Lifetime Air Pollution Exposure and Asthma in a Pediatric Birth Cohort

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Capsule Summary

In this area where air quality is within EPA standards, living close to a major road was associated with early- and mid-childhood asthma, and lifetime exposure to particles was associated with early asthma, and also, mid-childhood asthma among girls only.

Keywords

air pollution; traffic; asthma; wheeze; reactive airways; black carbon; particulate matter; childhood

To the Editor

While it is clear that ambient pollution triggers symptoms among asthmatics, there remains uncertainty about whether long-term exposure to pollution, at relatively low levels in the US, increases a child’s risk of developing asthma. While studies have generally concluded that...
pollution increases risk of asthma, the duration of exposure assessment varies substantially between these studies, and few have examined lifetime exposures. Some studies have concluded that ambient pollution primarily increases risk of early onset (by age 3) asthma and wheeze, while others have found associations that emerge later, at school-age. In Project Viva, a pre-birth cohort in the Boston area with lifetime residential histories, we have previously found that living closer to a major road, and lifetime exposure to particulate matter (PM$_{2.5}$), and black carbon (BC, a traffic-related PM$_{2.5}$ constituent) are associated with lower lung function in mid-childhood. We hypothesized that these exposures would be associated with asthma in early- and mid-childhood.

Study participants were 1,522 children whose mothers enrolled during their initial prenatal visit April 1999–July 2002. Further details about the cohort, exposure and outcome assessment, and statistical analyses are described in this article’s Online Repository. We geocoded each participant’s home address at birth and at each study visit and assigned distance to the nearest major roadway and census tract (year 2000) characteristics using ArcGIS. We examined categories of distance and also the natural logarithm of proximity to major roadway. We estimated daily BC exposure at home address using a validated spatiotemporal land-use regression model for traffic particles developed for the Boston area. We estimated daily community-level PM$_{2.5}$ by a hybrid model using satellite-derived measures of particle abundance and land use terms. We calculated first year of life and lifetime BC and PM$_{2.5}$ exposure, using each child’s time-averaged full residential history.

The following asthma-related outcomes were ascertained by maternal questionnaire: (1) early asthma/reactive airways: a doctor diagnosis of asthma, wheeze or reactive airway disease anytime between birth and the early childhood questionnaire (age 3–5); (2) recurrent wheeze at early childhood: wheezing in the past 12 months (at age 3–5) and wheezing at age 1 or 2; (3) ever asthma: a doctor diagnosis of asthma since birth, reported on the mid-childhood questionnaire (age 7–10); and (4) current asthma in mid-childhood: ever asthma and taking asthma medications or wheezing in the past 12 months.

We analyzed associations between exposures and asthma outcomes using logistic regression models, adjusting for potential confounders that we selected a priori. All models were adjusted for child sex, age, race/ethnicity, parental history of asthma, breastfeeding duration, birthweight for gestational age z score, bronchiolitis in infancy, current household income and smoking, census tract income and education (using 2000 U.S. census data), season of birth and date of visit. We tested whether associations between pollution and asthma differed by parental atopy, child sex and early atopic dermatitis, and individual- and neighborhood-level socioeconomic indicators, as described in the Online Repository.

Participant characteristics and exposures are described in Tables E1 and E2 in the Online Repository. Associations between measures of pollution exposure and asthma are shown in Table 1. Proximity to roadway, and exposure to BC and community-level PM$_{2.5}$ were all associated with early childhood (age 3–5) asthma/reactive airways. There were log-linear associations between proximity to roadway at birth (and also at the time of assessment) and odds of early asthma/reactive airways. Lifetime averages of BC and PM$_{2.5}$ had greater associations with odds of early asthma than exposures in the first year of life.
Proximity of the home to a major roadway at time of assessment, but not birth, was associated with mid-childhood (age 7–10) asthma overall and in sensitivity analyses confined only to those who moved (62%). There was a log-linear association of proximity to roadway with ever asthma. Children living < 100 m from a major road had nearly 3 times the odds of current asthma in mid-childhood compared to those >400 m away (OR 2.86, 95% CI 1.25, 6.55) (Table E3). BC and PM$_{2.5}$ exposures were not associated with mid-childhood asthma in primary analyses.

However, sex modified several associations between pollution and asthma in early- and mid-childhood (Figure 1). Among girls, but not boys, lifetime exposure to BC and PM$_{2.5}$ were each associated with greater odds of early and mid-childhood asthma. Children with parental atopy or early atopic dermatitis each had stronger associations between BC exposure and recurrent wheeze (Figure E1, Online Supplement). Complete results of tests of effect modification and additional sensitivity analyses are in the Online Supplement.

Our findings are in agreement with Brunst et al. (2015), who concluded that higher lifetime exposure to traffic-related pollution during childhood, not just early life exposure, increases risk of asthma. The large European ESCAPE study concluded that early life exposure to ambient pollution contributes disproportionately to asthma risk, but did not account for changing pollution levels and addresses over each child’s lifetime.

We found more consistent associations of pollution with early childhood asthma/reactive airways than with mid-childhood asthma. There may be less exposure misclassification for younger children, who generally spend a larger proportion of their time at home than school-aged children. Also, an inflammatory response to pollution may be more likely to produce wheeze in a younger child, whose airways are smaller. An important finding of this study is a consistent pattern of stronger associations between pollutant exposures and odds of asthma among girls compared to boys. While some have found boys to be more likely to wheeze by age 4 in association with pollution exposure, girls appear more likely to develop asthma in association with tobacco smoke exposure. It is plausible that differences in immune responses, including more exuberant inflammatory immune responses among girls, could explain the sex differences we found. Our observation that children with a parental history of atopy are more likely to wheeze in association with air pollution is consistent with other publications.

In this area where pollution levels are relatively low and within EPA standards, traffic-related pollution appears to increase risk of pediatric asthma, especially in early childhood.

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expressed in this article do not necessarily represent the views of the US Government, the Department of Health and Human Services or the National Institutes of Health.

**Abbreviations**

- **PM$_{2.5}$**: Particulate matter less than 2.5 microns in diameter
- **BC**: Black carbon

**References**

Sex-Specific Associations of Lifetime Pollution Exposure and Asthma

Results scaled per 0.2 μg/m$^3$ for BC, and per 2 μg/m$^3$ for community-level PM$_{2.5}$. Associations with PM$_{2.5}$ are adjusted for local primary PM$_{2.5}$ generated within 100 m of home. All models adjusted for child age, sex, race/ethnicity, breastfeeding duration, birth weight for gestational age z-score, infantile bronchiolitis, maternal and paternal asthma, household income and smoking, census tract income and education, season of birth and date of visit.

Figure 1.

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<table>
<thead>
<tr>
<th></th>
<th>Early Childhood (3–5 years)</th>
<th>Mid-Childhood (7–10 years)</th>
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<tbody>
<tr>
<td></td>
<td>N=1,444</td>
<td>N=1,242</td>
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<tr>
<td></td>
<td>Asthma/Reactive airways</td>
<td>Recurrent wheeze</td>
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<td></td>
<td>(OR (95% CI) per unit increase in exposure)</td>
<td>(OR (95% CI) per unit increase in exposure)</td>
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<tr>
<td>Proximity to Roadway</td>
<td></td>
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<tr>
<td>At birth</td>
<td><strong>1.21</strong> (1.00, 1.48)</td>
<td><strong>0.97</strong> (0.73, 1.27)</td>
</tr>
<tr>
<td>At visit</td>
<td><strong>1.22</strong> (0.99, 1.50)</td>
<td><strong>0.88</strong> (0.66, 1.18)</td>
</tr>
<tr>
<td>Black Carbon</td>
<td></td>
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<tr>
<td>1st year of life</td>
<td><strong>1.13</strong> (0.93, 1.38)</td>
<td><strong>1.14</strong> (0.87, 1.49)</td>
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<tr>
<td>Lifetime</td>
<td><strong>1.32</strong> (1.03, 1.69)</td>
<td><strong>1.26</strong> (0.91, 1.74)</td>
</tr>
<tr>
<td>Community-Level PM2.5</td>
<td></td>
<td></td>
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<tr>
<td>1st year of life</td>
<td><strong>2.25</strong> (1.17, 4.33)</td>
<td><strong>2.62</strong> (1.08, 6.34)</td>
</tr>
<tr>
<td>Lifetime</td>
<td><strong>2.35</strong> (1.12, 4.91)</td>
<td><strong>2.89</strong> (1.06, 7.85)</td>
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Home address location changed at least once after birth for 46% of participants by the early childhood visit, and for 62% of participants by the mid-childhood visit. Results scaled from the 75th to the 25th percentile of the log transformed distance to major roadway, per 0.2 μg/m$^3$ for BC, and per 2 μg/m$^3$ for community-level PM$^{2.5}$. Associations with PM$^{2.5}$ are adjusted for local primary PM$^{2.5}$ generated within 100 m of home. All models adjusted for child age, sex, race/ethnicity, breastfeeding duration, birth weight for gestational age z-score, infantile bronchiolitis, maternal and paternal asthma, household income and smoking, census tract income and education, season of birth and date of visit.