

Traffic exposure and allergic sensitization against latex in children

Thomas Hirsch, MD,^a Volker Neumeister, MSc,^b Stephan K. Weiland, MD, MSc,^c
Erika von Mutius, MD,^d Dörte Hirsch, MD,^a Heinz Gräfe, MSc,^e Heinrich Duhme,
MD,^c and Wolfgang Leupold, MD^a *Dresden, Münster, Munich, and Radebeul, Germany*

Background: Latex antigens have been found in urban air samples and in debris deposited near freeways.

Objective: We investigated whether exposure to road traffic in a large city is associated with allergic sensitization against latex in children.

Methods: A population-based sample of 2505 children aged 5 to 11 years was examined in a cross-sectional study in the city of Dresden, Germany. Specific IgE levels against latex and a panel of common aeroallergens (timothy grass, rye, birch, mugwort, *Cladosporium herbarum*, *Dermatophagoides pteronyssinus*, cat dander, and dog dander) was determined by using a fluorescence immunoassay (CAP Pharmacia). Traffic exposure was assessed by (1) parental self-report on traffic frequency and truck traffic in the street of residence, (2) traffic counts, and (3) measurements of benzene at 182 points on a 1-km² grid for 1 year, which were used to estimate the children's individual exposure at the home address.

Results: Eight hundred fifty-four (34.1%) of the children were sensitized against any of the allergens, and 150 (6.0%) had specific IgE (>0.35 kU/L) against latex. The prevalence of latex sensitization was not positively associated with self-reported traffic exposure, traffic counts in the street of residence (adjusted prevalence odds ratio for >5000 vs ≤5000 cars/d, 0.7; 95% confidence interval, 0.4-1.3), or benzene exposure (adjusted prevalence odds ratio for an increase of 1 µg/m³ air, 0.8; 95% confidence interval, 0.7-1.05).

Conclusion: The data suggest that exposure to road traffic is not associated with allergic sensitization to latex in children. (*J Allergy Clin Immunol* 2000;106:573-8.)

Key words: Traffic, road dust, rubber tires, wheels, latex particles, allergic sensitization, children, International Study of Asthma and Allergies in Childhood

Abbreviation used

SPT: Skin prick test

Increased exposure to particulate matter is associated with respiratory tract illness in children.¹⁻³ However, little is known about the specific pathogenetic constituents of these particles. Other studies found an association between automobile^{4,5} or truck⁶⁻⁸ traffic frequency and wheezing or allergic rhinoconjunctivitis. Whether these symptoms reflect a nonspecific irritative effect in the mucosa or atopic sensitization is controversial.^{2,4,9-11} Latex antigens have been found on respirable particles in urban air samples and in debris deposited near freeways.^{12,13} Airborne latex allergens can be inhaled and may therefore lead to sensitization and provoke respiratory symptoms.^{14,15} We investigated whether exposure to road traffic in a large city is associated with allergic sensitization against latex in children. Because exposure in early infancy might also have a profound effect on the risk of sensitization, traffic exposure in the first year of life and at present was investigated.

METHODS

Study population

In Dresden, Germany, a city with 480,000 inhabitants, all children participating in the school entry examinations (n = 3866) and a random sample of schoolchildren attending classes of fourth grade (n = 3668) were studied between August 1995 and June 1996 by using the Phase II modules of the International Study of Asthma and Allergies in Childhood.¹⁶ Because all school beginners were between 5 and 7 years of age at the time of investigation and most fourth grade pupils were between 9 and 11 years old (98.8%), we will refer in the following text to 5- to 7-year-old and 9- to 11-year-old children, respectively.

Parental questionnaires were distributed through the community health department, which conducts the school entry examinations, for 5- to 7-year-old children and through the schools for 9- to 11-year-old children. A completed questionnaire was returned by parents of 3312 (85.7%) children aged 5 to 7 years and by parents of 3045 (83%) children aged 9 to 11 years. The focus of this analysis is on responses to questions on traffic exposure and on responses regarding living conditions and other potential confounders of the association between traffic exposure and latex sensitization.

Determination of specific IgE against latex and other allergens

All 9- to 11-year-old children and a random subsample of 5- to 7-year-old children (n = 2328) were invited to take part in blood sampling. Blood samples were stored in a cooler during the field-

From ^athe University Children's Hospital and ^bthe Institute for Clinical Chemistry and ^cthe Laboratory of Medicine of the University of Technology, Dresden; ^dthe Institute of Epidemiology and Social Medicine, University of Münster; and ^ethe University Children's Hospital Munich and the Saxon State Authority for Environment and Geology, Radebeul.

The cross-sectional study was supported by the German Ministry of Education, Science, Research, and Technology, and the emission monitoring program was supported by the European Union project "Phare."

Received for publication Apr 3, 2000; accepted for publication June 12, 2000.

Reprint requests: Thomas Hirsch, MD, Kinderklinik im Universitätsklinikum Carl Gustav Carus der Technischen Universität Dresden, Fetscherstr 74, D-01307 Dresden, Germany.

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0091-6749/2000 \$12.00 + 0 1/109430

doi:10.1067/mai.2000.109430

work in the schools and were centrifuged and stored at -70°C in the laboratory later in the day. Specific serum IgE against latex was determined in the laboratory of the Institute for Clinical Chemistry of the Technical University Dresden. The cumulative concentrations of specific IgE against a panel of aeroallergens (timothy grass, rye, birch, mugwort, *Cladosporium herbarum*, *Dermatophagoides pteronyssinus*, cat dander, and dog dander [SX1 panel]) were determined in the laboratory of the Free University of Berlin because this examination was part of a multicenter study.¹⁶ Specimens were sent to Berlin on dry ice. In both laboratories a fluorescence enzyme immunoassay (CAP-FEIA; Pharmacia & Upjohn Diagnostics, Uppsala, Sweden) was used. The intra-assay coefficient of variation for the latex assay was 2.1%, and that for the interassay coefficient of variation was 5.1%. Blood samples were drawn from 2083 (56.8%) children in the older and 954 (41.0%) children in the younger age groups. IgE concentrations above the level of detection (0.35 kU/L) were considered as evidence of allergic sensitization in both assays.

Self-reported traffic exposure

Information on traffic exposure was obtained by asking the following 4 questions in the questionnaire:

1. How often is the traffic congested or jammed in your residential street (never/seldom/frequently/constantly)?
2. How often do trucks pass through your residential street on weekdays (never/seldom/frequently/constantly)?
3. What is the maximally allowed speed in your residential street (walking speed/30 km per hour/50 km per hour/>50 km per hour/upper speed limit)?
4. Does your child live on a main road/side street?

All 4 questions were asked with regard to the present situation and to the situation in the child's first year of life. The reliability of these answers was tested in 74 subjects by administration of the same questionnaire a second time within a 12-month period ($\kappa = 0.53$ - 0.91 for reports on present traffic exposure and $\kappa = 0.51$ - 0.70 for reports on traffic exposure in the first year of life).

Assessment of exposure to road traffic by traffic counts

Census data on traffic density on main streets in Dresden in 1994 were obtained by continuous counts at 8 main junctions and discontinuous counts at 160 additional monitoring points.¹⁷ The distance of the home addresses of the children to streets with high traffic counts (>5000 cars per day) was calculated by means of a geographic information system (Arc Info; Environmental Systems Research Institute, Redlands, Calif). Living in such streets has been associated with an increased risk for medical care visits in asthmatic children.¹⁸ On the basis of data indicating a steep decrease of traffic-dependent gaseous pollutants from the source of emission,¹⁹⁻²¹ children were categorized as those living a distance of 30 m or less or greater than 30 m from such streets.

Estimation of individual exposure to benzene

Benzene exposure was used as another marker of traffic exposure. In Dresden pollutant measurements have been related to actual traffic counts in 1994-1995. In these estimates 87% of the total benzene emission was attributable to road traffic.²² The method of the estimation of the individual annual mean exposure to benzene has been described in detail elsewhere.²³ In brief, benzene emission in the populated area of Dresden was monitored in a 1 km $\times$ 1 km grid between April 1994 and April 1995.²⁴ The children's exposure at the home address was estimated by using the annual mean emission values from the nearest monitoring station in each of the 4 geographic directions. These 4 values weighted by the inverse of the

square of their distance to the respective address were averaged. The benzene exposure estimates could be validated by continuous measurements between October 1994 and March 1995 at a street monitor in the city of Dresden. During this period, the measured benzene concentration was $6.5 \mu\text{g}/\text{m}^3$ air, whereas the exposure estimate for the position of the monitor was $7.1 \mu\text{g}/\text{m}^3$ air.

Data analysis

The analyses were restricted to children with complete addresses who had lived in Dresden for at least 12 months and from whom enough serum was obtained to determine both specific IgE against latex and a panel of aeroallergens (SX1 assay). In addition, data from children with foreign nationality ($n = 11$) were not evaluated because of concerns regarding the validity of the questionnaire responses and to avoid confounding by ethnic background.²⁵ This reduced the number of children to 834 and 1671 (82.5% of all children with blood examinations) in the younger and older age groups, respectively.

Because nearly all children sensitized against latex were also sensitized against other allergens (see the "Results" section), it would have been difficult to disentangle the effect of traffic exposure on specific sensitization from that on general atopic sensitization by comparing children sensitized against latex with children without allergic sensitization. Therefore the association of traffic exposure and latex sensitization was analyzed in the subgroup of children with atopic sensitization (specific serum IgE >0.35 kU/L either against latex or against the SX1 panel, $n = 854$).

The crude prevalence of atopic sensitization against latex and the SX1 panel was calculated for different categories of IgE concentrations (>0.35 kU/L, >0.7 kU/L, and >3.5 kU/L) in both age groups. Then children with different levels of traffic exposure were compared with respect to the prevalence of latex sensitization defined by these categories (χ^2 test for $2 \times n$ tables). The same comparisons were made by using the serum concentration of antilatel IgE as a continuous variable (Kruskal-Wallis H test). Furthermore, adjusted prevalence odds ratios for the association of latex sensitization with traffic exposure were calculated in a multivariate logistic regression model, including potential confounders like sex, age, parental atopy, social status, direction of the window in the child's room (toward the street or not), and floor level of the dwelling ($\leq$ first floor vs >first floor). The two latter items were included because they were associated both with latex sensitization and several traffic exposure variables in bivariate analyses ($P < .25$). The other potential confounders were associated only with traffic exposure, but they were treated as potential confounders in comparable analyses.^{2,6}

The power of this study to detect significant associations between traffic exposure and sensitization against latex ($\alpha < .05$; $1 - \beta = .80$) was estimated for 2×2 tables for the whole group of children with blood examinations ($n = 2505$) and for the atopic subgroup ($n = 854$). For example, for two equally large groups of children with different exposures, this study would have detected a difference in the prevalence of latex sensitization of 3.0% versus 5.5% in the whole population and a difference of 10% versus 17% among the atopic children.

Software used for all calculations was SPSS (Chicago, Ill) for Microsoft Windows 6.01, except for power calculations, which were made with Statcalc Epi Info Version 5.01 a (March 1991; World Health Organization, Geneva, Switzerland).

RESULTS

The mean age $\pm$ SD of the children was 5.4 ± 0.5 years in the younger age group and 9.7 ± 0.6 years in the older age group. Fifty-three percent of the subjects were boys. The 2505 children with results from serum analyses

TABLE I. Allergic sensitization against latex and common aeroallergens (SX1 assay) in the study population

	5- to 7-year old children (n = 834)	9- to 11-year-old children (n = 1671)
Latex		
Specific IgE in serum >0.35 kU/L	38 (4.6%)	112 (6.7%)*
Specific IgE in serum >0.7 kU/L	27 (3.2%)	76 (4.5%)
Specific IgE in serum >3.5 kU/L	5 (0.6%)	18 (1.1%)
Common aeroallergens (SX1)		
Specific IgE in serum >0.35 kU/L	221 (26.5%)	628 (37.6%)†
Specific IgE in serum >0.7 kU/L	184 (22.1%)	536 (32.1%)†
Specific IgE in serum >3.5 kU/L	122 (14.6%)	360 (21.5%)†

* $P < .05$ for χ^2 test for comparison of age groups.

† $P < .0005$ for χ^2 test for comparison of age groups.

TABLE II. Prevalence of latex sensitization* among atopic children (n = 854) by categories of self-reported traffic exposure

Self-reported traffic exposure‡	Never	Seldom	Frequent	Constant	Statistical significance†
Traffic jam in residential street					
First year of life	97/557 (17.4%)	26/156 (16.7%)	13/68 (19.1%)	3/13 (23.1%)	$P = .69$
At present	98/491 (20.0%)	26/173 (15.0%)	17/139 (12.2%)	5/34 (14.7%)	$P = .03$
Truck traffic in residential street					
First year of life	22/110 (20.0%)	65/378 (17.2%)	30/197 (15.2%)	23/114 (20.2%)	$P = .90$
At present	13/86 (15.1%)	74/359 (20.6%)	40/237 (16.9%)	20/158 (12.7%)	$P = .14$
	Walking speed (km/h)				
		30	50	>50	Statistical significance†
Speed limit in residential street					
First year of life	4/35 (11.4%)	40/205 (19.5%)	90/519 (17.3%)	5/23 (21.7%)	$P = .78$
At present	8/41 (19.5%)	67/322 (20.8%)	69/432 (16.0%)	2/28 (7.1%)	$P = .04$
	No	Yes			Statistical significance†
Residence in main street					
First year of life	94/530 (17.7%)	46/264 (17.4%)			$P = .91$
At present	105/547 (19.2%)	41/280 (14.6%)			$P = .10$
Other indicators of traffic exposure	≤5000 cars per day	>5000 cars per day			Statistical significance†
Traffic counts in residential street					
At present	133/727 (18.3%)	17/127 (13.4%)			$P = .18$
	3 $\mu\text{g}/\text{m}^3$	3.5 $\mu\text{g}/\text{m}^3$	4 $\mu\text{g}/\text{m}^3$	4.5 $\mu\text{g}/\text{m}^3$	>4.5 $\mu\text{g}/\text{m}^3$ Statistical significance†
Estimated yearly mean exposure to benzene§					
At present	17/114 (14.9%)	35/148 (23.6%)	35/200 (17.5%)	46/228 (20.2%)	17/164 (10.4%) $P = .16$

*Prevalence of latex sensitization (specific IgE >0.35 kU/L) in the subgroup of atopic children (n = 854).

† χ^2 Test for 2×2 tables for two exposure categories and Mantel-Haenszel χ^2 test for linear trend for more than two exposure categories.

‡The total numbers of individuals with self-reports on traffic exposure differ from 854 because of missing answers in the questionnaire.

§Estimation of individual exposure of 854 atopic children on the basis of emission monitoring in a 1-km² grid in the city of Dresden. Categories of benzene exposure are characterized by their upper limit except for the highest category.

comprised more 9- to 11-year old children (66.9% vs 35.6%; $P < .0005$, χ^2 test for 2×2 tables), more children with parental atopy (46.9% vs 43.9%, $P < .05$), and fewer children whose parents reported traffic jams in the child's first year of life (9.4% vs 13.3%, $P < .0005$) than the 3852 children with completed questionnaires but without serum analyses. Among the 854 atopic children, more boys (61.4% vs 48.8%, $P < .0005$), more 9- to 11-year-old children (73.9% vs 63.2%, $P < .0005$), and more children with parental atopy (54.1% vs 42.9%, $P < .0005$) were found compared with the 1651 children without

allergen-specific IgE. In addition, the atopic children had lived more frequently at main streets in the first year of life (33.2% vs 29.1%, $P < .05$).

One hundred fifty (6.0%) children had specific IgE of greater than 0.35 kU/L against latex. Only 5 of them were monosensitized against latex; the others were also sensitized against other allergens, as measured by the SX1 assay. Results of the SX1 assay were positive (>0.35 kU/L) in 849 children (33.9%). Table I shows the prevalence of different levels of sensitization to latex and common aeroallergens.

TABLE III. Adjusted odds ratios of latex sensitization among atopic children (n = 854)* by categories of traffic exposure

Self-reported traffic exposure	Never	Seldom	Frequent	Constant
Traffic jam in residential street: first year of life	1	1.1 (0.6-1.8)	1.1 (0.5-2.2)	1.7 (0.4-6.5)
Traffic jam in residential street: at present	1	0.7 (0.4-1.2)	0.7 (0.4-1.2)	0.8 (0.3-2.1)
Truck traffic in residential street: first year of life	1	0.9 (0.5-1.6)	0.7 (0.4-1.4)	1.0 (0.5-2.1)
Truck traffic in residential street: at present	1	1.6 (0.8-3.3)	1.2 (0.6-2.6)	1.0 (0.5-2.4)
Walking speed (km/h)				
		30	50	>50
Speed limit in residential street: first year of life	1	2.0 (0.6-7.2)	2.0 (0.6-6.7)	2.3 (0.4-11.6)
Speed limit in residential street: at present	1	1.1 (0.4-2.6)	0.9 (0.4-2.2)	0.4 (0.1-2.1)
		No	Yes	
Residence in main street: first year		1	1.0 (0.6-1.5)	
Residence in main street: at present		1	0.8 (0.5-1.3)	
Other indicators of traffic exposure	≤5000 cars per day		>5000 cars per day	
Traffic counts in residential street: at present	1	0.7 (0.4-1.3)		
			+1 µg/m³	
Estimated yearly mean exposure to benzene: at present†			0.8 (0.7-1.05)	

*Adjusted prevalence odds ratios and 95% confidence intervals of latex sensitization (specific IgE >0.35 kU/L) were calculated for the subgroup of atopic children (n = 854) by logistic regression analysis, adjusting for the following putative confounding variables: sex, age group, parental atopy, parental education, direction of child's room's window, and floor level of dwelling.

†Estimation of individual exposure of 854 atopic children on the basis of emission monitoring in a 1-km² grid in the city of Dresden, Germany.

The estimated individual yearly mean exposures to benzene at the home address ranged from 2.0 µg/m³ to 8.3 µg/m³ air (mean, 3.9 µg/m³), thus being well below the German national ambient air quality standard of 10 µg/m³ air.²⁶ Self-reports and objective measures of traffic exposure showed a reasonable and statistically significant correlation (ranges of Kendall rank correlation coefficient τ_b = 0.25-0.52 for categories of self-reports and traffic counts and 0.12-0.17 for categories of self-reports and benzene exposure; categories as indicated in Table II).

Self-reported exposure to traffic in the first year of life was not positively associated with allergic sensitization to latex (Table II). Current traffic exposure tended to even be negatively associated with sensitization to latex. Very similar results were obtained if a cut-off level of greater than 0.7 kU/L was chosen (data not shown). "Present residence in a main street" was associated with a higher prevalence of antilatel IgE of greater than 3.5 kU/L (yes: 13/280 [4.6%] vs no: 10/547 [1.8%], P = .02). However, for all other exposure variables, no significant association of traffic exposure and IgE levels above 3.5 kU/L was found. No significant differences of antilatel IgE concentrations as a continuous variable were found for any estimate of traffic exposure.

Stratified analysis of two subpopulations of children with presumably higher exposure to traffic exhaust (children in dwellings at low floors and children whose rooms had windows opening toward the street) showed similar trends. In addition, when evaluating the associations between latex sensitization and traffic exposure in the whole population, including atopic and nonatopic children (n = 2505), the results were similar (data not shown).

In a multivariate analysis including potential confounders, none of the surrogates of traffic exposure was significantly associated with the prevalence of antilatel IgE levels above 0.35 kU/L (Table III) or 0.7 kU/L (data not shown). In keeping with the positive bivariate associations, a significantly increased odds ratio for levels above 3.5 kU/L was found for children "living in a main street at present" (adjusted prevalence odds ratio, 3.7; 95% confidence interval, 1.4-10.0), but none of the other exposure variables was associated with high IgE levels.

DISCUSSION

This study examined a number of subjective and objective indicators of traffic exposure at birth and at present and allergic sensitization against latex in a large population-based sample of 5- to 7-year-old children and 9- to 11-year-old children. Low levels of specific IgE against latex (>0.35 kU/L) were found in the sera of 150 (6.0%) children. The observed prevalence of antilatel IgE of greater than 3.5 kU/L (23/2505 [0.9%]) was similar to the prevalence in a sample of adult Italian blood donors (6/1025 [0.6%]).²⁷ In an unselected population of 1175 Italian children of comparable age, positive skin prick test (SPT) responses against latex were found in 8 (0.7%) of 1175 individuals.²⁸ However, the comparability of IgE determinations and SPT reactions in samples from the general population has not been evaluated yet.

Our study does not find a consistent association between traffic exposure and any degree of latex sensitization. In contrast, a Swiss group reported a nonsignificantly increased odds ratio for latex sensitization in ado-

lescents reporting exposure to constant car and truck traffic.²⁹ However, the authors of this pilot study did not disentangle the effects of traffic exposure on atopic sensitization in general and against latex specifically, and no objective markers of traffic exposure were considered.

Clinically relevant sensitization to latex is strongly associated with a high degree of direct cutaneous or mucosal exposure to natural rubber in, for example, health care workers³⁰ or children with spina bifida and urogenital abnormalities.³¹ It is not known whether lower exposures (eg, through pacifiers) are also associated with an elevated risk of latex sensitization.³² Tire treads made from blends of crumbled natural and synthetic rubbers contain 200-fold less latex allergen per unit of protein than latex gloves, a dipped product.¹³ Exposure to natural rubber in the air near freeways is thus estimated to be orders of magnitude lower than in the rubber-manufacturing industry, where a Dutch study of 208 workers found no elevated IgE levels against latex.³³ Miguel et al³⁴ did not find substantial amounts of latex in dust vacuumed from paved roads. Reflecting their own contradictory results reported earlier,¹³ the authors speculated that the vicinity of curves may be contaminated with latex particles, whereas straight sections of roads are not. In conclusion, the reason for the lack of an association between traffic exposure and latex sensitization may be that traffic exposure is not always associated with exposure to sufficient amounts of sensitizing latex particles.

Exposure to diesel exhaust particles might play a specific adjuvant role, favoring sensitization to latex particles derived from truck tires.³⁵ We found no association between self-reported truck traffic and latex sensitization. However, diesel exhaust was not measured in our study.

Some possible methodical reasons for the failure to detect an association must be discussed as well. Data from children who underwent surgery suggest that latex exposure in early life has the most profound effect on the risk of sensitization.³⁶ In this study traffic exposure in the first year of life has only been assessed by parental self-report. Parental reports of earlier traffic exposure had a slightly lower reliability than self-reports of current traffic exposure. Because the serologic status of the latex-sensitized children was not known to their parents when they answered the questionnaire, differential misclassification of traffic is unlikely. Nondifferential misclassification may have attenuated estimates of early effects.

The increased risk of latex sensitization in health care workers suggests that latex sensitization can also be acquired in later life. However, we found a rather inverse association between traffic exposure at present and latex sensitization. This might be explained by selective migration of families with sensitized children. However, the migration in our sample could be assessed by a follow-up contact after 2 years when the laboratory results were mailed to the families participating in blood examinations; 5.4% had moved, and 94.6% still lived at the same address. Children with positive results for allergy tests (SX1 assay and antilatel IgE) had not moved more often than others.

The sample evaluated in this study comprised more children with atopic parents and more 9- to 11-year-old children than the primary population-based sample. This subgroup might be more susceptible to allergic sensitization. In a more susceptible group, any effect on sensitization would be overestimated rather than underestimated. It is therefore unlikely that our results can be explained by selection bias. In addition, our study had a power of 80% ($\alpha = .05$) to detect a difference of 3.0% versus 5.5% in the prevalence of latex sensitization in the general population and 10% versus 17% in the risk group of atopic children. It is therefore unlikely that our study missed a relevant traffic effect on latex sensitization caused by too small a sample size.

The determination of antilatel IgE in serum has a good predictive value for latex allergy in patients with spina bifida³⁷ but is less specific than the SPT with natural rubber milk in populations at lower risk,³¹ possibly because of cross-reactivity between latex and pollen allergens.^{38,39} In this study we have analyzed several degrees of sensitization because we wanted to detect sensitization rather than clinical allergy against latex. One might argue that the lack of an association of lower levels of antilatel IgE and traffic exposure is caused by the limited specificity of this measurement. However, we also did not find a consistent association between traffic exposure and higher concentrations of antilatel IgE (0.7 or 3.5 kU/L), which may be more specific for sensitization against latex.

Although we did not examine clinical latex allergy in this study, the lack of an association between traffic exposure and sensitization to latex makes it unlikely that traffic exposure is an important determinant for the development of latex allergy. This notion is further supported by the fact that higher concentrations of antilatel IgE, which are more strongly associated with clinical latex allergy,⁴⁰ were also not associated with traffic exposure.

In summary, this study investigated the association between exposure to traffic in the street of residence and allergic sensitization to latex in a large population-based sample of atopic children. Our findings do not confirm the hypothesis that traffic exposure is associated with allergic sensitization to latex.

We thank B. Vollheim from the Communal Department for Protection of the Environment for providing data on traffic counts and U. Olunczek from the Institute of Photogrammetry and Remote Sensing, University of Technology, Dresden, for his helpful assistance in the assignment of the pollution data to the children's addresses.

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