



# Data Sources and Methods: Air Health Indicator - Ozone and Fine Particulate Matter

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# 1 Introduction

The Air Health Indicator (AHI) is part of the Canadian Environmental Sustainability Indicators (CESI) (http://www.ec.gc.ca/indicateurs-indicators) program that provides data and information to track Canada's performance on key environmental sustainability issues.

Canadians are regularly exposed to air pollution from outdoor sources such as transportation and industrial activities. This exposure can lead to the development of chronic lung disease, heart attacks and strokes, and to the onset or worsening of breathing difficulty. These health effects contribute to lost productivity, doctors' and emergency room visits, hospital admissions and mortality. The AHI has been developed as a tool to monitor the impacts of outdoor air pollution exposure over time on the health of Canadians.

# 2 Description and rationale of the Air Health Indicator

The AHI monitors the percentage of all cardiopulmonary mortalities (deaths from heart- and lung-related diseases) that can be attributed to exposure to two important outdoor air pollutants: ground-level ozone and fine particulate matter ( $PM_{2.5}$ ).

# 3 Data

Canadian communities for which the ground-level ozone and fine particulate matter (PM<sub>2.5</sub>) concentrations were used for the national Air Quality Indicator of CESI were considered. The AHI is based on the criteria of having a reasonably complete time series of pollution and weather measurements, and enough daily mortality.

For each community there were three types of data used for the AHI: daily numbers of causespecific deaths, air pollution concentrations, and potential confounders to the mortality-air pollution association.

#### 3.1 Data source

#### Daily numbers of cause-specific deaths

The daily numbers of cause-specific deaths (non-accidental mortality data) were obtained from the national mortality database (Vital Statistics Database-Death 2004) maintained by Statistics Canada. Based on the International Classification of Diseases (ICD), the mortality data included only deaths from internal causes (ICD-9 code < 800 and ICD-10 code A00-R00), excluding external causes such as injuries. Regarding cause-specific deaths, in particular, we were interested in cardiopulmonary mortality related to the circulatory or respiratory system. For this specification, our mortality data were categorized into a cardiopulmonary group (ICD-10 code between I20-I50 and J10-J67). The cardiopulmonary mortality data were extracted by Statistics Canada for a specified census division only where the census division of residence was the same as the census division of death occurrence.

#### Air pollution concentrations

The daily ozone and PM<sub>2.5</sub> (the latter measured by the tapered element oscillating microbalance method or TEOM) concentration data were obtained from the National Air Pollution Surveillance (NAPS) Network (http://www.ec.gc.ca/rnspa-naps/Default.asp?lang=En&n=5C0D33CF-1) operated by Environment Canada. Established in 1969, NAPS provides accurate and long-term air quality data of a uniform standard across Canada to monitor the quality of ambient (outdoor) air in populated regions by specific procedures for the selection and positioning of monitoring stations. For each

NAPS monitoring station, the daily average concentration for a certain day was calculated only if at least 75% of 24 hourly concentrations for that day (i.e. at least 18 hourly concentrations) were available. Otherwise, it was recorded as missing. For each census division, the daily average concentration was averaged over monitoring stations if there were 2 or more stations located in that census division. For the metric of air pollutions, the daily 8-hour maximum was selected for ozone and the daily mean for PM<sub>2.5</sub>.

#### Potential confounders to the mortality-air pollution association

As for potential confounding variables to the exposure-mortality association, three factors were considered: time; temperature; and indicators for days of the week. Calendar time is included to control both temporal and seasonal variations. Daily temperature controls for the short-term effect of weather on daily mortality; and day of the week accounts for mortality that varies by day of the week. Specifically, to account for the weather effect, daily mean temperature data were obtained from the National Climate Data and Information Archive

(http://climate.weatheroffice.gc.ca/Welcome\_e.html) of Environment Canada. As for lifestyle factors such as smoking or cholesterol in the community, they do not vary meaningfully from day to day and thus can be ignored as confounders.

#### 3.2 Spatial coverage

Twenty Canadian communities (Saint John, Québec, Montréal, Ottawa, York, Toronto, Peel, Oakville, Hamilton, Niagara Falls, Kitchener, Windsor, Sarnia, Sault Ste. Marie, Winnipeg, Regina, Saskatoon, Calgary, Edmonton, and Vancouver) were selected for ozone. Eighteen communities (Saint John, Québec, Montréal, Ottawa, Toronto, Peel, Oakville, Hamilton, Niagara Falls, London, Windsor, Sarnia, Waterloo, Winnipeg, Regina, Calgary, Edmonton, and Vancouver) were selected for PM<sub>2.5</sub>.

Each community's geographic boundaries were defined by the census division associated with the city.

#### 3.3 Temporal coverage

Yearly data for the years 1990 to 2008 were used for ozone and yearly data for the years 2000 to 2008 were used for  $PM_{2.5}$ .

#### 3.4 Data timeliness

Mortality data are difficult to obtain and are a few years behind the other data. Raw 2007 data are now available but only the 2004 data were available in the correct format and details for use with the AHI. Consequently, the years 2005 to 2008 were approximated from the average of national annual risk (mortality data).

### 4 Methods

To identify the cardiopulmonary mortality-air pollution association, a Bayesian hierarchical model was applied. First, the city-specific risk for each city (census division) was estimated. Annual city-specific risks of cardiopulmonary mortality due to ozone or  $PM_{2.5}$  were estimated by a generalized Poisson model for each census division. A generalized additive over-dispersed Poisson regression model was applied to the daily mortality counts. Second, the national risk for each year was estimated by pooling over the city-specific risks. Third, testing was done of the resulting data for annual national attributable risks (due to ozone or  $PM_{2.5}$ ) to determine whether a time trend could be detected.

The AHI is based on two temporal functions: annual air pollutant concentrations and annual mortality risks of that air pollutant at the national level. The annual air pollutant concentrations were obtained from CESI's national Air Quality Indicator data (http://www.ec.gc.ca/indicateurs-indicators/default.asp?lang=en&n=B1385495-1#aq\_chart1\_o3\_en), and the annual mortality risks were estimated in the second stage above.

The annual mortality risk estimates showed no time trend. It was therefore possible to assert that the annual mortality risk was constant over the periods analysed. The average of 15 annual national risk estimates for ozone (0.012 per 10 ppb for 1990 to 2004) and the average of 5 annual national risk estimates for  $PM_{2.5}$  (0.032 per 10 ppb for 2000 to 2004) were used. It was assumed that this constancy persisted and each of these averages were projected for the years 2005 to 2008, for which yearly risk estimates were not yet available due to the unavailability of mortality data for these years. The overall annual risk was derived from the product of annual air pollutant concentrations and the average of annual mortality risk estimates of ozone and  $PM_{2.5}$ . Finally, to detect a time trend, the Sen's test, a non-parametric linear time-trend test, was applied. There were no time trends found in ozone and  $PM_{2.5}$  risks.

A time-trend analysis was also performed on the AHI using the Sen's test. A trend was detected at the 95% confidence interval for the ozone AHI but not for  $PM_{2.5}$ .

### 5 Caveats and limitations

The AHI is an indicator in development. It is now solely concentrating on the mortality risk aspect from cardiopulmonary diseases as a whole for ozone and fine particulate matter in communities where we had the best available data.

Future AHI work will include assessments of the potential reasons behind changes in mortality and morbidity attributable to air pollutant exposure, including factors that influence Canadians' health risk, such as demographics (e.g., age, sex), health and socioeconomic status. Incorporating these variables, together with linkages to region, province and municipality, will help to pinpoint the most vulnerable population groups—e.g., the elderly, the very young, and those with existing respiratory or cardiac conditions (or a predisposition to same).

### 6 References and further reading

Huang Y *et al.* (2005) Bayesian hierarchical distributed lag models for summer ozone exposure and cardio-respiratory mortality. *Environmetrics.* 16:547-562.

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