Association of traffic-related air pollution with cognitive development in children

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ABSTRACT

Background Air pollution from traffic has been associated with cardiorespiratory diseases in children and adults, but there is little information on its potential neurotoxic effects. This study aimed to investigate the association between exposure to nitrogen dioxide (NO_2), as a marker of traffic-related air pollution, and cognitive development in children.

Methods A population-based birth cohort from southern Spain was followed from the age of 4 years for 1 year. Complete data for analyses were gathered on 210 children living in urban and rural areas. NO_2 exposure was predicted by means of land use regression models. A standardised version of the McCarthy Scales of Children's Abilities (MSCA) was used to assess children's motor and cognitive abilities. Multivariate analyses were performed to evaluate the relation between exposure to NO_2 and MSCA outcomes, adjusting for potential confounders.

Results A negative effect of NO_2 was found across all MSCA subscales, despite low predicted NO_2 exposure levels (5–36 $\mu g/m^3$). Children exposed to higher NO_2 (>24.75 $\mu g/m^3$) showed a decrease of 4.19 points in the general cognitive score and decreases of 6.71, 7.37 and 8.61 points in quantitative, working memory and gross motor areas, respectively. However, except for gross motor function, associations were not statistically significant.

Conclusion Although results were not statistically significant, the associations found between exposure to NO_2 and cognitive functions suggest that traffic-related air pollution may have an adverse effect on neurodevelopment, especially early in life, even at low exposure levels.

Air pollution is associated with a number of short and long-term adverse respiratory and cardiovascular health effects.[1 2] These effects have largely been related to exposure to fine and ultrafine particles,[3] whose main source in urban air is transportation emissions. Epidemiological studies have repeatedly found a positive correlation between particulate air pollution levels and increased morbidity and mortality rates in children and adults.[3 4] Exposure to traffic-related air pollution early in life has been associated with an increased risk of infant mortality, adverse reproductive outcomes, cancer, the development of atopy and asthma and other adverse respiratory effects.[5]

Animal studies have shown that inhaled particles can be translocated from the respiratory system directly to the central nervous system, providing evidence that the brain is a target for airborne particulate matter.[6 7] In children and adults residing in large urban areas, exposure to severe air pollution has been associated with pathological lesions in brain tissues,[8 9] and children are at special risk because childhood is a crucial period of brain development. Nevertheless, only two studies have examined the relationship between chronic exposure to traffic-related air pollution and infant cognitive development. Calderon-Garcidueñas *et al*[10] reported that children with no known risk factors for cognitive disorders from a polluted urban environment (Mexico City) exhibited significant deficits in a combination of cognition tasks. A prospective birth cohort study by Suglia *et al*[11] in Boston (USA) reported a relationship between exposure to black carbon (the major component of particles from traffic) and reduced neurocognitive functioning in urban 8–11-year-old children. Given these findings and the scarce information on the neurotoxic effect of air pollution in humans, there is a need to investigate further the possible association between air pollution and neurodevelopmental disorders.

The aim of this study was to investigate the association of exposure to nitrogen dioxide (NO_2), as a surrogate for traffic-related air pollution, with cognitive development at the age of 4 years in a birth cohort in southern Spain, controlling for sociodemographic, physical and psychological determinants of this outcome.

METHODS

Study population

The study sample was drawn from a cohort established in Granada province (southern Spain)[12] as part of the INMA (Environment and Childhood) study, a population-based cohort study in Spain that focuses on prenatal environmental exposures in relation to growth, development and health from early fetal life until childhood.[13] From October 2000 to August 2002, 700 eligible mother–son pairs registered at the San Cecilio University Hospital of Granada were recruited. The inclusion and exclusion criteria were published elsewhere.[14] In the INMA study protocol, the medical follow-up of the children at the age of 4 years included a neuropsychological evaluation. Briefly, between September 2005 and September 2006, one out of three mothers (n=250) were randomly contacted by phone and invited to participate in the neurocognitive testing of the children. A total of 220 (88%) boys was evaluated over this 1-year period. Complete outcome data, information on exposure and other variables were available for a subset of 210 subjects. Written informed consent was obtained

from parents of children before the study, which was approved by the Ethics Committee of the San Cecilio University Hospital.

Study area

The study area was the health district of San Cecilio University Hospital of Granada, which has a total population of 512 000 inhabitants, a surface area of 4000 km^2, and includes the city of Granada (236 000 inhabitants, 87.8 km^2 and altitude of 740 m) and 50 towns and villages. The children's residence was classified by dividing the study area into four sub-areas: (a) urban area, corresponding to the central districts of the city, with high population and traffic densities; (b) metropolitan area, towns within the ring road surrounding Granada; (c) suburban area, towns and villages with more than 10 000 inhabitants; and (d) rural area, where population resides mainly in small villages (<5000 inhabitants). For modelling purposes, study sub-areas were collapsed into two categories: urban area (sub-area 'a') and non-urban area (sub-areas 'b–d').

NO_2 exposure

Home outdoor NO_2 levels were estimated as a proxy of individual exposure to traffic-related air pollution. Following a geographical information system-based methodology previously applied in the INMA study by Aguilera *et al*,[15] a land use regression (LUR) model[16] was built using NO_2 measurements from 76 sampling locations. Twenty-six monitoring sites were located in the city (urban area) and 50 in each town centre in the non-urban area.

Sampling was done in two 7-day periods (November 2005 and September 2006) during the same year as the evaluation of the children. Ambient NO_2 was monitored using Radiello passive samplers (Environmental Research Centre, S Maugeri Foundation, Padova, Italy), and concentrations were determined at the Centro Nacional de Sanidad Ambiental of the Instituto de Salud Carlos III (Madrid, Spain). The average of the two sampling periods represented annual mean NO_2 levels.[17] The mean NO_2 concentration at the measurement sites was 20.75 $\mu g/m^3$ (range 3.25–59 $\mu g/m^3$).

The LUR technique was used to predict the home outdoor concentration of NO_2 at children's home addresses (n=220), using the mean annual NO_2 level at each sampling site as a dependent variable. Two different models were obtained for the urban and non-urban areas (data not shown). Four categories of geographical data were collected: land use (urban, residential, industrial or agricultural); altitude of sampling site; road type (major, secondary or minor/residential roads) and length; and population density. Predictors included in the urban model were road type at a given location and percentage of residential land cover within a 200-m buffer zone around a given site. In the LUR model for the non-urban area, the universal *kriging* technique was first used to obtain a smoothed surface for the global trend of NO_2 concentration, and the predictors were the *kriging*-estimated value at a given location and road length within a 1500-m buffer zone. The adjusted R^2 value was 0.45 for the urban area and 0.75 for the non-urban area. A cross-validated R^2 of 0.64 was found between the mean annual NO_2 measurement for the whole study area and the corresponding predictions obtained from fitting the model to the data. Children's home addresses at the age of 4 years were geocoded and assigned a NO_2 estimate using the corresponding LUR model (urban or non-urban). The predicted annual mean NO_2 concentration at the addresses of the 210 children was 20.88 $\mu g/m^3$ (range 5.15–36.09 $\mu g/m^3$). There were no differences between children with complete cognitive measures (n=210) and those without (n=10).

Neuropsychological assessment

Cognitive and motor abilities were assessed by means of a standardised Spanish adaptation of the McCarthy Scales of Children's Abilities (MSCA),[18] which gives standardised test scores for five domains (quantitative, verbal, memory, perceptual performance and motor). A general cognitive score, which estimates global intellectual function, was calculated by combining the verbal, perceptual performance and quantitative scores. A higher score indicates a better performance. Two neuropsychologists were trained to administer and interpret the MSCA, which was carried out at the Paediatrics Department of our hospital. A strict protocol was applied to avoid inter-observer variability.[19] At the same time as the children were evaluated, the parents completed a self-reported questionnaire on parent-to-infant attachment and another on mental health, considered as effect modifiers on infant mental development.[20] The parent-to-infant attachment questionnaire consists of 19 items that assess the emotional bond of affection experienced by the parent towards the infant.[21] The general mental health questionnaire is designed to identify psychological distress and short-term changes in mental health in community and primary care settings and the 12-item version was used.[22] Staff involved in the neuropsychological testing were blinded to the degree of the child's exposure to air pollution.

In accordance with previous INMA studies,[19 23] MSCA items were reorganised into subscales for tasks highly associated with specific neurocognitive functions as follows: verbal memory (MSCA items 3 and 7II), working memory (MSCA items 5 and 14II), memory span or short-term memory (MSCA items 6, 7I and 14I), gross motor (MSCA items 9, 10 and 11), fine motor (MSCA items 12 and 13) and executive (MSCA items 2, 5, 6,14II, 15, 17 and 18) functions.

Covariates

Information on the characteristics of the study population was obtained by means of standardised questionnaires administered by trained interviewers at enrolment (after delivery) and at the 4-year follow-up visit. Sociodemographic covariates in the present analysis included place of residence, maternal and paternal education, maternal occupational status, parity, duration of breastfeeding, type of delivery, marital status, smoking during pregnancy and age of mothers and children. Information on birth weight and length and gestational age (physical covariates) was gathered by the paediatricians responsible for recruitment. Parents' mental health scale scores and parent-to-infant attachment scale scores were used as psychological covariates (table 1).

Statistical analysis

Except for working memory function, neurodevelopment scores followed a normal distribution and were treated as continuous variables. Working memory was transformed into a normally distributed variable by applying the formula $1/(x)^3$, inversely transforming the outcome coefficients after the multivariate models were fitted. Neurodevelopmental outcomes were standardised to a mean of 100 points with a standard deviation of 15 to homogenise all scales. Predicted NO_2 for the 210 children did not follow a normal distribution and was categorised into three groups according to tertile values (≤15.40, >15.40–24.75, >24.75 $\mu g/m^3$). Linear regression analysis was used to examine maternal, paternal and child variables in relation to NO_2 exposure levels.

Multivariate regression analyses were used to estimate the effect of NO_2 exposure on cognitive functions. Separate models were run to control for possible confounding factors. A 'crude' model was obtained for the general cognitive score after adjusting for the children's age, the psychologist and the school term in which the test was administered. Then, the influence of physical

Table 1 Characteristics of study population by exposure to NO_2 in the cohort INMA—Granada, 2000—6

	Exposure to NO_2 ($\mu g/m^3$)			
	<15.40	15.40—24.75	>24.75	All
(Sample size at age 4 years, n)	(68)	(70)	(72)	(210)
Covariates from birth to 4th year				
Place of residence at age 4 years (%)				
Rural (R)	45.6	0.0	1.4	15.2
Suburban	50.0	12.9	0.0	20.5***
Metropolitan	4.4	74.3	63.9	48.1***
Urban	0.0	12.9	34.7	16.2***
CHILD				
Birth weight (kg) (mean)	3.3	3.2	3.4	3.3*
Birth length (cm) (mean)	50.6	50.8	50.8	50.7
Gestational age (weeks) (mean)	39.3	39.0	39.4	39.2
School term at evaluation (%)				
3rd year, 3rd term (R)	32.4	34.3	33.3	33.3
4th year, 1st term	26.5	28.6	26.4	27.1
4th year, 2nd term	30.9	27.1	29.2	29.0
4th year, 3rd term	10.3	10.0	11.1	10.5
Age at evaluation (months) (mean)	51.2	51.5	51.2	51.3
MOTHER				
Educational level (%)				
University (R)	8.8	11.4	22.2	14.3
Secondary school	73.5	67.1	65.3	68.6**
Only primary school	17.6	21.4	12.5	17.1**
Occupation status (%)				
Unemployed (R)	64.7	50.0	37.5	50.5
Employed	35.3	50.0	62.5	49.5**
Parity at child's birth (%)				
0 (R)	47.1	50.0	45.8	47.6
1	35.3	37.1	43.1	38.6
2 or more	17.6	12.9	11.1	13.8
Breastfeeding (%)				
≥28 weeks (R)	2.9	2.9	6.9	4.3
16—27.9 weeks	33.8	28.6	37.5	33.3
2—15.9 weeks	33.8	44.3	34.7	37.6
<2 weeks	29.5	10.0	12.5	13.8
Type of delivery (%)				
Vaginal (R)	77.9	72.9	73.6	74.8
Caesarean	11.8	17.1	16.7	15.2
With forceps or other	10.3	10.0	9.7	10.0
Marital status at child age of 4 years (%)				
With a stable partner (R)	98.5	100.0	95.8	98.1
Without a stable partner	1.5	0.0	4.2	1.9*
Smoking during pregnancy (%)				
No (R)	80.9	71.4	79.2	77.1
Yes	19.1	28.6	20.8	22.9
Mental health (mean score)†	10.8	11.9	11.3	11.4
Mother-to-infant attachment (mean score)‡	74.4	74.6	73.9	74.3
Age at child's birth (years)	31.7	31.6	32.7	32.0
FATHER				
Educational level (%)				
University (R)	10.3	14.3	23.6	16.2
Secondary school	50.0	65.7	61.1	59.0**
Only primary school	39.7	20.0	11.1	22.9***
Mental health (mean score)†	9.6	10.4	11.2	10.2*
Father-to-infant attachment (mean score)‡	74.4	75.2	73.1	74.5

*p Value ≤0.10; **p value ≤0.05; ***p value ≤0.001.
†Mean score for the general mental health scale; a higher score indicates greater psychological distress.
‡Mean score for the parent-to-infant attachment scale; a higher score indicates a closer bond of affection.
(R) Reference category.
Smoking during pregnancy: at least one cigarette/day in the third trimester.
NO_2, nitrogen dioxide.

Table 2 McCarthy Scale scores by exposure to NO_2 (n=210) in the cohort INMA-Granada, 2000-2006.

McCarthy scores (median)†	Exposure to NO_2 ($\mu g/m^3$)			
	<15.40	15.40-24.75	>24.75	All
General cognitive	97.9	100.1	102.4*	100.5*
Perceptual-performance	98.4	100.9	102.2*	100.1*
Verbal	99.9	101.8	103.8	101.8
Memory	96.9	101.2	102.2	101.2
Quantitative	95.3	99.0	102.7	99.0
Motor	101.8	101.8	101.8	101.8*

*p-value≤0.05, in comparison to NO_2<15.40 $\mu g/m^3$
†The mean score for the McCarthy scales is 100, with a standard deviation of 15. A higher score indicates a better performance.

variables and in utero exposure to tobacco was assessed. A fully adjusted model was obtained by adjusting for these variables and sociodemographic and psychological variables. Covariates were included in the model if their inclusion modified the estimate of NO_2 effect on neurodevelopment by 10% or greater, regardless of their statistical significance. The same models were constructed for all psychological scores. Potential interaction between levels of exposure and parental education was assessed and retained in the model if it modified the NO_2 effect on cognitive development by 10% or greater. SPSS version 15.0 and STATA version 9.0 software packages were used for the analyses.

RESULTS

Table 1 lists the characteristics of the study population as a function of predicted exposure to NO_2 during the study period. A higher predicted NO_2 exposure level was found in urban (29.71 $\mu g/m^3$) compared with non-urban (9.17 $\mu g/m^3$) children ($p<0.001$). In bivariate analysis, covariates associated ($p\leq0.10$) with exposure to NO_2 were: place of residence at age 4 years ($p<0.001$, coefficient $\beta=4.74$, 15.54 and 20.61 for suburban, metropolitan and urban areas, respectively), birth weight ($p=0.06$, $\beta=0.002$), maternal education ($p=0.02$, $\beta=-5.15$ for 'only primary school'; $p=0.03$, $\beta=-3.60$ for 'secondary school'), paternal education ($p<0.001$, $\beta=-8.78$ for 'only primary school'; $p=0.02$, $\beta=-3.69$ for 'secondary school'), maternal occupation ($p=0.006$, $\beta=3.28$), maternal marital status ($p=0.09$, $\beta=7.31$), and paternal mental health score ($p=0.08$, $\beta=0.01$).

Table 2 shows MSCA cognitive measures as a function of NO_2 exposure. Bivariate analysis showed that exposure to NO_2 greater than 24.75 $\mu g/m^3$ was significantly associated with general cognitive ($p=0.05$, $\beta=4.94$) and perceptual performance scores ($p=0.04$, $\beta=5.22$), taking as reference the group of children exposed to NO_2 less than 15.40 $\mu g/m^3$. When NO_2 was treated as a continuous variable, associations were found for general cognitive ($p=0.02$, $\beta=0.27$), perceptual performance ($p=0.01$, $\beta=0.29$) and motor areas ($p=0.05$, $\beta=0.44$).

Table 3 shows crude and adjusted effects of NO_2 exposure on the general cognitive score. In the crude model, a positive association was found with exposure to NO_2 greater than 24.75 $\mu g/m^3$. A similar tendency and strength of association were shown when adjusting for birth weight and length, gestational age and smoking during pregnancy. Importantly, a negative effect of NO_2 was seen after adjustment for these variables and sociodemographic and psychological characteristics, although associations were not statistically significant. Exposure to higher NO_2 (>24.75 $\mu g/m^3$) had a negative effect on the general cognitive score (−4.19 points). A lower effect was seen for children exposed to NO_2 in the range 15.40—24.75 $\mu g/m^3$ (−1.07 points). The interaction between

exposure level and parental education did not attenuate the effect of exposure on cognitive scores.

Fully adjusted models for the remaining cognitive outcomes are shown in Table 4. Predicted exposure to NO_2 was negatively associated with all MSCA areas except for fine motor skills. The magnitude of the effect was stronger for gross motor function, working memory and quantitative skills. Exposure to NO_2 in the range 15.40–24.75 $\mu g/m^3$ and exposure greater than 24.75 $\mu g/m^3$ were related to decreases of −8.30 (p=0.08) and −8.61 points (p=0.10) in gross motor function, respectively.

DISCUSSION

Predicted exposure to ambient NO_2 was negatively associated with the cognitive development of 4-year-olds in the cross-sectional fully adjusted analysis of this cohort study. Although results were not statistically significant, the associations found suggest that traffic-related air pollution may have a detrimental effect on neurodevelopment. The study area has little industrial activity, and vehicle traffic appears to be the main source of air pollution, chiefly in the city and towns near main roads. Predicted outdoor NO_2 at child's home locations was used as a marker of exposure to air pollution, which was associated with decreases across all cognitive functions after adjustment for confounding variables. Association was stronger for gross motor function, especially in children with higher exposure to NO_2 (upper tertile). The association found with gross motor function may be explained by the earlier development of the brain regions involved in motor performance (eg, coordination, balance, posture control) compared with those involved in learning, memory and language.[24] Deficits in gross motor skills may thus be detectable before other deficits in cognitive function.[25] Psychological follow-up of these children is currently under way to test this proposition.

Unlike the investigation by Suglia *et al*,[11] the present study was conducted in two well-differentiated sub-areas (urban/rural). The small number of subjects from each area and the relatively low exposure levels may have limited the potential of the study and contributed to the absence of stronger associations with reductions in cognitive scores. Compared with ambient NO_2 and predicted exposure levels described in other Spanish urban areas,[15 26] Granada is not a highly air polluted area, and none of the predicted NO_2 concentrations (5–36 $\mu g/m^3$) exceeded the annual mean limit value of 40 $\mu g/m^3$ set by the European Commission for human health protection.[27] Another limitation is the potential misclassification of exposure, because children's exposure was based on estimated concentrations at their home location and commuting patterns were not considered, which could lead to a bias in the exposure measurement. Nevertheless, NO_2 exposure assignment was based on extensive field measurements, and individual exposure levels were predicted by the LUR technique, considered a valuable approach for estimating spatial patterns of traffic-related pollution[16] and increasingly used for exposure assessment.[28 29] Moreover, as indicated by time-activity studies,[30] children spend most of their time at home and near home, and residential indoor concentrations of NO_2 of outdoor origin are highly correlated with outdoor concentrations.[31] Data in the present study did not allow us to control for pollutants that may be found indoors, such as polycyclic aromatic hydrocarbons (PAH) and environmental tobacco smoke, which could also affect children's neurodevelopment.[32]

Despite the study limitations, our findings are in agreement with the only two studies[10 11] that previously examined the impact of air pollution on children's cognition. Suglia *et al*[11] found that exposure to black carbon predicted a three-point decrease in the Kaufman brief intelligence test intelligence quotient and four-point decrease in the wide range assessment of memory and learning test general index. They also estimated exposure to black carbon by using LUR modelling. In the other study, Calderón-Garcidueñas *et al*[10] reported a negative impact of air pollution on fluid cognition, memory and executive functions on the Wechsler intelligence scale for children—revised. The results of these studies are comparable to our findings, despite being carried out in larger urban areas with greater air pollution levels. Therefore, in the present study, exposure to greater air pollution was associated with a four-point decrease in the MSCA general cognitive score. The instruments used in the previous and present studies have been described as reliable and well-standardised child tests that facilitate comparisons among them.[33]

Strengths of this study include its inclusion of inner-city and rural settings, allowing comparisons between children with different exposures. Moreover, account was taken of physical and sociodemographic factors signalled as potential confounders in previous studies.[34] We thus controlled for socioeconomic status (inferred from home address, educational level and maternal occupation status) and for parental attachment and distress. Very few studies on exposure to environmental neurotoxicants and cognitive development in children have measured parental psychological characteristics such as maternal intelligence or mental health.[34 35]

Besides the well-documented association between brain damage and particulate air pollution, cognitive impairment has also been related to exposure to other urban airborne pollutants, with neurodevelopmental toxicity, such as PAH[35 36] or manganese,[37] as well as to traffic noise.[38] Therefore, it is biologically plausible to assume that air pollution can adversely affect brain development in children exposed to contaminants that are commonly present in urban areas (eg, particles, PAH).[35] This hypothesis is supported by the recent finding of an association between lung function and cognition,[39] indicating that both outcomes may operate under common regulatory processes and

Table 3 Adjusted effects of exposure to NO_2 ($\mu g/m^3$) on child's general cognitive score of McCarthy Scales at 4 years of age (Granada, n=210)§

General cognitive model	Exposure to NO_2 ($\mu g/m^3$)					
	15.40 - 24.75			> 24.75		
	β	95% CI	p	β	95% CI	p
Crude analysis†	3.32	-1.52, 8.15	0.18	4.25	-0.55, 9.05	0.08
Adjusted for BW + BL + GA + smoking during pregnancy	3.25	-1.59, 8.09	0.19	3.63	-1.13, 8.40	0.13
Fully-adjusted model‡	-1.07	-9.99, 7.85	0.81	-4.19	-14.02, 5.64	0.40

§Each row is a different multivariate model taking as reference group "children exposed to <15.40 $\mu g/m^3$ NO_2"
CI: confidence interval.
†Adjusted for child's age (months) and school term and evaluator (psychologist).
BW: birth weight (<2,500 g); BL: birth length (<49 cm); GA: gestational age (<37 weeks); smoking during pregnancy: at least one cigarette/day in the third trimester.
‡Adjusted for BW, BL, GA, smoking during pregnancy, place of residence (rural/sub-urban/metropolitan/urban), maternal and paternal educational level, maternal occupational status at child age of 4, maternal marital status, mother's parity at childbirth (no. of older siblings), breastfeeding (weeks), mother-to-infant attachment score and maternal mental health score.

Table 4 Fully-adjusted associations of the different cognitive sub-areas of McCarthy Scales with exposure to NO_2 ($\mu g/m^3$) for the INMA-Granada cohort, 2000-2006§

	Exposure to NO_2 ($\mu g/m^3$)					
Psychological outcomes†	**15.40 − 24.75**			**> 24.75**		
McCarthy areas	**β**	**95% CI**	**p**	**β**	**95% CI**	**p**
Perceptual-performance	0.45	-9.17; 10.06	0.93	-2.17	-12.76; 8.41	0.69
Verbal	-0.25	-9.53; 9.03	0.96	-3.09	-13.31; 7.13	0.55
Quantitative	-4.16	-14.33; 6.02	0.42	-6.71	-17.91; 4.49	0.24
Memory	-2.28	-11.95; 7.38	0.64	-5.52	-16.18; 5.13	0.31
Motor	-3.36	-13.04; 6.31	0.49	-5.30	-15.96; 5.36	0.33
Executive function	-1.93	-10.99; 7.13	0.67	-4.93	-14.90; 5.05	0.33
Memory span	-0.10	-9.61; 9.41	0.98	-3.46	-13.93; 7.01	0.51
Verbal memory	-0.36	-10.61; 9.90	0.95	-2.71	-14.02; 8.59	0.64
Working memory	-5.72	-16.26; 4.83	0.29	-7.37	-18.98; 4.24	0.21
Gross motor	-8.30	-17.69; 1.09	0.08	-8.61	-18.96; 1.74	0.10
Fine motor	3.28	-6.83; 13.40	0.52	0.91	-10.22; 12.05	0.87

§Each row is a different fully-adjusted model taking as reference group "children exposed to NO_2 less than 15.40 $\mu g/m^3$".
CI: confidence interval.
†Mean score for psychological outcomes is 100, with a standard deviation of 15. A higher score indicates a better performance.

share vulnerability to environmental factors. Although NO_2 appears unlikely to represent the causal agent at ambient concentrations, it is an appropriate marker for traffic pollution, which correlates well with numerous other components of automobile emissions.[40]

Given the increasing reports on the neurotoxic effects of particulate air pollution, it is important not only to understand the potential mechanisms underlying these effects but also to investigate whether air pollution can affect the developing brain and adversely affect cognition. The present results suggest that air pollution associated with vehicular traffic may have a negative effect on infant cognitive development, even at low exposure levels, supporting demands for the implementation of preventive measures.

What is already known on this subject

- Air pollution from traffic has been associated with cardiorespiratory diseases in children, but there is little information to date on its potential neurotoxic effects.
- Only two studies have shown an association between traffic pollution and cognition in children.
- Given the ubiquitous presence of traffic-related air pollution, there is a need to investigate further whether air pollution can affect the developing brain and impair children's cognitive development.

What this study adds

- This is one of the few available studies evaluating the association between air pollution and cognitive development in children.
- This study suggests that exposure to higher NO_2 may have a negative effect on cognition, especially early in life, even at low exposure levels.
- Epidemiological studies on the effects of urban air pollution on children's development should consider potential neurological damage.

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Competing interests None.

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