Environment International 121 (2018) 23–30

Contents lists available at ScienceDirect

Environment International

journal homepage: www.elsevier.com/locate/envint

A population-based birth cohort study of the association between childhood-onset asthma and exposure to industrial air pollutant emissions

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ARTICLE INFO

Handling Editor: Xavier Querol

Keywords:
Air pollution
Cohort study
Asthma
Industry
Children
Fine particles
Sulfur dioxide

ABSTRACT

Background: Studies of the association between air pollution and asthma onset have mostly focused on urban and traffic-related air pollution. We investigated the associations between exposure to industrial emissions and childhood-onset asthma in a population-based birth cohort in Quebec, Canada, 2002–2011.

Methods: The cohort was built from administrative health databases. We developed separately for $PM_{2.5}$ and SO_2 different metrics representing children's time-varying residential exposure to industrial emissions: 1) yearly number of tons of air pollutant emitted by industries located within 2.5 km of the residence; 2) distance to the nearest "major emitter" (≥100 tons) of either $PM_{2.5}$ and SO_2 within 7.5 km of the residence, and; 3) tons of air pollutant emitted by the nearest "major emitter" within 7.5 km, weighted by the inverse of the distance and the percentage of time that the residence was downwind. To handle the large number of zeros (i.e., children unexposed) we decomposed the exposure variable into two covariates simultaneously included in the regression model: a binary indicator of exposure and a continuous exposure variable centered at the mean value among exposed children. We performed Cox models using age as the time axis, adjusted for gender, material and social deprivation and calendar year. We indirectly adjusted for unmeasured secondhand smoke.

Results: The cohort included 722,667 children and 66,559 incident cases of asthma. Across the different exposure metrics, mean percentage changes in the risk of asthma onset in children exposed to the mean relative to those unexposed ranged from 4.5% (95% CI: 2.8, 6.3%) to 10.6% (95% CI: 6.2, 15.2%) for $PM_{2.5}$ and, from 1.1% (95% CI: −0.1, 3.3%) to 8.9% (95% CI: 7.1, 11.1%) for SO_2. Indirect adjustment for secondhand smoke did not substantially affect the associations. In children exposed, the risk of asthma onset increased with the magnitude of the exposure for all metrics, except the distance to the nearest major emitter of SO_2.

Conclusions: In this population-based birth cohort, residential exposure to industrial air pollutant emissions was associated with childhood-onset asthma. Additional studies with improved models for estimating exposure to industrial point-sources are needed to further support the observed associations.

1. Introduction

Asthma is the most common chronic disease among children in Canada (Radhakrishnan et al., 2014) and worldwide (WHO, 2016). According to the Public Health Agency of Canada, in 2011–2012 the prevalence of diagnosed asthma among Canadian children aged 1–4 and 5–9 years old was 6.2% and 14.0%, respectively (PHAC, 2017). The substantial increase in the incidence of asthma in children over past decades and the geographic variation in its prevalence supports the hypothesis that environmental factors play an important role in the development of childhood asthma (Gershon et al., 2010; Subbarao et al., 2009a; Subbarao et al., 2009b; von Mutius, 2000). In addition, a

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https://doi.org/10.1016/j.envint.2018.08.040
Received 21 March 2018; Received in revised form 15 August 2018; Accepted 16 August 2018

number of mechanisms and gene–environment interactions have been postulated by which air pollution could contribute to the development of asthma (Braback and Forsberg, 2009).

Epidemiological studies have used various designs and exposure assessment methods to investigate the effects of outdoor air pollution on asthma (Braback and Forsberg, 2009). Together they have provided sufficient evidence that exposures to ambient air pollutants, including ozone (O_3), particulate matter of aerodynamic diameters $\leq 2.5\,\mu m$ ($PM_{2.5}$) and $\leq 10\,\mu m$; (PM_{10}), sulfur dioxide (SO_2), and nitrogen oxides (NO_2), can exacerbate pre-existing asthma (Andersen et al., 2008; Gent et al., 2003; Holguin, 2008; O'Connor et al., 2008; Trasande and Thurston, 2005; von Mutius, 2000). However, whether air pollution plays a role in the initial development of the disease is less clear. Earlier reviews have suggested that the available evidence was inconsistent (Guarnieri and Balmes, 2014; Koenig, 1999); however meta-estimates from the most up-to-date review of 41 studies (of which 24 were birth cohorts) showed positive associations between the development of childhood asthma and exposure to traffic-related air, including $PM_{2.5}$, PM_{10}, NO_2 (Khreis et al., 2017).

Despite the fact that industries may be important sources of air pollution, particularly on a local scale, previous cohort studies investigating the potential role of air pollution on asthma onset have essentially focused on urban or traffic-related air pollution. In the only cohort study (Clark et al., 2010) that investigated exposure to air pollution from industries, incidence of asthma diagnosis in children aged up to 3–4 years was found to be associated with the inverse-distance weighted summation of emissions from industrial emitters within a 10 km radius of the residence at birth and during the first year of life. In some other studies exposure to air pollution from industrial sources, often represented by residential proximity, was associated with acute respiratory health effects in children, including emergency department visits and hospitalizations for asthma or respiratory symptoms (Brand et al., 2016; de Marco et al., 2010; Labelle et al., 2015; Lewin et al., 2013; Marchetti et al., 2014; Pope, 1989; Pope and Dockery, 1992; Rava et al., 2012; Rava et al., 2011; Rovira et al., 2014; Smargiassi et al., 2014; Smargiassi et al., 2009; Wichmann et al., 2009).

The objective of this study was to estimate whether yearly residential exposure to air pollution emissions from industrial point-sources was associated with childhood-onset asthma using a population-based birth cohort in Quebec (Canada), 2002–2011. Specifically, our focus was on industrial emissions of fine particles (i.e., particulate matter of aerodynamic diameters $\leq 2.5\,\mu m$; $PM_{2.5}$) and sulfur dioxide (SO_2), for which we assessed individual time-varying residential exposure based on a series of indicators that we developed from geographical, emissions and meteorological data.

2. Methods

2.1. The cohort of children born in Quebec, 2002–2011

We used an open birth cohort constructed with information from the Quebec Integrated Chronic Disease Surveillance System (QICDSS) of the Quebec Public Health Institute (Blais et al., 2014). This system was developed based on the linkage of five health administrative datasets including the registration file from the Quebec medicare system (Régie de l'assurance maladie du Québec, RAMQ), the hospital discharge file, drug file, the fee-for medical service file and the mortality file. The QICDSS covers approximately 95% of the Quebec population (Blais et al., 2014) and includes demographic (e.g., sex, date of birth) information as well as time-varying information about participants' six-character residential postal code. The Canadian six-digit postal code represents a block face in cities but can cover a substantially larger area in rural settings where populations are less dense.

The target population comprised all children born in Quebec from April 1, 2002, to March 31, 2011. During that period, newborns were identified as new entries of children in the RAMQ Inscription/Insurance Registry file. Newborns were followed from birth until they developed asthma (see Section 2.2 for the definition), died, moved out of Quebec or reached the end of the study period without developing the disease. Although the cohort was dynamic, children who moved out of Quebec could not re-enter the cohort if they moved back into the study area, given they may have developed the disease in the meantime. Censoring was done at time of loss to follow-up according to the RAMQ data.

2.2. Definition of asthma onset

We identified new cases of asthma in children using an algorithm that was previously validated (Gershon et al., 2010). This definition has also been used in a previous cohort study (Tétreault et al., 2016) and is currently used by public health institutions for asthma surveillance in Quebec and in Canada. Specifically, case definitions consisted of: i) at least two medical services (i.e., visits to the emergency room or physician's office) with a diagnosis of asthma within a two-year period, or; ii) one hospital discharge with a primary or secondary diagnosis of asthma. Asthma diagnoses were identified using ICD-9 and ICD-10 codes 493 and J45-J46, respectively. When two diagnoses were used to identify a case, the medical services had to be on different days and, the indexing date was the date of the second service.

2.3. Development of exposure metrics to industrial emissions

The Canadian National Air Pollution Surveillance network (NAPS) of fixed-site monitors is too sparse to adequately represent exposure of the population to industrial emissions of air pollutants, particularly outside of urban areas where the number of monitoring stations is very limited. Therefore, we developed a series of metric of exposure that may reflect children's exposure to industrial emissions based on their residence location. We focused on industrial emissions of $PM_{2.5}$ and SO_2. Specifically, we made use of information about location of industries and children's residence, emissions of air pollutant by industries and meteorological data to develop, for each pollutant, the following metrics of exposure:

1) The yearly amount in tons of single air pollutant (i.e., either $PM_{2.5}$ or SO_2) emitted by all industries located within a 2.5 km radius of the residence;
2) The distance between the residence and the closest "major" industrial emitter of $PM_{2.5}$ and SO_2 within 7.5 km of the residence. As in a previous study (Loyo-Berrios et al., 2007), we defined "major" industries as those emitting > 100 tons of either $PM_{2.5}$ or SO_2. Note that it is possible for an industry to be a major emitter of one pollutant, but not another (i.e. an industry can be a major emitter of SO_2, but not $PM_{2.5}$ or vice versa). Also, for each pollutant if more than one major emitter is located within 7.5 km, only the closest one was considered.
3) The *wind and inverse-distance weighted emissions*, defined as: the yearly number of tons of $PM_{2.5}$ or SO_2 emitted by the closest major industrial emitter located within a 7.5 km radius of the child's residence multiplied by the inverse of the distance (i.e., 1/distance, where the distance is in km) between the child's residence and the closest major industry and, by the percentage of hours per day that the child's residence was downwind of the closest major industry. We used the inverse of the distance because the distance on its original scale is expected to be negatively related to exposure (i.e., increase in the distance to the industry is expected to decrease the exposure to emissions from that industry), whereas the other indicators (i.e., tons emitted and percent of time downwind) should increase the exposure.

Emission data were extracted from the National Pollutant Release Inventory (NPRI) (Environment and Climate Change Canada, 2016), which is a legislated, publicly-accessible database of pollutant releases,