

Exposure to Air Pollution from Traffic and Childhood Asthma Until 12 Years of Age

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Background: There are limited prospective data on long-term exposure to air pollution and effects on childhood respiratory morbidity. We investigated the development of asthma and related symptoms longitudinally over the first 12 years of life in relation to air pollution from road traffic.

Methods: The Swedish birth cohort BAMSE (Children, Allergy, Milieu, Stockholm, Epidemiological Survey) includes 4089 children who were followed up with repeated questionnaires and blood samples for up to 12 years of age. Residential, daycare, and school addresses, time-activity patterns, emission databases, and dispersion models were used to estimate individual exposure to particulate matter with aerodynamic diameter $<10\ \mu\text{m}$ (PM_{10}) and nitrogen oxides (NO_x) from traffic.

Results: Overall, the data suggested possible associations between exposure to air pollution during the first year of life and asthma and wheezing in children up to 12 years of age. Asthma risks seemed to be particularly increased in children age 8 to 12 years; the overall odds ratio was 2.0 (95% confidence interval = 1.1–3.5), and for nonallergic asthma, the odds ratio was 3.8 (0.9–16.2) for a 5th to 95th percentile increase in time-weighted average exposure to PM_{10} (corresponding to $7.2\ \mu\text{g}/\text{m}^3$). Results were similar using exposure to traffic- NO_x .

Conclusions: We found modest positive associations between air pollution exposure from traffic during infancy and asthma in children during the first 12 years of life, with stronger effects suggested for nonallergic asthma.

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Respiratory tract symptoms are prevalent in children.¹ Several epidemiologic studies have found an association between long-term air pollution exposure and asthma^{2–6} or wheeze.^{2,7,8} However, other studies show no association between air pollution and respiratory symptoms.^{9–11} Differences in study design, and in exposure assessment and outcome definitions, may contribute to the apparently inconsistent findings. Furthermore, certain susceptibility factors may play a role, including early-life exposure, sex, and genetics.^{12–15}

Air pollution exposure during the first years of life seems to be particularly relevant to respiratory disorders later in childhood.^{8,13,16,17} Specific subtypes of asthma, such as allergic and nonallergic asthma, may be caused by air pollution, but results are inconsistent.^{4,8,13,18} Many studies have looked at the effects of air pollution on prevalence of asthma symptoms or their exacerbation, but only a few prospective studies have analyzed the contribution of air pollution exposure to the onset of disease.^{4,8,10} Given that the prevalence of respiratory symptoms changes with age, and that not all children who experience symptoms in early life develop asthma at older ages, it may be important to study both prevalence and incidence of asthma symptoms throughout childhood.

We have previously reported an association between exposure to traffic-related air pollution during the first year of life and development of respiratory symptoms at the age of 4 years in the population-based birth cohort BAMSE.⁸ Recently, the 12-year follow-up of this cohort was completed, which enables us to study the association between traffic-related air pollution and the development of asthma and related symptoms longitudinally up to 12 years of age.

METHODS

Study Population

The BAMSE study is based on a well-characterized cohort of children followed up prospectively from birth. Study design, recruitment, criteria for inclusion, and data

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collection procedures have been described elsewhere.¹⁹ In brief, 4089 children born between 1994 and 1996 in four municipalities in Stockholm County, representing urban and suburban environments, were enrolled. Data on residential characteristics and socioeconomic factors and parental allergic diseases, pet contact, and others were collected with a baseline questionnaire sent to parents when the child was approximately 2 months old. Repeated follow-ups were carried out at 1, 2, 4, 8, and 12 years; parents filled in similar questionnaires with a main focus on the children's symptoms related to wheezing and allergic diseases. Information on various exposures was also collected. Response rates were 96%, 94%, 91%, 84%, and 82%. Blood was obtained from 2614 children (64%) at the age of 4 years and from 2480 children (61%) at the age of 8 years. The study was approved by the ethical committee of Karolinska Institutet.

Air Pollution Exposure Assessment

The assessment of exposure to locally emitted air pollution from traffic was based on a successively refined methodology for estimating individual long-term source-specific exposure, as has been described in detail elsewhere.²⁰ In brief, the lifetime residential, daycare, and school addresses have been georeferenced, and time-weighted average outdoor levels of selected pollutants were calculated by use of emission inventories and dispersion models of the Stockholm and Uppsala Air Quality Management Association.

The local contribution to annual mean levels of traffic-PM₁₀ and traffic-NO_x (not including regional background) was calculated using a Gaussian air quality dispersion model and a wind model, both part of the Airviro Air Quality Management System (SMHI, Norrköping, Sweden; <http://airviro.smhi.se>). Air pollution concentrations were calculated for all addresses in the years 1994 to 2008, that is, from the year when the first child was born until the end of the 12-year follow-up. Emission databases for NO_x were available for the years 1990, 1995, 2000, 2002, 2003, 2004, 2006, and 2010. The model calculations of NO_x concentrations were interpolated to obtain estimates for all years during the period of interest. PM₁₀ model calculations were performed only for the year 2004 and applied to all years during the study period. There were 106 addresses in the most polluted street segments in the Stockholm inner city with multistory buildings on both sides. For these addresses, a street-canyon contribution was calculated using the SMHI-Airviro street-canyon model (<http://airviro.smhi.se>). To obtain air pollution levels that are comparable with measured levels of PM₁₀ and NO_x, the regional background of 10 µg/m³ PM₁₀ and 3 µg/m³ NO_x, respectively, should be added to our estimates.²¹

Definition of Health Outcomes

Wheezing was based on parental reporting in the questionnaires. We classified wheeze as one or more episodes and

as three or more episodes, during the 12 months before each questionnaire.

For children at 1 and 2 years, asthma was defined as three or more episodes of wheeze in combination with treatment with inhaled corticosteroids or signs of bronchial hyperactivity without concomitant respiratory infection. At 4, 8, and 12 years, at least four episodes of wheeze in the last 12 months, or at least one episode in combination with prescription of inhaled corticosteroids, was required for a diagnosis of asthma.²² Incident asthma was defined as asthma at a given age without having fulfilled the definition of asthma at any previous time. Incident wheeze was defined in the same way. Furthermore, children with asthma were classified as having allergic asthma if the child fulfilled the criteria of asthma at 4 or 8 years and was also sensitized to common inhalant allergens at the corresponding age and as having nonallergic asthma if not. Sensitization status was defined as blood immunoglobulin E (IgE) level of ≥0.35 kU_A/L to common inhalant allergens, analyzed with Phadiatop® (a mix of common inhalant allergens: birch, timothy, mugwort, cat, dog, horse, mold [*Cladosporium herbarum*], and house dust mite [*Dermatophagoides pteronyssinus*]).

Statistical Analyses

We assessed associations between air pollution exposure and asthma outcomes combined with sensitization using multinomial logistic regression; the results are presented as odds ratios (ORs) with 95% confidence intervals (CIs). Longitudinal associations between air pollution levels and repeated questionnaire reports of binary asthma outcomes were analyzed using logistic regression models applying generalized estimating equations with stationary correlation structure to account for correlations among repeated observations in the same subject.²³ The models incorporated terms for an interaction between time indicator variable and exposure to evaluate the effect of exposure over time. To assess effect modification by sex and allergic heredity, we entered into the models variables representing the product of air pollutant exposure and the potential modifiers. All results are presented across the same unit of change in exposure (value of 5th to 95th percentile difference in the estimated outdoor levels during the first year of life, which was 7.2 µg/m³ for PM₁₀ and 46.8 µg/m³ for NO_x) to make the associations comparable across the various exposure periods. The final models were adjusted for covariates that were selected based on earlier literature and changed the OR by >5%: municipality, socioeconomic status, heredity (mother or father with doctor's diagnosis of asthma and asthma medication or with doctor-diagnosed hay fever in combination with furred animals or pollen allergy), and the year the house was built. Parental socioeconomic status was classified as blue-collar, white-collar, and others (students, unemployed) according to the Nordic standard occupational classification and the Swedish socioeconomic classification.²⁴

Exposure time windows were defined as the first year of life, current exposure (12 months before the date of a questionnaire), and average exposure since the date of the previous follow-up. For analyses of the effects of exposure during infancy, 3633 children (89%) were included with information on exposure, confounders, and outcomes under study at a minimum three of five time points. There were

3477 subjects (85%) available for analyses of the effects of average air pollution exposure since the previous follow-up. A total of 2518 and 2378 children were included in the analyses of asthma in combination with IgE sensitization at the ages of 4 and 8 years, respectively. All statistical analyses were performed with STATA Release 11.1 (StataCorp, College Station, TX).

RESULTS

Table 1 shows the distribution of selected risk factors by asthma status across the first 12 years of life. Children who had ever experienced asthma symptoms up to 12 years of age more often came from families of blue-collar workers and more often had parents with allergies. The distribution of risk factors was similar in the study population and the original cohort (eTable 1; <http://links.lww.com/EDE/A625>). For the first five time periods of life (the first year of life, second year of life, and averages of 2–4, 4–8, and 8–12 years), the mean local contributions above regional background to the children's outdoor air pollution exposure levels were 4.2, 3.9, 3.6, 3.5, and 4.6 $\mu\text{g}/\text{m}^3$ for traffic- PM_{10} and 21.4, 18.6, 13.4, 10.6, and 7.8 $\mu\text{g}/\text{m}^3$ for traffic- NO_x (Fig. 1). Children who lived more centrally (Stockholm municipality), in older buildings, and who came from families of high socioeconomic status, experienced an average higher air pollution levels (data not shown). The correlations between estimated PM_{10} and NO_x concentrations in the various time periods were high, and therefore, multipollutant models were not feasible (eg, $r = 0.96$ between NO_x and PM_{10} exposure levels during the first year of life).

A total of 3.9%, 5.9%, 7.0%, 6.2%, and 6.7% of the children included in the analyses met the asthma definition criteria at 1, 2, 4, 8, and 12 years of age, respectively (Table 2). Among asthma cases, 34% were sensitized to common inhalant allergens at the age of 4 years and 58% at the age of 8 years. At least one episode of wheezing during the last 12 months was

TABLE 1. Distribution of Risk Factors at Birth by the Asthma Status During the First 12 Years of Life in the BAMSE Cohort

Covariates	No Asthma Ever No. (%)	Asthma Ever ^a No. (%)
Socioeconomic status of parents ^b		
Blue-collar workers	371 (15)	126 (20)
White-collar workers	2,123 (84)	491 (78)
Other	20 (1)	13 (2)
Year the house was built ^c		
Before 1940	865 (34)	160 (25)
1940–1975	1,007 (40)	281 (45)
After 1975	647 (26)	192 (30)
Heredity ^d		
No parental allergy or asthma	1,791 (72)	361 (58)
One parent with allergy or asthma	644 (26)	218 (35)
Both parents with allergy or asthma	59 (2)	43 (7)
Municipality ^e		
Stockholm	817 (33)	148 (24)
Järfälla	683 (27)	222 (35)
Solna	663 (26)	144 (23)
Sundbyberg	348 (14)	116 (18)

^aA child fulfills asthma definition at least at one of the follow-up occasions.

^bData available for 2,514 in first column and 630 in second column.

^cData available for 2,519 in first column and 633 in second column.

^dData available for 2,494 in first column and 622 in second column.

^eData available for 2,511 in first column and 630 in second column.

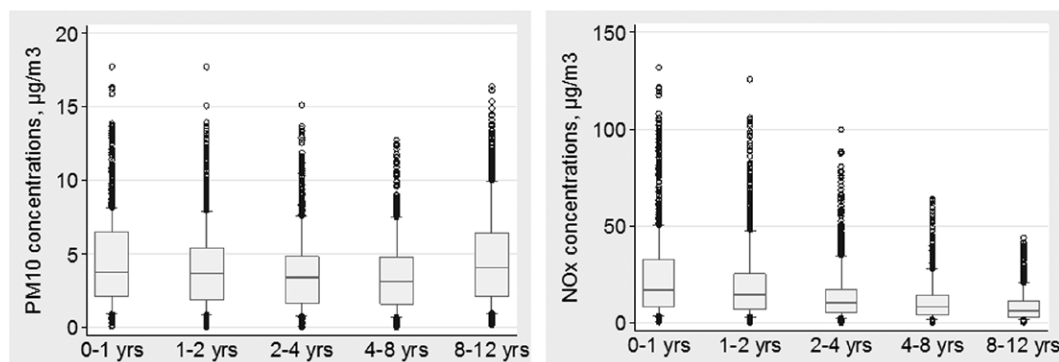


FIGURE 1. Distribution of the estimated average air pollution exposure levels up to 12 years of life in children from the BAMSE birth cohort in Stockholm (recruited from 1994 to 1996). Each box contains the middle 50% of the data or the interquartile range. The upper edge (hinge) of the box indicates the 75th percentile and the lower one the 25th percentile. The line in the box represents the median. The ends of the vertical lines ("whiskers") indicate 5th and 95th percentiles. Individual values outside this range are marked. Exposure time intervals: 0–1 yrs, average exposure between birth and 1 year of age; 1–2 yrs, average exposure between 1 and 2 years of age; 2–4 yrs, average exposure between 2 and 4 years of age; 4–8 yrs, average exposure between 4 and 8 years of age; 8–12 yrs, average exposure between 8 and 12 years of age.

TABLE 2. Distribution of Health Outcomes in Children up to 12 Years of Age in the BAMSE Birth Cohort in Stockholm ($n = 3,633$)

Outcome	Prevalent Cases ^a			Incident Cases ^b		
	Total No. (%)	Girls No.	Boys No.	Total No. (%)	Girls No.	Boys No.
Asthma						
1 year	139 (4)	53	86	139 (4)	53	86
2 years	211 (6)	86	125	132 (4)	54	78
4 years	247 (7)	92	155	132 (4)	51	81
8 years	203 (6)	82	121	91 (3)	41	50
12 years	213 (7)	77	136	82 (3)	33	49
Wheeze (≥ 1 episode)						
1 year	528 (15)	212	316	528 (15)	212	316
2 years	668 (19)	274	394	380 (13)	163	217
4 years	520 (15)	215	305	190 (7)	81	109
8 years	326 (10)	132	194	93 (4)	45	48
12 years	292 (9)	113	179	72 (4)	38	34
Wheeze (≥ 3 episodes)						
1 year	246 (7)	104	142	246 (7)	104	142
2 years	365 (10)	146	219	240 (7)	95	145
4 years	161 (4)	60	101	65 (2)	28	37
8 years	113 (5)	46	67	50 (2)	23	27
12 years	166 (5)	59	107	72 (3)	29	43

Percentages calculated in relation to the total number of subjects who contributed to the analyses at the respective occasion.

^aPrevalent cases defined as total number of cases at respective follow-up occasion.

^bIncident cases defined as first-time outcome in the respective age without fulfilling the definition at any previous time point.

reported by approximately 15% during the first 4 years of life, with a decreasing trend to the age of 12 years. The incidence of wheeze symptoms seemed to be highest during the first 2 years of life. All symptoms were more prevalent in boys than girls.

The associations between air pollution exposure and wheeze and asthma are presented in Figures 2 and 3. Detailed quantitative results are found in eTable 2 (<http://links.lww.com/EDE/A625>). Exposure during the first year of life tended to have positive overall effects on both prevalent and incident wheeze and asthma symptoms during the first 12 years of life; the OR for a 5th to 95th difference in traffic-PM₁₀ exposure and prevalence of asthma was 1.3 (95% CI = 0.7–2.2) and for at least one episode of wheeze was 1.2 (95% CI = 0.8–1.6). The association seemed strongest for asthma at 12 years of age (2.0 [1.1–3.5]). The same pattern was seen for asthma incidence.

Table 3 shows the association between exposure to air pollution during the first year of life and development of allergic and nonallergic asthma symptoms at 4 and 8 years of age. Air pollution exposure during infancy was associated with nonallergic asthma at 8 years of age (ORs for a 5th to 95th percentile difference in traffic-PM₁₀ = 3.8 [95% CI = 0.9–16]). A suggestion of increased risk was observed at the age of 4 years (1.6 [0.5–5.3]). This association seemed stronger when using traffic-NO_x as an indicator of exposure (2.4 [1.0–5.6]). No significant interactions with air pollution were seen for sex ($P = 0.21$) or heredity ($P = 0.57$ and 0.48, for one and two affected parents, respectively).

No overall effect was observed with average air pollution exposure since the date of previous follow-up (ORs for a 5th to 95th difference in traffic-PM₁₀ exposure = 0.9 [95% CI = 0.6–1.3] and 0.9 [95% CI = 0.6–1.4] for prevalent and incident asthma, respectively; Fig. 3). Similar absence of effects was seen in relation to exposure during the preceding 12 months (data not shown).

DISCUSSION

In our study, we found a suggestion of positive associations between exposure to traffic-related air pollution during infancy and overall risks of respiratory symptoms in children over the first 12 years of life. Associations seemed strongest for the oldest children and for nonallergic asthma. No overall association was observed between air pollution exposure after the first year of life and development of asthma symptoms.

This study is an extension of previous analyses within the BAMSE cohort at 4 years, in which exposure to traffic-related air pollution was associated with various indicators of airway disease, and in particular, with persistent wheeze.⁸ In the current analyses, we have added information on air pollution exposure at subsequent home addresses, and at daycare and school addresses, together with health data from repeated questionnaires up to 12 years of age and clinical examinations at 4 and 8 years. Response rates were high during the entire follow-up, with 82% of the subjects continuing in the study at the age of 12 years. Availability of health data from the

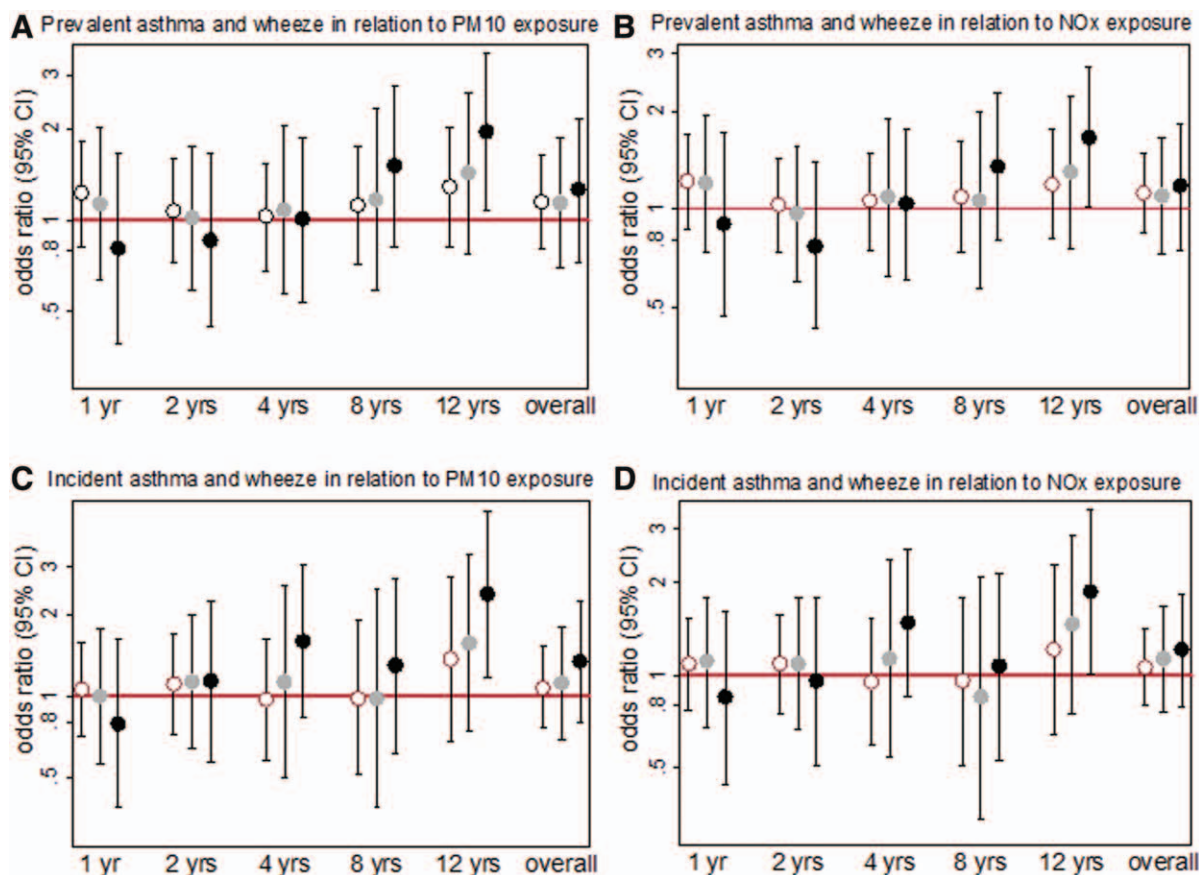


FIGURE 2. A–D, Associations between exposure to PM₁₀ and NO_x from road traffic during the first year of life and prevalent or incident asthma and wheeze during the first 12 years of life among children in the BAMSE birth cohort in Stockholm ($n = 3,633$). Odds ratios are calculated for a 5th to 95th percentile difference in the air pollution exposure levels during the first year of life, corresponding to 7.2 $\mu\text{g}/\text{m}^3$ for PM₁₀ and 46.8 $\mu\text{g}/\text{m}^3$ for NO_x. Adjusted for municipality, socioeconomic status, heredity, and the year the house was built. • Wheeze ≥ 1 episode; • wheeze ≥ 3 episodes; asthma.

repeated questionnaires at several points in time allowed us to assess incident disease and longitudinal effects. Furthermore, objective measures, such as serum levels of IgE, were obtained from more than 60% of the original cohort at 4 and 8 years, which enabled us to investigate various asthma phenotypes.

It has been suggested that timing of the exposure might be important for childhood respiratory morbidity.²⁵ We investigated several time aspects of long-term exposure, including early-life exposure and current exposure (during the last year), and average exposure between follow-ups. In general, the strongest effect was observed in relation to exposure during infancy, which is in line with previous studies indicating that prenatal and early-life periods represent critical windows for the effects of exposure on development of childhood asthma and related symptoms.^{8,13,16,17,26} An alternative explanation might be that the exposure classification method based on home address has less error early in life, before children start to go to daycare and schools.

Our findings suggest that traffic-related air pollution primarily increases the risk of nonallergic asthma symptoms. Experimental research suggests that such effects may be

linked to changes in the formation of reactive oxygen species, alterations in antioxidant defense, and increased nonallergic inflammation.²⁷ Furthermore, other epidemiologic studies have also shown higher risks of nonallergic asthma in relation to air pollution exposure compared with allergic asthma,^{4,13} supporting the hypothesis that air pollutants stimulate non-specific irritative rather than allergic inflammatory changes in the airways. Compared with allergic asthmatics, nonallergic asthmatics have a higher sensitivity of the nasal and bronchial epithelia to nonallergic stimuli such as air pollutants, strong smells, cold air, respiratory viruses, and others.²⁸ Children with an allergic predisposition may spend more time indoors compared with those without an allergic predisposition, and therefore, their exposure to air pollution could differ.²⁹

The evidence regarding effects of long-term exposure to air pollution on respiratory symptoms is limited because only a few studies have investigated the development of disease up to school age, with estimates of outdoor air pollution exposure based on small spatial scales and by timing exposure before the development of symptoms. A cohort study in the Netherlands demonstrated positive associations between levels of

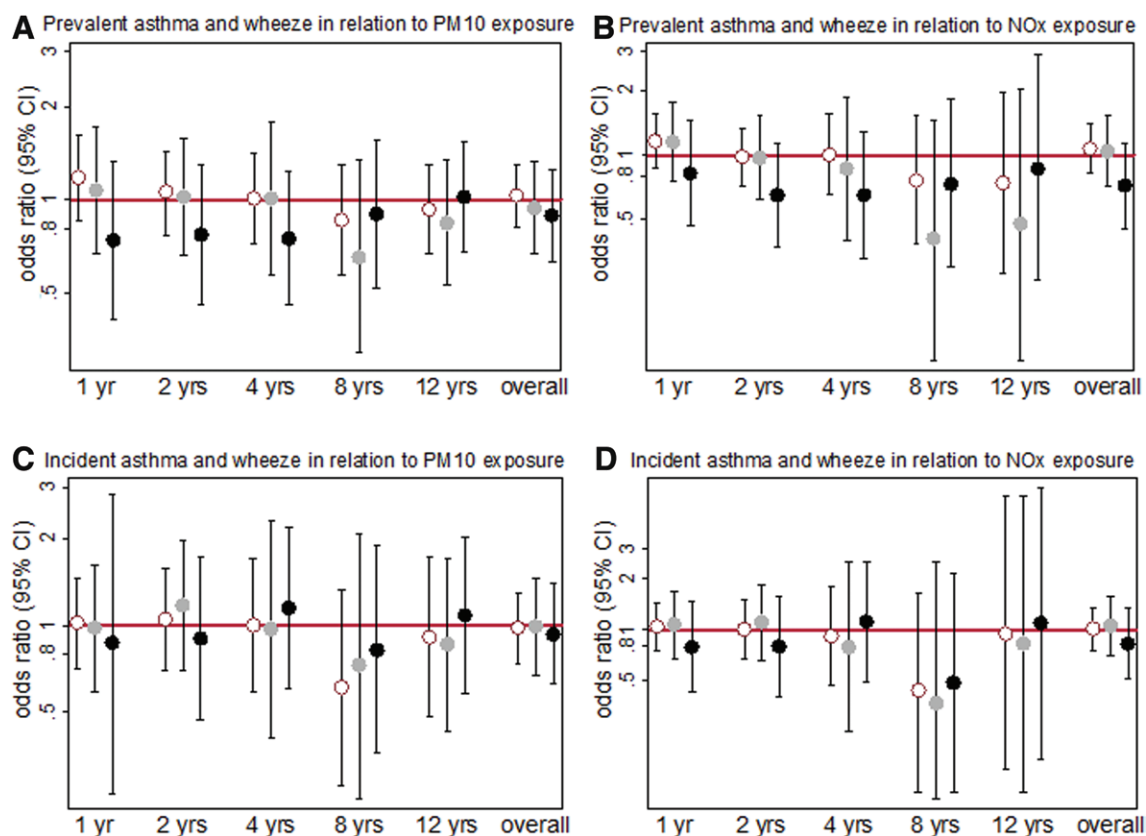


FIGURE 3. A–D, Associations between average exposure to PM₁₀ and NO_x from road traffic since the previous follow-up and prevalent or incident asthma and wheeze during the first 12 years of life among children in the BAMSE birth cohort in Stockholm ($n = 3,477$). Odds ratios are calculated for a 5th to 95th percentile difference in the air pollution exposure levels during the first year of life, corresponding to 7.2 $\mu\text{g}/\text{m}^3$ for PM₁₀ and 46.8 $\mu\text{g}/\text{m}^3$ for NO_x. Adjusted for municipality, socioeconomic status, heredity, and the year the house was built. • Wheeze ≥ 1 episode; • wheeze ≥ 3 episodes; asthma.

TABLE 3. Associations Between Exposure to PM₁₀ and NO_x During the First Year of Life and Asthma in Relation to Sensitization at 4 and 8 Years of Age in the BAMSE Cohort in Stockholm

	No.	PM ₁₀		NO _x	
		Crude ^a OR (95% CI)	Adjusted ^b OR (95% CI)	Crude ^a OR (95% CI)	Adjusted ^b OR (95% CI)
At age 4 years					
No asthma and no sensitization ^c	2,008	1.0	1.0	1.0	1.0
Nonallergic asthma	126	1.5 (0.5–4.8)	1.6 (0.5–5.3)	2.1 (0.9–5.0)	2.4 (1.0–5.6)
Allergic asthma ^d	65	1.3 (0.3–5.9)	1.4 (0.3–6.8)	1.3 (0.4–4.4)	1.5 (0.4–5.1)
At age 8 years					
No asthma and no sensitization ^c	1,692	1.0	1.0	1.0	1.0
Nonallergic asthma	73	3.1 (0.7–13.1)	3.8 (0.9–16.2)	2.3 (0.7–7.1)	2.6 (0.9–8.1)
Allergic asthma ^d	100	1.0 (0.3–3.5)	1.1 (0.3–3.8)	0.7 (0.2–2.2)	0.8 (0.2–2.4)

^aOdds ratios are calculated with adjustment for municipality only (design variable) for a 5th to 95th percentile difference in the air pollution exposure levels during the first year of life, corresponding to 7.2 $\mu\text{g}/\text{m}^3$ for PM₁₀ and 46.8 $\mu\text{g}/\text{m}^3$ for NO_x.

^bOdds ratios adjusted for municipality, socioeconomic status, heredity, and the year the house was built.

^cReference category.

^dSensitization to inhalant allergens is defined as an IgE antibody level of ≥ 0.35 kU_A/L.

traffic-related air pollution at the birth address and incidence and prevalence of asthma in children who were followed up from birth until 8 years of age. (For an interquartile range difference in $PM_{2.5}$ exposure at birth address corresponding to $3.2 \mu g/m^3$, the OR for prevalence and incidence of asthma was 1.26 [95% CI = 1.04–1.51] and 1.28 [95% CI = 1.10–1.49], respectively).⁴ The authors observed similar effects for NO_2 and soot. In contrast, a Norwegian study did not find any association of either early-life or lifetime average traffic-related exposures with asthma onset or with current respiratory symptoms in 9- to 10-year-old children in Oslo (a difference in NO_2 exposure during the first year of life, equivalent to $27.3 \mu g/m^3$, was associated with a risk of 0.82 [95% CI = 0.67–1.02], for doctor-diagnosed asthma onset).¹⁰ We found no overall association between average air pollution exposure after the first year of life and asthma symptoms, which could be partly attributable to declining trends in air pollution levels throughout the study period. The introduction of catalytic converters in private cars during the early 1990s, which coincided with the initiation of our cohort, led to a subsequent reduction of the emissions for several of the air pollutants—a decline that continued as older vehicles were replaced.³⁰ In the Norwegian study with levels of outdoor air pollution comparable to those in Sweden, it has been suggested that the exposure levels might have been too low to identify notable associations with asthma symptoms.¹⁰

Our exposure assessment methodology aimed to estimate individual exposure to exhaust particles and road dust, which have fundamentally different origins and characteristics. We used NO_x as an indicator for exhaust particles because NO_x (or NO_2) levels in the Stockholm area have been shown to correlate well with both soot³¹ and particle number concentrations,³² whereas PM_{10} mostly represents particles mechanically generated from road dust.³³ For the current study, the individual exposure estimates for each study subject were obtained from a time- and space-resolved dispersion model enhanced by addition of street-canyon contribution for addresses in the most polluted street segments. Cross-validation of dispersion models against ambient measurements suggests fairly good model performance in predicting ambient concentrations of traffic-related pollutants.³⁴ The exposure assessment was also enhanced by including not only residential addresses but also addresses of daycares and schools.

There are several limitations in this study. One is that PM_{10} concentrations could be calculated only for the year 2004. However, studded tire use in the Stockholm area and traffic work in the inner city have been stable during the last decades, suggesting that the emissions of PM_{10} may not have changed much.³³ Furthermore, the influence of road moisture on the yearly variations of PM_{10} concentrations, which has crucial impact on PM_{10} , could not be resolved with available data.³⁵ In the current study, PM_{10} and NO_x concentrations were highly correlated. Therefore, we were not able to separate the

effects of these pollutants. Some misclassification of air pollution exposure is possible, partly because modeling of air pollution may give error in the exposure estimates and because outdoor exposure may not measure true personal exposure, although children (especially infants) are likely to spend most of their time in their own neighborhoods. In addition, because all traffic-related exposures were assessed independently from health outcomes and potential confounders, systematic error is unlikely, and random error would probably attenuate exposure-response associations.

Misclassification of the outcome is often an issue in studies in which asthma and wheeze are assessed by parental questionnaires, particularly in young children who have less distinctive symptoms. Furthermore, reporting could be influenced by differing diagnostic practices among doctors. Childhood asthma is not a homogeneous disease, and various phenotypes of wheeze and asthma have been defined based on prognosis, allergic status, and lung function.^{36,37} Questionnaire reports have generally been used in large epidemiologic studies for which more accurate diagnostics are often not feasible, and asthma is usually defined according to a certain number of wheezing episodes in the last 12 months. In young children, the term asthma is often replaced by wheezing, which may also include symptoms over time and classifications such as transient, persistent, or late-onset wheezing.³⁸ Because many children who experience wheeze early in life will outgrow their symptoms, manifest asthma is difficult to define before school age.³⁹ In this study, we observed the highest risk of asthma related to air pollution exposure in older children (ie, 8 or 12 years) when asthma presumably can be defined more reliably. Similar trends were also seen in the Prevention and Incidence of Asthma and Mite Allergy study, in which the strongest associations between early exposure to air pollutants and asthma were seen in 7- to 8-year-old children.⁴ Further studies are, however, needed to confirm that exposure to air pollutants early in life is associated with asthma development in school age.

In conclusion, the current study found a suggestion of positive overall associations between exposure to air pollution from road traffic during the first year of life and risk of respiratory symptoms in children over the first 12 years of life, with the strongest effect suggested for nonallergic asthma.

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