Active and uncontrolled asthma among children exposed to air stack emissions of sulphur dioxide from petroleum refineries in Montreal, Quebec: A cross-sectional study

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BACKGROUND: Little attention has been devoted to the effects on children’s respiratory health of exposure to sulphur dioxide (SO2) in ambient air from local industrial emissions. Most studies on the effects of SO2 have assessed its impact as part of the regional ambient air pollutant mix.

OBJECTIVE: To examine the association between exposure to stack emissions of SO2 from petroleum refineries located in Montreal’s (Quebec) east-end industrial complex and the prevalence of active asthma and poor asthma control among children living nearby.

METHODS: The present cross-sectional study used data from a respiratory health survey of Montreal children six months to 12 years of age conducted in 2006. Of 7964 eligible households that completed the survey, 842 children between six months and 12 years of age lived in an area impacted by refinery emissions. Ambient SO2 exposure levels were estimated using dispersion modelling. Log-binomial regression models were used to estimate crude and adjusted prevalence ratios (PRs) and 95% CIs for the association between yearly school and residential SO2 exposure estimates and asthma outcomes. Adjustments were made for child’s age, sex, parental history of atopy and tobacco smoke exposure at home.

RESULTS: The adjusted PR for the association between active asthma and SO2 levels was 1.14 (95% CI 0.94 to 1.39) per interquartile range increase of SO2 and the prevalence of active asthma and poor asthma control among children living nearby was greater (PR=1.39 per interquartile range increase in modelled SO2[95% CI 1.00 to 1.94]).

CONCLUSIONS: Results of the present study suggest a relationship between exposure to refinery stack emissions of SO2 and the prevalence of active and poor asthma control in children who live and attend school in proximity to refineries.

Key Words: Asthma; Children; Cross-sectional study; Dispersion modelling; Point source emissions; Sulphur dioxide

Epidemiological studies suggest that exposure to air pollution is associated with adverse consequences on children’s respiratory health (1). Sulphur dioxide (SO2), a gaseous respiratory irritant, is among the air pollutants of public health concern in urban and industrialized environments.

Most epidemiological investigations on the effects of SO2 have assessed its acute and chronic effects as a component of the regional ambient air pollutant mix. Panel studies involving children and time series analyses have documented associations between short-term (daily) exposure to regional SO2 levels and respiratory effects (e.g., lung function changes, increased respiratory symptoms, emergency department visits and hospital admissions for asthma and other respiratory causes) in healthy and asthmatic children (2-10). An association between long-term (years) exposure to regional SO2 levels and respiratory effects (increased prevalence of symptoms and respiratory diseases in children) has also been reported in cross-sectional and semi-ecological cohort studies in which urban regional SO2 levels were compared (11-18). In other studies, little or no evidence supporting a relationship between regional ambient SO2 levels and adverse acute or chronic respiratory effects was found (13,19-22). Many factors may explain this discrepancy, including the varying mix of air pollutants and misclassification of exposure.

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Methods

Study design and population
Between April and July 2006, a cross-sectional, population-based survey on the determinants of respiratory health in children was conducted on the Island of Montreal by the Montreal Public Health Department (25). Study participants were recruited from a random list of names and addresses of 17,697 Montreal households with at least one child between six months and 12 years of age living in the vicinity of these industrial facilities. Data from a cross-sectional survey performed across the Island of Montreal on the prevalence of asthma was linked to dispersion model data from another study on acute effects performed in the same region (23). Dispersion modeling provides an approximation of ground-level concentrations of air pollutants around point sources and near housing, and can be used to quantify an individual’s exposure, particularly over the long term, using averaged meteorological data (24).

Two studies have assessed the respiratory health effects of an individual’s exposure within a community to industrial emissions of SO₂. Refineries and power plants are among the well-known industrial facilities that contribute locally and regionally to ambient SO₂ levels. The present study aimed to examine the effects of stack emissions of SO₂ from petroleum refineries located in Montreal’s (Quebec) east-end industrial complex on the prevalence of active asthma and poor asthma control among children six months to 12 years of age living in the vicinity of these industrial facilities. Data from a cross-sectional survey performed across the Island of Montreal on the prevalence of asthma was linked to dispersion model data from another study on acute effects performed in the same region (23). Dispersion modeling provides an approximation of ground-level concentrations of air pollutants around point sources and near housing, and can be used to quantify an individual’s exposure, particularly over the long term, using averaged meteorological data (24).

Asthma control was established in subjects with ‘active asthma’ and was assessed on the basis of five specific symptom-based criteria and was considered poorly controlled if one or more of the following features during the 12 months preceding the survey: wheezing or whistling in the chest, a dry cough at night, an asthma attack or use of bronchodilators. The definition of active asthma in Health Canada’s Respiratory Health Survey (ECHRHS) standardized written questionnaires, as well as the Quebec Child and Adolescent Health and Social Survey, to ensure comparability between Canadian and International results. Survey and asthma experts were asked to review the questionnaire, and a small-scale pretest was performed for content validation. Questions focused on the child’s respiratory and allergy symptoms and illnesses, use of health care services, personal and family medical history, home environmental exposures, lifestyle factors and sociodemographic characteristics. Home postal codes (six characters) were requested from the respondents. Within the study area, a six-digit postal code often corresponds to a single segment of road on which fewer than 50 individuals reside. A question about the location of the school the child attended (when applicable) was also included. Written informed consent was obtained from the child’s parent or legal guardian. The study protocol was approved by the Montreal Public Health Research Ethics Board.

Study variables

Health outcomes: The definition of active asthma in Health Canada’s 1995-1996 Student Lung Health Survey was used (26). ‘Active asthma’ was defined as ever having being diagnosed with asthma by a physician and having reported one or more of the following features during the 12 months preceding the survey: wheezing or whistling in the chest, a dry cough at night, an asthma attack or use of bronchodilators. ‘Asthma control’ was defined as ever having being diagnosed with asthma by a physician and having reported one or more of the following features during the 12 months preceding the survey: wheezing or whistling in the chest, a dry cough at night, an asthma attack or use of bronchodilators. ‘Asthma control’ was established in subjects with ‘active asthma’ and was assessed on the basis of five specific symptom-based criteria that are similar to those in the Canadian Pediatric Asthma Consensus guidelines (27). Asthma was considered poorly controlled if one or more of the following features was reported: daytime symptoms (eg, wheezing or whistling in the chest ≥3 times a week); night-time symptoms (eg, wheezing or whistling in the chest ≥3 times a week).
The geographical location of a child's home and school within the seven FSAs was estimated using the centroid coordinates of the six-digit postal codes. Yearly ambient SO2 levels from refinery stack emissions were estimated at the locations of the centroid coordinates of these six-digit postal codes. They were computed from hourly SO2 levels using the AERMOD air dispersion model as described in an earlier study that assessed their acute respiratory effects (23). This model is recommended by the United States Environmental Protection Agency for estimating the concentration of pollutants at specific ground-level receptors surrounding an emission source (28). Refinery emissions of SO2 and meteorological data for 2004 were used because 2005 emission data were not available.

Briefly, data for several point-source emissions of the two refineries were used to model hourly SO2 levels at receptor locations corresponding to the residential six-digit postal code centroids in the east end of Montreal (in the FSAs H1A, H1B, H1K, H1L, H1C, H1E and H1J). The point-source emissions included those from main vents and stacks. They were computed from hourly SO2 levels using the AERMOD air dispersion model as described in an earlier study that assessed their acute respiratory effects (23). This model is recommended by the United States Environmental Protection Agency for estimating the concentration of pollutants at specific ground-level receptors surrounding an emission source (28). Refinery emissions of SO2 and meteorological data for 2004 were used because 2005 emission data were not available.

The inputs to the dispersion model also included hourly meteorological records from the Pierre Elliott Trudeau Montreal International Airport, approximately 25 km from the study area, and upper air data from a rural monitoring site descriptive of the greater Montreal region. All meteorological data were acquired from Environment Canada (30). The topographical characteristics across the area of interest were considered to be constant. Allowances in the model were made for the nature of the local terrain, including both vegetated (grass) and paved surfaces.

Attempts were made to account for the time-activity patterns of all selected study children. For children younger than than five years of age on September 30, 2005 (compulsory school attendance age cut-off date each year), who were assumed to spend most of their time at home, hourly SO2 exposure estimates included only home exposure values. For school-age children (between five and 12 years of age), the time spent at home and at school was considered: it was assumed that 8 h a day (08:00 to 16:00) was spent at school on weekdays (Monday to Friday) from September to June and that the remaining time was spent at home. A missing SO2 value for school exposure was assigned to children attending schools outside the area of east-end Montreal (11.7%). Background ambient SO2 levels were not considered because only one average value at the monitoring station was available for the Island of Montreal (a constant for all the children in the present study). For the analyses, intra-urban geographical variations in pollutant levels emitted by industrial stacks were available only for SO2.

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TABLE 1
Characteristics of children from east-end Montreal (Quebec), 2006

<table>
<thead>
<tr>
<th>Variable</th>
<th>Active asthma (n=142)</th>
<th>No active asthma (n=679)</th>
<th>Poor asthma control* (n=51)</th>
<th>Active asthma with acceptable control* (n=86)</th>
<th>No asthma or controlled asthma (n=765)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, years, mean ± SD</td>
<td>7.6±2.7</td>
<td>7.5±3.3</td>
<td>7.2±2.7</td>
<td>7.6±2.7</td>
<td>7.5±3.3</td>
</tr>
<tr>
<td>Sex, n (%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>72 (50.7)</td>
<td>345 (50.8)</td>
<td>31 (60.8)</td>
<td>37 (43.0)</td>
<td>382 (49.9)</td>
</tr>
<tr>
<td>Female</td>
<td>70 (49.3)</td>
<td>334 (49.2)</td>
<td>20 (39.2)</td>
<td>49 (57.0)</td>
<td>383 (50.1)</td>
</tr>
<tr>
<td>Parental history of atopy, n (%)</td>
<td>90 (63.4)</td>
<td>224 (33.0)</td>
<td>34 (66.7)</td>
<td>51 (59.3)</td>
<td>275 (36.0)</td>
</tr>
<tr>
<td>Household income/year, n (%)††</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;$75,000</td>
<td>96 (73.8)</td>
<td>393 (64.5)</td>
<td>36 (75.0)</td>
<td>57 (73.1)</td>
<td>450 (65.5)</td>
</tr>
<tr>
<td>≥$75,000</td>
<td>34 (26.2)</td>
<td>216 (35.5)</td>
<td>12 (25.0)</td>
<td>21 (26.9)</td>
<td>237 (34.5)</td>
</tr>
<tr>
<td>Maternal educational level, n (%)**</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Secondary and lower</td>
<td>54 (39.1)</td>
<td>212 (32.0)</td>
<td>19 (37.3)</td>
<td>33 (40.2)</td>
<td>245 (32.9)</td>
</tr>
<tr>
<td>Postsecondary</td>
<td>84 (60.9)</td>
<td>451 (68.0)</td>
<td>32 (62.7)</td>
<td>49 (59.8)</td>
<td>500 (67.1)</td>
</tr>
<tr>
<td>Passive smoke exposure, n (%)</td>
<td>28 (19.7)</td>
<td>125 (18.4)</td>
<td>10 (19.6)</td>
<td>16 (18.6)</td>
<td>141 (18.4)</td>
</tr>
<tr>
<td>Yearly SO2 exposure, µg/m³, mean ± SD</td>
<td>4.75 (3.34)</td>
<td>4.37 (3.17)</td>
<td>5.37 (3.50)††</td>
<td>4.55 (3.09)</td>
<td>4.39 (3.2)</td>
</tr>
</tbody>
</table>

*Assessment of asthma control was not possible for five active asthmatic subjects, thus, the number of controlled and uncontrolled asthmatics does not sum to 142; †Significant difference from the group of children without active asthma (χ² P<0.05); ‡Significant difference from the group of children without active asthma (χ² P<0.001); §Significant difference from the group of children with no asthma or controlled asthma (χ² P<0.001); Eighty-two individuals had missing values (n=739); **Twenty individuals had missing values (n=801). ††Significant difference from the group of children with no asthma or controlled asthma (t test P<0.05). SO₂ Sulphur dioxide

TABLE 2
Prevalence ratios (PR) for the association between yearly ambient sulphur dioxide exposure levels (AERMOD estimates) and prevalence of active asthma and poor asthma control

<table>
<thead>
<tr>
<th>Health outcomes</th>
<th>Yes</th>
<th>No</th>
<th>PR (95% CI)</th>
<th>PR (95% CI)*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Active asthma</td>
<td>124</td>
<td>679</td>
<td>1.15 (0.94–1.41)</td>
<td>1.14 (0.94–1.39)</td>
</tr>
<tr>
<td>Poor asthma control†</td>
<td>51</td>
<td>765</td>
<td>1.45 (1.03–2.03)</td>
<td>1.39 (1.00–1.94)</td>
</tr>
</tbody>
</table>

*Expressed as an interquartile range (IQR) increase and adjusted for child's age, sex, parental atopy and environmental tobacco smoke exposure at home; †There were missing data for the questions used for the assessment of asthma control in five children.

Montreal, and ambient school and residential exposure to refinery stack emissions of SO₂. In the present study, SO₂ exposure was estimated using a dispersion model, which provided the intra-urban geographical variation of ambient SO₂ levels. In earlier studies, the association between industrial SO₂ emissions and respiratory health effects among children was assessed by comparing large geographical areas with different SO₂ levels or areas in varying proximity to industrial facilities that burn coal or oil, such as power plants, refineries, incinerators, petrochemical complexes and other industrial sources of SO₂ (31).

Our results concur with those of Charpin et al (32), Dales et al (33) and Yang et al (34) who reported an increased prevalence of respiratory symptoms in industrial-polluted communities with higher levels of SO₂ compared with low-pollution areas. Our results are also in agreement with studies reporting an increased prevalence of asthma (35-37) or asthma-related symptoms (37-39) among children living in proximity to industrial areas, including refineries and petrochemical plants, where SO₂ emissions occur.

The prevalence of parental atopy was quite high in our study (more than 60% in children with active asthma and poor control of their disease, and 33% in children without asthma). Some previous studies that measured parental atopy using questionnaires reported widely varying prevalences of positive parental history of atopy in asthmatic children, for example, ranging from 15% (family history of asthma) to 28% (mother has asthma) among Canadian children with asthma (40,41). In these studies, different approaches were used to define the presence of atopy (42). The differences observed between our study and previous work could partly result from the fact that we used a more inclusive definition (ie, a reported history of asthma, allergic rhinitis and/or eczema in the child’s biological mother or father) to capture a variety of parental atopy profiles associated with asthma. The higher prevalence of parental atopy in this population could also be due to a much lower proportion of immigrants living in the area (the lowest on the Island of Montreal). In fact, the prevalence of declared parental atopy among immigrants was approximately one-half of that among the domestic population (North American origin) (25).

Furthermore, other studies did not find an association between proximity to areas with SO₂-emitting industrial facilities and asthma-related outcomes (43,44). These inconsistencies may be due to the fact that most studies failed to properly classify exposure. Exposure may be better estimated with the use of dispersion modelling than by proximity to industrial facilities.

While a dispersion model was used to estimate exposure, our study was still subject to several limitations. First, definitive information regarding the residency of the children was not available. Our school-residence weighted time may not have been an adequate representation of exposure. We assumed that children who were younger than five years of age were at home; however, some were likely attending daycare, but the location of the daycare centres was not available. Nevertheless, school and daycare hours are small compared with home hours, and would have limited influence on annual exposure estimates. In support of this, when we used home exposure data only (ie, without school exposure), similar results were observed.

Second, the exposure estimates did not include other sources of SO₂. We focused on the contribution of SO₂ emissions from the refineries because it was the main source of this pollutant in the study area. Background SO₂ emissions from other local industrial sources or from diverse urban sources were considered to be negligible.

Third, we do not know whether the effects observed in our study were due to SO₂, to other pollutants or to a combination of both. Stack emissions of other pollutants such as fine particles, which have been associated with asthma, occur concurrently with SO₂. Fugitive volatile organic compound (VOC) and stack emissions are dispersed to residential areas by winds and expose the population. Unfortunately, we could not address the effects of VOC and fine particulate emissions in our analyses because the levels of these pollutants were not modelled. Furthermore, we do not know whether the effects were due to cumulative or recent exposure. Pollutant levels and refinery
emissions were higher in the past and, for older children, exposure might have been higher in the early years of life. However, asthma control is probably more influenced by recent exposure.

Given the multifactorial etiology of asthma, it is also possible that pollutants derived from indoor sources contribute to the aggravation of the disease. Yet, we controlled for ETS exposure in the home – the most probable confounder or effect modifier among environmental and lifestyle risk factors for asthma. Analyses controlling for the presence of reported mold or humidity in the house, as well as road traffic density on the street of the residence were also performed, with no confounders to the associations with SO2 levels observed (data not shown).

It is also worth noting that in the current study, asthma diagnosis and asthma status (active asthma and disease control) were not validated with objective measurements such as lung function tests. Nevertheless, the methods we used were similar to those of other national and international epidemiological studies that used validated and standardized questionnaires. We also assessed asthma symptoms and control using criteria and questions that are widely used in clinical practices involving children. We should also point out that asthma cannot be definitely confirmed in children younger than six years of age, and that the diagnosis of asthma is not based on physiological criteria for that age group (27,45).

Finally, the high level of public concern about the health impacts of refinery pollution might have led subjects to move away from the industrial sector, which might have influenced our prevalence results. To limit the effect of moving, we studied only children who resided at the same address in the study area for at least one year before the 2006 survey or since birth. Furthermore, public concern might have led subjects living near the refineries to over-report respiratory health symptoms. While differential reporting bias was not ruled out, the initial study was presented as a respiratory health survey of Montreal children rather than an assessment of the respiratory health effects from exposure to refinery emissions, thereby reducing such bias. Furthermore, the use of a dispersion model to estimate exposure, rather than only proximity to the industrial complex, renders differential reporting of asthma symptoms according to exposure category less likely.

CONCLUSION

Results of the present study suggest an association between exposure to SO2 from refinery stack emissions and the prevalence of active and poor asthma control. Additional studies are needed to understand whether the observed associations were due to repeat acute or chronic exposure or to both, and if industrial emissions are associated with the development of asthma.

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