Parental Stress Increases the Detrimental Effect of Traffic Exposure on Children's Lung Function

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***Rationale*: Emerging evidence indicates that psychosocial stress enhances the effect of traffic exposure on the development of asthma.**

***Objectives*: We hypothesized that psychosocial stress would also modify the effect of traffic exposure on lung function deficits.**

***Methods*: We studied 1,399 participants in the Southern California Children's Health Study undergoing lung function testing (mean age, 11.2 yr). We used hierarchical mixed models to assess the joint effect of traffic-related air pollution and stress on lung function.**

***Measurements and Main Results*: Psychosocial stress in each child's household was assessed based on parental response to the perceived stress scale (range, 0–16) at study entry. Exposures to nitric oxide, nitrogen dioxide, and total oxides of nitrogen (NO_x), surrogates of the traffic-related pollution mixture, were estimated at schools and residences based on a land-use regression model. Among children from high-stress households (parental perceived stress scale >4) deficits in FEV_1 of 4.5 (95% confidence interval, -6.5 to -2.4) and of 2.8% (-5.7 to 0.3) were associated with each 21.8 ppb increase in NO_x at homes and schools, respectively. These pollutant effects were significantly larger in the high-stress compared with lower-stress households (interaction *P* value 0.007 and 0.05 for residential and school NOx, respectively). No significant NOx effects were observed in children from low-stress households. A similar pattern of association was observed for FVC. The observed associations for FEV_1 and FVC remained after adjusting for sociodemographic factors and after restricting the analysis to children who do not have asthma.**

***Conclusions*: A high-stress home environment is associated with increased susceptibility to lung function effects of air pollution both at home and at school.**

Keywords: parental stress; traffic exposure; lung function; children

Exposure to air pollution, especially to local particulate and the traffic-related air pollution (TRP) mixture near children's homes, has been associated with asthma prevalence, incidence, and exacerbation, and with lung function deficits (1–8). Genetic and toxicologic studies suggest that these relationships are causal (9–12). Emerging evidence indicates that the susceptibility to health effects caused by air pollution is greater among socially deprived populations (13–15), which might be because of psychosocial stress (13). Although the exact mechanism for this susceptibility remains unknown, one possible explanation is synergistic psychosocial stress-induced and TRP-induced oxidative stress, a common biologic effect of both exposures, resulting in airway inflammation (11, 16).

Psychosocial stress is associated with high-level endogenous steroid production, which leads paradoxically to steroid resistance and a diminished antiinflammatory effect of cortisol (17). Diminished production of antiinflammatory IL-5 and IFN-γ from peripheral blood mononuclear cells, and an increase in eosinophils (18), has been reported from children with asthma who perceived low parental support (a proxy for psychosocial stress). Because psychosocial stress can provide a milieu that can augment the effect of inflammatory and oxidative stressors present in TRP (19–21), we hypothesized that children exposed to high level of chronic stress would be more susceptible to the detrimental effect of TRP on lung function deficits in children.

We investigated our hypothesis in a large cohort of Children's Health Study (CHS) participants, for who detailed sociodemographic household information, markers of perceived parental stress as a proxy for children's psychosocial stress, lung function measurements, and validated residential and school TRP exposure estimates were available. Some of the results of these studies have been previously reported in the form of an abstract (22).

AT A GLANCE COMMENTARY

Scientific Knowledge on the Subject

Traffic exposure has been shown to be associated with asthma risk and lung function in children. Psychosocial stress increases susceptibility to the detrimental effects of traffic exposure–mediated asthma risk in children. Currently, the role of stress on lung function levels in healthy children remains unknown.

What This Study Adds to the Field

This study shows that children who grow up in households with high levels of psychosocial stress are more susceptible to the detrimental effects of traffic exposure on lung function compared with children who grow up in households with low parental stress.

METHODS

Study Population

Characteristics of the study population have been described previously (6). The current analysis involves 1,399 children (mean age,11.2; SD 0.63) from eight communities in Southern California. These

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communities were selected to reflect a broad range of regional air pollutant exposures across communities and large gradients in traffic exposure within communities. Children underwent spirometric lung function testing during the 2008–2009 school year. Information about respiratory illness and environmental exposures was provided by parents in a questionnaire sent home with children at the time of spirometric testing. Additional information, including sociodemographic characteristics and parental psychosocial stress, was available from a questionnaire parents completed at the time of enrollment of this cohort in 2002–2003. Parents provided informed consent (*see* the online supplement).

Parental Stress

The perceived stress scale (PSS), which was used to measure parental stress, has been validated as a measure of negative affective states and physical symptoms of stress (23). We used a four-item version of the scale that has been used previously to predict incidence of wheeze and asthma in children (24, 25). Items included: "In the last month, how often have you felt" (*1*) "that you were unable to control the important things in your life"; (*2*) "confident about your ability to handle your personal problems"; (*3*) "that things were going your way"; and (*4*) "your difficulties were piling up so high that you could not overcome them." Each item was scored on a scale of 0–4, with larger values indicating higher stress levels. The PSS gives equal weight to each item, resulting in an overall score ranging from 0–16. The PSS was also dichotomized at its sample median into high (>4) and low (≤ 4) parental stress.

Other Covariates

Sociodemographic characteristics (i.e., race, income, and insurance), secondhand exposure to tobacco smoke, health insurance, housing characteristics, history of allergy, and child and parental asthma were assessed by questionnaire (*see* the online supplement).

Local TRP

Exposure to nitric oxide (NO), nitrogen dioxide (NO_2), and total oxides of nitrogen (NO_x) was estimated based on measurements of these pollutants at 940 locations in CHS communities during 2 weeks in each of the two Southern California seasons. A prediction model was developed based on distance to traffic corridors, traffic volume, average vehicle NOx emission rates, wind speed and direction and height of the mixing layer, and elevation and population density (26). Cross-validation analysis showed that this model was able to predict 68%, 61%, and 72% of the local variation in average levels of NO_2, NO, and NOx, respectively (26). This model was used to predict concentrations of these pollutants at the homes and schools of study participants.

Lung Function Measurements

Maximal-effort spirometry and standing height and weight were measured by trained field technicians, using previously described procedures (7). We evaluated three measures of lung function in this analysis: (1) FVC, (2) FEV_1, and (3) forced expiratory flow over the mid-range of expiration ($FEF_{25–75}$).

Asthma

Lifetime asthma at study entry was assessed based on parental report of physician-diagnosed asthma. Children who were asthma-free at study entry were classified as having new-onset asthma when a parent reported physician-diagnosed asthma on the annual follow-up questionnaire. Because low lung function is associated with asthma and we have previously reported that TRP exposure was associated with increased risk of new-onset asthma in children residing in high-stress households (25), we conducted sensitivity analyses restricted to children without any history of asthma.

Statistical Methods

We evaluated the determinants of high parental stress using univariate logistic regression models. We used hierarchical two-stage multivariate regression models to assess the joint effect of TRP and stress on lung function levels with interaction terms after accounting for clustering at community and school levels with adjustments for known determinants of lung function (*see* the online supplement). Heterogeneity of TRP effects by PSS was assessed by comparing nested models using a partial likelihood ratio test with and without appropriate interaction terms (i.e., for PSS with TRP). We fitted the following model:

$$LF = \alpha + \beta_1 TRP_i + \beta_2 TRP_s + \beta_3 PSS + \gamma_1 PSS^* TRP_i + \gamma_2 PSS^* TRPs + C + \varepsilon_s, \quad (Eq.1)$$

where LF represented one of the lung function measurements, and TRP_i (residential TRP centered to local school TRP) and TRP_s represented one of the TRPs at the residence and school level, respectively. The parameters γ_1 and γ_2 quantify modifying effects of stress on the association between LF and residential and school level TRP, respectively. The model included school-specific (ε_s) random effects and adjustments for several potential confounders (C) including community of residence as fixed effects. The approximately orthogonal relationship between school and deviated residential TRPs allowed us to assess the joint effect of both exposures in the same model. Effect estimates for both residential and school TRPs were scaled to an increase in exposure equivalent to 2 SD based on the distribution of residential TRP.

We were concerned that the modifying effects of PSS (captured by γ_1 and γ_2) could be confounded by other social factors. We addressed this issue by including additional interaction terms between TRP and other social factors in the above model. We determined whether the estimates of γ_1 and γ_2 changed by at least 10% on including any of these additional interaction terms.

All analyses were performed using Statistical Analysis System software (SAS version 9.2; SAS Institute Inc., Cary, NC). A two-tailed alternative hypothesis was assumed in all analyses with significance being claimed at the 0.05 probability level.

RESULTS

We observed a wide range of variation in parental stress among the participants' parents. The observed PSS values ranged from 0–16, with mean of 3.9 (SD 2.8) and median of 4. Approximately 11.5% (n = 156) of parents reported no stress in their life (PSS = 0). Less than 25% of the parents reported PSS less than two (23%) or PSS greater than seven (20%). High parental PSS varied by sociodemographic and personal factors (Table 1). Compared with whites, Asian and Hispanic households had higher levels of PSS. Among Hispanics, completion of the questionnaire in Spanish was associated with an increased risk of high parental stress. Neither parental nor child's asthma status was associated with parental stress. Characteristics suggestive of low socioeconomic status were associated with increased parental stress, including low parental education, annual household income less than $30,000, lack of health insurance, and no air-conditioner in the home. In this cohort, 6.4% of children did not have health insurance, 17.4% had parents with less than a high school education, and 18.8% did not live in a single-family home, characteristics that were associated with high parental stress. About 5.3% of the parents reported at least one smoker at home and 6.1% of children had *in utero* exposure to maternal smoking. Although maternal smoking during pregnancy was not associated with parental stress, a smoker in the home was associated with a 1.7-fold increased risk of high parental stress.

The TRPs had a wide range of variability for both homes and schools. For example, the mean residential and school NO_x were 46.7 ppb (SD 19.6) and 49.7 (SD 19.2) ppb, with ranges of 101.6 and 61.8, respectively (Table 2). In the neighborhood of children's homes around each school, residential NO_x ranged from 55.5 ppb less than to 52.7 ppb greater than the school estimate (SD 10.9; data not shown). Similar ranges in the distribution of NO and NO_2 were observed.

TABLE 1. SUBJECT CHARACTERISTICS AND ASSOCIATIONS WITH PARENTAL STRESS

Risk Factor	N (%)	High Parental Stress OR (95% CI)
Subject characteristics		
Male	672 (48)	0.90 (0.73–1.12)
Race and ethnicity		
White	629 (45)	1.00
Asian	64 (4.6)	2.67 (1.5–4.6)
African American	22 (1.6)	0.84 (0.4–2)
Do not know	149 (10.7)	2.41 (1.7–3.5)
Mixed	194 (13.9)	0.95 (0.7–1.3)
Other or Native American Indian	341 (24.4)	1.83 (1.4–2.4)
Hispanicity		
Not Hispanic	595 (42.5)	1.00
Hispanic	769 (55)	1.52 (1.2–1.9)
Do not know	35 (2.5)	2.99 (1.4–6.3)
Spanish language questionnaire (among Hispanic whites)	288 (38)	1.79 (1.3–2.4)
Asthma in children	173 (13.1)	0.91 (0.7–1.3)
Parental history of asthma	293 (22.3)	1.06 (0.8–1.4)
Sociodemographic factors		
Parental education		
At least college diploma	419 (30.8)	1.00
High school or some college	704 (51.8)	2.04 (1.5–2.8)
Did not finish high school	236 (17.4)	3.04 (2.3–4.5)
Household income		
≥$30,000	913 (74.7)	1.00
<$30,000	309 (25.3)	2.44 (1.9–3.2)
Health insurance		
Yes	1,231 (88)	1.00
No	93 (6.6)	1.49 (1–2.2)
Home characteristics		
Mildew at home	348 (27.2)	1.20 (0.9–1.5)
Cats at home	248 (18.1)	0.68 (0.5–0.9)
Dogs at home	388 (28.4)	0.87 (0.7–1.1)
Cockroaches in home	141 (10.4)	1.79 (1.3–2.6)
In utero smoking	83 (6.1)	1.06 (0.7–1.7)
Any smoker inside home	73 (5.3)	1.72 (1.1–2.8)
Air conditioning at home	900 (65.2)	0.53 (0.4–0.7)
Type of home		
Single family house	1,114 (81.2)	1.00
Apartment (2–10 units)	191 (13.9)	2.46 (1.8–3.4)
Apartment (>10 units)	45 (3.3)	2.20 (1.2–4.1)
Mobile home	22 (1.6)	1.92 (0.8–4.6)

Definition of abbreviations: CI = confidence interval; OR = odds ratio.

TABLE 2. DISTRIBUTION OF ANNUAL AVERAGE RESIDENTIAL TRAFFIC MODELED POLLUTION

TRP*	Location	Mean (SD)	Median (IQR)	Min	Max	Range
NOx	Home	46.7 (19.6)	44.3 (31.7)	6.15	107.7	101.5
	School	49.7 (19.2)	49.1 (27.8)	16.6	78.3	61.8
NO_2	Home	23.2 (6.96)	24.3 (10.6)	3.70	41.5	37.8
	School	23.8 (6.73)	24.7 (7.8)	8.25	33.7	25.5
NO	Home	23.4 (14.3)	19.1 (20.3)	2.11	77	74.9
	School	25.8 (14)	21.4 (24.2)	5.84	51.4	45.5

Definition of abbreviations: IQR = interquartile range; NO = nitric oxide; NO_2 = nitrogen dioxide; NO_x = total oxides of nitrogen.

*Traffic-related pollution (oxides of nitrogen) in parts per billion.

On average the lung volume and flows were larger for boys than girls; however, the effect of TRP did not differ by sex (interaction P value > 0.10 for all TRP), so we combined girls and boys in analyses of lung function. In models adjusted for sex and other relevant covariates, we observed a detrimental effect of TRPs on lung function. For example, each 21.8 ppb (2 SD [10.9] of average school-deviated residential NOx) increase in residential NO_x was associated with 2.31% (P value < 0.001) and 1.85% (P value < 0.001) decrease in FVC and FEV_1, respectively (Table 3). No statistically significant association was observed between any TRP and $FEF_{25–75}$. A similar pattern of associations was observed for NO and NO_2; however, the effect of NO was modestly larger and more significant than NOx or NO_2. We did not observe any statistically significant associations between lung function measures and TRP at school.

We did not observe any statistically significant associations between PSS and lung function levels (*see* Table E1 in the online supplement). However, strong modifying effects of PSS on the association between FVC and FEV_1 and both home and school TRPs were observed (Table 3). The adverse effect of TRP was restricted to children from high-stress households and these associations were most strongly associated with predicted NO. For example, among children with high parental stress, each 21.8 ppb increase of residential and school NOx was associated with 4.5% (95% confidence interval [CI], −6.7 to −2.4) and 2.8% (95% CI, −5.7 to 0.28) deficits in FEV_1, respectively. A stronger and more significant interaction was observed between household stress and NO level at homes and schools. No significant associations with TRP were observed in low-stress households. Although a similar pattern was observed for $FEF_{25–75}$, the interactions between stress and TRPs were not statistically significant.

We evaluated whether the modifying effect of PSS could be explained by other factors that were associated with PSS (Table 1). Lack of health insurance (n = 93) modified the association of FEV_1 with residential NOx (interaction P value = 0.04) but not with school NO_x (interaction P value = 0.59; *see* Table E2). For each 21.8 ppb increase in NOx, the FEV_1 deficit was 9.03% (95% CI, −13.4 to −3.8) for children without health insurance and −1.3% (95% CI, −2.9 to 0.25) for children with health insurance. However, the joint effects of health insurance and NOx did not fully explain the observed modifying effect of PSS on lung function. The interaction between PSS and NOx remained significant (P value = 0.01) and the interaction between health insurance and NOx was attenuated and marginally significant (P value = 0.07) when both terms were included in the same model. Even after restricting the analysis to children with health insurance, we observed that each 21.8 ppb increase in NOx was associated with 3.7% (95% CI, −5.86 to −1.49) deficit in FEV_1 among children from high-stress households compared with those from low-stress households (*see* Table E3).

Another possible explanation of our findings is that the lung function deficits were caused by differential risk of TRP by asthma status in this population (25). To address this concern, we performed sensitivity analysis by excluding all prevalent (n = 173) and new-onset asthma cases (n = 165). Among the children without any asthma diagnosis (n = 1,061), we observed a modifying effect of household stress on the association between lung function and TRP exposure at home and school (Table 4) that was similar to that observed in the entire sample. Each 21.8 ppb increase of residential and school NOx was associated with a 4.3% (95% CI, −6.55 to −2.07) and 3.5% (95% CI, −6.68 to −0.14) decrease in FEV_1, respectively, among children with high parental stress.

DISCUSSION

To our knowledge, this is the first study demonstrating that growing up in a stressful household was associated with larger TRP-induced lung function deficits in children. Children whose parents reported a stressful life early in the child's school years experienced a detrimental effect associated with exposure to both residential and school TRP on their lung function volume and flow in large airways. The observed pattern of effects in these children could not be explained by asthma or by a range

TABLE 3. EFFECT OF TRP ON LUNG FUNCTION MEASUREMENTS, STRATIFIED BY PARENTAL STRESS LEVEL (HIGH OR LOW)*

TRP	Location	Main Effect % Change (*95% CI*)	Low Parental Stress % Change (*95% CI*)	High Parental Stress % Change (*95% CI*)	Interaction *P* Value
			FVC		
NOx	Home	−2.31 (−3.8 to −0.8)	0.02 (−2.1 to 2.2)	−4.73 (−6.7 to −2.7)	<0.01
	School	−0.98 (−3 to 1)	1.04 (−1.7 to 3.9)	−3.01 (−5.9 to −0.1)	0.07
NO	Home	−2.60 (−4.2 to −1)	−0.04 (−2.4 to 2.4)	−4.84 (−6.9 to −2.8)	<0.01
	School	−1.39 (−3.5 to 0.8)	0.82 (−2.2 to 4)	−3.30 (−6.3 to −0.2)	0.04
NO_2	Home	−2.00 (−3.4 to −0.6)	0.07 (−1.9 to 2)	−4.55 (−6.5 to −2.6)	0.02
	School	−0.59 (−2.5 to 1.3)	1.29 (−1.3 to 4)	−2.68 (−5.4 to 0.1)	>0.10
			FEV_1		
NOx	Home	−1.85 (−3.3 to −0.4)	0.62 (−1.6 to 2.9)	−4.47 (−6.5 to −2.4)	<0.01
	School	−0.15 (−2.2 to 1.9)	2.52 (−0.3 to 5.5)	−2.77 (−5.7 to 0.3)	0.07
NO	Home	−2.09 (−3.7 to −0.5)	0.75 (−1.7 to 3.3)	−4.61 (−6.7 to −2.5)	<0.01
	School	−0.40 (−2.6 to 1.8)	2.62 (−0.5 to 5.8)	−3.03 (−6.1 to 0.2)	0.04
NO_2	Home	−1.60 (−3 to −0.2)	0.54 (−1.4 to 2.5)	−4.31 (−6.3 to −2.3)	0.02
	School	0.06 (−1.9 to 2)	2.43 (−0.3 to 5.2)	−2.48 (−5.3 to 0.4)	>0.10
			MMEF		
NOx	Home	−0.72 (−3.7 to 2.4)	0.85 (−3.4 to 5.3)	−2.57 (−6.7 to 1.8)	>0.10
	School	1.21 (−2.9 to 5.5)	3.28 (−2.4 to 9.2)	−0.82 (−6.8 to 5.6)	>0.10
NO	Home	−1.09 (−4.3 to 2.2)	0.77 (−4 to 5.8)	−2.90 (−7.2 to 1.6)	>0.10
	School	0.95 (−3.4 to 5.5)	3.39 (−2.7 to 9.9)	−1.15 (−7.5 to 5.6)	>0.10
NO_2	Home	−0.55 (−3.4 to 2.4)	0.88 (−3 to 4.9)	−2.60 (−6.7 to 1.7)	>0.10
	School	1.19 (−2.7 to 5.3)	2.99 (−2.3 to 8.6)	−0.76 (−6.5 to 5.4)	>0.10

Definition of abbreviations: CI = confidence interval; MMEF = $FEF_{25–75}$; NO = nitric oxide; NO_2 = nitrogen dioxide; NO_x = total oxides of nitrogen; TRP = traffic-related pollution.

*Models adjusted for log height and square term for log height, body mass index and square term for body mass index, sex, age, age–sex interaction, race, Hispanic ethnicity, respiratory illness at time of lung function, field technician, and community. Percent (%) change was scaled to 2 SD (NOx = 21.8 ppb; NO = 16.2 ppb; and NO_2 = 7.3 ppb) of the TRP difference between home and school (averaged across schools).

of behavioral, socioeconomic, or environmental factors associated with stress.

Over the last decade intensive investigation indicates that traffic-related particulate or gaseous pollutants result in adverse effects on respiratory health in children (3, 7, 11, 12, 27, 28). In the CHS we have shown that particulate matter and ambient NO_2 (28), and living close to a freeway, were associated with a significant decrease in lung function growth in children 10–18 years of age (7). Emerging evidence indicates that these exposures result in formation of radical oxygen species leading to inflammation-mediated injury to lung tissue (29, 30). Furthermore, TRP exposure has been shown to have direct proinflammatory effects by enhancing mast cell degranulation and cytokine release (31, 32).

A potential explanation for the stress-related pattern of TRP respiratory effects is the biologic pathways common to effects of TRP and stress (13). Psychosocial stress has been linked to inflammation and oxidative stress (16). These common biologic pathways may explain the recent epidemiologic studies demonstrating that the risk of TRP-associated asthma was larger in children in high-stress homes or with a history of exposure to violence (20, 25). Although the epidemiologic studies have largely examined modifying effects of stress on the relationship between TRP and asthma, one toxicologic study found that stress modified the effect of pollution on lung function (21). In a rat model, low respiratory flows and volumes resulting from exposure to ambient fine particles occurred only in stressed animals. Moreover, markers of systemic inflammation, such as tumor necrosis factor-α, C-reactive protein, white blood count, and absolute monocyte and lymphocyte counts, were on average higher among the stressed animals compared with the nonstressed control animals. Psychosocial stress-induced heightened inflammatory status in CHS participants is one possible explanation for the findings of the current study. Further studies are warranted to delineate the involved biologic pathways.

Another unique finding of this study was that stress-related susceptibility was related to TRP exposure effects both at home and at school. Children in this age group spend almost one-third of their daytime hours at school so exposure at school is an important contributor to total exposure. Because the residential TRP levels were deviated from the corresponding school TRP level, effects could be assessed jointly in the regression model. The observed consistent pattern of TRP susceptibility among children growing up in a high-stress household regardless of the location of TRP exposure also strengthens a causal interpretation of the observed lung function associations with TRP exposure. Furthermore, the higher effect estimates and lower *P* values in the order of NO, NOx, and NO_2 suggest that reactive primary traffic emissions were better reflected by NO and it might be of etiologic importance.

Emerging evidence suggests that social deprivation promotes the adverse effect of air pollution on respiratory health outcomes (20, 33, 34), and psychosocial stress associated with deprivation is one possible explanation for this effect (9, 13, 19, 35, 36). In this study markers of social deprivation (i.e., low socioeconomic status; lack of health insurance; and housing characteristics, such as secondhand smoke exposure, cockroaches in the home, and lack of air conditioning) were associated with higher household stress (Table 1). However, none of those factors explained the large TRP-related health effects we observed in children with high household stress. Although lack of health insurance, a marker of low socioeconomic status, increased the susceptibility to TRP exposure (Table E3), the modifying effect of stress was still evident even among children who had health insurance (Table E4).

A major strength of the study was the use of predicted TRP exposures that were calibrated from models based on actual measurement of NO, NO_2, and NOx at a large number of homes and schools in the study communities. The final exposure model accounted for traffic distance and volume, meteorology, and other characteristics of nearby land use (26). Approximately two-thirds of the within-community variability in these TRPs was explained by the model.

There are some limitations to these data. The TRP exposure was estimated at the center point of the school buildings, but

TABLE 4. EFFECT OF TRP ON LUNG FUNCTION MEASUREMENTS AMONG CHILDREN WITHOUT ANY HISTORY OF ASTHMA (N = 1061), STRATIFIED BY PARENTAL STRESS LEVEL (HIGH OR LOW)*

TRP	Location	Main Effect % Change (*95% CI*)	Low Parental Stress % Change (*95% CI*)	High Parental Stress% Change (*95% CI*)	Interaction *P* Value
			FVC		
NOx	Home	−2.49 (−4.1 to −0.9)	−0.47 (−2.9 to 2)	−4.72 (−6.9 to −2.5)	0.05
	School	−0.85 (−3.1 to 1.4)	1.39 (−1.7 to 4.6)	−3.74 (−6.9 to −0.4)	0.05
NO	Home	−2.93 (−4.7 to −1.2)	−0.80 (−3.5 to 2)	−4.94 (−7.2 to −2.6)	0.06
	School	−1.32 (−3.7 to 1.1)	1.05 (−2.3 to 4.6)	−4.12 (−7.5 to −0.7)	0.04
NO_2	Home	−2.01 (−3.5 to −0.5)	−0.18 (−2.3 to 2)	−4.39 (−6.6 to −2.2)	0.09
	School	−0.39 (−2.5 to 1.8)	1.66 (−1.3 to 4.7)	−3.26 (−6.4 to −0.1)	>0.10
			FEV_1		
NOx	Home	−1.88 (−3.5 to −0.2)	0.59 (−1.8 to, 3.1)	−4.34 (−6.6 to −2.1)	0.05
	School	0.09 (−2.2 to 2.4)	3.32 (0.1 to 6.7)	−3.46 (−6.7 to −0.1)	0.03
NO	Home	−2.24 (−4 to −0.5)	0.55 (−2.2 to 3.4)	−4.57 (−6.8 to −2.2)	0.06
	School	−0.23 (−2.6 to 2.2)	3.37 (−0.2 to 7)	−3.81 (−7.2 to −0.3)	0.02
NO_2	Home	−1.50 (−3 to 0.1)	0.63 (−1.6 to 2.9)	−4.06 (−6.3 to −1.8)	0.06
	School	0.37 (−1.8 to 2.6)	3.22 (0.2 to 6.4)	−3.06 (−6.2 to 0.2)	>0.10
			MMEF		
NOx	Home	0.24 (−3.1 to 3.7)	2.33 (−2.5 to 7.4)	−1.31 (−6.1 to 3.7)	>0.10
	School	2.13 (−2.5 to 7)	5.43 (−1 to 12.3)	−0.83 (−7.6 to 6.5)	>0.10
NO	Home	−0.10 (−3.7 to 3.7)	2.40 (−3.1 to 8.1)	−1.64 (−6.5 to 3.5)	>0.10
	School	1.89 (−3.1 to 7.1)	5.74 (−1.2 to 13.2)	−1.20 (−8.3 to 6.4)	>0.10
NO_2	Home	0.32 (−2.8 to 3.6)	2.12 (−2.2 to 6.6)	−1.35 (−6 to 3.5)	>0.10
	School	2.12 (−2.3 to 6.8)	4.93 (−1.1 to 11.3)	−0.72 (−7.3 to 6.3)	>0.10

Definition of abbreviations: CI = confidence interval; MMEF = $FEF_{25–75}$; NO = nitric oxide; NO_2 = nitrogen dioxide; NO_x = total oxides of nitrogen; TRP = traffic-related pollution.

*Models adjusted for log height and square term for log height, body mass index and square term for body mass index, sex, age, age–sex interaction, race, Hispanic ethnicity, respiratory illness at time of lung function, field technician, and community. Percent (%) change was scaled to 2 SD (NOx = 21.8 ppb; NO = 16.2 ppb; and NO_2 = 7.3 ppb) of the TRP difference between home and school (averaged across schools).

these TRP can have high variability on a small spatial scale. Therefore, children's true exposure at school may have been substantially different when they were outdoors exercising, a period of likely greatest vulnerability to TRP exposure because of increased ventilation rates and consequent increased lung dose. If the observed associations were causal, it is likely that accounting for this exposure uncertainty would have resulted in a larger estimated health effect. The use of parental stress as a proxy for psychosocial stress in the child limits our ability to differentiate between the effects of household and personal stress in these children. Parental stress has been shown to be indicative of stress in children (37, 38), and at the time of assessment of early life stress at study enrollment, these children were too young to provide reliable questionnaire information on their own stress levels. Studies with longitudinal measurement of parental stress, personal stress, and lung function measurement are needed to clarify the role of the psychologic stressors on lung function and lung function growth.

Findings of this study suggest that psychosocial stress may increase the susceptibility of the lung to the detrimental effect of TRP, resulting in decreased lung volume (FVC) and flow in the larger airways (FEV_1). Further research is warranted to identify the biologic pathways involved in the augmentation of the detrimental effect of TRP in children exposed to household stress. Beyond this etiologic importance, our findings also have potentially important public health implications. The magnitude of the TRP-associated deficits in FEV_1 and FVC levels in children growing up in high-stress households was larger than deficits reported for children exposed to maternal smoking during pregnancy and secondhand tobacco smoke (39, 40). Hispanic white children living in low income households have been reported to be exposed to high levels of TRP (41) and in our sample they also had higher levels of household stress. Therefore, these children may be a group at particularly high risk of preventable TRP-associated deficits in lung function. Furthermore, children spend a substantial amount of time at school where they are also exposed to TRP. In California, more than 10% of the public schools are within 150 m of major roadways with more than 25,000 daily vehicular traffic (42) and a similar pattern of heavy school-related traffic exposure has been reported in other major metropolitan areas across the United States (43). Our findings suggest that by regulating TRP levels around residential areas and schools, the adverse effect of TRP on lung function among vulnerable children could be reduced.

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