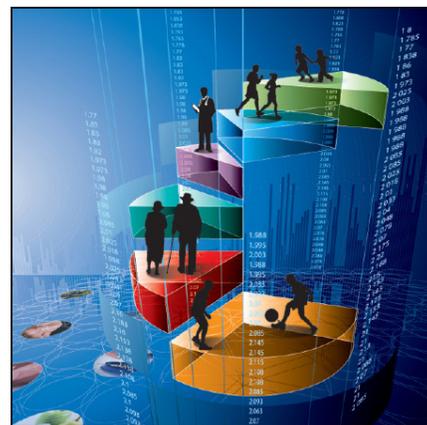


Health Reports

Exposure to industrial air pollutant emissions and lung function in children: Canadian Health Measures Survey, 2007 to 2011

by Suzy L. Wong, Allan L. Coates and Teresa To

Release date: February 17, 2016



Statistics
Canada

Statistique
Canada

Canada

How to obtain more information

For information about this product or the wide range of services and data available from Statistics Canada, visit our website, www.statcan.gc.ca.

You can also contact us by

email at STATCAN.infostats-infostats.STATCAN@canada.ca

telephone, from Monday to Friday, 8:30 a.m. to 4:30 p.m., at the following toll-free numbers:

- Statistical Information Service 1-800-263-1136
- National telecommunications device for the hearing impaired 1-800-363-7629
- Fax line 1-877-287-4369

Depository Services Program

- Inquiries line 1-800-635-7943
- Fax line 1-800-565-7757

Standards of service to the public

Statistics Canada is committed to serving its clients in a prompt, reliable and courteous manner. To this end, Statistics Canada has developed standards of service that its employees observe. To obtain a copy of these service standards, please contact Statistics Canada toll-free at 1-800-263-1136. The service standards are also published on www.statcan.gc.ca under “Contact us” > “Standards of service to the public.”

Note of appreciation

Canada owes the success of its statistical system to a long-standing partnership between Statistics Canada, the citizens of Canada, its businesses, governments and other institutions. Accurate and timely statistical information could not be produced without their continued co-operation and goodwill.

Standard table symbols

The following symbols are used in Statistics Canada publications:

- . not available for any reference period
- .. not available for a specific reference period
- ... not applicable
- 0 true zero or a value rounded to zero
- 0^s value rounded to 0 (zero) where there is a meaningful distinction between true zero and the value that was rounded
- ^P preliminary
- ^r revised
- X suppressed to meet the confidentiality requirements of the *Statistics Act*
- ^E use with caution
- F too unreliable to be published
- * significantly different from reference category ($p < 0.05$)

Published by authority of the Minister responsible for Statistics Canada

© Minister of Industry, 2016

All rights reserved. Use of this publication is governed by the Statistics Canada [Open Licence Agreement](#).

An HTML version is also available.

Cette publication est aussi disponible en français.

Exposure to industrial air pollutant emissions and lung function in children: Canadian Health Measures Survey, 2007 to 2011

by Suzy L. Wong, Allan L. Coates and Teresa To

Abstract

Background: Long-term exposure to ambient air pollution has been associated with adverse effects on children's lung function. Few studies have examined lung function in relation to industrial emissions of air pollutants.

Data and methods: This cross-sectional study was based on 2,833 respondents aged 6 to 18 for whom spirometry data were collected by the Canadian Health Measures Survey, 2007 to 2011. The weighted sum of industrial air emissions of nitrogen oxides (NO_x) and fine particulate matter (PM_{2.5}) within 25 km of the respondent's residence was derived using National Pollutant Release Inventory data. Multivariate linear regression was used to examine the relationship between NO_x and PM_{2.5} emissions and forced vital capacity (FVC), the forced expiratory volume in 1 sec (FEV₁), and the ratio of the two (FEV₁/FVC).

Results: Industrial air emissions of NO_x were not significantly associated with lung function among males or females. Emissions of PM_{2.5} were negatively associated with FEV₁ and FEV₁/FVC, but not FVC, among males. PM_{2.5} was not significantly related to lung function among females.

Interpretation: The associations that emerged between lung function and industrial emissions of PM_{2.5} among males were consistent with airway obstruction. Further research is warranted to investigate the gender differences observed in this study.

Keywords: Ambient air pollution, particulate matter, nitrogen dioxide, National Pollutant Release Inventory, outdoor air pollution

Lung function is an objective measure of respiratory health and a predictor of cardiorespiratory morbidity and mortality.¹ Long-term exposure to ambient air pollution has been associated with adverse effects on children's lung function.²⁻⁴ These pollutants include, but are not limited to, nitrogen dioxide (NO₂) and fine (less than 25 micrometres in diameter) particulate matter (PM_{2.5}).

Human production of NO₂ and PM_{2.5} is primarily from combustion, notably, from vehicles and industrial processes. Numerous studies have examined lung function in relation to long-term exposure to ambient levels of these air pollutants or to traffic emissions, but few have examined industrial emissions.² Of those studies, some have observed reductions in lung function among children living near industrial facilities,^{5,6} but others have not.⁷

This article examines the relationship between long-term exposure to industrial air emissions of nitrogen oxides (NO_x) and PM_{2.5} and lung function in a nationally representative sample of Canadian children and youth aged 6 to 18. The data are from the Canadian Health Measures Survey and the National Pollutant Release Inventory.

Methods

Canadian Health Measures Survey

The Canadian Health Measures Survey (CHMS) is an ongoing survey designed to provide direct health measures at the national level for people living in private households. Cycle 1 was conducted from March 2007 through February 2009, and collected information from respondents aged 6 to 79. Cycle 2 took place from August 2009 through November 2011, and collected

data from respondents aged 3 to 79. Residents of First Nations Reserves or other Aboriginal settlements, institutions and some remote regions, and full-time members of the Canadian Forces were excluded. More than 96% of the population was represented. Ethics approval for the CHMS was obtained from Health Canada's Research Ethics Board.

The CHMS involves an in-home interview during which a questionnaire is administered. This is followed by a visit to a mobile examination centre (MEC) where physical measures (including spirometry to assess lung function) are taken, and additional questionnaires are administered. Participation is voluntary; respondents can opt out or refuse any part of the survey at any time. Written informed consent is obtained from respondents aged 14 or older. For younger children, a parent or legal guardian provides written consent, in addition to written assent from the child (where possible). After adjustments for the sampling strategy, the combined response rate for cycles 1 and 2 was 53.5%. Details about the survey, including the sampling strategy, are available at www.statcan.gc.ca/chms.

Spirometry was performed using a Fleisch pneumotachograph type spirometer (KoKo™, nSpire Longmont Co, USA) in accordance with the testing procedure in the revised joint American Thoracic Society/European Respiratory Society guidelines.⁸ Technician training for the MEC was the same for all operators; ongoing quality control assessment was done manually and electronically. Only spirometry tracings meeting international standards⁸ were accepted at the time of testing. All tracings were reviewed by a qualified pulmonary function technician who made the final decision on acceptance or rejection of tracings from the field.⁹ The lung function parameters examined in this study are the conventional spirometric indices used to

detect impairment¹⁰: forced vital capacity (FVC), which measures the total volume exhaled after a maximum inspiration; 1-sec forced expiratory volume, which measures the maximum volume that can be exhaled within 1 sec (FEV_1); and the ratio of the two— FEV_1/FVC .

Respondents were not eligible for spirometry if they were younger than 6, older than 79, or 27 or more weeks' pregnant; had a heart attack or major surgery in the chest or abdomen in the previous three months or eye surgery in the past six weeks; reported taking medication for tuberculosis; had an acute respiratory tract infection (for example, cold, flu); or had other conditions that could make spirometry unsafe for the respondent⁸ or yield results that were unreliable or unrepresentative of their usual lung function.

Respondents were classified as white or non-white based on their self-reported cultural or racial background. Education was classified into three categories (less than secondary school graduation, secondary school graduation or some postsecondary education, and postsecondary graduation) based on the highest level attained by a member of the respondent's household. Total household income was classified into three categories (low, middle, high), adjusted for household size. Respondents were considered to have a respiratory condition/symptom if they replied "yes" to one or more questions about diagnosed chronic conditions, wheezing, coughing, phlegm, and shortness of breath (Text table 1). Those who replied "no" to each question were classified as not having a respiratory condition/symptom.

Respondents were "regularly" exposed to second-hand smoke if they reported that they were exposed to second-hand smoke in their home every day or almost every day. To determine maternal smoking, mothers of respondents younger than age 12 were asked if they had smoked while pregnant with the respondent. Respondents

had "a history of smoking" if they met one or more of the following conditions: reported smoking 100 or more cigarettes in their lifetime; reported currently being a daily or occasional smoker; or their cotinine concentration was more than 50ng/mL.¹¹ Free cotinine was measured from a spot midstream urine sample collected at the MEC and sent to the testing laboratory at the Institut national de santé publique du Québec (accredited under ISO 17025).

Age was calculated by subtracting the self-reported birth date from the clinic examination date. Standing height was measured to the nearest 0.1 cm using a ProScale M150 digital stadiometer (Accurate Technology Inc., Fletcher, USA). Weight was measured to the nearest 0.01 kg using a Mettler Toledo digital scale. Respondents were classified as obese or not obese according to World Health Organization criteria based on age, sex and body mass index (kg/m^2).¹²

Ambient temperature at the hour of data collection at the MEC was obtained from Environment Canada's National Climate and Data Information Archive (www.climate.weatheroffice.gc.ca). The concentration of NO_2 and $PM_{2.5}$ at the hour of data collection was obtained from the air monitoring station of the National Air Pollution Surveillance Network (www.ec.gc.ca/rnsps-naps) nearest to the MEC that recorded hourly measurements.

National Pollutant Release Inventory

The National Pollutant Release Inventory (NPRI) contains data on industrial emissions of air pollutants. The NPRI is Canada's legislated, publicly accessible inventory of pollutant releases into air, water and land, disposals, and transfers for recycling. It compiles information reported by facilities and published by Environment Canada under the authority of the *Canadian Environmental Protection Act, 1999*. In 2011, more than 8,000 facilities reported to the NPRI on more than 300 listed substances, including NO_x and $PM_{2.5}$. NO_x includes nitric oxide (NO) and NO_2 . Nitrous oxide (N_2O) was not included when calculating NO_x releases. Since NO_x is a mixture, both NO and NO_2 were expressed on an NO_2 -equivalent basis before individual quantities were combined for reporting the total NO_x release.¹³ Facilities are required to report releases of these substances if they exceed the specified reporting threshold of 20 tonnes for NO_x and 0.3 tonnes for $PM_{2.5}$. Details are available at: <http://www.ec.gc.ca/inrp-npri/>

Facility types were identified by North American Industry Codes in the NPRI. Of the 5,763 facilities required to report $PM_{2.5}$ emissions that were included in this study (within 25 km of CHMS respondents' residences), 62% were Manufacturing, 19% were Agriculture,

Text table 1

Lung function questions from Canadian Health Measures Survey used to define "respiratory condition or symptom"

Question	Age range
We are interested in "long-term conditions" which are expected to last or have already lasted 6 months or more and that have been diagnosed by a health professional.	
Do you have asthma? (yes/no)	6 to 18
Do you have chronic bronchitis? (yes/no)	6 to 18
Has your child had wheezing or whistling in the chest in the last 12 months? (yes/no)	6 to 12
In the last 12 months, has your child had a dry cough at night, apart from a cough associated with a cold or a chest infection? (yes/no)	6 to 12
Do you cough regularly? (yes/no)	13 to 18
Do you cough up phlegm regularly? (yes/no)	13 to 18
During the past year, have you had a cough where you brought up phlegm that lasted 3 months or more? (yes/no)	6 to 18
Do even simple chores make you short of breath? (yes/no)	6 to 18

**Exposure to industrial air pollutant emissions and lung function in children:
Canadian Health Measures Survey, 2007 to 2011 • Research Article**

Forestry, Fishing and Hunting, and 5% were Utilities. The largest numbers of facilities were Oil and Gas Extraction (n = 698), followed by Non-Metallic Mineral Mining and Quarrying (n = 375), Cement and Concrete Product Manufacturing (n = 346), Petroleum and Coal Product Manufacturing (n = 281), and Electric Power Generation, Transmission and Distribution (n = 169). Of the 3,776 facilities required to report NO_x emissions that were included in this study, 57% were Mining, Quarrying, and Oil and Gas Extraction, 35% were Manufacturing, and 2% were Transportation and Warehousing. The largest numbers of facilities were Oil and Gas Extraction (n = 1,802), Electric Power Generation, Transmission and Distribution (n = 188), Basic Chemical Manufacturing (n = 144), Pulp, Paper and Paperboard Mills (n = 140), and Water, Sewage and Other Systems (n = 84).

Annual total air emissions of NO_x and PM_{2.5} for each facility for each year from 2007 to 2011, and their geographic coordinates were obtained from the NPRI database. For each pollutant, annual exposure from emissions was calculated for industrial sites within a radius of 25 km of respondents' residences. The geographic co-ordinates of their residences were determined from their six-digit postal code and PCCF+ software.¹⁴ For each respondent, emissions

from industrial sites within the specified radius were weighted [weight = exp(-0.5*(d/25)²); d = distance from respondent's residence to industrial site]¹⁵ and then summed. Respondents were assigned the annual exposure for the calendar year in which they participated in the MEC component of the CHMS. Year-to-year emissions were highly correlated (Table 1).

Statistical analysis

Respondents were excluded from the analysis if they were not aged 6 to 18, or if their spirometry measures were of insufficient quality. Lung function reference equations differ by ethnic group, but sample sizes were too small to enable analysis of ethnicity other than white. Therefore, respondents were excluded if they were not white. This resulted in a final sample of 2,833: 1,429 (50.4%) males and 1,404 (49.6%) females.

Almost all (2,691 or 95%) of the 2,833 respondents had emissions of both NO_x and PM_{2.5} within 25 km of their residence; 60 had only emissions of PM_{2.5} within 25 km of their residence; and 82 did not have emissions of NO_x or PM_{2.5} within that distance.

Descriptive statistics were calculated, overall and by sex. Univariate linear regressions were performed to identify significant associations between lung function parameters and industrial air emissions of PM_{2.5} and NO_x. Lung

function parameters were modelled as percent predicted based on the Global Lung Initiative prediction equations.¹⁰ Separate analyses were performed for males and females.

For lung function parameters significantly associated with industrial air emissions (p < 0.05), multivariate linear regressions were performed to control for potential confounders. There were five nested models for each lung function parameter. Model 1 was the unadjusted model; Model 2 added respiratory condition/symptom; Model 3 added household income; Model 4 added short-term PM_{2.5}; and Model 5 added age.

Other potential confounders were: education, regular exposure to second-hand smoke in the home, maternal smoking while pregnant with the respondent, height, and obesity. However, because univariate linear regressions showed that they were not associated with the lung function parameters at the 0.10 level, they were not included in the adjusted models. Univariate linear regressions with lung function parameters showed that respiratory condition/symptom had the lowest p-value, followed by household income, short-term PM_{2.5}, and age; these variables were added to the nested models accordingly.

All estimates were based on weighted data. Survey weights for combining cycles 1 and 2 were used. Statistical analyses were performed with SAS and SUDAAN software. Standard errors, coefficients of variation, and 95% confidence intervals were calculated with the bootstrap technique.^{16,17} The number of degrees of freedom was specified as 24 to account for the CHMS sample design.¹⁸

Table 1
Correlation matrix of year-to-year emissions for sum of industrial air emissions of nitrogen oxides and fine particulate matter within 25-km radius of residence of 2007 to 2011 Canadian Health Measures Survey respondents aged 6 to 18

Industrial air emission/Year	Year				
	2007	2008	2009	2010	2011
Nitrogen oxides					
2007	...	0.99	0.94	0.95	0.90
2008	0.99	...	0.96	0.97	0.91
2009	0.94	0.96	...	0.99	0.97
2010	0.95	0.97	0.99	...	0.97
2011	0.90	0.91	0.97	0.97	...
Fine particulate matter					
2007	...	0.92	0.88	0.87	0.79
2008	0.92	...	0.95	0.93	0.84
2009	0.88	0.95	...	0.96	0.81
2010	0.87	0.93	0.96	...	0.83
2011	0.79	0.84	0.81	0.83	...

... not applicable

p < 0.0001 for all correlations

Sources: National Pollutant Release Inventory; 2007 to 2009 and 2009 to 2011 Canadian Health Measures Survey, combined.

Results

Mean percent predicted lung function and industrial air emissions values, overall and by sex, are shown in Table 2.

Results of the unadjusted and adjusted regression models examining the association between industrial air emissions and lung function parameters are shown in Table 3. Emissions of NO_x were not significantly associated with lung func-

Exposure to industrial air pollutant emissions and lung function in children: Canadian Health Measures Survey, 2007 to 2011 • Research Article

tion for males or females. By contrast, for males, emissions of PM_{2.5} were significantly associated with FEV₁ and FEV₁/FVC, but not with FVC. For females,

industrial air emissions of PM_{2.5} were not associated with lung function.

The association between industrial air emissions of PM_{2.5} and FEV₁ and FEV₁/FVC among males remained significant when adjusting for respiratory condition/symptom, household income, short-term PM_{2.5} levels, and age. An increase of 190 tonnes of industrial air emissions within 25 km of residence was associated with a 1% reduction in percent predicted FEV₁; an increase of 370 tonnes was associated with a 1% reduction in percent predicted FEV₁/FVC.

Table 2

Mean percent predicted lung function and mean amount of industrial air emissions within 25 km of residence, by sex, household population aged 6 to 18, 2007 to 2011

	Total			Males			Females		
	Mean	95% confidence interval		Mean	95% confidence interval		Mean	95% confidence interval	
		from	to		from	to		from	to
Percent predicted lung function parameters[†]									
FVC	103.07	102.27	103.87	103.21	102.30	104.12	102.93	101.92	103.93
FEV ₁	98.86	98.07	99.64	98.63	97.70	99.56	99.09	98.00	100.18
FEV ₁ /FVC	95.47	94.97	95.97	95.13	94.39	95.87	95.81	95.17	96.45
Industrial air emissions									
NO _x (tonnes)	2,372.34 ^E	1,547.02	3,197.66	2,431.43 ^E	1,589.65	3,273.22	2,312.64 ^E	1,487.75	3,137.53
PM _{2.5} (tonnes)	250.27	169.08	331.46	250.62	169.08	331.46	249.92	166.30	333.54

^E interpret with caution

[†] based on Global Lung Initiative reference equations

FVC = forced vital capacity

FEV₁ = 1-sec forced expiratory volume

NO_x = nitrogen oxides

PM_{2.5} = fine particulate matter

Source: 2007 to 2009 and 2009 to 2011 Canadian Health Measures Survey, combined.

Table 3

Regression coefficients relating percent predicted lung function parameters to industrial air emissions within 25 km of residence, by sex, household population aged 6 to 18, 2007 to 2011

Industrial airemission/ Sex	FVC				FEV ₁				FEV ₁ /FVC			
	beta	95% confidence interval		p-value	beta	95% confidence interval		p-value	beta	95% confidence interval		p-value
		from	to			from	to			from	to	
PM_{2.5}												
Males												
Model 1	-0.003	-0.007	0.001	0.133	-0.006	-0.010	-0.002	0.005*	-0.003	-0.005	0.000	0.021*
Model 2	-0.005	-0.009	-0.002	0.004*	-0.003	-0.005	-0.001	0.017*
Model 3	-0.005	-0.009	-0.001	0.008*	-0.003	-0.005	0.000	0.025*
Model 4	-0.005	-0.009	-0.001	0.009*	-0.003	-0.005	0.000	0.028*
Model 5	-0.005	-0.009	-0.001	0.010*	-0.003	-0.005	0.000	0.025*
Females												
Model 1	-0.002	-0.005	0.001	0.157	-0.003	-0.006	0.000	0.060	-0.001	-0.004	0.002	0.546
NO_x												
Males												
Model 1	0.000	-0.001	0.000	0.307	0.000	-0.001	0.000	0.101	0.000	0.000	0.000	0.155
Females												
Model 1	0.000	-0.001	0.000	0.646	0.000	-0.001	0.000	0.080	0.000	0.000	0.000	0.099

... not applicable

* significant at p < 0.05

FVC = forced vital capacity

FEV₁ = 1-sec forced expiratory volume

PM_{2.5} = fine particulate matter

NO_x = nitrogen oxides

Model 1 = unadjusted; Model 2 = Model 1 + respiratory condition/symptom; Model 3 = Model 2 + household income; Model 4 = Model 3 + short-term PM_{2.5}; Model 5 = Model 4 + age

Source: 2007 to 2009 and 2009 to 2011 Canadian Health Measures Survey, combined.

Discussion

The association between exposure to industrial air emissions of NO_x and PM_{2.5} and lung function was examined in a nationally representative sample of Canadian children and youth aged 6 to 18, using data from the NPRI and the CHMS. The significant negative association between emissions of PM_{2.5} and FEV₁ and FEV₁/FVC among males suggests that such emissions are related to airway obstruction in this group.

These findings are consistent with previous research. For example, a study in Argentina found that children aged 6 to 12 living near petrochemical plants had lower lung function (13% lower FEV₁ percent predicted) than those in two relatively unpolluted areas.⁵ Levels of particulate matter, including PM_{2.5}, were higher near the petrochemical plants than in other parts of the city.¹⁹ An analysis in Spain that compared the lung function of children aged 6 to 14 living in a municipality near a large oil refinery and liquid fuel gasification plant with that of children in a nearby rural municipality found that those in the petrochemical industry area had lower lung function (10.3% lower FEV₁).⁶

Not all research has reported significant associations. A study of 13- to 14-year-olds in Spain did not find differences in lung function between those living near petrochemical plants, those living in a city with medium vehicular traffic, or those in an area with low vehicular traffic and no industry.

What is already known on this subject?

- Long-term exposure to ambient air pollution, such as fine particulate matter and nitrogen dioxide, has been associated with adverse effects on children's lung function.
- Few studies have examined lung function in relation to industrial emissions of air pollutants.

What does this study add?

- The National Pollutant Release Inventory, Canada's legislated, publicly accessible inventory of pollutant releases, was used as a source of data for industrial air emissions.
- At ages 6 to 18, a significant association between industrial air emissions of fine particulate matter and lung function was apparent for males, but not females.

These inconsistent results may be due to differences in characteristics of the petrochemical sites in various studies, such as wind direction and speed, humidity, precipitation, crude oil quality, production technology, pollution control equipment, and other nearby industrial activities and sources of pollution. As well, the composition of PM_{2.5} can vary substantially with its origin, and particles from various sources may have different toxicities.²⁰

A strength of the current study is that industrial air emissions were assigned at the individual level, rather than at the community or municipal level based on proximity to a petrochemical plant.

Whether a gender difference exists in the relationship between air pollution and children's lung function is unclear. This study found a significant association for boys, but not girls, which is consistent with several other studies.^{3,21-24}

However, some research has reported stronger associations for girls,²⁵⁻²⁷ or no differences.^{4,28,29}

Gender differences in the health effects of exposure to particulate matter may be related to differences in particle deposition in the respiratory tract due to anatomical differences and ventilation dynamics, and this effect may depend on particle size.³⁰ In addition, even moderate physical activity can result in a total lung deposition rate three to five times greater than at rest owing to higher minute ventilation and a greater prevalence of oral breathing,^{31,32} which bypasses the particle filtering that occurs when breathing through the nose. According to data for 2007 to 2009, boys were more physically active than girls,³³ which has been associated with time spent outdoors.^{34,35} Variations in time outdoors, and resultant exposure to air pollutants, may have contributed to the gender differences reported in this study and others.

No significant association between industrial emissions of NO_x and lung function was observed. Although most studies have examined NO₂ in relation to respiratory health,² some have shown a significant association with NO_x and lung function.^{4,36} However, those studies examined traffic-related NO_x, rather than industry-related NO_x. Studies employing residential proximity to highways and major roads as a measure of traffic-related air pollutants have used cut-points of 50m to 200m to identify those with greater exposure.^{37,38} In the present analysis, facilities located much farther away (25 km) were considered relevant sources of industrial emissions of NO_x. Results based on a smaller radius might be different.

Limitations

This analysis has a number of limitations. Concentrations of industrial air emissions were not measured. NPRI data, which were used as a proxy for exposure to industrial emissions, reflect only emissions from facilities required to report to the NPRI. Facilities were required to report to the NPRI if one or more NPRI

substances were manufactured, processed or otherwise used at the facility during the year, and the total number of hours worked at the facility exceeded the 20,000-hour employee threshold (about 10 full-time employees). However, there were exceptions (<http://www.ec.gc.ca/inrp-npri/>). Of facilities required to report, those with emissions below the reporting thresholds would not be included in the derivation of the industrial air emissions variables. Thus, emissions are underestimated to the extent that facilities emitting pollutants below the reporting threshold are located near respondents' residence.

Long-term exposure to industrial air emissions based on NPRI data is a variable that has not been validated. At-source monitors could theoretically be used to validate the amount of emissions reported by each facility, but could not validate respondent exposure. And while personal air monitors could measure respondent exposure, they would not be able to distinguish between industrial emissions and other sources of air pollution.

Directional effects of air pollution due to weather and climate, the effect of stack height, or the temporal patterns of releases were not taken into account. Further, information was not available about how long respondents had lived at the reported place of residence or the locations of respondents' schools.

The full effect of industrial emissions could not be examined because no CHMS respondents may have been living near some reporting facilities. The distribution of the total number of facilities that reported both NO_x and PM_{2.5} emissions, NO_x emissions only, and PM_{2.5} emissions only was approximately equal.¹³ By contrast, the majority of respondents lived within 25 km of facilities that reported emissions of both NO_x and PM_{2.5}; none lived near facilities that reported just NO_x emissions. Only areas with a population of at least 10,000 and a maximum respondent travel distance of 50 km in urban areas and 100 km in rural areas were considered as potential CHMS collection sites.⁹ The distribution of types of reporting facilities might have differed if

the survey had included respondents from areas with lower population densities. Alternatively, some reporting facilities may be located more than 25 km from residential areas. Determining the location of reporting facilities in relation to residential areas and population densities was beyond the scope of this study.

Conclusion

NPRI data have been used in the past to study environmental issues, such as pollution emissions by population socio-economic status and socio-cultural characteristics.^{39,40} This is the first time they have been used to examine the relationship between industrial air pollution and measures of lung function.

A significant association emerged between industrial emissions of air pollutants and lung function. Specifically, a negative association was observed between $PM_{2.5}$ and FEV_1 and FEV_1/FVC for young males, but not young females. No association was apparent between emissions of NO_x and lung function. Further analyses of the gender differences reported in this study are warranted. ■

References

- Sin DD, Wu L, Man SF. The relationship between reduced lung function and cardiovascular mortality: a population-based study and a systematic review of the literature. *Chest* 2005; 127:1952-9.
- Gotschi T, Heinrich J, Sunyer J, Kunzli N. Long-term effects of ambient air pollution on lung function. *Epidemiology* 2008; 19: 690-701.
- Gao Y, Chan EYY, Li LP, et al. Chronic effects of ambient air pollution on lung function among Chinese children. *Archives of Diseases in Childhood* 2013; 98: 128-35.
- Gehring U, Bruzиеva O, Agius RM, et al. Air pollution exposure and lung function in children: the ESCAPE project. *Environmental Health Perspectives* 2013; 121: 1357-64.
- Wichmann FA, Muller A, Busi LE, et al. Increased asthma and respiratory symptoms in children exposed to petrochemical pollution. *Journal of Allergy and Clinical Immunology* 2009; 123: 632-8.
- Rusconi F, Catelan D, Accetta G, et al. Asthma symptoms, lung function, and markers of oxidative stress and inflammation in children exposed to oil refinery pollution. *Journal of Asthma* 2011; 48: 84-90.
- Rovira E, Caudras A, Aguilar X, et al. Asthma, respiratory symptoms and lung function in children living near a petrochemical site. *Environmental Research* 2014; 133: 156-63.
- Miller MR, Hankinson JL, Brusasco V, et al. Standardisation of spirometry. *European Respiratory Journal* 2005; 26: 319-38.
- Statistics Canada. *Canadian Health Measures Survey (CHMS) Data User Guide: Cycle 2*. Ottawa: Statistics Canada, 2013.
- Quanjer PH, Stanojevic S, Cole TJ, et al. Multi-ethnic reference values for spirometry for the 3-95-yr age range: the global lung function 2012 equations. *European Respiratory Journal* 2012; 40: 1324-43.
- Society for Research on Nicotine and Tobacco Subcommittee on Biochemical Verification. Biochemical verification of tobacco use and cessation. *Nicotine and Tobacco Research* 2002; 4: 149-59.
- WHO Multicentre Growth Reference Study Group. *WHO Child Growth Standards: Length/Height-for-age, Weight-for-age, Weight-for-length, Weight-for-height and Body Mass Index-for-age: Methods and Development*. Geneva: World Health Organization, 2006.
- National Pollutant Release Inventory. *National Pollutant Release Inventory Reporting Guide: Reporting for Part 4 Substances - Criteria Air Contaminants*. Available at: <http://www.ec.gc.ca/inrp-npri/default.asp?lang=En&n=1FAA2366-1>
- Wilkins R, Peters PA. *PCCF+ Version 5K User's Guide*. Ottawa: Statistics Canada, 2012.
- Fotheringham AS, Brunsdon C, Charlton M. *Geographically Weighted Regression: The Analysis of Spatially Varying Relationships*. West Sussex, England: John Wiley and Sons, Ltd., 2002: 210-1.
- Rao JNK, Wu CFJ, Yue K. Some recent work on resampling methods for complex surveys. *Survey Methodology* (Statistics Canada, Catalogue 12-001) 1992; 18(2): 209-17.
- Rust KF, Rao JNK. Variance estimation for complex surveys using replication techniques. *Statistical Methods in Medical Research* 1996; 5(3): 283-310.
- Statistics Canada. *Instructions for Combining Cycle 1 and Cycle 2 Canadian Health Measures Survey (CHMS) Data*. Ottawa: Statistics Canada, 2013.
- Massolo L, Mueller A, Tueros M, et al. Assessment of mutagenicity and toxicity of different size fractions of air particulates from La Plata, Argentina, and Leipzig (Germany). *Environmental Toxicology* 2002; 17: 219-31.
- Laden F, Neas LM, Dockery DW, Schwartz J. Association of fine particulate matter from different sources with daily mortality in six U.S. cities. *Environmental Health Perspectives* 2000; 108: 941-7.
- Hwang BF, Chen YH, Lin YT, et al. Relationship between exposure to fine particulates and ozone and reduced lung function in children. *Environmental Research* 2015; 137C: 382-90.
- Jedrychowski W, Flak E, Mroz E. The adverse effect of low levels of ambient air pollutants on lung function growth in preadolescent children. *Environmental Health Perspectives* 1999; 107: 669-74.
- Brunekreef B, Janssen NA, de Hartog J, et al. Air pollution from truck traffic and lung function in children living near motorways. *Epidemiology* 1997; 8: 298-303.
- Schultz E, Gruzиеva O, Bellander T, et al. Traffic-related air pollution and lung function in children at 8 years of age – a birth cohort study. *American Journal of Respiratory and Critical Care Medicine* 2012; 186: 1286-91.
- Frye C, Hoelscher B, Cyrus J, et al. Association of lung function with declining ambient air pollution. *Environmental Health Perspectives* 2003; 111: 383-7.
- Oftedal B, Brunekreef B, Nystad W, et al. Residential outdoor air pollution and lung function in schoolchildren. *Epidemiology* 2008; 19: 401-8.
- Peters JM, Avol E, Gauderman WJ, et al. A study of twelve Southern California communities with differing levels and types of air pollution. II. Effects on pulmonary function. *American Journal of Respiratory and Critical Care Medicine* 1999; 159: 768-75.
- Raizenne M, Neas LM, Damokosh AI, et al. Health effects of acid aerosols on North American children: pulmonary function. *Environmental Health Perspectives* 1996; 104: 506-14.

**Exposure to industrial air pollutant emissions and lung function in children:
Canadian Health Measures Survey, 2007 to 2011 • Research Article**

29. Gauderman WJ, Avol E, Gilliland F, et al. The effect of air pollution on lung development from 10 to 18 years of age. *New England Journal of Medicine* 2004; 351: 1057-67.
30. Health Canada. *Canadian Smog Science Assessment Volume 2: Health Effects*. Ottawa: Health Canada, 2013.
31. Daigle CC, Chalupa DC, Gibb FR, et al. Ultrafine particle deposition in humans during rest and exercise. *Inhalation Toxicology* 2003; 15: 539-52.
32. Chalupa DC, Morrow PE, Oberdorster G, et al. Ultrafine particle deposition in subjects with asthma. *Environmental Health Perspectives* 2004; 112: 879-82.
33. Colley RC, Garriguet D, Janssen I, et al. Physical activity of children and youth: Accelerometer results from the 2007 to 2009 Canadian Health Measures Survey. *Health Reports* 2011; 22: 1-9.
34. Schaefer L, Plotnikoff RC, Majumdar SR, et al. Outdoor time is associated with physical activity, sedentary time, and cardiorespiratory fitness in youth. *Journal of Pediatrics* 2014; 165: 516-21.
35. Stone MR, Faulkner FEJ. Outdoor play in children: associations with objectively-measured physical activity, sedentary behavior and weight status. *Preventive Medicine* 2014; 65: 122-7.
36. Urman R, McConnell R, Islam T, et al. Associations of children's lung function with ambient air pollution: joint effects of regional and near-roadway pollutants. *Thorax* 2014; 69: 540-7.
37. Riley S, Wallace J, Nair P. Proximity to major roadways is a risk factor for airway hyper-responsiveness in adults. *Canadian Respiratory Journal* 2012; 19: 89-96.
38. Middleton N, Yiallourous P, Nicolaou N, et al. Residential exposure to motor vehicle emissions and the risk of wheezing among 7-8 year-old schoolchildren: a city-wide cross-sectional study in Nicosia, Cyprus. *Environmental Health* 2010; 9: 1-17.
39. Premji S, Bertrand F, Smargiassi A, Daniel M. Socio-economic correlates of municipal-level pollution emissions on Montreal Island. *Canadian Journal of Public Health* 2007; 139: 138-42.
40. Kershaw S, Gower S, Rinner C, Campbell M. Identifying inequitable exposure to toxic air pollution in racialized and low-income neighbourhoods to support pollution prevention. *Geospatial Health* 2013; 7: 265-78.