Traffic-related Air Pollution near Busy Roads
The East Bay Children's Respiratory Health Study

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Recent studies, primarily in Europe, have reported associations between respiratory symptoms and residential proximity to traffic; however, few have measured traffic pollutants or provided information about local air quality. We conducted a school-based, cross-sectional study in the San Francisco Bay Area in 2001. Information on current bronchitis symptoms and asthma, home environment, and demographics was obtained by parental questionnaire (n = 1,109). Concentrations of traffic pollutants (particulate matter, black carbon, total nitrogen oxides [NO_X], and nitrogen dioxide [NO_2]) were measured at 10 school sites during several seasons. Although pollutant concentrations were relatively low, we observed differences in concentrations between schools nearby versus those more distant (or upwind) from major roads. Using a two-stage multiple-logistic regression model, we found associations between respiratory symptoms and traffic-related pollutants. Among those living at their current residence for at least 1 year, the adjusted odds ratio for asthma in relationship to an interquartile difference in NO_X was 1.07 (95% confidence interval, 1.00–1.14). Thus, we found spatial variability in traffic pollutants and associated differences in respiratory symptoms in a region with good air quality. Our findings support the hypothesis that traffic-related pollution is associated with respiratory symptoms in children.

Keywords: air pollution; asthma; bronchitis; epidemiology; vehicle emissions

Numerous epidemiologic studies have documented adverse effects of air pollution on health (1). The majority of these population-based studies have used pollutant concentrations measured at central monitoring sites to estimate exposures and have not, in general, considered local spatial variability in pollutant levels. However, motor vehicle emissions, the principal source of ambient air pollution in most urban areas, are likely to vary substantially within a given community, and researchers have begun to document differences in traffic-related pollutants on a neighborhood scale (2, 3).

Recently, a number of epidemiologic studies have reported associations between residential proximity to busy roads and a variety of adverse respiratory health outcomes in children, including respiratory symptoms, asthma exacerbations, and decrements in lung function (4–12). In some reports, truck traffic has been more strongly associated with these adverse outcomes than total vehicular traffic (6, 7, 10, 11).

Most studies have used metrics of proximity to traffic as surrogates of exposure to traffic pollution (e.g., residential proximity to major roads, traffic volume at the nearest road, or modeled levels of traffic pollution). Few have measured pollutant concentrations as part of the exposure assessment or provided information on local air quality (7, 10–12). The majority of studies have been conducted in Europe and Japan, where fleet composition (diesel versus gasoline), emissions factors, fuel specifications, land use, and population distributions near busy roads differ from those in the United States. Regional and microenvironmental concentrations of particulate matter (PM) may be higher in European cities compared with many parts of the United States (13). Therefore, it is important to evaluate the extent to which proximity to traffic may be associated with health impacts in the United States. Previous studies in the United States were conducted in areas of Southern California and the Northeast with significant local air-quality problems; both used metrics of proximity to traffic, not measured pollutant concentrations (8, 14).

The objective of this study was to explore associations between respiratory symptoms and exposures to traffic-related air pollutants among children living and attending schools near busy roads in an urban area with high traffic density but good regional air quality. Some of the results of this study have been previously reported in the form of abstracts (15).

METHODS

Study Design and Health Assessment

We conducted a school-based, cross-sectional study in the San Francisco metropolitan area (Alameda County, CA) in 2001. The study area was comprised of 10 neighborhoods that span a busy traffic corridor. School sites were selected to represent a range of locations upwind and downwind of major roads (Figure 1).

In spring 2001, we enrolled children (grades 3–5) in participating classes (n = 64) using methods similar to those used in other school-based studies (16–18). We obtained information on health outcomes (bronchitis symptoms in the past 12 months and physician-confirmed asthma in the past 12 months), demographics, home environmental factors, and activity factors using parental questionnaires (English and Spanish) (for additional information on the study design and health assessment, *see* the online supplement). The study protocol was approved by the Committee for the Protection of Human Subjects, California Health and Human Services Agency.

Air Pollution from Traffic

We measured concentrations of traffic pollutants (particulate matter [PM_{10}, $PM_{2.5}$], black carbon [BC], total nitrogen oxides [NO_X], and nitrogen dioxide [NO_2]) at the school sites. PM_{10} and $PM_{2.5}$ mass concentrations were measured using filter-based samples, whereas BC concentrations were determined on the PM_{10} filter samples using an established light attenuation method that we validated for fiberfilm filters (19, 20). NO_X and NO_2 concentrations were determined with passive diffusion samplers (Ogawa, Inc., Pompano Beach, FL). Nitric oxide (NO) concentrations were calculated as the difference between NO_X and NO_2.

Pollutant monitoring was conducted simultaneously at all school sites for 11 1-week intervals in the spring (March–June) and for 8 weeks

(*Received in original form March 4, 2004; accepted in final form May 31, 2004*)

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This article has an online supplement, which is accessible from this issue's table of contents online at www.atsjournals.org

Am J Respir Crit Care Med Vol 170. pp 520–526, 2004
Originally Published in Press as DOI: 10.1164/rccm.200403-281OC on June 7, 2004
Internet address: www.atsjournals.org

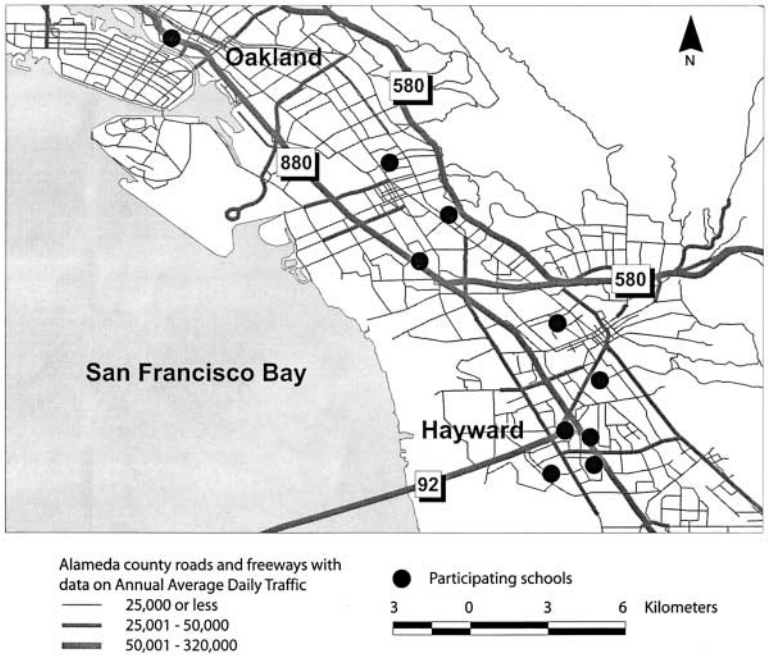

Figure 1. East Bay Children's Respiratory Health Study area. The study region is to the east and across the bay from the city of San Francisco.

in the fall (September–November) of 2001. NO_X and NO_2 were sampled during all weeks at each school. PM_{10} and $PM_{2.5}$ and the BC concentrations were not measured every week. Study-averaged air pollution concentrations were calculated at each school by first normalizing the data to account for occasional missing values. Additional details are described in the online supplement and elsewhere (21). In preliminary analyses, we also used school location in relationship to prevailing winds and proximity to busy roads as an additional traffic metric.

Data Analysis

We examined associations between pollutants and health outcomes using a two-stage hierarchical modeling strategy. This method has been used in other epidemiologic studies of air pollution when pollutants were measured at the group level (18, 22). In our study, the exposure groups were represented by the neighborhood schools. In the first stage, we initially identified potential confounders (demographic, host, or home environmental variables) associated with health outcomes in this dataset. We then performed exploratory stepwise logistic regressions to develop a model in which individual-level characteristics best predicted the odds of each health outcome. Explanatory variables that remained significant at $p < 0.15$ were retained in the model. We then fit a logistic regression model that included an indicator variable for each school in addition to the individual-level covariates.

In the second stage, the adjusted school-level logits or prevalence rates determined in the first stage were regressed on the school-specific ambient pollutant concentrations. In this manner, we obtained the log odds ratios (ORs) relating asthma or bronchitis symptoms to air pollution, after adjusting for individual-level risk factors.

We calculated adjusted ORs for a change in measured pollutant concentration equal to the interquartile ranges of the pollutant distributions. Analyses were conducted using SAS version 8.2 for Windows (Cary, NC) and STATA, version 8 (College Park, TX).

RESULTS

We distributed 1,574 questionnaires in 64 participating classrooms in the 10 schools. Three children were excluded because their parents spoke neither English nor Spanish. Among the remaining students, there was a response rate of 70.7% (1,111/1,571). Participation rates across schools ranged from 61–83%. Approximately 30% completed the questionnaire in Spanish. Two children with reported cystic fibrosis were excluded from the analysis. The final analysis sample consisted of 1,109 questionnaires.

Table 1 summarizes the participants' demographic characteristics, prevalence of selected personal and home environmental characteristics, and respiratory health outcomes. Our study population was racially diverse. Approximately 30% of households had incomes below the federal poverty line. Fourteen percent of the parental respondents reported having been told by a doctor that their child had asthma in the preceding 12 months. This represents a measure of period prevalence of asthma and would include some incident cases. Twelve percent of children had bronchitis symptoms in the past year. Of those reporting bronchitis symptoms in the past 12 months, 43% also reported having asthma. Using a slightly different definition of asthma (physician-diagnosed ever, and asthma symptoms, including wheezing, in the past 12 months), 11% of our study population had current asthma.

The estimated pollutant concentrations at the schools are summarized in Table 2. Concentrations of several pollutants (i.e., BC, NO_X, NO, and, to a lesser extent, NO_2) were higher at schools located within 300 m downwind of a freeway compared with those at schools upwind or further from major traffic sources. There was less variation in $PM_{2.5}$ and PM_{10}. Concentrations of BC, NO_X, and NO were highly correlated (r^2 = approximately 0.9 for each interpollutant correlation). The study average $PM_{2.5}$ (12 $\mu g/m^3$) was similar to the annual average concentration of $PM_{2.5}$ at the central monitoring station, located approximately 15 km south of the study area. NO_X and NO_2 measurements at the school sites away from traffic were similar to levels measured at the regional site (21).

Table 3 summarizes the results of the two-stage hierarchical logistic regression models of the odds of asthma and bronchitis symptoms in the previous year in relationship to six different pollutants, each examined in separate regressions. Results are shown for all subjects, for long-term residents only (1 year or longer at the current address), and for the latter group stratified by sex. In addition to the traffic metric, explanatory variables

TABLE 1. DEMOGRAPHIC, FAMILY, AND HOME CHARACTERISTICS OF THE EAST BAY CHILDREN'S RESPIRATORY HEALTH STUDY RESPONDENTS

Characteristics	All Subjects (*n = 1,109*) (%)	Subjects Attending Schools: Near and Downwind of Major Roads (*Four Schools, n = 402*) (%)	Subjects Attending Schools: Far or Upwind of Major Roads (*Six Schools, n = 707*) (%)
Sex			
Female	52.6	51.8	53.1
Race/ethnicity			
White	12.6	11.0	13.5
Black, African American	11.1	7.0	13.4
Hispanic	43.5	47.6	41.2
Asian	14.0	15.5	13.1
Other/multiracial	18.9	18.8	18.9
SES indicators			
Household at/below federal poverty level	31.3	31.8	31.0
Parent's education: high school or less*	48.7	51.4	47.1
Family history			
Biological mother with asthma	12.2	9.5	13.7
Maternal smoking during pregnancy	10.3	7.8	11.7
Home indoor environment			
Smoker in the household, since child's birth	17.9	13.1	20.6
Smoker in the household, current	7.2	3.2	9.5
Furry pet	37.3	36.0	38.1
Pests, past 12 mo	63.1	65.4	61.8
Gas stove	63.1	63.6	62.9
Indicator of mold/mildew presence, past 12 mo	44.6	43.5	45.3
Health outcomes			
Chest illness before age 2	23.3	18.8	25.9
Asthma, past 12 mo	14.0	13.9	14.1
Bronchitis, past 12 mo	12.1	13.2	11.5

* Parent responding to the questionnaire.

retained in all of the final models for asthma and bronchitis included chest illness before age 2 years, household mold/moisture, and pests observed in the home in the preceding 12 months. The final models for asthma also included maternal history of asthma. The addition of other potential confounders such as race/ethnicity, socioeconomic variables, maternal smoking during pregnancy, current smoker in the home, air conditioning, and gas stove use yielded similar pollutant effect estimates.

For the full sample, associations were observed between both asthma in the past 12 months and bronchitis symptoms in the past 12 months and the pollutants, especially NO_X, NO, and BC. The effect estimates for $PM_{2.5}$ and PM_{10} were smaller, which may have been due in part to the smaller concentration ranges among the 10 sites for these pollutants. No multipollutant models were evaluated because of the high interpollutant correlations. Restricting the analysis based on duration of residence (i.e., at

TABLE 2. NEARBY TRAFFIC SOURCES AND AVERAGE POLLUTANT CONCENTRATIONS AT TEN SCHOOLS

School	Major Traffic Source†	AADT (*#/d*)	Distance‡ (*m*)	< 300 m Downwind	PM_{10} ($\mu g/m^3$)	$PM_{2.5}$ ($\mu g/m$)	BC ($\mu g/m^3$)	NO_X (*pbb*)	NO_2 (*pbb*)	NO§ (*pbb*)
1	No				30	12	0.7	42	22	19
2	Yes	90,000	230	Yes	29	13	0.9	55	24	31
3	Yes	210,000	360	No	32	12	0.8	49	21	29
4	No				30	12	0.8	41	19	22
5	Yes	210,000	130	Yes	30	12	0.9	62	26	36
6	No				30	12	0.7	39	21	17
7	No				29	11	0.7	33	20	11
8	Yes	130,000	350	No	29	12	0.7	45	23	21
9	Yes	210,000	200	Yes	30	12	0.9	57	26	31
10*	Yes	190,000	60	Yes	32	15	1.1	69	31	38
Study average					30	12	0.8	49	23	25

Definition of abbreviations: AADT = annual average daily traffic; BC = black carbon; NO = nitric oxide; NO_X = total nitrogen oxides; NO_2 = nitrogen dioxide; $PM_{2.5}$ = particulate matter of aerodynamic diameter 2.5 μm or less; PM_{10} = particulate matter of aerodynamic diameter 10 μm or less.

Estimated average pollutant concentration at each school based on normalized concentrations (*see text*). Monitoring was conducted for 11 weeks in the spring (March–June) and 8 weeks in the fall (September–November of 2001). The number of weeks underlying our estimates of chronic exposure varied for each measured pollutant: NO_X (18), NO_2 (19), BC (11), $PM_{2.5}$ (10), and PM_{10} (9).

* There is also a shopping center and a parking lot abutting the school grounds to the south and a freeway off ramp less than 50 m to the northwest.

† Includes roads with AADT above 50,000 vehicles per day located within 1,000 m of school. AADT estimate provided by the California Department of Transportation (Cal Trans).

‡ Distances were estimated using a geographic information system. Latitude and longitude of the monitors were determined using a global-positioning system device (Garmin GPS II). In some cases, distances were estimated using aerial photographs or measured using a distance wheel.

§ $NO = NO_X - NO_2$

TABLE 3. ODDS RATIOS (95% CONFIDENCE INTERVAL) OF RESPIRATORY ILLNESS BY SCHOOL-BASED AMBIENT AIR POLLUTANT CONCENTRATIONS USING TWO-STAGE MODEL

		All Subjects		LTR Subjects		LTR Females		LTR Males	
		(*n = 1,109*)		(*n = 871*)		(*n = 462*)		(*n = 403*)	
	Exposure	OR	CI	OR	CI	OR	CI	OR	CI
Bronchitis*		n = 93/797		n = 79/635		n = 38/341		n = 41/291	
	NO_X	1.05	(1.01, 1.08)	1.06	(1.03, 1.09)	1.07	(1.03, 1.11)	1.03	(0.98, 1.09)
	NO_2	1.02	(0.99, 1.06)	1.03	(1.00, 1.06)	1.04	(1.01, 1.08)	1.02	(0.98, 1.06)
	NO	1.05	(1.02, 1.09)	1.06	(1.03, 1.09)	1.07	(1.03, 1.11)	1.04	(0.98, 1.10)
	PM_{10}	1.03	(0.99, 1.07)	1.02	(0.98, 1.07)	1.04	(1.01, 1.09)	1.01	(0.95, 1.06)
	$PM_{2.5}$	1.02	(1.00, 1.05)	1.03	(1.01, 1.05)	1.04	(1.02, 1.05)	1.02	(0.99, 1.05)
	BC	1.04	(1.00, 1.08)	1.05	(1.01, 1.08)	1.06	(1.02, 1.10)	1.03	(0.98, 1.08)
Asthma†		n = 101/705		n = 78/562		n = 42/297		n = 36/263	
	NO_X	1.04	(0.97, 1.11)	1.07	(1.00, 1.14)	1.17	(1.06, 1.29)	1.02	(0.93, 1.11)
	NO_2	1.02	(0.97, 1.07)	1.04	(0.98, 1.10)	1.09	(1.03, 1.15)	1.00	(0.94, 1.07)
	NO	1.05	(0.98, 1.12)	1.08	(1.00, 1.15)	1.19	(1.03, 1.36)	1.02	(0.94, 1.12)
	PM_{10}	1.02	(0.96, 1.09)	1.04	(0.97, 1.12)	1.09	(0.92, 1.29)	1.02	(0.94, 1.10)
	$PM_{2.5}$	1.00	(0.97, 1.04)	1.01	(0.97, 1.06)	1.06	(0.99, 1.15)	0.99	(0.95, 1.04)
	BC	1.02	(0.96, 1.09)	1.05	(0.99, 1.13)	1.12	(0.95, 1.33)	1.00	(0.93, 1.09)
Asthma (no outlier, school 5)†		n = 96/641		n = 73/507		n = 38/271		n = 35/233	
	NO_X	1.08	(1.00, 1.17)	1.10	(1.00, 1.20)	1.14	(1.02, 1.28)	1.07	(0.96, 1.19)
	NO_2	1.06	(0.99, 1.13)	1.07	(0.98, 1.17)	1.09	(0.97, 1.22)	1.05	(0.96, 1.16)
	NO	1.08	(1.00, 1.17)	1.09	(1.00, 1.19)	1.14	(1.03, 1.26)	1.07	(0.96, 1.18)
	PM_{10}	1.06	(0.97, 1.16)	1.08	(0.98, 1.19)	1.09	(0.96, 1.24)	1.08	(0.97, 1.19)
	$PM_{2.5}$	1.04	(0.96, 1.12)	1.03	(0.94, 1.13)	1.03	(0.91, 1.17)	1.03	(0.94, 1.14)
	BC	1.07	(0.98, 1.17)	1.09	(0.99, 1.19)	1.14	(1.02, 1.27)	1.06	(0.95, 1.18)

Definition of abbreviations: BC = black carbon; CI = confidence interval; LTR = long-term resident; OR = odds ratio; NO = nitric oxide; NO_X = total nitrogen oxides; NO_2 = nitrogen dioxide; $PM_{2.5}$ = particulate matter of aerodynamic diameter 2.5 μm or less; PM_{10} = particulate matter of aerodynamic diameter 10 μm or less.

Odds ratios are calculated per IQR of average pollutant concentrations as follows: NO_X =14.9 ppb; NO_2 = 3.6 ppb; NO = 11.6 ppb; PM_{10} = 1.4 μg/m^3; $PM_{2.5}$ = 0.7 μg/m^3; BC = 0.15 μg/m^3.

For hierarchical analyses of asthma in long-term residents (current address for 1 year or more), only 9 schools were included in the analysis; one school had no cases (due to low numbers and missing values).

* First stage model adjusted for: child's respiratory illness before age 2; pests, indicator of mold presence.

† First stage model adjusted for: child's respiratory illness before age 2; pests, indicator of mold presence; maternal history of asthma.

least 1 year at current residence) tended to increase the effect estimates slightly in relationship to asthma, especially when the sample was restricted to girls. Stratification by duration of residence or sex did not change the results for bronchitis. Results were similar when nonnormalized pollution values were used (data not shown).

We conducted additional sensitivity analyses, including (*1*) dropping the one school that was an outlier with respect to the proportion of Hispanic students (89% vs. 21–53% at other schools), (*2*) using a different definition for current asthma, and (*3*) stratifying bronchitis by a reported history of asthma. When the "outlier" school was dropped, the magnitude of the ORs for bronchitis did not change much, but the confidence intervals were wider. In the asthma analyses, dropping the outlier school resulted in similar or slightly greater effect estimates. Applying different questionnaire-based asthma definitions showed little change but slightly larger confidence intervals. After stratifying students by whether they also "ever" had asthma, the results suggested that those with a history of asthma were driving the results for bronchitis, but the sample size became too small to make clear inferences. Figures 2 and 3 depict the associations between BC and bronchitis and asthma.

DISCUSSION

To our knowledge, this is the first epidemiologic study in the United States to evaluate relationships between measured traffic-related pollutants and respiratory symptoms. For children residing at their current address for at least 1 year, we found modest but significant increases in the odds of bronchitis symptoms and physician-diagnosed asthma in neighborhoods with higher concentrations of traffic pollutants. These results are consistent with previous reports of positive associations between proximity to traffic and various respiratory outcomes (4–12). Furthermore, our findings were observed in a region with relatively clean air (low concentrations of ozone and PM) (*see* the online supplement for details). Although previous epidemiologic studies in the United States exploring chronic respiratory effects of air pollution in children have shown inconsistent results, this might be due in part to exposure misclassification, as these studies used air quality measurements conducted at single fixed-site monitors in each city (17, 18, 22, 23).

Our findings were robust to multiple sensitivity analyses using different questionnaire-based definitions of current asthma and wheezing in the past 12 months. The slight increase in effect estimates for associations between asthma after restricting the analysis to those with longer duration at current residence may be due to a reduction in exposure measurement error. Our study population was very mobile (23% had moved in the preceding 12 months, and only 32% had lived at the same address since before the age of 2 years).

We considered whether there might be bias due to nonresponse or self-reporting. We saw no significant difference in proportions of questionnaires returned in Spanish versus English by school, but there was a modest inverse correlation between pollution concentrations measured at each school and response rate. However, the response rate for individual classrooms within each school varied as well and appeared to depend on the willingness of teachers to encourage participation. Dropping the school closest to a freeway (which also had the highest measured pollutant concentrations, a high percentage of Hispanic students, and the lowest response rate) did not change the effect estimates for

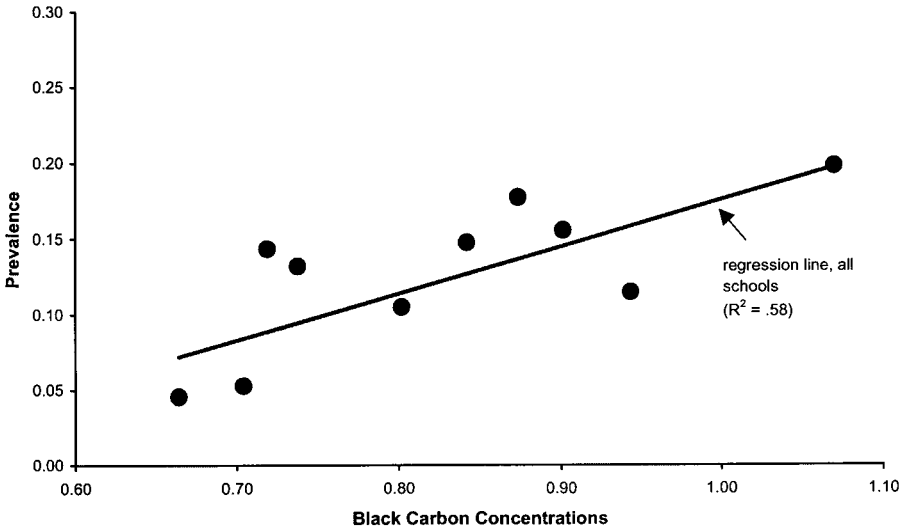

Figure 2. Adjusted school-specific bronchitis prevalence rates versus black carbon, long-term residents.

bronchitis and increased the estimates for asthma. This would suggest that knowledge of potential high traffic exposure probably did not affect parental reporting of the children's respiratory histories. This study was not undertaken in response to public concerns about traffic nor, at the time the study was conducted, was there much local interest in potential health hazards of proximity to traffic. Therefore, reporting and nonresponse biases were unlikely to have unduly influenced our results.

We found increased association with asthma (but not bronchitis) with exposure to traffic air pollutants for girls who had lived at their current addresses at least 1 year compared with boys (Table 3). Several investigators have also reported greater traffic-associated effect estimates for girls versus boys (7, 8, 10, 24, 25). Previous air pollution studies examining the sex-specific effects of air pollution on lung function and lung function growth have been mixed (26, 27). The reasons for the observations in our study are unclear and deserve attention in future studies.

Exposures

We found spatial variability in exposure due specifically to roads with heavy traffic within a relatively small geographic area for BC, NO_X, NO, and to a lesser extent NO_2. There was less variation in $PM_{2.5}$ across schools; this is consistent with previous observations that $PM_{2.5}$ is more likely to reflect regional air quality (2). The higher effect estimates with BC, NO_X, and NO compared with NO_2 and $PM_{2.5}$ suggest that primary or fresh traffic emissions may play an etiologic role in these relationships. Although NO_X, NO, and BC may serve as indicators of exposure to traffic-related pollutant mixtures, they may also act as etiologic agents themselves (28).

We found that downwind direction was an important determinant of increased exposure to traffic pollutants and that a simple traffic indicator (school location downwind and $<$ 300 m from a major road) gave estimates of ORs similar to or greater than pollutant measurements in preliminary analyses using a one-stage model (data not shown). Within a geographic area with flat terrain and low-rise buildings, the direction of wind in relationship to the traffic source is the most important weather parameter. Other parameters important in air dispersion of traffic pollutants (e.g., atmospheric stability, wind speed, and surface topography) would be relatively similar at the different school sites.

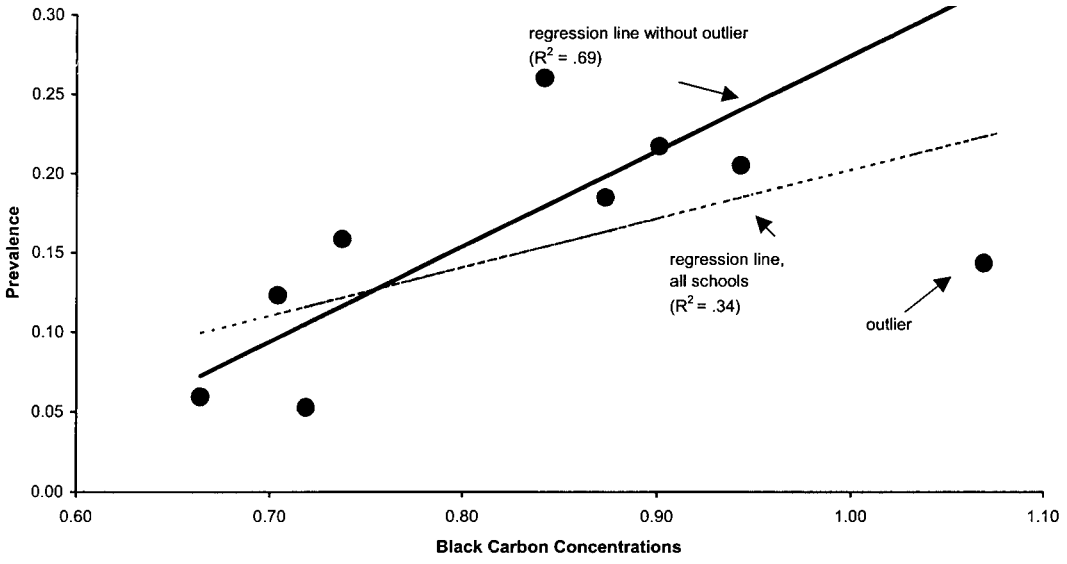

Figure 3. Adjusted school-specific asthma prevalence rates versus black carbon, long-term residents.

A simple single-stage logistic model using pollutant measurements also yielded positive associations between pollutants and symptoms with a much larger effect estimate and smaller confidence intervals.

We assumed that traffic-related pollutants measured at the neighborhood schools would be a good proxy for the children's overall exposure to such pollutants. Children attending the schools in this study generally lived within walking distance and did not use school buses. Therefore, pollutant concentrations in the children's neighborhoods probably tracked those at their schools. The most plausible exposure error in an urban setting would be that subjects who attend schools with very high traffic exposures from a nearby freeway would tend to have similar or lower home exposures, whereas children with low school exposures would tend to live in homes with similar or only slightly higher traffic exposures. This pattern of measurement error would tend to underestimate the association between exposure and outcome (29).

Alternatively, repeated daily exposures for 6–8 hours during the school year may themselves represent biologically important influences on some children's respiratory health, analogous to occupational exposures for susceptible adults. In a recent study of proximity to traffic and respiratory health, Janssen and colleagues found that effect estimates based on the school-to-highway distance were comparable or greater than those based on residence-to-highway distance (11).

The average measurements at each school were used to estimate long-term average traffic air pollutant concentrations. We measured pollutants at each of the 10 sites concurrently (to avoid concerns of week-to-week variability) in two different periods that reflect the major seasonal wind patterns for the area. We found that the rank order (relative values) of the schools did not vary from week to week or season to season, supporting the validity of this approach. Additionally, the NO_x and NO_2 concentrations at schools upwind or further from high traffic roads were similar to NO_X and NO_2 concentrations measured at the closest fixed-site monitor (21). Although there may have been some changes in the absolute traffic volume on major roads in recent years, the principal traffic patterns in the area have not changed. Thus, the relative values (rank order) of the site-specific pollutant concentrations measured in our study are likely to be representative of those in recent years.

The cross-sectional nature of our study design is a further limitation on causal inference, but we observed the same or modest increase in effect estimates for current asthma and bronchitis when we restricted our analysis to those who had lived at their present address for at least a year. Most studies on proximity to traffic and respiratory symptoms have been cross-sectional, and further longitudinal studies are needed to elucidate the role of traffic-related air pollution in the development and exacerbation of asthma and other respiratory symptoms.

Another limitation was that the exposures were assigned at the group level (n = 10); however, the multilevel analysis allows adjustment for individual confounders in the first stage of analysis. Moreover, in this respect, this study is comparable with other epidemiologic investigations (e.g., the Harvard Six Cities Study and the Children's Health Study in Southern California) (n = 12 communities). Another recent cross-sectional study of traffic-related air pollution and respiratory symptoms included 13 schools (18, 22, 23).

We also lacked information on indoor measurements of traffic-related pollutants. However, recent studies have found high correlations between personal exposures to NO_2 and traffic parameters (30). Others have found that indoor concentrations and exposure to soot (PM from diesel exhaust) is highly correlated with outdoor levels (2).

Other Covariates

Maternal asthma, household mold/moisture, pests, and chest illness before the age of 2 years were important explanatory variables in the final model for current asthma, consistent with previous studies (31–33). We explored whether current levels of traffic pollution could modify the risk of current asthma symptoms depending on past history of chest illness; however, there was not sufficient power to explore interactions based on early medical history. Race/ethnicity and indicators of socioeconomic status were not important predictors of health outcomes in our study. This may be due, in part, to our study design (i.e., the schools were selected to have relatively similar measures of socioeconomic status).

We did not find associations between exposure to environmental tobacco smoke and current asthma; the results of previous cross-sectional studies in school-aged children have been mixed (34). The prevalence of current household smokers in our study was small, however, limiting study power. It is possible that there is some underreporting of household smoking (7% in our study vs. 19% statewide). (35) Alternatively, a substantial portion of our study population was less acculturated Hispanics (30% of parents responded in Spanish), and only 3.6% of Hispanic households reported a history of maternal smoking. Other investigators have also observed very low smoking rates (less than 5%) among less acculturated Hispanics (B. Eskenazi, personal communication) (36). If underreporting does exist, it is possible that residual confounding might have affected our estimates of pollutant/respiratory health outcome relationships. However, the addition to the regression model of variables correlated with exposure to environmental tobacco smoke (e.g., socioeconomic status and race–ethnicity) did not change the pollutant effect estimates, suggesting that significant confounding by environmental tobacco smoke was not likely.

In summary, we found associations between traffic-related pollutants and asthma and bronchitis symptoms in the past 12 months in a highly urbanized region of the United States with good regional air quality, where local air pollution is dominated by vehicular sources. Although the cross-sectional study design, exposure assignment at the group level, small geographic area, and possible unmeasured covariates may limit the generalizability of the study, our findings are consistent with previous investigations in Europe and the United States (11, 14, 37). In addition, our results underscore the limitations of using central air monitoring stations for assigning population exposures. Concentrations of air toxics such as diesel exhaust particles or surrogates such as BC or soot should be more widely monitored. Measurement of personal exposures to traffic pollutants is not feasible in large population-based studies; the use of geographic modeling approaches to estimate exposures for individuals may be a good alternative (38). Future studies that can better characterize exposures to traffic pollutants, and their sources (i.e., diesel versus gasoline engines) will be important to understand better the public health impacts of motor vehicle emissions.

Conflict of Interest Statement: J.J.K. does not have a financial relationship with a commercial entity that has an interest in the subject of this manuscript; S.S. does not have a financial relationship with a commercial entity that has an interest in the subject of this manuscript; M.L. does not have a financial relationship with a commercial entity that has an interest in the subject of this manuscript; B.C.S. does not have a financial relationship with a commercial entity that has an interest in the subject of this manuscript; A.T.H. does not have a financial relationship with a commercial entity that has an interest in the subject of this manuscript; B.O. does not have a financial relationship with a commercial entity that has an interest in the subject of this manuscript.

Acknowledgment: The authors thank Jackie Hayes and Donna Eisenhower, Survey Research Center, University of California, Berkeley, for coordinating the survey; Toshifumi Hotchi, Lawrence Berkeley National Laboratories, for work on the air monitoring study; and Bob McLaughlin, California Department of Health Services,

for work using a geographic information system. Shelley Green, Rob McConnell, Ed Avol, Patricia van Vliet, Bob Gunier, and Paul English provided helpful discussions. The authors also thank the school administrators and teachers for their support and acknowledge study participants and their families.

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