Burden of Mortality Due to Ambient Fine Particulate Air Pollution (PM_{2.5}) in Interior and Northern BC

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ABSTRACT

Objectives: Our objective was to estimate the burden of mortality attributable to long-term exposure to ambient fine particulate matter (PM_{2.5}) among adults in two rural regions of British Columbia, the Interior and the North, in order to provide information for public health professionals setting health priorities.

Methods: We used the standard method to calculate the fraction of all-cause mortality among adults (≥30 years old) attributable to long-term exposure to ambient PM_{2.5} as described in the World Health Organization global burden of disease study. PM_{2.5} concentration was either locally measured using tapered element oscillating microbalance (TEOM) continuous monitors or estimated for unmonitored cities. For the base case, we used a conservative PM_{2.5} threshold (5.0 ug/m³); for sensitivity analysis, we set the threshold to estimate all mortality attributable to anthropogenic PM_{2.5} (3.1 ug/m³). We conducted sensitivity analysis for PM_{2.5} concentrations estimated in unmonitored cities.

Results and Conclusions: Mean annual PM_{2.5} concentration ranged from 3.1 to 7.4 ug/m³ across local health areas in Northern and Interior BC. Sixty percent of the adult population lived in monitored regions. PM_{2.5} was estimated to cause 0.20% of all-cause mortality among adults (16 deaths/year). Anthropogenic PM_{2.5} was estimated to cause 0.93% of all-cause mortality among adults (74 deaths/year). Estimates were sensitive to both PM_{2.5} threshold and estimated PM_{2.5} in unmonitored communities. This demonstrates that ambient PM_{2.5} air pollution does have an important mortality burden, even in a region with relatively low PM_{2.5} concentrations.

Key words: Air pollution; particulate matter; public health; British Columbia; Canada; mortality

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

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It is widely accepted that air pollution is detrimental to health. The relationship between mortality and short-term exposure to high concentrations of fine particulate matter and sulphur oxides was demonstrated in the Great London Smog in 1952,¹ and subsequently replicated in several natural experiments.^{2,3} Recent epidemiologic research suggests that long-term exposure to low-level air pollution may be associated with a greater population health burden than short-term exposure. Prospective longitudinal cohort studies have demonstrated that the risk of mortality from long-term exposure to levels of ambient fine particulate matter (PM_{2.5}) found in Western cities is up to ten times greater than the risk from short-term exposure to the same concentration increment,⁴⁻¹⁴ and that life expectancy improves as PM_{2.5} concentrations are reduced.¹⁵

Nevertheless, measures used to inform public health action and policies in Canada, such as the air quality health index¹⁶ and burden of disease estimates,¹⁷ rely on effect measures for short-term exposure. There is no information about burden of disease associated with long-term exposure to air pollution in rural British Columbia, despite major industrial sources in some communities corresponding to higher pollutant levels than those found in urban areas. Our analysis uses existing data to estimate the burden of mortality from long-term exposure to fine particulate air pollution in rural BC.

METHODS

Setting, population and exposure assessment

Interior Health Authority and Northern Health Authority are rural health authorities which make up the majority of the geographic area of British Columbia, but only contain 16% (n=733,285) and 6% (285,493) of the population, respectively.¹⁸ The major contributors to fine particulate air pollution vary by community and include traffic, industries, forest fires and wood-burning stoves.

Mortality data for 2005 to 2009 for each local health area (LHA) were obtained from BC Vital Statistics Agency. Outdoor ambient PM_{2.5} concentrations ([PM_{2.5}]) were measured by the BC Ministry of Environment using tapered element oscillating microbalance (TEOM) continuous monitors, corrected to compensate for losses of volatile material. Monitors closest to the main populations within

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Table 1. Population and Annual Mean PM_{2.5} Concentration in Interior and Northern BC, 2005-2009

Local Health Area	Population ≥30 Years Old Mean (%)	Annual Mean [PM _{2.5}]
Golden	4448 (0.94)	6.7
Grand Forks	6335 (1.3)	6.9
Kamloops	68,818 (15)	5.1
Central Okanagan	113,888 (24)	4.9
Nelson	16,115 (3.4)	4.4
Southern Okanagan	14,447 (3.1)	4.5
Vernon	42,611 (9.0)	5.7
Cariboo-Chilcotin	16,368 (3.5)	6.3
Unmonitored Interior Health	189,361 (40)	n/a
Burns Lake	4637 (2.8)	4.6
Fort Nelson	3309 (2.0)	3.4
Kitimat	6570 (3.9)	3.6
Prince George	57,311 (34)	7.2
Quesnel	14,832 (8.9)	7.0
Terrace	12,067 (7.2)	3.1
Unmonitored Northern Health	68,845 (41)	n/a

each monitored LHA were applied to the population of that LHA. We estimated the [PM_{2.5}] for unmonitored LHAs as the median of monitored communities in the corresponding health authority (HA) and conducted sensitivity analyses for this estimation.

Calculation of burden of mortality

Since the relationship between [PM_{2.5}] and mortality is widely accepted to be log-linear over the range of concentrations observed in BC,¹⁹ the mortality attributable to each increment of [PM_{2.5}] can be estimated using concentration response functions derived from longitudinal cohort studies. The largest such study using data collected by the American Cancer Society (ACS) followed 300,000 participants in 51 cities over 25 years¹³ and modeled relative risk estimates that were robust to analytic scrutiny.¹⁰

Biological plausibility for these effects has been demonstrated through pathophysiologic mechanisms including oxidative stress and inflammation in the lungs and leading to accelerated atherosclerosis and altered autonomic activity in the heart.¹⁹ Other air pollutants have been associated with mortality, however none have the weight of evidence that exists for PM_{2.5}.

We used the relative risk for all-cause mortality from the ACS study (1.06 per 10 ug/m³) and locally measured 5-year mean [PM_{2.5}] to calculate a relative risk for each local health area in the Interior and Northern Health Authorities (equation 1). The attributable fraction – the proportion of mortality from all causes that is due to air pollution – is derived from this relative risk (equation 2). For ambient air pollution, the attributable fraction and the population attributable fraction are identical since the entire population is exposed. The burden of mortality attributable to long-term exposure to PM_{2.5} is the product of the attributable fraction and measured all-cause mortality.

$$RR' = e^{\beta(C-C_0)} \text{ [equation 1]}$$

Where β is the regression coefficient, derived from $RR (\beta = [(\log RR)/10])$. C is the five-year mean observed [PM_{2.5}] and C_0 is the counterfactual concentration, the concentration below which no mortality effects are assumed.

In order to calculate the attributable fraction (AF), RR' was used in the classic attributable risk calculation:²⁰

$$AF = [1 - (1/RR')] \text{ [equation 2]}$$

Attributable mortality (AM) is the product of the attributable fraction and the five-year mean all-cause mortality.

Figure 1. Ambient PM_{2.5} monitoring network in Interior and Northern BC

$$AM = AF \times (\text{all-cause mortality}) \text{ [equation 3]}$$

We used mortality for adults 30 and older, since this corresponds to the population in the ACS study. Confidence intervals were calculated using the method of Greenland et al. (1987).²¹

Using this method, mortality is estimated only for PM_{2.5} concentrations above a reference concentration that is set *a priori* (counterfactual concentration, C_0). We set C_0 to the lowest concentration measured in the ACS (5 ug/m³) for the base case scenario, since the effects of PM_{2.5} have not been studied below this concentration.

Anthropogenic Burden of Mortality

In order to estimate mortality burden due to pollution (i.e., [PM_{2.5}] above natural levels), we set the counterfactual concentration to that found in the absence of emissions from human sources, by using the lowest observed concentration in BC (3.1 ug/m³, in Terrace). This is similar to the minimum [PM_{2.5}] used in the WHO burden of disease estimates (3 ug/m³) and the minimum background [PM_{2.5}] observed in the United States.^{22, p.1401}

We conducted a sensitivity analysis for the concentration estimated for unmonitored communities, by setting their concentration to background levels (3.1 ug/m³). This results in no mortality contribution from these communities.

RESULTS

The mean annual [PM_{2.5}] in Interior and Northern BC LHAs ranged from 3.1 to 7.4 ug/m³, with higher concentrations in cities with forest processing, particularly those in valleys where winter inversions trap pollutants (e.g., Prince George, Quesnel; Table 1). Most of the adult population (60%) lived in monitored LHAs.

In our base case, we estimate that fine particulate air pollution causes 0.1% (8 deaths/year) and 0.4% (6 deaths/year) of all-cause mortality among adults in Interior and Northern Health, respectively. For the entire region, an estimated 0.20% (16 deaths/year) of mortality was caused by fine particulate air pollution. The anthropogenic burden of mortality was much higher at 0.93% (74 deaths/year) for the region (Table 2). This demonstrates the sen-

Table 2. Estimates of Attributable Fraction and Annual Mortality Due to Air Pollution in Interior and Northern BC Under Different Scenarios (2005-2009)

Case	All Local Health Authorities		Monitored Local Health Authorities	
	Attributable Fraction (%)	Annual Mortality Count (95% CI)	Attributable Fraction (%)	Annual Mortality Count (95% CI)
Base case	0.20 (0.19, 0.21)	16 (15, 16)	0.20 (0.20, 0.21)	10 (15, 17)
Anthropogenic burden of mortality	0.93 (0.89, 0.97)	74 (71, 77)	0.84 (0.80, 0.87)	43 (41, 44)

sitivity of estimates to the counterfactual concentration, which was set to 5.0 ug/m³ in the base case and 3.1 ug/m³ for the anthropogenic burden of mortality.

DISCUSSION

We estimate that 0.20% of mortality among adults in Interior and Northern Health Authorities is due to fine particulate air pollution, corresponding to 8 and 6 deaths each year in each health authority respectively.

The attributable fraction for PM_{2.5} ranged from 0.20 to 0.93% depending on the case scenario. Our base case is lower than the World Health Organization global burden of disease estimates for mortality fraction attributable to urban PM_{2.5} pollution for this region of North America (0.42%),²² due to low pollution levels in BC relative to other regions of North America.

Our estimates are very sensitive to the counterfactual concentration – the PM_{2.5} concentration above which we attribute mortality. This method assumes a threshold concentration below which no mortality effects occur, set to 5 ug/m³ in the base case. The threshold model is debated among scientists. Mortality effects of ambient PM_{2.5} have been demonstrated down to the lowest measured levels (~5 ug/m³) and there is biological plausibility that they occur at even lower levels.¹⁹ Furthermore, the relationship between concentration and mortality is non-linear, with incremental effects greater at lower concentrations.²³ Therefore the true fraction of mortality due to air pollution is likely closer to our estimates for all anthropogenic PM_{2.5}, which is 0.93%.

Our estimates fall within a credible range relative to other attributable causes of mortality. The estimates of PM_{2.5} attributable mortality (0.20 to 0.93%) are less than global estimates for population attributable fraction (PAF) for mortality due to second-hand smoke exposure (1%)²⁴ and are a fraction of the population attributable fraction for mortality due to smoking estimated for Northern and Interior BC (19%).²⁵

This type of analysis has inherent uncertainties. The major limitations of the methodology relate to: the exposure assessment and the appropriateness of the effect estimate for the target population.

Exposure assessment is restricted because 1) [PM_{2.5}] from one central monitor was applied to the population of each LHA and 2) [PM_{2.5}] was not measured in all LHAs. This method does not account for individual variation in exposure due, for example, to spatial variations in PM_{2.5}, penetration indoors, and individual time spent outdoors. It is, however, comparable to the exposure assessment used in the cohort studies where the effect estimates were derived.²⁶ Since monitors were selected for proximity to major population centres, it would underestimate exposures for those living near pollution hotspots and overestimate exposures for those living outside the city. The overall effect on the estimates is uncertain. [PM_{2.5}] estimates for unmonitored LHAs have a large influence on attributable fraction, and since 40% of the population lives in unmonitored regions, this is a major limitation. [PM_{2.5}] in moni-

tored sites may not be representative of those in unmonitored ones since, for example, monitors may be deliberately sited near sources of emissions. PM_{2.5} measurement by TEOM underestimates [PM_{2.5}] at lower temperatures, causing a conservative bias in our estimates. Our exposure estimates are for current exposure, however, the current burden is due to past exposure which was higher in this BC region. However, a method to incorporate past exposures into burden of disease estimations has not been established.²²

Effect estimates are never completely transferable from one population to another; however, suitability can be assessed based on 1) [PM_{2.5}] and 2) population. Differences between the physico-chemical properties of PM_{2.5} between the 51 cities in the ACS and these BC sites are uncertain. Furthermore, even if these differences were quantified, a method to apply this information to burden of disease estimates has not been developed. Our sites included small cities and rural towns, where the main pollution sources include forest-related industry, forest fires, wood-burning stoves, and traffic. Although ACS included smaller cities with similar pollution profiles, the ideal effect estimate for our estimations would be derived from a cohort study with emissions profiles and population characteristics better matched to BC; however, these studies have not occurred. The age range of the BC population was matched to that of the ACS study and the populations had similar smoking rates (21-26% vs. 22%); however, population susceptibility to air pollution may differ between these groups. Therefore the ACS effect estimate is the most suitable, albeit imperfect, effect estimate available. It is important to note that effect estimates are remarkably similar across cohort studies across different populations in North America,^{11,13,27,28} therefore the contribution of these uncertainties is likely to be small.

In conclusion, the findings of this study demonstrate that fine particulate air pollution does have an important mortality burden in a region of Canada with relatively low pollution levels. The magnitude of health impact is a critical piece of information that can be considered along with other factors (e.g., preventability, public acceptability of interventions) when assessing public health priorities.

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RÉSUMÉ

Objectifs : Estimer le fardeau de la mortalité attribuable à l'exposition à long terme aux particules fines dans l'air ambiant (PM_{2.5}) chez les adultes de deux régions de la Colombie-Britannique, l'Intérieur et le Nord, afin de donner de l'information aux professionnels de santé publique qui établissent les priorités de santé.

Méthode : Nous avons utilisé la méthode standard pour calculer la fraction de la mortalité toutes causes confondues chez les adultes (≥30 ans) attribuable à l'exposition à long terme aux PM_{2.5} dans l'air ambiant comme décrite dans l'étude de l'Organisation mondiale de la Santé sur la charge mondiale de morbidité. La concentration en PM_{2.5} a été soit mesurée localement à l'aide d'échantillonneurs en continu à microbalance à oscillation, soit estimée (pour les villes sans relevé local). Pour le scénario de base, nous avons utilisé un seuil modeste de PM_{2.5} (5,0 µg/m³); pour l'analyse de sensibilité, nous avons fixé le seuil de manière à estimer la mortalité toutes causes confondues attribuable aux PM_{2.5} d'origine anthropique (3,1 µg/m³). Nous avons réalisé des analyses de sensibilité pour les concentrations estimatives en PM_{2.5} dans les villes sans relevé local.

Résultats et conclusions : La concentration annuelle moyenne en PM_{2.5} s'échelonnait de 3,1 à 7,4 µg/m³ dans les régions sanitaires locales du Nord et de l'Intérieur de la C.-B. Soixante pour cent de la population adulte vivait dans des régions avec relevé local. Nous estimons que les PM_{2.5} et les PM d'origine anthropique ont causé respectivement 0,20 % et 0,93 % de la mortalité toutes causes confondues chez les adultes (respectivement 16 et 74 décès/an). Les estimations étaient sensibles à la fois au seuil des PM_{2.5} et au seuil estimatif dans les communautés sans relevé local. Cela démontre que la pollution de l'air ambiant par les PM_{2.5} contribue à la charge de mortalité, même dans les régions à concentration relativement faible en PM_{2.5}.

Mots clés : pollution de l'air; particules; santé publique; Colombie-Britannique; Canada; mortalité