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Associations of Children's Lung Function with Ambient Air Pollution: Joint Effects of Regional and Near-roadway Pollutants

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Abstract

Background—Prior studies have reported adverse effects of either regional or near-roadway air pollution (NRAP) on lung function. However, there has been little study of the joint effects of these exposures.

Objectives—To assess the joint effects of NRAP and regional pollutants on childhood lung function in the Children's Health Study.

Methods—Lung function was measured on 1,811 children from eight Southern Californian communities. NRAP exposure was assessed based on (1) residential distance to the nearest freeway or major road and (2) estimated near-roadway contributions to residential nitrogen dioxide (NO₂), nitric oxide (NO), and total nitrogen oxides (NO_x). Exposure to regional ozone (O₃), NO₂, particulate matter with aerodynamic diameter less than 10 μm (PM₁₀) and 2.5 μm (PM_{2.5}) was measured continuously at community monitors.

Results—A 17.9 ppb (two standard deviation) increase in near-roadway NO_x was associated with deficits of 1.6% in FVC (p=0.005) and 1.1% in FEV₁ (p=0.048). Effects were observed in all communities and were similar for NO₂ and NO. Residential proximity to a freeway was associated with a reduction in FVC. Lung function deficits of 2–3% were associated with regional PM₁₀ and PM_{2.5} (FVC and FEV₁) and with O₃ (FEV₁), but not NO₂, across the range of exposure between communities. Associations with regional pollution and NRAP were independent in models adjusted for each. Effects of NRAP were not modified by regional pollutant concentrations.

Conclusions—Results indicate that NRAP and regional air pollution have independent adverse effects on childhood lung function.

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COMPETING INTERESTS

Frederick Lurmann is employed by Sonoma Technology, Inc (Petaluma, CA). Rob McConnell has received research support from an air quality violations settlement agreement between the South Coast Air Quality Management District, a California state regulatory agency, and BP. The other authors declare they have no actual or potential competing interests.

Keywords

traffic; lung function; air pollution; children; land-use regression

INTRODUCTION

Reduced lung function has been associated with subsequent increased risk of overall mortality, including coronary artery disease and respiratory disease in adults [1] and with asthma in children.[2] Therefore, identifying factors that reduce lung function but are modifiable could lead to interventions with large public benefits.

Regional air pollutants have been associated with reduced lung function in both adults and children.[3–4] Studies examining lung function in children exposed to local residential near-roadway air pollution (NRAP) have not found consistent associations,[5–11] although exposure metrics differed across studies. However, there has been little investigation of the joint effects of regional and NRAP exposures.

In this study, we assessed the joint effects of NRAP and regional exposures to ozone (O₃), particulate matter with aerodynamic diameter of less than 10 μm and 2.5 μm (PM₁₀ and PM_{2.5}), and nitrogen dioxide (NO₂) on childhood lung function in the Children's Health Study (CHS). We examined associations with both traffic proximity measures and land-use regression modeled NRAP based on a prior dense air monitoring study of NO_x conducted within CHS communities.

METHODS

Study Subjects

The CHS has enrolled over 11,000 children in a series of cohorts investigating the health effects of air pollution. The current analysis includes a cohort established in 2002–2003 when participants were 5–7 years of age.[12] During the 2007–2008 school year, lung function was measured on 1,811 cohort participants (82% of the active cohort) from eight communities, as described in detail in the Online Supplement.

Questionnaires

Questionnaires completed by parents or guardians at study enrollment provided information on participants' health, socio-demographic and other exposures, which was updated yearly. A complete list of covariates is described in the Online Supplement.

Lung Function

Trained technicians measured lung function, weight, and height, and collected information about recent acute respiratory illness. Using pressure-transducer-based spirometers (Screenstar Spirometers, Morgan Scientific, Haverhill, MA), we identified the maximal forced expiratory volume during the first second (FEV₁) and forced vital capacity (FVC) from a series of seven efforts from each child, as described previously.[13]

Air Pollution Exposure

NRAP exposures at each child's residence and school were based on estimates of surrogates, including distance to freeways, highways, and large surface streets. Spatial land use regression models were developed based on an extensive monitoring campaign of nitrogen oxides (NO_x) and nitrogen dioxide (NO_2) and by subtraction nitrogen oxide (NO) at over 900 locations in CHS communities, as described previously.[14] Key predictors included distance to freeways and major roads, traffic volumes and their emissions-weighted dispersion estimates, with lesser contributions from population density and local variation in elevation. The resulting annual average predicted residential concentrations of near-roadway NO , NO_2 , and NO_x , incrementally increased above regional background, was used in analyses, as described below.

The regional level of NO_2 , $\text{PM}_{2.5}$, PM_{10} , and O_3 was computed as the mean of the six years of each pollutant measured continuously at a central monitoring location in each community from cohort recruitment (2002) to the recording of lung function tests (2007).

Additional details of NRAP and regional pollutant exposure assessment are provided in the Online Supplement.

Statistical Methods

We fitted linear regression models (with fixed effects for each study community) to investigate associations of FVC and FEV_1 with NRAP and a mixed model that included a random intercept for community to assess associations with regional pollutants and joint effects with NRAP. Each pulmonary function outcome was log transformed to satisfy the assumptions of the models. All models were adjusted for demographic and anthropomorphic characteristics (eg. height) and selected other potential confounders (eg. spirometry technician). In sensitivity analyses, other potential confounders and effect modifiers were examined using standard methods described in further detail in the Online Supplement.

The NRAP NO_x (and NO and NO_2) predicted residential exposures were deviated from a community-specific mean. Conceptually, this allowed examination of the effect of the complex NRAP mixture, for which the nitrogen oxides are only a surrogate, and to distinguish it from the regional NO_2 effect, which was assessed based on the continuous measurements at the community monitor so as to be comparable to other regional pollutant assessments. This procedure was also necessary to make the NRAP NO_x approximately orthogonal (uncorrelated) to cross-community regional exposures in the mixed models. Health effect estimates were scaled to the range of long-term average regional pollution across all communities and to two standard deviations in the predicted NRAP nitrogen oxides.

Based on our final model, we also computed estimated lung function representative of different combinations of high and low regional and NRAP environments. Low regional pollution was based on the minimum value of regional $\text{PM}_{2.5}$ while low NRAP was defined as one standard deviation below the mean value for deviated NO_x . Conversely, high regional pollution was based on the maximum value of regional $\text{PM}_{2.5}$ and high NRAP was defined as one standard deviation above the mean value for deviated NO_x . We expressed the

predicted lung function in these different environments as percentages relative to those in the cleanest environment (low regional and low NRAP).

RESULTS

The average age at lung function measurement was 11.2 years (SD=0.6). A plurality of participants was White (40%) and a majority was of Hispanic ethnicity (57%, Table 1). Household income less than \$30,000 and parental education less than high school were common, and secondhand tobacco smoke exposure was uncommon.

Overall, 27% of children lived within 500 m of a freeway, while 20%, 15% and 38% lived 500–1,000 m, 1,000–1,500 m, or >1,500 m from a freeway, respectively (Supplemental Material, Table S-1). There was 14% of children who lived within 75 m of a major road (mostly non-freeway), 17% between 75 and 150 m, 28% between 150 and 300 m, and 40% at least 300 m. The distributions of residential proximity to freeways and major roads varied substantially from community to community. Predicted residential near-roadway NO_x, NO, and NO₂ showed wide variation within most study communities (Figure 1). Correlations among regional pollutant levels ranged from 0.06 (between PM₁₀ and NO₂) to 0.80 (between PM₁₀ and PM_{2.5}; Supplemental Material, Table S-2). O₃ had relatively strong positive correlations with PM_{2.5} and PM₁₀. The correlation between predicted near-roadway NO, NO₂ and NO_x (within communities) exceeded 0.90 (Supplemental Material, Table S-3).

The means of FEV₁ and FVC for males were 2,474 ml and 2,902 ml, respectively, and the corresponding means for females were 2,442 ml and 2,783 ml. Living within 500 m of a freeway was associated with a nearly 2 percent deficit in FVC (−1.96%; 95% CI: −3.41%, −0.49%; p=0.009) compared to those living at least 1,500 m from a freeway (Table 2). Mean FEV₁ was also lower for children living within 500 m of a freeway but the association was not statistically significant. Although close proximity to a major road was negatively associated with each measure of lung function, these associations were not statistically significant.

Near-roadway residential NO_x, NO, and NO₂ had statistically significant negative associations with both FVC and FEV₁ (Table 2). For example, a two standard deviation increase in near-roadway NO_x exposure (17.9 ppb) was associated with a 1.56% deficit in FVC (−2.62, −0.49; p=0.005), and a 1.10% deficit in FEV₁ (−2.19, −0.01; p=0.048). Negative associations between near-roadway NO_x and lung function were observed within six of the eight study communities for FEV₁ (Figure 2A) and within all eight study communities for FVC (Figure 2B). There was not significant heterogeneity of near-roadway NO_x effects across the eight communities for either FEV₁ (p=0.61) or FVC (p=0.64).

Adjustment for potential confounding variables resulted in only small changes to the estimated effects of near-roadway residential NO_x on FEV₁ and FVC (Table 3). For example, across models that included various additional adjustments, the near-roadway NO_x-related deficits ranged from −0.96% to −1.12% (main model: −1.10%) for FEV₁, and from −1.40% to −1.60% (main model: −1.56%) for FVC. In an analysis restricted to children without asthma, the effect of near-roadway NO_x was similar to that in the entire

study population (1.19% decline in FEV₁ and 1.51% decline in FVC). The difference in effects between children with and without asthma was not statistically significant. There was also no significant heterogeneity in near-roadway NO_x effects on lung function in girls compared to boys. Although we have observed associations of lung function with exposure at schools of participants in this study in conjunction with psychosocial stress,[15] we observed no main effects of exposure in schools in this analysis (results not shown).

Deficits in FEV₁ of approximately 3% were observed across the range of community O₃ and PM_{2.5} levels (p=0.006 for O₃ and 0.001 for PM_{2.5}, Table 4 and Figure 3). A greater than 2% deficit was observed across the range of PM₁₀ exposure. Deficits in FVC of over 2% were also observed across the range of both PM_{2.5} and PM₁₀ (Table 4 and Figure 4); however, a single community (Mira Loma) appears to have driven the association between FVC and PM₁₀.

In models assessing the joint effects of regional and NRAP, there was little change in the strength of the regional pollutant associations with either FVC or FEV₁, after adjusting for near-roadway NO_x (Table 5). For FEV₁, there was little change in the unadjusted association of near-roadway NO_x (1.10% deficit in Table 2) after adjusting for regional pollutants effects (1.04% to 1.14% deficits in Table 5). For FVC, the unadjusted association with near-roadway NO_x (1.56% deficit in Table 2) was somewhat attenuated after adjusting for regional pollutants (1.40% to 1.49% deficits in Table 5), although the associations remained significant. Similar patterns of lung function deficits in two-pollutant models were observed for near-roadway NO and NO₂ (results not shown). The patterns of effects of freeway proximity associations were also similar in models including a regional pollutant and in models unadjusted for regional pollution (results not shown). We examined the possibility that background pollutant exposures might up-regulate pulmonary response to near-roadway pollutants resulting in larger lung function deficits in communities with high regional pollutants. However, none of the regional pollutants significantly modified the association between near-roadway residential NO_x and each of the lung function endpoints (results not shown).

DISCUSSION

These results indicate that exposure to near-roadway air pollution adversely affects childhood lung function. Strengths of the study were the ability to demonstrate consistent effects of NRAP using both roadway proximity and validated predicted NO_x markers for the NRAP mixture in communities with differing regional air quality, roadway networks, and geographical characteristics. The study design offered an unusual opportunity to demonstrate that associations of lung function with NRAP pollutant variation were independent of associations also observed with regional air pollution.

NRAP is a complex mixture of particles and reactive gases with oxidant and pro-inflammatory properties that could plausibly cause the observed lung function deficits.[16–17] Oxides of nitrogen were selected to develop prediction models for likely near-roadway variation of the mixture because they are inexpensive to measure with the spatial density needed to develop valid models. NO₂ also has known oxidant and immune-modulatory

properties and could contribute to the near-roadway lung functions effects,[18] although in our analysis it was not possible to distinguish NRAP NO₂ effects from other components of the mixture. The association of regional PM_{2.5} and PM₁₀ with both FEV₁ and FVC, and no effect of regional NO₂, suggests that there were independent effects of transported or secondary regional particulate matter and of the NRAP mixture (rather than NO₂). In addition, previous reports from the CHS (and other studies) showing associations of NRAP, but not regional pollutants, with prevalent and incident asthma [12 19–20] also are consistent with separate and independent effects of these diverse pollutant mixtures.

It is also possible that more complex combinations of regional and NRAP account for the observed associations, as toxicological and experimental studies indicate that interaction with other pollutants may enhance the effects of particle exposure.[21–22] Although the study design allowed us to examine the heterogeneity of NRAP health effects across multiple communities, we found little evidence for interaction between regional pollutants and NRAP. Rather, the adverse effects were relatively consistent in all eight study communities, although there was limited precision to each estimate because of limited community-specific sample size.

We have previously observed associations of regional PM [23] and traffic proximity [7] with growth of FVC, but accompanied by larger effects in FEV₁ in an older cohort of CHS participants. Other studies of traffic and lung function in elementary school and adolescent children have also found larger associations with flow rates than with FVC.[8–9 24] However, the current results are consistent with an observed effect of regional pollutants on FVC in a cross sectional analysis of prior CHS cohorts.[13] Additional follow up of this cohort is ongoing and may help elucidate these relationships.

Some previous studies that have looked at associations between residential traffic related pollution and lung function were performed in multiple geographical regions,[5 7–8 10–11] but many of these studies used only roadway proximity or traffic count/density metrics rather than validated exposure models. Other studies that have used land-use regression to estimate the relationship between NRAP and childhood lung function were performed in relatively limited geographical regions.[6 9] Results have not been consistent across studies.

These inconsistencies in the strength of association between near-roadway residential traffic exposure and respiratory health across several prior studies[5–11] may result in part from the use of different types of NRAP measures, with differing degrees of uncertainty as proxies for pollution exposure. A strength of this study was the use of quantitative residential NO_x exposure assignments derived from a spatial land-use regression model calibrated to measurements at well characterized locations in study communities.[14] Additionally, the association between lung function and predicted NO_x was consistent with the inverse relationship between residential distance to a freeway and lung function, which was also observed in an earlier CHS cohort,[7] as concentrations of NRAP decrease with increasing distance from a freeway.[25] Comparable, high quality, exposure assessment across studies would facilitate qualitative comparisons or pooled analyses and might lead to more consistent epidemiologic findings.

The adverse associations of lung function with O₃, PM_{2.5}, and PM₁₀ are consistent with other studies.[3] In earlier CHS cohorts we reported associations of lung function with PM_{2.5} and PM₁₀, as well as NO₂, but not with O₃. [7–13] However, O₃ and PM were correlated across communities of the current cohort, and it was therefore not possible to distinguish effects of each.

This study replicates the general design and general age range of a cross-sectional report from a previous CHS cohort [19] but expands the scope of that earlier work by examining both between and within-community pollutant effects. The amount of between-community regional variation in the present study is less than that found in previous CHS studies due to our focus on more-urban communities with larger gradients in NRAP. However, a nearly two-fold difference in the six-year averaged regional pollution concentrations (Figures 3 and 4) exists between the highest and lowest polluted communities, which allowed us to identify between and within-community effects. We have been collecting additional lung function data and will examine longitudinal pollutant effects separately.

We considered the possibility that bias explained our results. Participants and non-participants from the cohort were generally similar across a broad range of demographic, social and housing characteristics (Supplemental Material, Table S-4). The only significant difference was for boys, who were more likely than girls to be non-participants. However, adjusting for sex and for other characteristics had little impact on the NRAP effect estimate (Table 3). Furthermore, the effect of NO_x on lung function in analyses restricted to girls was generally similar to the effect among all participants. Although selection bias and residual confounding by other factors cannot be excluded as an explanation for our results, these analyses provide little reason to believe that this occurred.

There are potentially large public health implications of these findings because NRAP exposure due to proximity of homes and other locations where children spend time is common [26–27] and lung function in childhood tracks into adult life.[28–30] Furthermore, the strong association between exposure and lung function in non-asthmatic children suggests that traffic-related pollution did not affect only a sensitive subgroup but rather has a potential impact on all children. Although direct comparison of the magnitude of effects of regional and near-roadway pollution is difficult, the deficits associated with near-roadway NO_x across a (two-standard deviation) range of within-community variation encompassing most children in our study communities was only modestly less than the effects of regional pollutants across the range of community-average exposure. Compared with a child living in a low NRAP environment in a low regional PM_{2.5} community, the results suggest that a child living in a high NRAP environment in a community with high PM in Southern California would experience a greater than 4% decrease in FEV₁ (Figure 5) For comparison with another common exposure, maternal secondhand smoking of 1 pack/day has been shown to be associated with a 0.4% deficit in childhood level of FEV₁. [31] Prevention of these large pollutant effects poses a challenge to the current air pollution regulatory framework, which historically has set standards using risk calculations that consider effects of regional air quality but not near-roadway traffic-related variation in exposure.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

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What is the key question?

Do residential near-roadway and regional air pollution cause reduced lung function?

What is the bottom line?

This study found that increased near-roadway and regional air pollutants were independently associated with lower FEV₁ and FVC.

Why read on?

A design including multiple communities and predicted near-roadway residential air pollution exposure from well-validated models allowed this study to demonstrate associations of lung function deficits with regional ozone and particulate matter that were independent of associations with indicators of the near-roadway pollutant mixture in multiple communities.

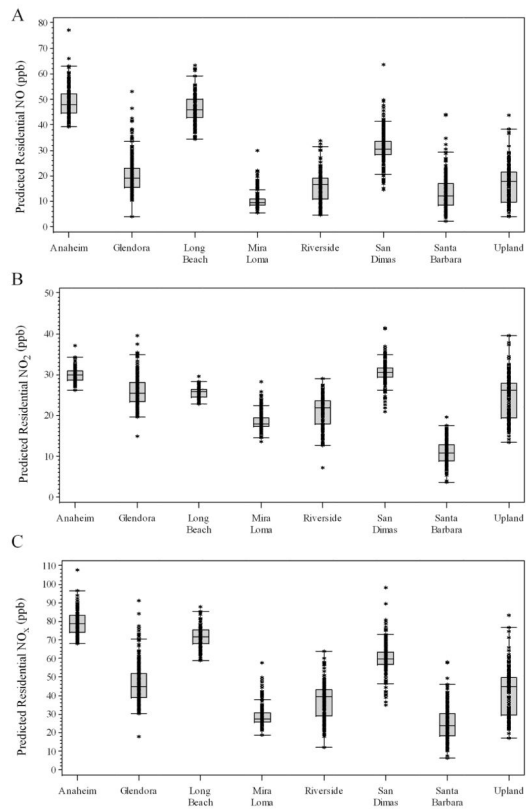


Figure 1. Distribution of predicted local (A) NO, (B) NO₂, and (C) NO_x within each of the eight study communities based on a spatial land-use regression model.

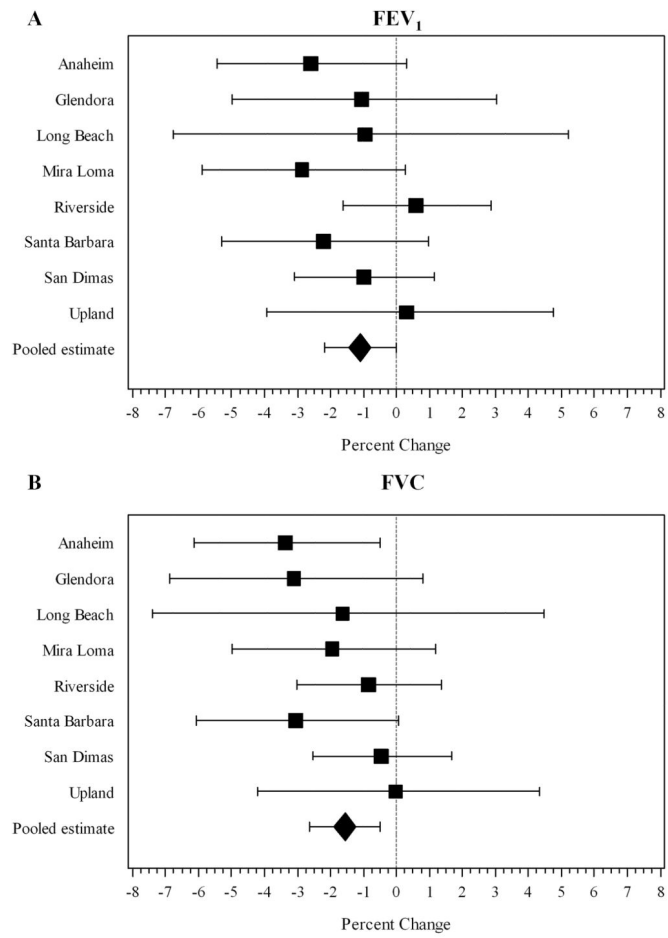


Figure 2. Associations of local NO_x with (A) FEV₁ and (B) FVC within each study community.

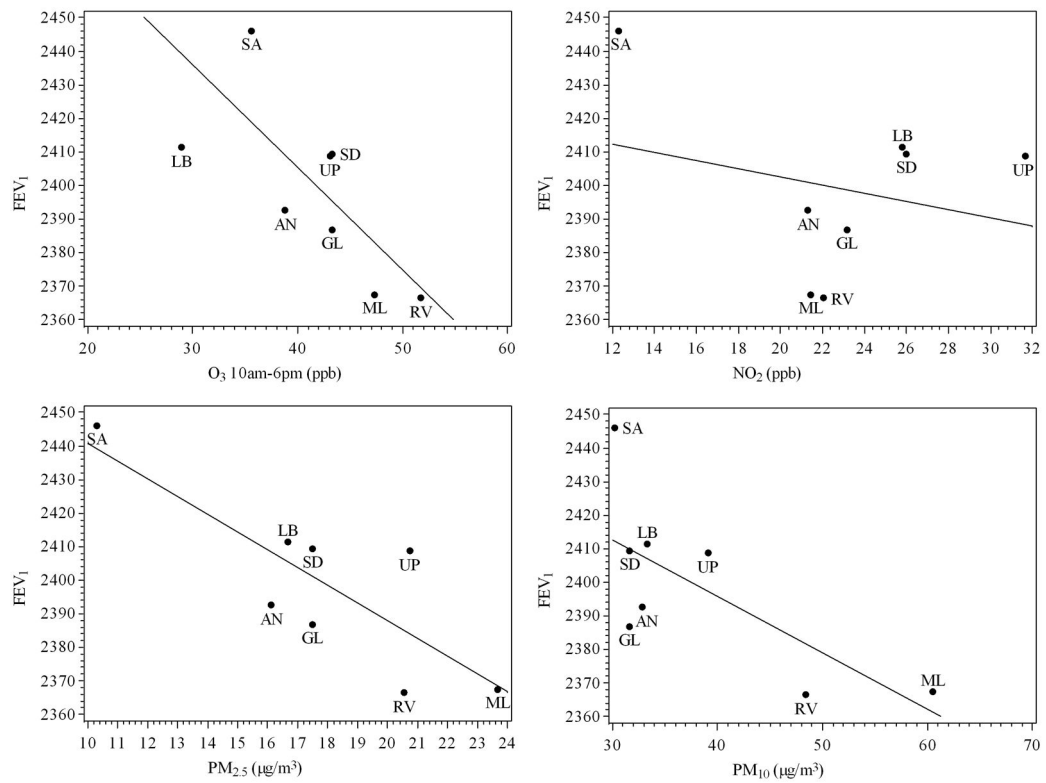


Figure 3. Adjusted average FEV₁ versus 2002–2007 community-average pollutant levels. Average FEV₁ values are referenced to a white, non-hispanic female of average height and BMI and without a respiratory infection on the day pulmonary function was examined.

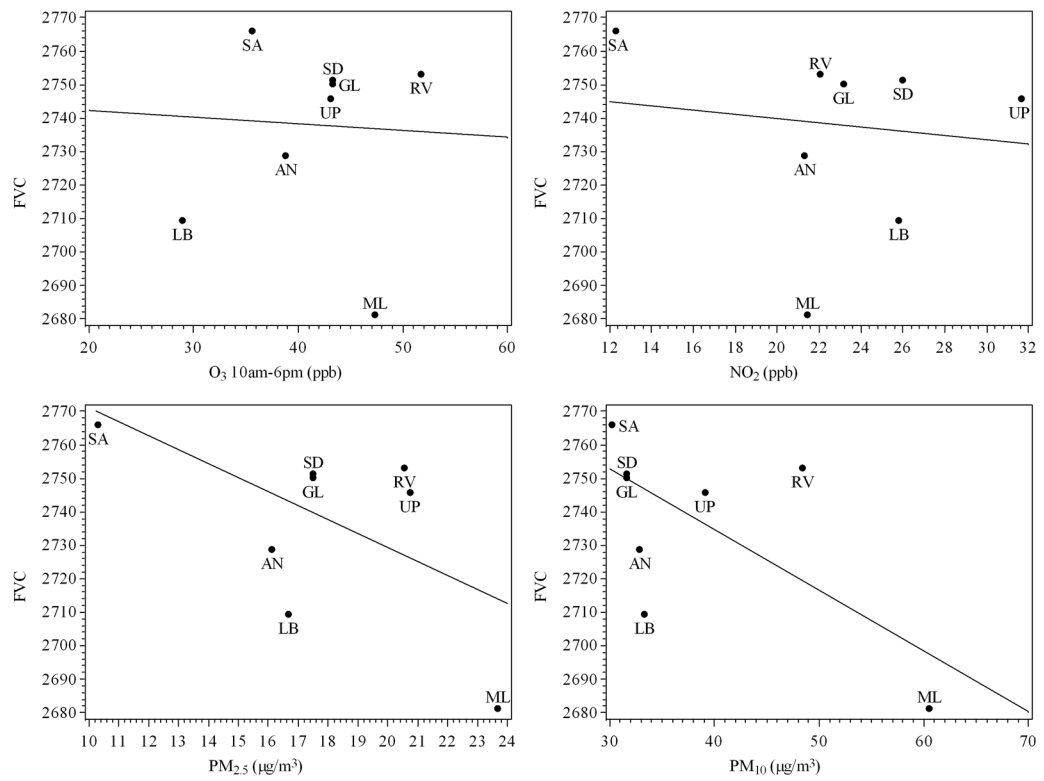


Figure 4. Adjusted average FVC versus 2002–2007 community-average pollutant levels. Average FVC values are referenced to a white, non-hispanic female of average height and BMI and without a respiratory infection on the day pulmonary function was examined.

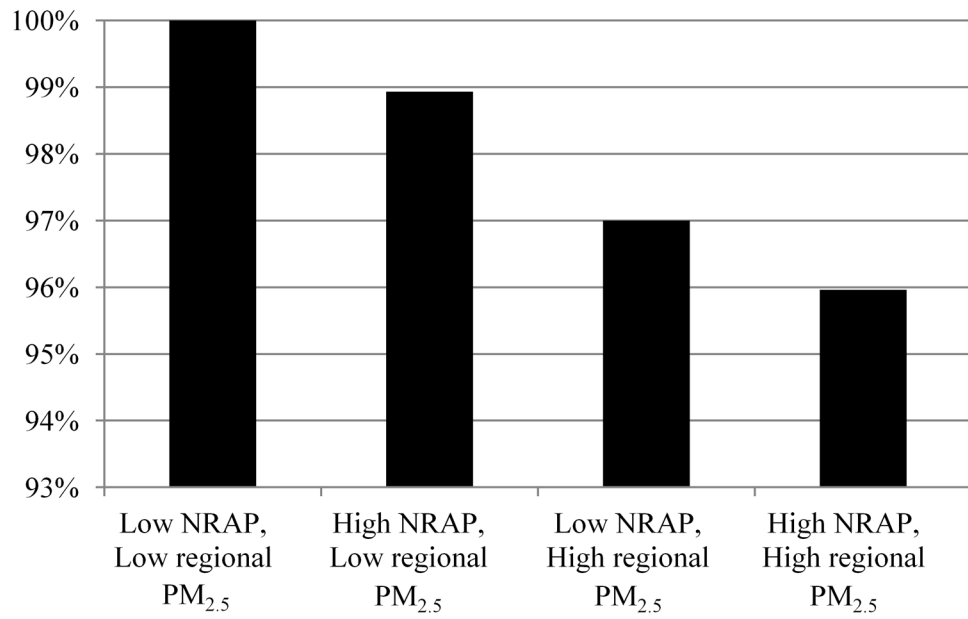


Figure 5. Joint effect of regional PM_{2.5} and NRAP on FEV₁. Percentages in different exposure environments are relative to a low regional PM_{2.5} and low NRAP environment as described in the Statistical Methods section.

Table 1

Characteristics of 1,811 CHS participants with lung function testing

	N (total=1811)	% [†]
Male	871	48.1
Race		
Asian	86	4.8
Black	39	2.2
Don't Know	239	13.2
Mixed	229	12.6
Other	486	26.8
White	732	40.4
Hispanic ethnicity		
Don't Know	92	5.1
Hispanic	1028	56.8
Not Hispanic	691	38.2
<u>SES</u>		
Household income		
<\$30,000	402	27.1
\$30,000 or more	1084	73.0
Parental education		
Did not finish high school	345	20.6
High school diploma or some college	854	51.0
College diploma or greater	477	28.5
Health insurance covers child	1508	89.3
<u>Home characteristics/Potential exposures</u>		
Gas stove	1462	86.5
Dog	599	35.8
Cat	312	18.8
Mold past 12 months	172	10.5
Secondhand smoke exposure	67	3.8
In-utero exposure to maternal smoking	99	5.8
<u>Health conditions</u>		
Acute respiratory illness	164	9.4
Medical diagnosis of asthma	334	19.5

[†] Due to missing values, denominators (n) for each percentage may differ.

Table 2

Effects of measures of near-roadway air pollution on lung function level.

	FEV₁[†]		FVC[†]	
	%Diff	95% CI	%Diff	95% CI
Freeway				
>1,500 m	Ref		Ref	
1,000–1,500 m	1.63	(–0.05, 3.34)	0.99	(–0.65, 2.66)
500–1,000 m	–0.50	(–2.05, 1.07)	–1.01	(–2.52, 0.53)
<500 m	–1.06	(–2.55, 0.45)	–1.96	(–3.41, –0.49)**
Trend (p-value)		0.09		0.004
Major Road				
>300 m	Ref		Ref	
150–300 m	–0.56	(–1.90, 0.79)	–0.69	(–2.00, 0.65)
75–150 m	–0.50	(–2.04, 1.06)	–0.82	(–2.32, 0.72)
<75 m	–1.58	(–3.21, 0.09)	–1.53	(–3.14, 0.11)
Trend (p-value)		0.09		0.06
Predicted Near-roadway Pollution[‡]				
NO ₂	–1.00	(–2.08, 0.09)	–1.40	(–2.46, –0.33)*
NO	–1.19	(–2.27, –0.09)*	–1.68	(–2.74, –0.60)***
NO _x	–1.10	(–2.19, –0.01)*	–1.56	(–2.62, –0.49)***

[†] All models include adjustments for log of height and its squared value, BMI and BMI², sex, age, sex*age interaction, race, Hispanic ethnicity, respiratory illness at time of test, field technician, and study community.

[‡] Near-roadway residential pollutants were scaled to two standard deviations of their respective community-mean centered distributions (6.4 ppb for NO₂, 12.3 ppb for NO, and 17.9 ppb for NO_x).

* p<0.05,

** p<0.01,

*** p<0.005

Table 3Sensitivity analysis for lung function effects of near-roadway residential NO_x.

	<u>FEV₁[†]</u>	<u>FVC[†]</u>
	% diff (95% CI)	% diff (95% CI)
Main model	-1.10 (-2.19, -0.01)	-1.56 (-2.62, -0.49)
Additional covariates		
Main model + family income	-1.04 (-2.13, 0.07)	-1.51 (-2.58, -0.43)
Main model + parental level of education	-0.96 (-2.05, 0.14)	-1.44 (-2.51, -0.36)
Main model + diagnosis of asthma by medical doctor	-1.06 (-2.14, 0.03)	-1.55 (-2.61, -0.47)
Main model + dogs in home	-0.97 (-2.06, 0.13)	-1.40 (-2.47, -0.33)
Main model + cats in home	-1.09 (-2.18, 0.00)	-1.55 (-2.61, -0.47)
Main model + exposure to gas stove	-1.10 (-2.18, -0.01)	-1.56 (-2.62, -0.49)
Main model + in-utero exposure to maternal smoking	-1.09 (-2.17, 0.01)	-1.60 (-2.66, -0.53)
Main model + exposure to tobacco smoke at home	-1.12 (-2.20, -0.02)	-1.57 (-2.63, -0.50)
Main model + exposure to mold	-1.12 (-2.21, -0.03)	-1.57 (-2.64, -0.50)
Main model + insurance coverage	-1.10 (-2.18, -0.01)	-1.55 (-2.61, -0.48)
Subgroup analysis		
Non-asthmatics	-1.19 (-2.41, 0.05)	-1.51 (-2.72, -0.29)
Asthmatics	-0.65 (-3.35, 2.14)	-1.20 (-3.91, 1.58)
Boys	-0.96 (-2.48, 0.58)	-1.13 (-2.60, 0.36)
Girls	-1.10 (-2.65, 0.48)	-1.81 (-3.34, -0.25)

[†]See Table 2 for adjustment variables and scaling factor for pollutant effects.

Table 4

Effect of averaged regional pollutants on lung function level.

	Regional Pollutant	% Diff [†]	95% CI
FEV₁	O ₃ (10am-6pm)	-3.10	(-5.24, -0.91) **
	PM _{2.5}	-2.94	(-4.65, -1.20) ***
	PM ₁₀	-2.19	(-3.98, -0.37) *
	NO ₂	-1.19	(-4.14, 1.85)
FVC	O ₃ (10am-6pm)	-0.31	(-3.11, 2.57)
	PM _{2.5}	-2.25	(-3.94, -0.52) *
	PM ₁₀	-2.05	(-3.54, -0.54) **
	NO ₂	-0.79	(-3.52, 2.02)

[†]See footnote to Table 2 for adjustment variables (community adjustment not included). Each pollutant was scaled to the range of the 24-hour average over the study period from 2002 until 2007 with the exception of O₃, which was scaled to the 8-hour average from 10am to 6pm (22.7 ppb for O₃ 10-6, 13.3 µg/m³ for PM_{2.5}, 30.3 µg/m³ for PM₁₀, 19.4 µg/m³ for NO₂).

* p<0.05,

** p<0.01,

*** p<0.005

Table 5

Joint analysis of regional air pollution and near-roadway NO_x on lung function.

	Regional Pollutant	Effect of Regional Pollutant [†]		Effect of Near-roadway NO _x [‡]	
		% Diff	95% CI	% Diff	95% CI
FEV ₁	O ₃ (10am-6pm)	-3.24	(-5.32, -1.11)***	-1.04	(-2.11, 0.05)
	PM _{2.5}	-3.00	(-4.76, -1.21)***	-1.07	(-2.14, 0.01)
	PM ₁₀	-2.24	(-4.04, -0.41)*	-1.14	(-2.22, -0.06)*
FVC	NO ₂	-1.22	(-4.23, 1.88)	-1.07	(-2.15, 0.02)
	O ₃ (10am-6pm)	-0.34	(-3.21, 2.63)	-1.47	(-2.53, -0.41)**
	PM _{2.5}	-2.35	(-4.09, -0.57)**	-1.40	(-2.46, -0.34)**
	PM ₁₀	-2.17	(-3.68, -0.63)**	-1.49	(-2.54, -0.43)**
	NO ₂	-0.78	(-3.62, 2.15)	-1.46	(-2.52, -0.39)**

[†] See Table 2 for adjustment variables (community adjustment not included) and scaling factor for pollutant effects of near-roadway NO_x. See footnote to Table 4 for scaling factor for regional pollutants.

* p<0.05,

** p<0.01,

*** p<0.005