[8] disability-adjusted life years (DALYs),[5] quality-adjusted life years (QALYs)[9] and monetary

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valuation.[3] Particularly with respect to mortality outcomes, there has been considerable debate as to which is the most meaningful metric that most accurately characterizes the true impact of air pollution on health.[10–12] Life expectancy has the advantage of capturing the effects of premature mortality on life span in a manner that is arguably more readily understood by a lay audience than QALYs and DALYs.

In this paper, we estimate the effects of actual observed changes in air pollution concentrations over an 11-year period, employ spatially comprehensive exposure estimates derived from satellite remote sensing and quantify the impacts on life expectancy, mortality and morbidity.

METHODS

Exposure assignment

Estimates of particulate matter of median aerodynamic diameter <2.5 μm ($PM_{2.5}$) were based on satellite remote sensing observations for the periods 1999–2001 and 2010–2012.[13] Decadal mean satellite-derived $PM_{2.5}$ concentrations were well correlated (R = 0.76, slope = 0.96) with data from ground-based monitoring across North America. Air pollution exposure surface grid cells were mapped to population by area weighting based on 478,780 dissemination blocks, and these were further weighted up to census divisions (n = 288) using population weighting by dissemination area. With respect to short-term exposures, changes in annual average exposure were assumed to equal the average change in daily exposure.

While the focus of this analysis is on the impacts of observed changes in $PM_{2.5}$ between 2000 and 2011, we also estimated the impacts of further reductions of $PM_{2.5}$ from 2011 levels to an estimated natural background concentration of 1.8 μg/m^3, which is based on a review of monitoring data from rural and remote monitoring sites during time periods classified as being influenced primarily by background air mass types.[4] This concentration is comparable in concept to the USEPA "policy-relevant background", defined as concentrations that would occur in the absence of anthropogenic emissions in continental North America.[14] It is also similar to the lowest concentrations observed in our data and to background concentrations employed in a recent US analysis, which ranged from 0.74 to 1.72 μg/m^3 by region.[15]

Estimation of health impacts

Change in frequency of health outcomes was estimated for 2011 as the product of the 2011 population, annual baseline rate of the health outcome and the proportional change in health outcome per specified change in $PM_{2.5}$ concentration. Counts of mortality and hospital admissions were obtained for each census division (CD) with the exception of Quebec, where these data were not available. Quebec mortality counts for each CD were derived by applying national age- and cause-specific rates to the population age distribution of individual CDs. For Quebec hospital admission counts, we employed age- and cause-specific provincial rates for Quebec, applied to the population age distribution of individual CDs. Rates were averaged over the three most recent years of available data to improve stability. Baseline rates of other health outcomes were applied uniformly to all CDs. Proportional change in health outcome was derived from linear, log relative risk or log odds ratio models as follows:

$$\pi_{\text{lin}} \cong \frac{\beta_{\text{lin}}}{\mu}\Delta x \qquad 1$$

$$\pi_{lrr} = e^{\beta_{lrr}\Delta x} - 1 \cong \pi_{lor} = e^{\beta_{lor}\Delta x} - 1 \qquad 2$$

where π_{lin}, π_{lrr} and π_{lor} are the proportional change and β_{lin}, β_{lrr} and β_{lor} are the literature-derived regression coefficients corresponding to linear, log relative risk and log odds ratio models respectively; μ is the mean baseline rate of the given outcome from the same source in the literature as $\beta_{\text{lin;}}$ and Δx is the change in $PM_{2.5}$ concentration.

Analyses of mortality and life expectancy employed cause-specific β values for mortality from ischemic heart disease, cerebrovascular disease, lung cancer and chronic obstructive pulmonary disease (COPD) for adults 25 years and over, in keeping with the approach employed in the Global Burden of Disease analysis.[5] These were derived from a meta-analysis of worldwide cohort studies.[16] As a sensitivity analysis, we also employed results for all internal cause mortality from a Canadian cohort study,[17] which was excluded from the meta-analysis because it lacked data on personal risk factors such as smoking. β values for other outcomes were based on individual studies. Values of π_{lin}, π_{lrr} and π_{lor} and their sources, as well as corresponding baseline rates of health outcomes and their sources, are summarized in Supplementary Table 1.

Methods for estimating changes in life expectancy have been described in detail elsewhere.[18] Briefly, we estimated changes in life expectancy in relation to $PM_{2.5}$ using standard life table methods such that, at each age group, cause- and age-specific mortality rates were modified by the proportional change in mortality associated with a specified change in $PM_{2.5}$. Changes in the probability of surviving to the next age group were propagated through the life table in order to estimate the change in life expectancy. We employed the 2009–2011 life table for Canada. As a sensitivity analysis, we used provincial life tables for Ontario, Manitoba, Saskatchewan, Alberta and British Columbia. Because we analyzed life expectancy impacts in relation to the excess risk from air pollution on COPD, cerebrovascular disease, ischemic heart disease and lung cancer mortality, we required mortality rates for these conditions by five-year age group to apply to the life table. These data were not available for Quebec, and province-specific counts by cause and five-year age group for smaller provinces were considered too small and likely to produce unstable rates.

Uncertainty

The analysis was conducted using the Air Quality Benefits Assessment Tool[7] in Microsoft Excel and the @Risk add-in. Monte Carlo simulations employing 10,000 iterations were used to propagate uncertainty in exposure response functions according to the input distributions shown in Supplementary Table 1. We report 95% confidence intervals (CIs) based on the 2.5$^{\text{th}}$ and 97.5$^{\text{th}}$ percentiles of the output distribution.

RESULTS

Maps of $PM_{2.5}$ concentration by 2006 census dissemination area are shown in Figures 1 and 2. The highest concentrations in 1999–2001 were observed in the southern prairie provinces as well as southern Ontario and Quebec. Between 1999–2001 and 2010–2012, substantial reductions were observed in eastern provinces, particularly in populous southern portions, and southern Alberta, while increases were observed elsewhere, particularly in the western provinces and territories (Supplementary Figure 1). The population-weighted average change in concentration across Canada was $-2.0\,\mu g/m^3$, representing a 23.4% reduction during this period. Population weighted average changes by province ranged from $-3.35\,\mu g/m^3$ (−37.0%) in Quebec to $+0.95\,\mu g/m^3$ (+14.7%) in Saskatchewan. Changes for all provinces and $PM_{2.5}$ concentrations in 2000 and 2011 for all CDs are provided in Supplementary Table 2.

The estimated health impacts of these changes are summarized in Table 1. An increase in life expectancy of 0.10 years (95% CI 0.03–0.23) was estimated, representing a 0.12% increase. The percentage change in other outcomes ranged from 0.13% for cardiac emergency room visits and hospital admissions to 3.55% for mortality. CDs with the greatest reductions in $PM_{2.5}$ and corresponding increases in life expectancy and those with the greatest increases in $PM_{2.5}$ and reductions in life expectancy are shown in Table 2. The top five reductions in $PM_{2.5}$ were observed in Quebec, representing about a 50%–60% reduction in concentrations and an estimated gain in life expectancy of about 1/3 of a year. In comparison, in Toronto there was a $4.7\,\mu g/m^3$ (33.8%) decrease in $PM_{2.5}$ associated with a gain in life expectancy of 0.23 years, and in Montreal there was a $4.0\,\mu g/m^3$ (37.1%) reduction in $PM_{2.5}$ associated with a gain in life expectancy of 0.20 years. The bottom five values were all in the prairies, where there were increases in $PM_{2.5}$ ranging from 50% to 80% with estimated losses in life expectancy of 0.14 to 0.20 years. The equivalent top and bottom five values for mortality impacts are shown in Table 3. Unlike life expectancy, these depend on both population and change in $PM_{2.5}$. Thus, for relatively large population centres with relatively large changes in $PM_{2.5}$, substantial reductions in the number of deaths were estimated. Again the top five CDs were in the east and the bottom five in the west. Increases in $PM_{2.5}$ and small numbers of increased deaths in the bottom five CDs may be more appropriately interpreted as a lack of improvement rather than an important deterioration in $PM_{2.5}$ concentrations and associated health impacts. Estimated life expectancy, mortality and morbidity impacts in all CDs are provided in Supplementary Tables 3–13.

Further reductions of $PM_{2.5}$ from 2011 levels (population weighted average $6.5\,\mu g/m^3$) to an estimated natural background concentration of $1.8\,\mu g/m^3$ were estimated to result in an additional gain in life expectancy of 0.27 years (95% CI 0.08–0.77, national population weighted average). Increases of up to 0.69 years (95% CI 0.20–2.11) were estimated in individual CDs. A reduction in the number of deaths of 5,600 (95% CI 1,800–14,000)

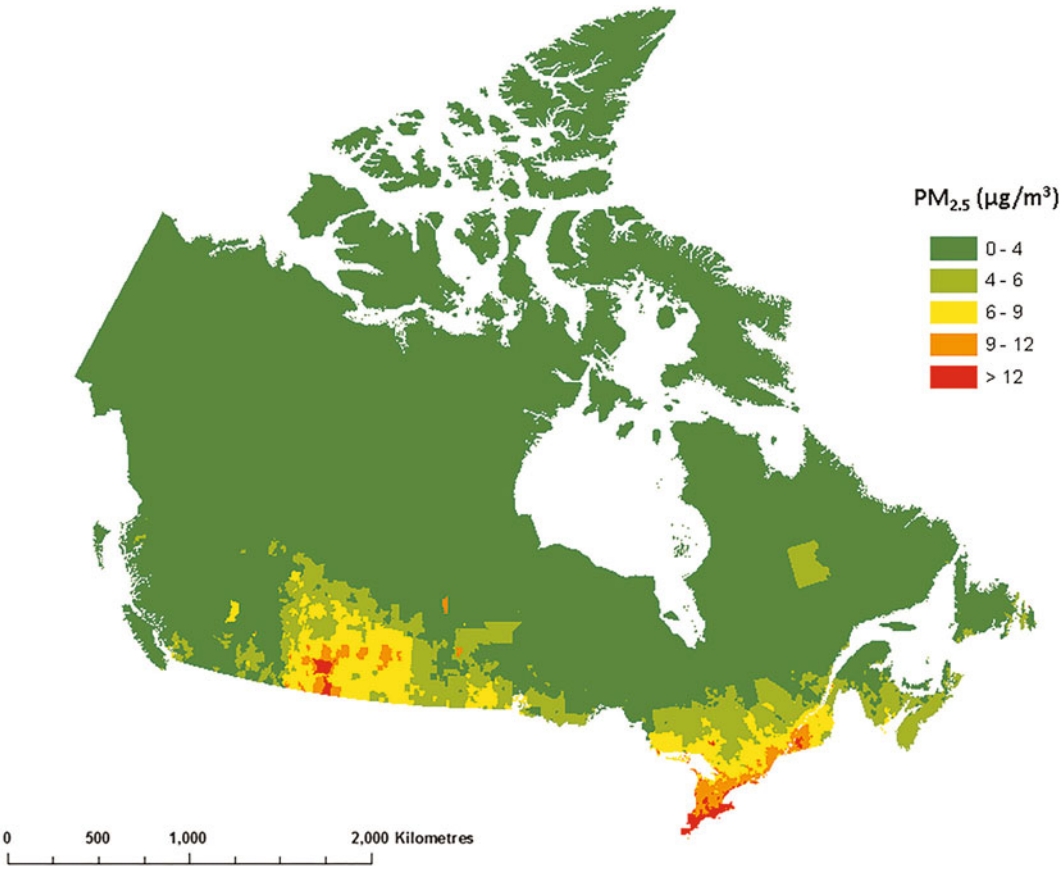

Figure 1. Estimated $PM_{2.5}$ concentration ($\mu g/m^3$) by census dissemination area, 1999–2001

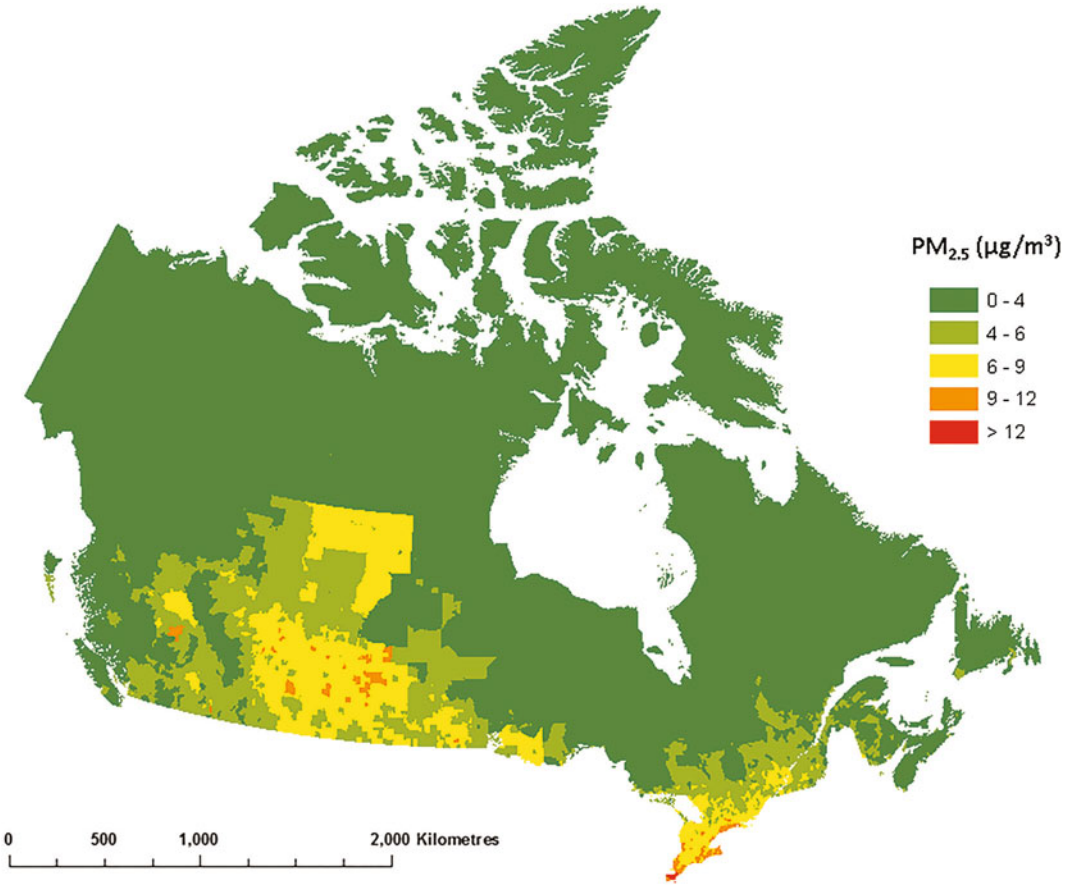

Figure 2. Estimated $PM_{2.5}$ concentration (µg/m^3) by census dissemination area, 2010–2012

Table 1. Estimated changes in health impacts associated with changes in $PM_{2.5}$ concentrations, Canada, 2000 to 2011

Outcome	Change*	95% CI*	Percentage	95% CI
Life expectancy (years)	0.10	0.03–0.23	0.1	0.0–0.3
Deaths†	−2500	−(780–6100)	−3.6	−(1.1–8.7)
Respiratory hospital admissions	−230	−(150–310)	−0.1	−(0.1–0.2)
Cardiac hospital admissions	−340	−(180–490)	−0.1	−(0.1–0.2)
Cardiac emergency room visits	−440	−(240–650)	−0.1	−(0.1–0.2)
Respiratory emergency room visits	−1200	−(770–1600)	−0.1	−(0.1–0.2)
Adult chronic bronchitis cases	−4000	−(0–7800)	−2.6	−(0.0–5.0)
Child acute bronchitis episodes	−19,000	−(0–41,000)	−1.7	−(0.0–3.8)
Asthma symptom days	−770,000	−(160,000–1,400,000)	−1.3	−(0.3–2.2)
Restricted activity days	−5,700,000	− (3,400,000–8,000,000)	−1.0	−(0.6–1.3)
Acute respiratory symptom days	−11,000,000	− (0–22,000,000)	−0.5	−(0.0–1.1)

* Values are rounded to two significant figures.
† From ischemic heart disease, cerebrovascular disease, lung cancer and chronic obstructive pulmonary disease.
CI = confidence interval.

or 8.1% was also estimated. A summary of life expectancy, mortality and morbidity impacts is provided in Supplementary Tables 14–16, and detailed results by CD are also provided in Supplementary Tables 17–27.

Estimates of life expectancy impacts were not sensitive to employing provincial vs. national life tables. However, impacts were sensitive to analysis of all internal causes of death rather than four selected causes. The estimated national population weighted average increase in life expectancy based on changes in $PM_{2.5}$ from 2000 to 2011 increased from 0.10 years (95% CI 0.03–0.23) to 0.16 years (95% CI 0.09–0.24) for all internal causes of death, and the estimated national reduction in mortality increased from 2,500 to 4,100 deaths. Estimates of the impacts of further reductions from 2011 concentrations to natural background also increased: gain in life expectancy from 0.27 years (95% CI 0.08–0.77, national population weighted average) to 0.40 years (95% CI 0.21–0.60), and national reduction in mortality from 5,600 to 9,500.

DISCUSSION

We estimated the impacts of changes in $PM_{2.5}$ concentrations in Canada between 2000 and 2011 using nationally comprehensive

Table 2. Census divisions (CDs) with the greatest reductions and increases in $PM_{2.5}$ concentrations, 2000 to 2011

Location	2011 population	Δ $PM_{2.5}$		Δ life expectancy (years) (95% CI)
		($\mu g/m^3$)	Percent	
Le Haut-Richelieu	114,565	−7.4	−58.9	0.34 (0.12–0.76)
Longueuil	406,878	−6.5	−49.2	0.31 (0.11–0.69)
La Vallée-du-Richelieu	111,776	−6.4	−50.6	0.30 (0.10–0.68)
Les Maskoutains	85,236	−6.3	−53.1	0.30 (0.10–0.67)
Rouville	32,895	−6.2	−52.4	0.29 (0.10–0.66)
Manitoba CD 20 (central west)	11,254	2.6	59.9	−0.14 –(0.04–0.38)
Saskatchewan CD 15 (Prince Albert*)	84,796	2.6	52.6	−0.15 –(0.05–0.39)
Saskatchewan CD 9 (Yorkton*)	37,373	2.8	58.4	−0.15 –(0.05–0.41)
Alberta CD 16 (Fort McMurray*)	61,017	2.9	81.6	−0.16 –(0.05–0.44)
Saskatchewan CD 14 (central east)	39,211	3.5	79.6	−0.20 –(0.06–0.54)

* Including the surrounding area.

Table 3. Census divisions (CDs) with the greatest estimated reductions and increases in mortality*, 2000 to 2011

Location	2011 population	Δ $PM_{2.5}$		Δ deaths (95% CI)	
		($\mu g/m^3$)	Percentage	Number†	Percentage
Toronto	2,773,516	−4.7	−33.8	−370 –(110–900)	−8.6 –(2.7–21.1)
Montreal	1,966,095	−4.0	−37.1	−360 –(110–890)	−7.2 –(2.2–17.8)
Longueil	406,878	−6.5	−49.2	−99 –(32–230)	−11.2 –(3.7–26.0)
Quebec	557,060	−3.2	−41.6	−85 –(26–210)	−5.9 –(1.8–14.7)
Ottawa	897,907	−3.1	−33.4	−84 –(27–210)	−5.8 –(1.8–14.2)
Alberta CD 10 (Lloydminster‡)	99,939	1.9	30.9	10 (3–28)	3.8 (1.0–11.0)
Manitoba CD 11 (Winnipeg)	692,572	0.4	5.4	11 (3–30)	0.8 (0.2–2.0)
Saskatchewan CD 15 (Prince Albert‡)	84,796	2.6	52.6	11 (3–31)	5.2 (1.5–14.8)
Alberta CD 8 (Red Deer‡)	201,604	2.1	38.0	16 (4–47)	4.3 (1.1–12.7)
Alberta CD 11 (Edmonton‡)	1,240,657	0.9	14.3	39 (11–110)	1.8 (0.5–5.0)

* From ischemic heart disease, cerebrovascular disease, lung cancer and chronic obstructive pulmonary disease.
† Values rounded to two significant figures.
‡ Including the surrounding area.

estimates of $PM_{2.5}$ derived from satellite remote sensing observations. A substantial population weighted average decrease in concentration of nearly 25% (2.0 $\mu g/m^3$) was observed over this period, with decreases as high as 7.4 $\mu g/m^3$ or nearly 60% in individual CDs, particularly in Quebec and Ontario. Increases of up to 3.5 $\mu g/m^3$ were also observed, particularly in the prairies. These changes were estimated to result in a national population weighted average increase in life expectancy of 0.10 years (up to approximately 1/3 of a year in the areas experiencing the greatest reductions in concentrations) and reductions in the frequency of mortality and morbidity of up to 3.6%. We also estimated that further reductions in $PM_{2.5}$ from 2011 concentrations to an estimated natural background level would result in an additional improvement in life expectancy of 0.27 years (up to 0.69 years in individual CDs) and a reduction in mortality of 5,600 or 8.1%.

The overall downward trend in $PM_{2.5}$ concentrations is consistent with trends in emissions of $PM_{2.5}$ precursors in both Canada and the US.[19,20] These may be attributable to numerous policy initiatives in both countries, including reduced sulphur content in fuels and controls on coal-fired power plant emissions, and to reduced economic activity after 2008.[21,22] Indeed, we found that the majority of the reduction in population weighted average $PM_{2.5}$ concentration across Canada occurred after 2006. It has also been suggested that localized upward trends in some western areas may be due to increased oil and gas development and wildfire activity.[21]

In a recent analysis based on regression of county-specific change in life expectancy and decreased $PM_{2.5}$ in 545 US counties, Correia et al. reported that between 2000 and 2007, life expectancy was estimated to have increased by 0.35 years per 10 $\mu g/m^3$ decline in $PM_{2.5}$,[8] which is proportionally comparable with our results. On average $PM_{2.5}$ concentrations had decreased by 1.6 $\mu g/m^3$ over this period in these US counties. The magnitude of estimated changes in life expectancy in relation to fine particle air pollution is comparable with that of other common risk factors. It has been estimated that a public smoking ban would increase life expectancy in Denmark by 0.33 years;[23] eliminating smoking would increase population average life expectancy in US states by 0.8–2.2 years;[24] and reduced serum cholesterol, diastolic blood pressure and body weight would increase US population average life expectancy by up to 0.8, 1.1 and 0.6 years respectively.[25]

With respect to mortality and morbidity impacts, in an extended analysis of the Harvard Six Cities Cohort Study, Laden et al. observed a decrease in $PM_{2.5}$ between 1979 and 1988 with an associated decrease in total mortality of 27% (95% CI 5–43%) per 10 $\mu g/m^3$, which is proportionally somewhat higher than our estimate.[26] Lim et al. recently estimated the global burden of disease associated with a variety of risk factors, including air pollution.[5] Over 3 million deaths worldwide were attributed to ambient particulate matter, representing 6% of deaths,[27] and ambient particulate matter ranked 9th of 67 risk factors worldwide and 14th in high-income North America on the basis

of attributable DALYs.[5] 7,200 attributable deaths were estimated for Canada,[28] which is higher than our estimate. However, this was based on a higher counterfactual exposure level and generally higher estimated $PM_{2.5}$ concentrations. In addition, $PM_{2.5}$ concentrations were randomly assigned to grid cells with modelled concentrations less than $10\,\mu g/m^3$ (which would include most of Canada), because the air pollution estimation model performed poorly relative to measured values at low concentrations.[29] This would tend to obscure findings in these areas. We also found that, not surprisingly, estimates of life expectancy and mortality impacts were substantially larger when based on an analysis of all internal causes of death rather than four selected causes, as employed in the Global Burden of Disease initiative. The latter approach was adopted because of potentially wide variation in underlying cause-specific mortality rates throughout the world, which could threaten the validity of estimating impacts based simply on all-cause mortality. While this approach facilitates international comparisons, it clearly represents a conservative estimate of impacts on mortality and life expectancy.

Health Canada has estimated that 4,200 deaths (5.7%) were attributable to long-term exposure to $PM_{2.5}$ in the eight largest Canadian cities where air pollution monitoring data were available (representing about 30% of the Canadian population).[4] This estimate was based on a change in exposure from a 3-year average for 1998–2000 (population weighted average $10.7\,\mu g/m^3$) to a natural background concentration of $1.8\,\mu g/m^3$. The Canadian Medical Association extrapolated monitoring data to all of Canada, estimating that 21,000 deaths could be attributed to $PM_{2.5}$ based on a change in exposure from 2008 levels to zero concentration.[3] Extrapolation from monitored to unmonitored areas is likely to overestimate concentrations in those areas, thus biasing impact estimates upwards.

There are varied opinions in the literature on the most appropriate metric to capture the public health impacts of air pollution. Attributable deaths in particular have been criticized because they do not account for the extent to which life is shortened, especially when based only on effects in studies of short-term exposure.[10–12] Life expectancy has been advanced as an alternative metric from the point of view of accounting for both risk and change in life span.[10–12] Disability-adjusted life expectancy and quality-adjusted life expectancy additionally account for change in health status and in principle can capture the effects of air pollution on morbidity. However, acute morbidity, including outcomes such as disease exacerbations resulting in emergency visits or hospital admissions, are not readily captured by these metrics because of their relatively short duration (days to weeks vs. years). In addition, DALYs and QALYs, while informative for decision scientists, are not intuitive for non-experts.

Strengths and limitations

The strengths of our study include estimation of the impacts of actual observed changes in air pollution concentrations over an 11-year period, use of spatially comprehensive exposure estimates derived from satellite remote sensing and quantification of impacts using life expectancy, as well as mortality and morbidity.

The limitations include uncertainty about whether the slope of concentration response functions we employed, which in some cases were based primarily on studies from the US or elsewhere at generally higher $PM_{2.5}$ concentrations, are applicable at the lower concentrations observed in Canada. However, a recent Canadian cohort study found that the magnitude of the risk was similar to that observed elsewhere, despite lower average $PM_{2.5}$ concentrations.[17] We also assumed that concentration–response functions were static over time. While several studies have attempted to discern trends in risk over time, the results have so far been inconsistent.[30–33]

CONCLUSIONS

A substantial population weighted average reduction in $PM_{2.5}$ of nearly 25% was observed between 2000 and 2011, with larger reductions in Quebec and Ontario, and increases in some locations in the prairies. At the national level, these were associated with estimated improvements in life expectancy and reductions in mortality and morbidity. Areas that failed to improve or worsened between 2000 and 2011 warrant additional scrutiny to attempt to identify options for reducing $PM_{2.5}$ concentrations.

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RÉSUMÉ

OBJECTIFS : Estimer les impacts sur la santé publique des changements dans la pollution de l'air en fines particules au Canada entre 2000 et 2011, en employant des estimations d'exposition complètes à l'échelle nationale et en quantifiant les impacts sur l'espérance de vie, la mortalité et la morbidité.

MÉTHODE : Nous avons employé des estimations d'exposition exhaustives dérivées de la télédétection satellitaire pour estimer les effets des changements réels observés dans les concentrations en fines matières particulaires (MP) de diamètre aérodynamique médian <2.5 µm ($MP_{2.5}$), entre 2000 et 2011. Nous avons estimé les changements dans l'espérance de vie à l'aide des méthodes standard des tables de survie, et les changements dans la fréquence des résultats sanitaires en fonction de la population, du niveau de référence des résultats sanitaires et du changement proportionnel dans les résultats sanitaires selon le changement spécifié de la concentration en $MP_{2.5}$.

RÉSULTATS : Une diminution moyenne des $MP_{2.5}$ de près de 25 % (2.0 µg/m^3), pondérée selon la population, a été observée entre 2000 et 2011. On estime que cela a entraîné une hausse moyenne nationale de l'espérance de vie, pondérée selon la population, de 0.10 an (intervalle de confiance de 95 % : 0.03–0.23; jusqu'à 0.34 an dans certains secteurs du recensement) et des baisses de fréquence de la mortalité et de la morbidité jusqu'à 3.6 %. Des augmentations maximales de 3.5 µg/m^3 des $MP_{2.5}$ ont été observées dans certains secteurs du recensement, en particulier dans les Prairies.

CONCLUSION : À l'échelle nationale, les changements dans les concentrations en $MP_{2.5}$ survenus entre 2000 et 2011 étaient associés à une amélioration estimative de l'espérance de vie moyenne nationale, pondérée selon la population, et à une baisse nette de la mortalité et de la morbidité. Les régions où la situation ne s'est pas améliorée ou s'est aggravée durant la période à l'étude devraient faire l'objet d'un examen approfondi afin de trouver des options pour réduire les concentrations en $MP_{2.5}$.

MOTS CLÉS : pollution de l'air; espérance de vie; mortalité; morbidité