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Short-term association between ambient air pollution and pneumonia in children: A systematic review and meta-analysis of time-series and case-crossover studies☆

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ABSTRACT

Ambient air pollution has been associated with respiratory diseases in children. However, its effects on pediatric pneumonia have not been meta-analyzed. We conducted a systematic review and meta-analysis of the short-term association between ambient air pollution and hospitalization of children due to pneumonia. We searched the Web of Science and PubMed for indexed publications up to January 2017. Pollutant-specific excess risk percentage (ER%) and confidence intervals (CI) were estimated using random effect models for particulate matter (PM) with diameter $\leq$ 10 (PM_{10}) and $\leq$2.5 μm ($PM_{2.5}$), sulfur dioxide (SO_2), ozone (O_3), nitrogen dioxide (NO_2), and carbon monoxide (CO). Results were further stratified by subgroups (children under five, emergency visits versus hospital admissions, income level of study location, and exposure period). Seventeen studies were included in the meta-analysis. The ER% per 10 μg/m^3 increase of pollutants was 1.5% (95% CI: 0.6%–2.4%) for PM_{10} and 1.8% (95% CI: 0.5%–3.1%) for $PM_{2.5}$. The corresponding values per 10 ppb increment of gaseous pollutants were 2.9% (95% CI: 0.4%–5.3%) for SO_2, 1.7% (95% CI: 0.5%–2.8%) for O_3, and 1.4% (95% CI: 0.4%–2.4%) for NO_2. ER% per 1000 ppb increment of CO was 0.9% (95% CI: 0.0%–1.9%). Associations were not substantially different between subgroups. This meta-analysis shows a positive association between daily levels of ambient air pollution markers and hospitalization of children due to pneumonia. However, lack of studies from low-and middle-income countries limits the quantitative generalizability given that susceptibilities to the adverse effects of air pollution may be different in those populations. The meta-regression in our analysis further demonstrated a strong effect of country income level on heterogeneity.

1. Introduction

The adverse effects of air pollution on cardiorespiratory diseases have been demonstrated in a series of studies (Fuertes et al., 2014; Künzli et al., 2000). The Global Burden of Disease Study 2013 estimated that ambient particulate matter (PM) pollution was responsible for nearly three million premature deaths worldwide (Forouzanfar et al., 2015). Other studies reveal that children living near roadways or wood industries are likely to have a higher risk of pneumonia, while a ban on diesel-powered motor vehicles may reduce the hospital admissions for the disease (El-Zein et al., 2007). Indeed, children are particularly susceptible to PM pollution, as they inhale more air per unit body weight than adults (World Health Organization, 2005). Furthermore, early exposure to ambient air pollution may affect children's normal growth and lung development, as the immune and organ systems are still evolving

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at this stage (Chen et al., 2015; Sunyer, 2008).

Pneumonia is the leading cause of childhood death, accounting for about 1.3 million deaths among children under five years of age in 2010–2011 (Walker et al., 2013). About 75% of the burden occurs among children from low-and-middle-income-countries, where poverty and severe ambient air pollution are common (Brauer et al., 2016). The current evidence suggests that many risk factors may be involved in the onset of pediatric pneumonia (Jackson et al., 2013; Sonego et al., 2015; Walker et al., 2013). These might include a broad range of personal (e.g. age or sex), family, and socio-economic characteristics; environmental factors (e.g. indoor air pollution, low quality of drinking water, second hand smoke, etc.); as well as comorbidities (e.g. HIV in children).

Acute effects of air pollution are efficiently captured by statistical time-series analyses that investigate the association between daily concentrations of air pollution and daily counts of health outcomes while controlling for other time-varying – thus potentially confounding – co-factors, such as meteorological conditions, seasonality, and other time-trends. So far, several systematic reviews have been conducted to evaluate the short-term association of ambient air pollution and lower respiratory tract infections in children (Atkinson et al., 2014; Mehta et al., 2013a; Romieu et al., 2002). However, there is no published meta-analysis of the acute effects of ambient air pollution on pediatric pneumonia to date. Meta-analyses play a vital role in the assessment of evidence, impact assessments, and public health policy making. Thus, we systematically evaluate this association using hospital admissions due to pneumonia as the main outcome.

2. Material and methods

2.1. Data sources and search strategy

We conducted a systematic review and meta-analysis of studies indexed in PubMed and Web of Science and published until January 3rd, 2017, that reported on the acute effects of outdoor air pollutants on hospitalization due to pneumonia. The search was not restricted by language, but we solely focused on original research articles published in peer reviewed journals. Database-specific search queries were developed using relevant keywords (See supplementary material A).

2.2. Eligibility criteria

Studies were included in the review if they (1) investigated the short-term association of ambient air pollutants with hospitalization due to pneumonia (whereby short-term effects were defined as those occurring within zero to seven days after exposure), (2) reported estimates for children up to 18 years of age, and (3) provided quantitative results for the effects (i.e. point estimate and confidence interval (CI)).

The following exclusion criteria were defined: (1) studies conducted among the general population without specific estimation for children, (2) studies of the long-term effects of air pollution on pneumonia, (3) studies that do not include pneumonia as an endpoint, (4) studies that do not measure pollutants, such as those comparing outcomes before and after an intervention like fuel bans, (5) review articles, and (6) studies limited to an abstract.

2.3. Study selection

First, two reviewers (NTTN and HA) independently screened the retrieved records. If there was a disagreement between the two reviewers, third and fourth reviewers (LP and NK) were called upon to help make a final decision. Afterwards, one reviewer (NTTN) examined the full text to evaluate eligibility according to the set criteria, which were then double checked by the second reviewer (HA). Finally, both reviewers (NTTN and HA) were involved independently in data extraction.

2.4. Data collection process

Data extraction forms were pre-designed to collect all relevant information. Data included citation information (title, author, and year of publication); the study setting (study location or country, and study period); the study design (time series, case-crossover analyses, etc.); daily mean number of events or total events; definition of health outcome; measurement of exposure, with air pollutant level; time of exposure; covariates in the models; and study results.

2.5. Meta-analysis approach

Meta–analysis was performed across available studies to summarize the short-term effects of pollutants on hospitalization of children due to pneumonia. Studies were eligible for meta-analyses if they used either time series or case-crossover analyses. Time series analyses use Poisson regressions to investigate associations of short-term changes in ambient concentrations of pollutants with daily changes in health outcomes. The case-crossover approach compares concentrations at the time of an event with those during a control time, within the same person. Both methods are widely used to assess short-term relationships between ambient levels of air pollution and the daily frequency of adverse health conditions (Yun Lu and Zeger, 2007).

Many studies have published a variety of estimates to accommodate various lags, multiple pollutants, or sub-groups defined by location (e.g. single cities versus multiple cities), among others. To prevent over-representation of a single study in the pooled estimation, only one estimate was selected from each study, according to the following rules: a) in the case of studies that reported several lags, we used the shortest one in the pooled estimate; b) cumulative lags (e.g. lag 0–2 or 0–4 days) were used if no single lag estimates were provided; c) in the case of multi-city studies, we included the single city results, one by one; and d) where more than one publication was available for a given population or region, we included the one that had the longest time-series period and/or the largest number of events.

To pool the effects, all relative risks (RR) were standardized to an increase of 10 $\mu g/m^3$ of PM_{10} and $PM_{2.5}$ concentration; 10 ppb of sulfur dioxide (SO_2), nitrogen dioxide (NO_2), and ozone (O_3) concentration; and 1000 ppb of carbon monoxide (CO). In case of small relative risks, odds ratios and relative risks can be considered the same. For studies reporting RR_u per U units instead of standard level (e.g. 10 units), the $RR_{standardised}$ was calculated by:

$$RR_{\text{standardised}} = RR_u^{\frac{increment\ unit\ (e.g.\ 10)}{u}}$$

where U is the increment used in the original study to estimate the effects.

For studies reporting the excess risk (ER), we transformed the ER to RR, using the formula: RR = 1 + ER. Some studies used $\mu g/m^3$ to describe the concentrations of gaseous pollutants; thus, we converted all estimates to ppb (Danish Centre For Environment And Energy).

For those pollutants where the effects were expressed for different exposure time metrics, such as one-hour or eight-hour instead of 24-h means, we converted the results to reflect a 24-h scale. This was the case for SO_2, NO_2 and CO. The relationship