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Traffic-related air pollution and noise and children's blood pressure: Results from the PIAMA birth cohort study

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Abstract

Aims: Elevation of a child's blood pressure may cause possible health risks in later life. There is evidence for adverse effects of exposure to air pollution and noise on blood pressure in adults. Little is known about these associations in children. We investigated the associations of air pollution and noise exposure with blood pressure in 12-year-olds.

Methods: Blood pressure was measured at age 12 years in 1432 participants of the PIAMA birth cohort study. Annual average exposure to traffic-related air pollution [NO_2, mass concentrations of particulate matter with diameters of less than 2.5 μm ($PM_{2.5}$) and less than 10 μm (PM_{10}), and $PM_{2.5}$ absorbance] at the participants' home and school addresses at the time of blood pressure measurements was estimated by land-use regression models. Air pollution exposure on the days preceding blood pressure measurements was estimated from routine air monitoring data. Long-term noise exposure was assessed by linking addresses to modelled equivalent road traffic noise levels. Associations of exposures with blood pressure were analysed by linear regression. Effects are presented for an interquartile range increase in exposure.

Results: Long-term exposure to NO_2 and $PM_{2.5}$ absorbance were associated with increased diastolic blood pressure, in children who lived at the same address since birth [adjusted mean difference (95% confidence interval) [mmHg] 0.83 (0.06 to 1.61) and 0.75 (−0.08 to 1.58), respectively], but not with systolic blood pressure. We found no association of blood pressure with short-term air pollution or noise exposure.

Conclusions: Long-term exposure to traffic-related air pollution may increase diastolic blood pressure in children.

Keywords

Air pollution, birth cohort, blood pressure, children, traffic noise

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Introduction

Cardiovascular diseases are the main cause of death in Europe, and high blood pressure is a major risk factor of cardiovascular disease.[1] There is growing concern about the medical and economic burden associated with elevated childhood pressure[2] as it can persist into adulthood and is associated with cardiovascular disease later in life.[3]

The impact of ambient air pollution on blood pressure, so far, has been mainly investigated in adults. Most of these adult studies investigated the association between short-term changes in air pollution levels and blood pressure (see, for example, references 4–9).

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Findings of these studies suggest that short-term increases in particulate matter can lead to acute increases in systolic and diastolic blood pressure. Fewer adult studies investigated the association between long-term exposure to air pollution and blood pressure in adults and provided evidence for a positive association between long-term air pollution exposure and blood pressure.[10–12]

Only two studies, so far, investigated the effect of traffic-related air pollution on childhood blood pressure. A study amongst 10-year-old school children living in two areas of Lahore, Pakistan with markedly different levels of particulate matter air pollution, found a substantially higher systolic and diastolic blood pressure in children living and attending school in the area with high compared to low levels of traffic-related air pollution.[13] In a cohort study comparing 6–13-year-old children from Mexico City exposed to high air pollution concentrations with children from a control city with lower levels of air pollution, a significant association was found between short-term 7-day cumulative levels of particulate matter with diameters of less than 2.5 μm ($PM_{2.5}$) and mean pulmonary arterial pressure.[14]

Motorized traffic is not only an important source of air pollution but also an important source of community noise.[15,16] Associations of chronic aircraft and road traffic noise exposure with childhood blood pressure have been examined in several, mainly cross-sectional studies. A recent review of the literature suggests that, in children, the evidence for a positive association with noise is stronger for systolic compared to diastolic blood pressure.[17]

Since very little is known about the effects of traffic-related air pollution on blood pressure in children and because air pollution and noise share traffic as a common source and have both been suggested to be positively associated with blood pressure, we investigated the associations of short- and long-term exposures to air pollution, as well as long-term exposure to traffic noise with blood pressure in 12-year-old children from the Prevention and Incidence of Asthma and Mite Allergy (PIAMA) birth cohort study.

Methods

Study design

The methods of the PIAMA birth cohort study have been described elsewhere.[18] In brief, pregnant women were recruited during their second trimester of pregnancy in 1996–1997 from a series of communities in the north, west, and centre of the Netherlands. The study started with 3963 participants. Postal questionnaires were sent to the parents during pregnancy, at the child's ages of 3 months and 1 year, and yearly thereafter up to the age of 8 years. At the age of 11 years, the parents and children received separate questionnaires. A medical examination including measurements of blood pressure took place during a home visit at the age of about 12 years. None of the children had evidence of acute infection on the day of the medical examination. The institutional review board of the participating institutes approved the study protocol, and written informed consent was obtained from all participants.

Long-term air pollution exposure assessment

Air pollution concentrations at the participants' home and school addresses at the time of blood pressure measurements were estimated by Land-Use Regression (LUR) models described elsewhere.[19] In brief, air pollution monitoring campaigns were performed between February 2009 and February 2010 in the study area. Three 2-week measurements of nitrogen dioxide (NO_2) were performed within that year at 80 sites during the warm, cold, and one intermediate season. Simultaneous measurements of 'soot' (determined as the reflectance of $PM_{2.5}$ filters), $PM_{2.5}$, and PM_{10} were performed at half of the sites.[20,21] Results from the three measurements were averaged.[21] Predictor variables on nearby traffic, population/household density and land use derived from Geographic Information Systems (GIS) were evaluated to explain spatial variation of annual average concentrations. LUR models were then used to estimate air pollution concentration at the participants' addresses, for which the same GIS predictor variables were collected.

Short-term air pollution exposure assessment

We used daily data on NO_2, PM_{10}, and ozone from routine background monitoring sites of the National Air Quality Monitoring Network of the National Institute for Public Health and the Environment (RIVM) located in the study area to estimate short-term exposures. Short-term exposure was defined as the average of the air pollution levels on the 7 days preceding blood pressure measurements at the monitoring site closest to a participant's home. Examples of time series of daily air pollution levels at selected sites are presented in Supplementary Figure S1.

Traffic noise exposure assessment

Road traffic is the major source of noise in our study area. Road traffic noise at the participants' home and school addresses at the time of blood pressure

measurements was estimated using the EMPARA noise mapping model for the Netherlands (resolution 25×25 m). Traffic noise was modelled as yearly average A-weighted day-evening-night 24-hour sound level (L_{den} dB(A)).[22] Input variables for the model are traffic intensity, traffic composition and traffic speed, distance to roads, land use, location of noise barriers, and quiet asphalt.[22] The most recent input data (which is representative for the year 2008) were used. Since, in general, the daily variation in noise levels is small in comparison with the variation in the Lden between study subjects,[23] associations with short-term noise exposures were not considered.

Blood pressure measurements

Blood pressure was measured in 1432 children using automatic blood pressure meters (Omron M6, Omron Healthcare Europe BV, Hoofddorp, The Netherlands) according to the recommendations of the American Heart Association.[24] The cuff (15–22 cm (small) or 22–32 cm (normal) dependent on the mid-upper arm circumference) was placed at the non-dominant arm. Systolic and diastolic blood pressure were measured at least twice with 5 minutes intervals according to a standard protocol while the child was seated. We used the mean of the measures in the present analysis.

Confounders

The following variables were selected a priori based on a literature review and considered as potential confounders: gender; age at time of blood pressure measurements; height and body mass index (BMI) defined as weight in kilograms divided by height squared in metres as measured at the time of blood pressure measurement (children were weighed and measured in their underwear by trained research staff); cuff size (small/normal); gestational age at birth and birthweight; weight gain during the first year of life as reported in the 1-year questionnaire, physical activity defined as number of days per week with at least 1 hour of sporting activity and puberty development scale[25] as reported by the child in the 11-year questionnaire; maternal education categorized as low, medium, and high; maternal smoking during pregnancy, parental smoking in the child's home, breastfeeding; maternal hypertension during pregnancy, respiratory infections (pneumonia and/or otitis media during the first 2 years of life as reported in the 1- and 2-year questionnaires); and ambient and room temperature. Daily data on ambient temperature were obtained from the national network of the Royal Dutch Meteorological Institute and averaged for the 7 days preceding blood pressure measurements. Room temperature was measured during the medical examination.

Statistical analysis

We compared the distributions of baseline characteristics between subsamples of children that were and were not included in the present study using Student's t-test for continuous and chi-square test for categorical variables. We investigated the functional unadjusted relationships between exposures and blood pressure variables by means of smoothing splines (GAM function; The R Project for Statistical Computing 2.8.0, www.r-project.org). For noise we assumed that only levels above 45 dB(A) would have an effect. Smoothing parameters were estimated by the Generalized cross-validation criterion using the mgcv-package. Since most of the associations were found to be linear or almost linear (Supplementary Figures S2 and S3), associations of systolic and diastolic blood pressure with air pollution and noise exposure were analysed by means of linear regression analyses with non-transformed exposures. For each exposure variable, we performed separate crude and adjusted regression analyses taking into account the potential confounders listed above. Associations are expressed as change in blood pressure per interquartile range increase in exposure. We performed stratified analyses by sex to investigate differential susceptibility of boys and girls to the effects of air pollutants and noise, and stratified analyses by changes of address since birth to investigate the relevance of chronic (non-movers) versus more recent exposures (movers). Moreover, we repeated our analysis without children with doctor-diagnosed asthma during the past 12 months and doctor-diagnosed diabetes ever (as reported in the 11-year questionnaire). Statistical analyses, except smoothing splines, were performed with the Statistical Analysis System version 9.2 (SAS, Cary, NC, USA) for Windows.

Results

We were able to geocode home addresses and assign air pollution and noise exposures for 1400 of the 1432 children with blood pressure measurements. Distributions of blood pressure and population characteristics are shown in Table 1. Participants included in the present study more often had highly educated mothers than non-participants (Supplementary Table S1).

Air pollution, noise, and temperature

The distributions of air pollution exposure variables, traffic noise, and temperature are presented in Table 2 and Supplementary Table S2. Long-term air pollution

Table 1. Distribution of systolic and diastolic blood pressure and potential confounders in the study population

	N	Study population
Systolic blood pressure (mmHg)	1400	114.8 ± 9.2 (92.0–149.0)
Diastolic blood pressure (mmHg)	1400	66.7 ± 6.5 (43.0–90.0)
Age (years)[a]	1400	12.7 ± 0.4 (12.0–13.8)
Height (cm)[a]	1399	160.1 ± 7.8 (134.3–193.2)
BMI (kg/cm^2)[a]	1397	18.7 ± 2.7 (11.8–33.7)
Puberty development scale	1361	1.5 ± 0.5 (1.0–4.0)
Gestational age at birth (weeks)	1398	39.9 ± 1.6 (28.7–43.6)
Birthweight (g)	1398	3532 ± 528 (1370–5000)
Weight gain during 1st year (g)	1349	6271 ± 995 (2790–14,000)
Female sex	1400	713 (50.9)
Maternal education	1398	
Low		239 (17.1)
Intermediate		583 (41.7)
High		576 (41.2)
Maternal hypertension during pregnancy	1162	126 (10.0)
Breastfeeding at 3 months	1398	790 (56.5)
Maternal smoking during pregnancy	1391	186 (13.4)
Pneumonia and/or otitis media during first 2 years of life	1368	471 (34.4)
Parental smoking in child's home	1365	148 (10.8)
No. of days with ≥1 h physical activity	1367	
0–2		164 (12.0)
3+		1203 (88.0)
Doctor-diagnosed asthma in the past 12 months	1347	55 (4.1)
Doctor-diagnosed diabetes ever	1363	4 (0.3)
Change of address since birth	1400	831 (59.4)

Values are mean ± SD (range) or *n* (%).; [a]At blood pressure measurement.

Table 2. Distribution of long-term air pollution and noise levels, short-term air pollution levels, and ambient and room temperature at the home addresses

	N	Minimum	Median	Maximum	IQR
Long-term air pollution					
NO_2 ($\mu g/m^3$)	1400	9.6	21.8	40.0	17.5–25.3
$PM_{2.5}$ absorbance ($10^{-5}/m$)	1400	0.8	1.2	2.1	1.0–1.3
$PM_{2.5}$ ($\mu g/m^3$)	1400	14.9	16.5	19.3	15.6–16.7
PM_{10} ($\mu g/m^3$)	1400	23.7	24.5	30.1	24.0–25.0
Short-term air pollution					
NO_2 ($\mu g/m^3$)	1400	4.0	16.6	78.8	12.0–25.3
PM_{10} ($\mu g/m^3$)	1400	8.9	21.4	73.6	16.6–27.7
O_3 ($\mu g/m^3$)	1400	4.9	39.6	78.9	27.6–51.2
Traffic noise (L_{den} dB(A))	1400	45.0	53.0	70.5	50.3–56.1
Temperature					
Ambient temperature (°C)	1400	−7.7	9.2	24.8	4.5–15.0
Room temperature (°C)	1380	15.0	21.0	28.7	19.9–22.7

IQR, interquartile range.

Table 3. Spearman correlation coefficients between air pollution and traffic noise exposure variables, and temperature

	Long-term air pollution				Short-term air pollution				Temperature	
	NO_2	$PM_{2.5}$ abs	$PM_{2.5}$	PM_{10}	NO_2	PM_{10}	O_3	Traffic noise	Ambient	Room
Long-term air pollution: home										
NO_2	1.00									
$PM_{2.5}$ absorbance	0.89	1.00								
$PM_{2.5}$	0.67	0.82	1.00							
PM_{10}	0.77	0.88	0.56	1.00						
Short-term air pollution										
NO_2	0.46	0.42	0.31	0.27	1.00					
PM_{10}	0.15	0.14	0.13	0.07	0.54	1.00				
O_3	−0.03	−0.04	−0.06	−0.01	−0.53	−0.21	1.00			
Traffic noise – home	0.45	0.47	0.52	0.51	0.11	0.01	0.01	1.00		
Temperature										
Ambient temperature	0.13	0.14	0.15	0.10	−0.37	−0.18	0.54	0.09	1.00	
Room temperature	−0.03	−0.05	−0.05	−0.01	−0.35	−0.15	0.44	0.00	0.59	1.00

($r = 0.79$–0.91), but not noise levels ($r = 0.34$) at home and school addresses were highly correlated. Correlations between air pollution, noise, and temperature variables are presented in Table 3. The annual average NO_2, PM, and $PM_{2.5}$ absorbance levels were highly correlated ($r = 0.65$–0.89). Correlations between long-term and short-term air pollution levels were moderate or low ($r \leq 0.46$). Correlations between air pollution exposures and noise were moderate to low ($r \leq 0.52$) Ozone was positively correlated with temperature.

Air pollution and blood pressure

Differences between crude and adjusted effect estimates were larger for short-term air pollution exposures than for long-term air pollution and noise (Table 4 and Supplementary Table S3). Part of the differences was attributable to differences in the study population (Supplementary Table S4). For short-term air pollution effects, differences were largely attributable to adjustment for temperature. In adjusted models, we observed that diastolic blood pressure tended to increase with increasing long-term air pollution exposure at home and school addresses. No association was found with average air pollution levels on the 7 days and 1 to 7 days preceding blood pressure measurements (Table 4 and Supplementary Figure S4). Associations with long-term air pollution exposure were not confounded by traffic-noise exposure (Supplementary Table S5). Associations with long-term exposures at home addresses were stronger for children who lived at the same address since birth compared to children who had moved at least once and (marginally) statistically significant ($p < 0.1$) associations were observed in the subgroup of movers only [adjusted difference (95% confidence interval) 0.83 (0.06 to 1.58) for NO_2; 0.75 (−0.08 to 1.58) for $PM_{2.5}$ absorbance]. Associations of diastolic blood pressure and long-term air pollution tended to be stronger in boys compared to girls, but confidence intervals largely overlap (Supplementary Table S6). Associations became slightly stronger and became (marginally) statistically significant for NO_2, $PM_{2.5}$ absorbance, and $PM_{2.5}$ after exclusion of children with doctor-diagnosed asthma and/or diabetes (Supplementary Table S7).

Traffic noise and blood pressure

In adjusted models, diastolic blood pressure tended to increase with traffic noise exposure at the school address (Supplementary Table S3). No other associations were observed between traffic-noise exposure and blood pressure (Table 4 and Supplementary Table S3).

Discussion

We demonstrated positive associations between long-term exposure to traffic-related air pollution and diastolic but not systolic blood pressure of children aged 12 years who lived at the same address since birth. Blood pressure was not associated with short-term air pollution or noise exposure.

Our finding of a positive association between long-term air pollution and diastolic blood pressure is consistent with the finding of a recent study in Pakistan, which revealed that 10-year-old children attending a

Table 4. Estimated crude and adjusted associations of systolic and diastolic blood pressure with long-term air pollution and traffic noise exposure at the home address, and short-term air pollution exposure

	Crude ($n = 1400$)		Adjusted ($n = 1147$)	
Exposure (increment)	β (95% CI)	p-value	β (95% CI)	p-value
Systolic blood pressure				
Long-term air pollution				
NO_2 (7.8 $\mu g/m^3$)	−0.19 (−0.84 to 0.46)	0.5701	−0.03 (−0.70 to 0.64)	0.9306
$PM_{2.5}$ absorbance (0.3×10^{-5}/m)	−0.05 (−0.74 to 0.65)	0.8984	0.03 (−0.69 to 0.74)	0.9434
$PM_{2.5}$ (1.2 $\mu g/m^3$)	−0.06 (−0.94 to 0.82)	0.8948	−0.07 (−0.97 to 0.82)	0.8708
PM_{10} (1.0 $\mu g/m^3$)	−0.05 (−0.57 to 0.48)	0.8528	0.03 (−0.49 to 0.55)	0.9050
Short-term air pollution				
NO_2 (13.3 $\mu g/m^3$)	0.46 (−0.11 to 1.03)	0.1163	0.08 (−0.55 to 0.71)	0.8017
PM_{10} (11.1 $\mu g/m^3$)	0.49 (−0.06 to 1.05)	0.0832	0.16 (−0.41 to 0.72)	0.5875
O_3 (23.6 $\mu g/m^3$)	−1.13 (−1.87 to −0.40)	0.0025	−0.06 (−0.94 to 0.83)	0.9017
Traffic noise (5.8 dB(A))	−0.04 (−0.66 to 0.58)	0.8983	−0.25 (−0.87 to 0.36)	0.4197
Diastolic blood pressure				
Long-term air pollution				
NO_2 (7.8 $\mu g/m^3$)	0.26 (−0.20 to 0.72)	0.2655	0.33 (−0.18 to 0.84)	0.2085
$PM_{2.5}$ absorbance (0.3×10^{-5}/m)	0.38 (−0.11 to 0.87)	0.1284	0.41 (−0.13 to 0.96)	0.1357
$PM_{2.5}$ (1.2 $\mu g/m^3$)	0.57 (−0.04 to 1.19)	0.0687	0.56 (−0.12 to 1.24)	0.1072
PM_{10} (1.0 $\mu g/m^3$)	0.15 (−0.21 to 0.52)	0.4113	0.19 (−0.21 to 0.58)	0.3590
Short-term air pollution				
NO_2 (13.3 $\mu g/m^3$)	0.54 (0.14 to 0.94)	0.0084	0.17 (−0.31 to 0.65)	0.4804
PM_{10} (11.1 $\mu g/m^3$)	0.26 (−0.13 to 0.65)	0.1938	−0.02 (−0.45 to 0.41)	0.9346
O_3 (23.6 $\mu g/m^3$)	−0.94 (−1.45 to −0.42)	0.0004	−0.46 (−1.13 to 0.21)	0.1803
Traffic noise (5.8 dB(A))	0.08 (−0.36 to 0.52)	0.7154	−0.06 (−0.53 to 0.41)	0.8063

Values are change in blood pressure (mmHg) for an interquartile range increase in exposure.; Adjusted for sex, age, height, and BMI, cuff size, gestational age at birth, birthweight, weight gain during the first year of life, breast feeding, maternal smoking during pregnancy, parental smoking in child's home, physical activity, puberty development scale, maternal education, maternal hypertension during pregnancy, pneumonia and/or otitis media during the first 2 years of life, ambient temperature, and room temperature.

primary school situated in an area with heavy traffic, had a significantly higher systolic and diastolic blood pressure (115.9 mmHg, 95% CI 114.0 to 117.9, and 70.9, 95% CI 69.2 to 72.7, respectively), than children attending a school situated in an area with less traffic (108.3 mmHg, 95% CI 106.1 to 110.6, and 66.4 mmHg, 95% CI 64.4 to 68.4, respectively).[13] However, effects are much smaller in the present study and limited to diastolic blood pressure. The about 10-fold higher air pollution concentrations in the Pakistan study compared to our study could be one explanation for the difference in effect sizes. In comparison, the mean long-term PM_{10} concentration was only $24.7 \pm 0.9\ \mu g/m^3$ in our study. The effect sizes observed in the present study are comparable, however, with the effect sizes for systolic blood pressure that have been reported from adult studies investigating long-term air pollution effects in areas with similar levels of exposure: for example, 0.9 mmHg (95% CI 0.4–1.4) per $2.4\ \mu g/m^3$ $PM_{2.5}$ in a German study[11] and 2.41 mmHg (95% CI 1.77–3.05) per $1\ \mu g/m^3$ black carbon in a study from the Greater Boston area.[10] The stronger effects in non-movers compared to movers suggest that chronic exposure is of greater relevance than more recent exposure.

Only few other studies investigated the association between air pollution exposure and blood pressure in children. In a cohort study comparing 6–13-year-old healthy children from Mexico City exposed to high air pollution concentrations with children from a control city with lower levels of air pollution, a significant association was found between short-term 7-day cumulative $PM_{2.5}$ levels and systolic pulmonary arterial and mean pulmonary arterial pressure.[26] The reported mean systolic pulmonary arterial pressure in the control group and children from the southern and northern parts of Mexico City were 20.7 ± 0.7, 24 ± 0.9, and 27.2 ± 1.4 mmHg, respectively. Another study investigated the association between household short-term 24-h $PM_{2.5}$ and black carbon from biomass combustion (geometric mean and range, 53, 14–393 $\mu g/m^3$, and 3.2,

Table 5. Associations of systolic and diastolic blood pressure with long-term air pollution and traffic noise exposure at the home address, and short-term air pollutant stratified analyses by moving since birth

	Non-movers ($n = 471$)		Movers ($n = 676$)	
Exposure (increment)	β (95% CI)	p-value	β (95% CI)	p-value
Systolic blood pressure				
Long-term air pollution				
NO_2 (7.8 $\mu g/m^3$)	0.14 (−0.84 to 1.12)	0.7835	−0.07 (−1.00 to 0.87)	0.8882
$PM_{2.5}$ absorbance (0.3×10^{-5}/m)	−0.03 (−1.08 to 1.02)	0.9593	0.19 (−0.80 to 1.17)	0.7115
$PM_{2.5}$ (1.2 $\mu g/m^3$)	−0.23 (−1.60 to 1.14)	0.7388	0.05 (−1.15 to 1.25)	0.9306
PM_{10} (1.0 $\mu g/m^3$)	0.03 (−0.76 to 0.82)	0.9469	0.14 (−0.56 to 0.84)	0.7049
Short-term air pollution				
NO_2 (13.3 $\mu g/m^3$)	0.38 (−0.57 to 1.33)	0.4302	−0.10 (−0.94 to 0.75)	0.8245
PM_{10} (11.1 $\mu g/m^3$)	−0.06 (−0.90 to 0.79)	0.8959	0.35 (−0.41 to 1.11)	0.3699
O_3 (23.6 $\mu g/m^3$)	−0.06 (−1.44 to 1.33)	0.9360	−0.19 (−1.35 to 0.97)	0.7506
Traffic noise (5.8 dB(A))	−0.14 (−1.08 to 0.81)	0.7774	−0.21 (−1.02 to 0.60)	0.6146
Diastolic blood pressure				
Long-term air pollution				
NO_2 (7.8 $\mu g/m^3$)	0.83 (0.06 to 1.61)	0.0362	−0.10 (−0.77 to 0.58)	0.7791
$PM_{2.5}$ absorbance (0.3×10^{-5}/m)	0.75 (−0.08 to 1.58)	0.0777	0.14 (−0.58 to 0.85)	0.7041
$PM_{2.5}$ (1.2 $\mu g/m^3$)	0.61 (−0.48 to 1.69)	0.2752	0.41 (−0.46 to 1.29)	0.3529
PM_{10} (1.0 $\mu g/m^3$)	0.49 (−0.14 to 1.11)	0.1302	0.00 (−0.51 to 0.51)	0.9996
Short-term air pollution				
NO_2 (13.3 $\mu g/m^3$)	0.21 (−0.55 to 0.96)	0.5942	0.20 (−0.41 to 0.82)	0.5135
PM_{10} (11.1 $\mu g/m^3$)	−0.43 (−1.10 to 0.24)	0.2135	0.33 (−0.22 to 0.88)	0.2436
O_3 (23.6 $\mu g/m^3$)	0.27 (−0.83 to 1.37)	0.6285	−0.98 (−1.82 to −0.14)	0.0221
Traffic noise (5.8 dB(A))	0.12 (−0.63 to 0.88)	0.7519	−0.10 (−0.69 to 0.49)	0.7322

Values are change in blood pressure (mmHg) for an interquartile range increase in exposure.; Adjusted for sex, age, height, and BMI, cuff size, gestational age at birth, birthweight, weight gain during the first year of life, breast feeding, maternal smoking during pregnancy, parental smoking in child's home, physical activity, puberty development scale, maternal education, maternal hypertension during pregnancy, pneumonia and/or otitis media during the first 2 years of life, ambient temperature, and room temperature.

2.0–9.6 $\mu g/m^3$ for $PM_{2.5}$ and black carbon, respectively) in 10-year-old Chinese schoolchildren and found no association.[27] Unlike this paediatric study and many studies in adults,[4–9] we found no associations of blood pressure with short-term air pollution exposure. Although the observed increases in blood pressure due to air pollution are small on the individual level, they can be meaningful as this shift the blood pressure distribution in the general population towards a more unfavourable distribution, this may result in significant increases in the number of subjects with high blood pressure or hypertension.

Several biological mechanisms have been proposed for the effect of ambient air pollution on blood pressure. The inhalation of particulate matter has been shown to induce changes in the autonomic balance favouring sympathetic activity, to mediate systemic oxidative stress and inflammation, and to promote vascular dysfunction leading to arterial vasoconstriction.[28]

We have no explanation for the differential association of air pollutants with systolic and diastolic blood pressure in our study. However, findings are in agreement with findings of two randomized double-blind crossover studies among healthy 18–50 years old non-smokers, where an increase in diastolic but not systolic blood pressure was found with $PM_{2.5}$.[29] In another study among an elderly population, short-term outdoor black carbon and $PM_{2.5}$ had a greater effect on diastolic than systolic blood pressure.[30]

Several studies found positive associations between aircraft and road traffic noise exposure and systolic blood pressure.[31–34] Findings for diastolic blood pressure in these studies are mixed. In two of these studies, noise exposure was also associated with diastolic blood pressure,[32,34] whereas in others there was no association with diastolic blood pressure.[31,33] The low level and limited range of noise exposure may be a potential explanation for the lack of an association in the present study. Moreover, noise levels at the façade of the child's

home may not be representative for the child's exposure at home if, for example, the child's bedroom is at the back of the home.

The strengths of our study are the individually home- and school-based air pollution and noise exposure assessment using state-of-the-art exposure models and the large size of the study population. We included exposures at the home and school addresses. However, a possible limitation of our exposure assessment may be that no information was available about time-activity pattern. Although we had detailed information on most potential confounders, we were unable in this study to take a number of factors into account that are associated with children's blood pressure, including genetic factors and parental cardiovascular risk factors.[35] We were able to exclude children with asthma, the most common chronic disease in childhood, and children with diabetes. Associations became slightly stronger.

In conclusion, long-term exposure to traffic-related air pollution may increase diastolic blood pressure in children.

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Conflict of interest

The authors declare that there is no conflict of interest.

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