Title: Traffic-related air pollution near busy roads: the East Bay Children's Respiratory Health Study

Authors and Affiliations

Janice J. Kim [1], Svetlana Smorodinsky[1,2], Michael Lipsett[1,2], Brett C. Singer[3], Alfred T. Hodgson[3], Bart Ostro[1*]

[1]Office of Environmental Health Hazard Assessment, California Environmental Protection Agency, Oakland, CA

[2] current affiliation: California Department of Health Services, Oakland, CA

[3]Atmospheric Sciences Department and [3]Indoor Environment Department, Environmental Energy Technologies Division, Lawrence Berkeley National Laboratory, Berkeley, CA

*Corresponding author:
Bart Ostro, Ph.D.
Office of Environmental Health Hazard Assessment
1515 Clay Street, 16th Floor, Oakland, CA 94612
Telephone: (510) 622-3157
Fax: (510) 622-3210
Email: Bostro@oehha.ca.gov

The contents and opinions expressed in this manuscript are those of the authors and do not represent the official position of the Office of Environmental Health Hazard Assessment, the California Environmental Protection Agency, or the California Department of Health Services.

Running title: Traffic and respiratory health

Word Count (body):3390

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

Abstract

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 were obtained

by parental questionnaire (n=1,109). Concentrations of traffic pollutants (particulate

matter (PM_{10}, $PM_{2.5}$), black carbon (BC), and nitrogen oxides (NO_X and NO_2)) were

measured at ten 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 one

year, the adjusted odds ratios (OR) for asthma in relation to an interquartile difference in

NO_X were OR = 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.

Word count: 196

Suggested MeSH: Air Pollution, Vehicle Emissions, Asthma, Bronchitis, Epidemiology

1

Introduction

Numerous epidemiological 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 epidemiological 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.(4-10) 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) (11) The majority of studies have been conducted in Europe and Japan, where fleet composition (diesel vs. gasoline), emissions factors, fuel specifications, land use, and population distributions near busy roads differ from those in the U.S. Regional and micro-environmental concentrations of particulate matter 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 U.S. 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 these studies have been previously reported in the form of abstracts.(15)

Methods: (word count 552)

Study design and health assessment

We conducted a school-based cross-sectional study in the San Francisco metropolitan area (Alameda County, California) in 2001. The study area was comprised of ten 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)(17,18) We obtained information on health outcomes (bronchitis symptoms in the past 12 months and

3

physician-confirmed asthma in the past 12 months), demographics, home environmental factors and activity factors using parental questionnaires (English and Spanish).(16-18) 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), and nitrogen oxides (NO_X and NO_2)) at the school sites. PM_{10} and $PM_{2.5}$ mass concentrations were measured using filter-based samples, while 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., USA). 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 one-week intervals in the spring (March - June) and for eight weeks 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 data supplement and elsewhere.(21)

In preliminary analyses, we also used school location in relation 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 epidemiological studies of air pollution when pollutants were measured at the group level.(22) (18) In our study, the exposure groups are 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 relating asthma or bronchitis symptoms to air pollution, after adjusting for individual-level risk factors.

We calculated adjusted odds ratio(s) (ORs) for a change in measured pollutant

concentration equal to the interquartile ranges (IQR) 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 to 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 1109

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. About 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 meters downwind of a freeway compared to 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 ($r^2 \sim 0.9$ for each inter-pollutant correlation). The study average $PM_{2.5}$ (12 µg/m^3) was similar to the annual average concentration of $PM_{2.5}$ at the central monitoring station, located approximately 15 kilometers 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 relation to six different pollutants, each examined in separate regressions. Results are shown for all subjects, for long-term residents only (one year or longer at the current address), and for the latter group stratified by gender. In addition to the traffic metric, explanatory variables retained in all the final models for asthma and bronchitis included chest illness before age two, 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. 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 multi-pollutant models were evaluated because of the high inter-pollutant correlations. Restricting the analysis based on duration of residence (i.e., at least one year at current residence) tended to increase the effect estimates slightly in relation to asthma, especially when the sample was restricted to girls. Stratification by duration of residence or gender did not change the results for bronchitis. Results were similar when non-normalized 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% versus 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 odds ratios 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 epidemiological 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 one 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. (4-10) (11).(7,10-12) Furthermore, our findings were observed in a region with relatively clean air (low concentrations of ozone and particulate matter). (See online supplement for details) Although previous epidemiological 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, only 32% had lived at the same address since before age

two).

We considered whether there might be bias due to non-response 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 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 non-response 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 one 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 gender-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. While 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 <300m from a major road) gave estimates of odds ratios 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 relation 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.

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

11

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 et al. found that effect estimates using 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 multi-level analysis allows adjustment for individual confounders in the first stage of analysis. Moreover, in this respect this study is comparable with other epidemiological 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.(22)

(18,23)

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 (particulate matter from diesel exhaust) is highly correlated with outdoor levels.(2)

Other covariates.

Maternal asthma, household mold/moisture, pests, and chest illness before age two were important explanatory variables in the final model for current asthma, consistent with previous studies (31).(32,33) 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 SES).

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 under-reporting of household smoking (7 % in our study vs. 19% statewide).(35) Alternatively, a substantial portion of our study population were 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 under-reporting 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 (ETS) (e.g., SES and race-ethnicity) did not change the pollutant effect estimates, suggesting that significant confounding by ETS 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 vs. gasoline engines) will be important to better understand the public health

15

impacts of motor vehicle emissions.

References

1. Brunekreef B, Holgate ST. Air pollution and health. Lancet   2002;360:1233-42.

2. Fischer P, Hoek G, van Reeuwijk H, Briggs D, Lebret E, van Wijnen J, Kingham S,
   and Elliott P. Traffic-related differences in outdoor and indoor
   concentrations of particles and volatile organic compounds in Amsterdam.
   Atmospheric Environment 2000;34:3713-22.

3. Roorda-Knape MC, Janssen N, de Hartog J, van Vliet P, Harssema H, and
   Brunekreef B. Air pollution from traffic in city districts near major
   motorways. Atmospheric Environment 1998;32(11):1921-30.

4. Wjst M, Reitmeir P, Dold S, Wulff A, Nicolai T, von Loeffelholz-Colberg EF, and
   von Mutius E. Road traffic and adverse effects on respiratory health in
   children [see comments]. BMJ 1993;307(6904):596-600.

5. Weiland SK, Mundt KA, Ruckmann A, and Keil U. Self-reported wheezing and
   allergic rhinitis in children and traffic density on street of residence. Ann
   Epidemiol 1994;4(3):243-7.

6. Ciccone G, Forastiere F, Agabiti N, Biggeri A, Bisanti L, Chellini E, Corbo G,
   Dell'Orco V, Dalmasso P, Volante TF et al. Road traffic and adverse
   respiratory effects in children. SIDRIA Collaborative Group. Occup Environ
   Med 1998;55(11):771-8.

7. van Vliet P, Knape M, de Hartog J, Janssen N, Harssema H, and Brunekreef B. Motor vehicle exhaust and chronic respiratory symptoms in children living near freeways. Environ Res 1997;74(2):122-32.

8. English P, Neutra R, Scalf R, Sullivan M, Waller L, and Zhu L. Examining associations between childhood asthma and traffic flow using a geographic information system. Environ Health Perspect 1999;107(9):761-7.

9. Edwards J, Walters S, and Griffiths RK. Hospital admissions for asthma in preschool children: relationship to major roads in Birmingham, United Kingdom. Arch Environ Health 1994;49(4):223-7.10. Brunekreef B, Janssen NA, de Hartog J, Harssema H, Knape M, and van Vliet P. Air pollution from truck traffic and lung function in children living near motorways. Epidemiology 1997;8(3):298-303.11. Janssen NA, Brunekreef B, van Vliet P, Aarts F, Meliefste K, Harssema H, Fischer P. The relationship between air pollution from heavy traffic and allergic sensitization, bronchial hyperresponsiveness, and respiratory symptoms in Dutch schoolchildren. Environ Health Perspect 2003;111(12):1512-8.

12. Kramer U, Koch T, Ranft U, Ring J, Behrendt H. Traffic-related air pollution is associated with atopy in children living in urban areas. Epidemiology 2000;11(1):64-70.

13. Hoek G, Brunekreef B, Goldbohm S, Fischer P, van den Brandt PA. Association between mortality and indicators of traffic-related air pollution in the

Netherlands: a cohort study. Lancet 2002;360(9341):1203-9.

14. Lin S, Munsie JP, Hwang SA, Fitzgerald E, Cayo MR. Childhood asthma hospitalization and residential exposure to state route traffic. Environ Res 2002;88(2):73-81.

15. Kim J, Smorodinsky S, Lipsett M, et al. Traffic-Related Air Pollution and Respiratory Health: The East Bay Children's Respiratory Health Study. [Abstract] Epidemiology 2002;13:(4 Supplement)S100

16. Ware JH, Ferris BG Jr, Dockery DW, Spengler JD, Stram DO, Speizer FE. Effects of ambient sulfur oxides and suspended particles on respiratory health of preadolescent children. Am Rev Respir Dis 1986;133(5):834-42.

17. Dockery DW, Cunningham J, Damokosh AI, Neas LM, Spengler JD, Koutrakis P, Ware JH, Raizenne M, Speizer FE. Health effects of acid aerosols on North American children: respiratory symptoms. Environ Health Perspect 1996;104(5):500-5.

18. Peters JM, Avol E, Navidi W, London SJ, Gauderman WJ, Lurmann F, Linn WS, Margolis H, Rappaport E, Gong H, et al. A study of twelve Southern California communities with differing levels and types of air pollution. I. Prevalence of respiratory morbidity. Am J Respir Crit Care Med 1999;159(3):760-7.

19. Gundel L, Dod R, Rosen H, and Novakov T. The relationship between optical

attenuation and black carbon concentration for ambient and source particles. The Science of the Total Environment 1984;36:197-202.

20. Edwards J, Ogren J, Weiss R, and Charlson R. Particulate air pollutants: a comparison of British "Smoke" with optical absorption coefficient and elemental carbon analysis. Atmospheric Environment 1983;17(11):2337-41.

21. Singer BC, Hodgson AT, Hotchi T., and Kim JJ. Passive measurement of nitrogen oxides to assess traffic-related pollutant exposure for the East Bay Children's Respiratory Health Study. Atmos Environ 2004;38(3):393-403 .

22. Dockery DW, Speizer FE, Stram DO, Ware JH, Spengler JD, Ferris BG Jr. Effects of inhalable particles on respiratory health of children. Am Rev Respir Dis 1989;139(3):587-94.

23. McConnell R, Berhane K, Gilliland F, London SJ, Vora H, Avol E, Gauderman WJ, Margolis HG, Lurmann F, Thomas DC, et al. Air pollution and bronchitic symptoms in Southern California children with asthma. Environ Health Perspect 1999;107(9):757-60.

24. Oosterlee A, Drijver M, Lebret E, and Brunekreef B. Chronic respiratory symptoms in children and adults living along streets with high traffic density. Occup Environ Med 1996;53(4):241-7.

25. Pershagen G, Rylander E, Norberg S, Eriksson M, and Nordvall SL. Air pollution involving nitrogen dioxide exposure and wheezing bronchitis in children. Int J Epidemiol 1995;24(6):1147-53.

26. Peters JM, Avol E, Gauderman WJ, Linn WS, Navidi W, London SJ, Margolis H, Rappaport E, Vora H, Gong H Jr, et al. A study of twelve Southern California communities with differing levels and types of air pollution. II. Effects on pulmonary function. Am J Respir Crit Care Med 1999;159(3):768-75.

27. Gauderman WJ, Gilliland GF, Vora H, Avol E, Stram D, McConnell R, Thomas D, Lurmann F, Margolis HG, Rappaport EB, et al. Association between air pollution and lung function growth in southern California children: results from a second cohort. Am J Respir Crit Care Med 2002;166(1):76-84.

28. Delfino RJ, Gong H Jr, Linn WS, Pellizzari ED, Hu Y. Asthma symptoms in Hispanic children and daily ambient exposures to toxic and criteria air pollutants. Environ Health Perspect 2003;111(4):647-56.

29. Wyler C, Braun-Fahrlander C, Kunzli N, Schindler C, Ackermann-Liebrich U, Perruchoud AP, Leuenberger P, Wuthrich B. Exposure to motor vehicle traffic and allergic sensitization. The Swiss Study on Air Pollution and Lung Diseases in Adults (SAPALDIA) Team. Epidemiology 2000;11(4):450-6. Comment in: Epidemiology 2001; 12(1) 137-8.

.

30. Rijnders E, Janssen NA, van Vliet PH, Brunekreef B. Personal and outdoor nitrogen dioxide concentrations in relation to degree of urbanization and traffic density. Environ Health Perspect 2001;109 Suppl 3:411-7.

31. Bornehag CG, Blomquist G, Gyntelberg F, Jarvholm B, Malmberg P, Nordvall L, Nielsen A, Pershagen G, Sundell J. Dampness in buildings and health. Nordic interdisciplinary review of the scientific evidence on associations between exposure to "dampness" in buildings and health effects (NORDDAMP). Indoor Air 2001;11(2):72-86.

32. Martinez FD. Maternal risk factors in asthma. Ciba Found Symp 1997;206:233-9.

33. Martinez F. Viral infections and the development of asthma. Am J Respir Crit Care Med 1995;151:1644-8.

34. Cook DG, Strachan DP. Health effects of passive smoking. 3. Parental smoking and prevalence of respiratory symptoms and asthma in school age children. Thorax 1997;52(12):1081-94.

35. California Department of Health Services. Tobacco Control Section. California Tobacco Control Update . November 2002. Available at URL: http://www.dhs.ca.gov/tobacco/documents/TCSupdate.PDF .

36. Klinnert MD, Price MR, Liu AH, Robinson JL. Unraveling the ecology of risks for

early childhood asthma among ethnically diverse families in the southwest.
. Am J Public Health 2002;92:792-8.

37. Delfino RJ. Epidemiologic evidence for asthma and exposure to air toxics: linkages between occupational, indoor, and community air pollution research. Environ Health Perspect 2002;110 Suppl 4:573-89.

38. Brauer M, Hoek G, van Vliet P, Meliefste K, Fischer P, Gehring U, Heinrich J, Cyrys J, Bellander T, Lewne M, et al. Estimating long-term average particulate air pollution concentrations: application of traffic indicators and geographic information systems. Epidemiology 2003;14(2):228-39.

39. Texas Transportation Insititute. 2002 Urban Mobility Report.   Available http://mobility.tamu.edu/ums/.

40. Rodes CE and Holland DM. Variations of NO, NO_2 and O_3 concentrations downwind of a Los Angeles freeway. Atmospheric Environment   1981;15: 243-50.

41. Zhu Y, Hinds WC, Kim S, Sioutas C. Concentration and size distribution of ultrafine particles near a major highway. J Air Waste Manag Assoc 2002;52(9):1032-42.

Figure Legends.

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.

Figure 2. Adjusted School-Specific Bronchitis Prevalence Rates Versus Black Carbon, Long-term Residents

Figure 3. Adjusted School-Specific Asthma Prevalence Rates Versus Black Carbon, Long Term Residents

Table1. Demographic, family, and home characteristics of the East Bay Children's Respiratory Health Study respondents.

Characteristics	All subjects (N = 1109) %	Subjects attending schools	
		Near and Downwind of major roads (4 schools, N =402) %	Far or Upwind of major roads (6 schools, N = 707) %
Gender			
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 months	63.1	65.4	61.8
Gas stove	63.1	63.6	62.9
Indicator of mold/mildew presence, past 12 months	44.6	43.5	45.3

Health outcomes

Chest illness before age 2	23.3	18.8	25.9
Asthma, past 12 months	14.0	13.9	14.1
Bronchitis, past 12 months	12.1	13.2	11.5

_____

*Parent responding to the questionnaire

Table 2: Nearby traffic sources and average pollutant concentrations at ten schools*

School	Major Traffic Source[†]	AADT (#/day)	Distance[‡] (m)	<300 m downwind	PM_{10} µg/m³	$PM_{2.5}$ µg/m	BC (µg/m³)	NO_X ppb	NO_2 ppb	NO[§] ppb
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[11]	Yes	190,000	60	Yes	32	15	1.1	69	31	38
Study average					30	12	0.8	49	23	25

28

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=1109)		(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	
NOx	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)

* 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

‡LTR = long term resident (current address for 1 year or more)

Odds ratios are calculated per interquartile range (IQR) of average pollutant concentration as follows:

NOx = 14.9 ppb, NO2 = 3.6 ppb, NO = 11.6 ppb, PM10 = 1.4 µg/m3, PM2.5 = 0.7 µg/m3, BC = 0.15 µg/m3
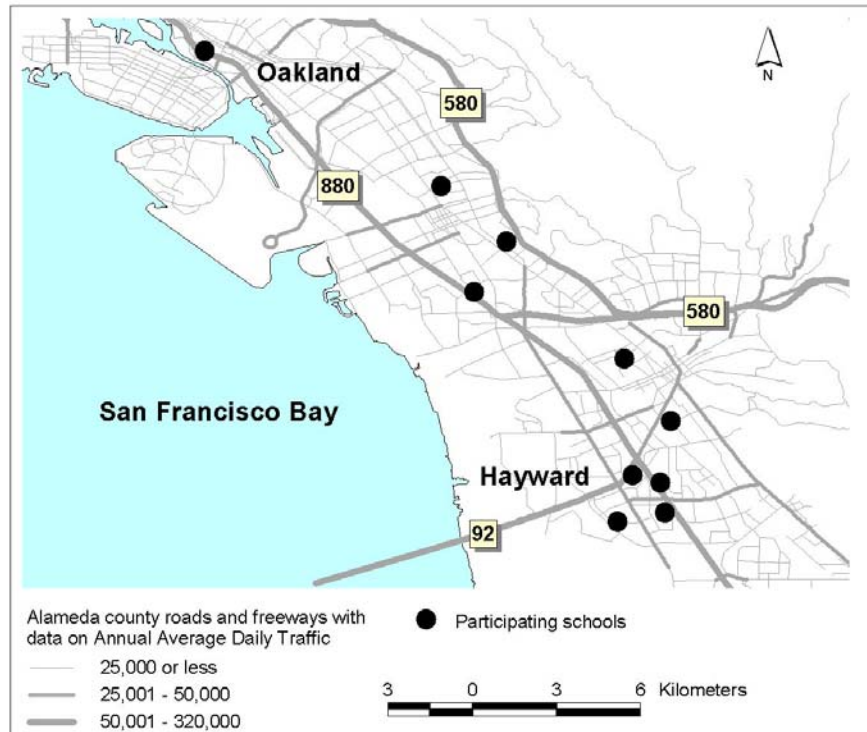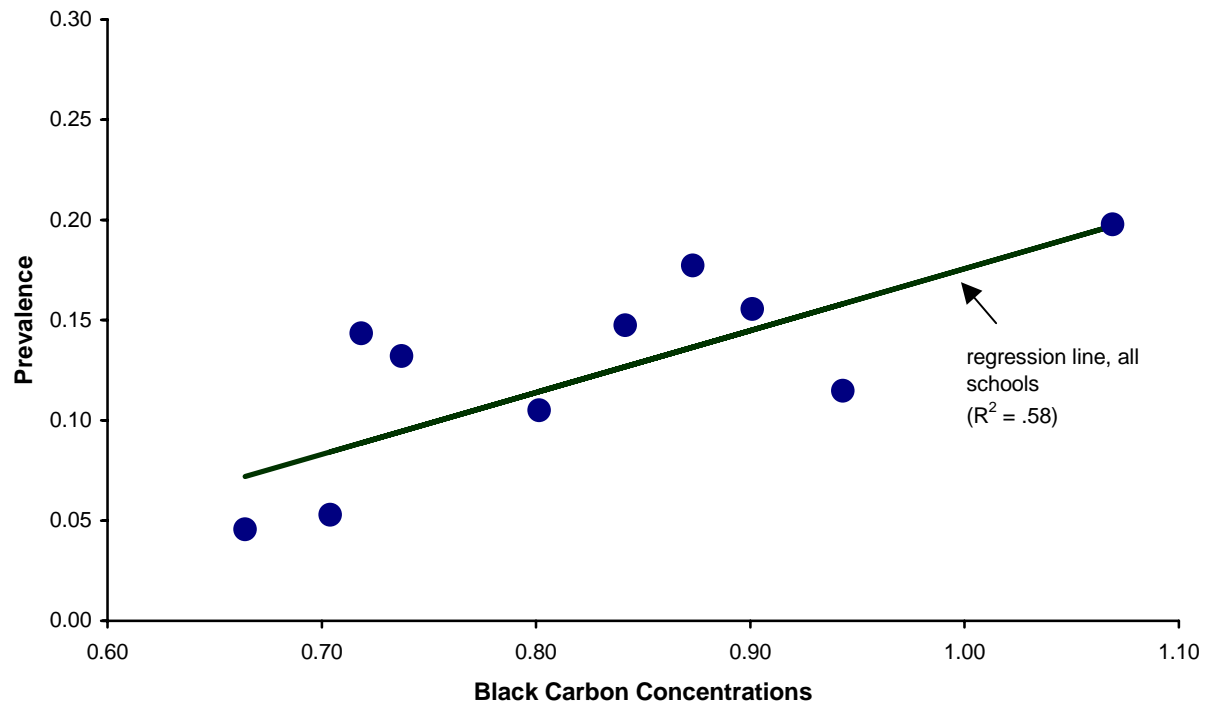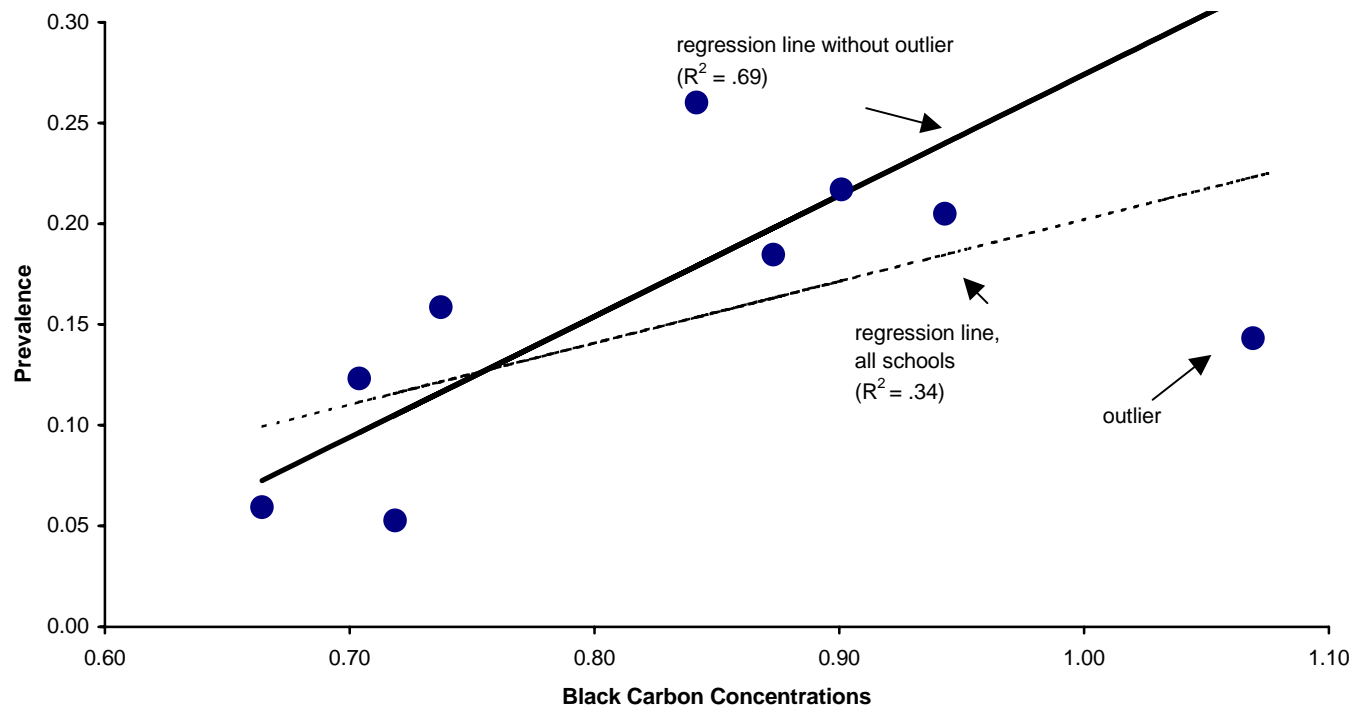
Figure 1.

Figure 2

Figure 3.

regression line without outlier
(R^2 = .69)

regression line,
all schools
(R^2 = .34)

outlier

Prevalence

Black Carbon Concentrations

Title: Traffic-related air pollution near busy roads: the East Bay Children's Respiratory

Health Study

Authors

Janice J. Kim , Svetlana Smorodinsky , Michael Lipsett , Brett C. Singer , Alfred T.

Hodgson , Bart Ostro

Online Data Supplement

Methods :

Study design and study location

The study area (approximately 21 km x 5 km) is across the bay from San Francisco, and includes 10 neighborhood elementary schools that span a busy traffic corridor. The topography is relatively flat and the urban landscape consists primarily of widely spaced, low-level buildings. Traffic congestion in the San Francisco-Oakland area ranks second in the United States,(39) but regional air quality is generally good due to ocean breezes from the southwest or west. At the nearby air quality monitoring station (Fremont), 15 km southeast of the study area, ozone concentrations rarely exceed the national or state standards (0-1days/ year above federal ozone standards for past 3 years). Annual-average concentrations of PM_{10} and $PM_{2.5}$ at the Fremont station were 23.4 and 12.2 $\mu g/m^3$, respectively in 2001. These values are below the PM_{10} and $PM_{2.5}$ National Ambient Air Quality Standards (50 $\mu g/m^3$ and 15 $\mu g/m^3$, respectively) and just above the more stringent California standards (20 $\mu g/m^3$ and 12 $\mu g/m^3$, respectively) .

School site selection

We used a public school database from the California Department of Education (CDE) containing statewide information on school locations, enrollment, and demographics (URL: http://www.cde.ca.gov). A traffic database was obtained from the California Department of Transportation (CalTrans) Highway Performance Monitoring System (HPMS, 1997). Traffic files included statewide freeway and major road network

geography and counts of the annual average daily traffic (AADT) on those roadways. We utilized Geographic Information Systems (GIS) software to integrate, display, and analyze data from these disparate data sources (ArcView 3.2, ESRI, Redlands, CA).

We selected elementary schools for possible recruitment such that the school locations would represent a range of distances upwind and downwind from major roads. Because concentrations of traffic pollutants decrease to background levels within 300 m downwind (40,41) and because several European studies have found adverse health effects in children residing or attending schools in close proximity to roads with AADT of 25,000 or more vehicles (4,9), we used these parameters to guide our selection process.

Briefly, to identify possible school sites for recruitment, school addresses were geo-coded and overlaid with the CalTrans road network. Candidate schools "near" a major road were identified based on a geo-coded location less than 350 m from any road with AADT $\geq$ 90,000 vehicles. Schools "farther" from local or heavy traffic were also identified using GIS (no local road with AADT $\geq$ 20,000 vehicles/day within 300 m and no major highway or freeway within 750 m). We identified eight schools that were "near" freeways and twenty-one schools that were "farther" from local or heavy traffic in the proposed study area. From this list, we sought to recruit schools that were demographically similar and that were likely to have a range of exposure to traffic pollutants based on proximity to major roads and prevailing wind directions.

Ten schools that reflected locations with a range of distances upwind and downwind from major roads were selected. There were no major non-roadway sources of air pollution in

the neighborhoods (CA Air Resources Board, private communication).

Population:

In Spring, 2001, we invited all children in grades 3-5 in participating classes (n=64) to join the study. Packets containing a health questionnaire in English or Spanish, an informed consent form, and study information were distributed in the classroom, completed by parents, and returned to the teacher. An option was available to complete the questionnaire by telephone with trained research staff. Up to three reminder notices were sent to non-responders. We gave a donation towards educational materials to classrooms and provided nominal compensation to parents completing the questionnaire. The study protocol was approved by the Committee for the Protection of Human Subjects, California Health and Human Services Agency.

Health Assessment:

Questions on respiratory symptoms and illnesses were modeled after those used in previous studies on air pollution and children's health.(16) (17,18) Subjects were defined as having current asthma if their parents selected asthma in response to the question, "During the previous 12 months, did a doctor say that your child had any of the following chest illnesses?" (Choices were pneumonia, asthma, reactive airway disease, and other chest illness.) Current bronchitis was defined as: (1) a positive response to the question: "During the past 12 months, did your child have bronchitis?" or (2) a report of cough and chest congestion or phlegm lasting at least three consecutive months of the past 12.

We also asked about personal factors potentially associated with asthma or bronchitis, including demographic variables (race/ethnicity and measures of socioeconomic status (SES) (e.g., parents' education, income, health insurance)); host factors (age, gender, chest illness before age two, maternal smoking during pregnancy, maternal or paternal history of asthma, premature birth, breastfeeding); home environmental factors (current smoker in the home, gas stove, evidence of mold, pets, problem with pests in the past year, carpet in the bedroom; and activity factors (sports participation, commute patterns to school). Pests included cockroaches, mice, rats, termites, spiders, or ants. Those with "chest illness before the age of two" were reported to have seen a doctor before age two for asthma, bronchiolitis, RSV, croup, reactive airway disease, or pneumonia.

Indicator of "mold" was a Yes to any of the following: history of residential water damage, visible mold/yildew, water condensation, or mold/musty smell in the past 12 months. In sensitivity analyses we used a slightly different definition of asthma (physician-diagnosed ever, and asthma symptoms, including wheezing, in the past 12 months), and duration of residence (at least one year at current address Yes/No).

Air Pollution from Traffic:

To estimate the children's overall exposure to traffic pollution, we measured concentrations of traffic-related pollutants at the schools. We also used school location with respect to prevailing winds and proximity to busy roads as a separate exposure metric.

(1) Traffic-related pollutant measurements: We determined PM_{10} and $PM_{2.5}$ mass

concentrations from weeklong filter samples (Pallflex fiberfilm, Pall/Gelman) collected

with small sampling pumps (Airchek 2000, SKC, Inc) and size-selective inlets (PEM,

MSP Corp). BC concentrations were determined on the PM_{10} filter samples using an

established light attenuation method that we validated for the fiberfilm filters.(19) (20)

Concentrations of NO_X and NO_2 were determined with passive diffusion samplers

(Ogawa, Inc., USA) deployed over a one-week sampling period. The NO concentration

was estimated as the difference between NO_X and NO_2. Additional details of the

monitoring protocol are described elsewhere.(21)

We estimated chronic exposures to traffic pollutants by measuring pollutant

concentrations simultaneously at all schools over several month. Specifically, monitoring

was conducted for 11 weeks in the spring (March - June) and eight weeks in the fall

(September-November) 2001. NO_X and NO_2 were sampled during all periods at each

school.(21) PM_{10} and $PM_{2.5}$ 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 data as follows: Because of

missing samples and week-to-week variability at each site, we obtained average site-

specific pollutant concentrations as follows: For each week and pollutant, we calculated

the ratio of each school's measured pollutant level relative to the mean across all schools

sampled that week. Then, for each school, the mean ratio over the entire study period

was calculated. The mean ratios are normalized concentrations and represent the relative

exposures across school locations. To obtain an estimate of the average pollutant

concentration at each school, we multiplied   the mean of the school-specific normalized

concentration by the study-average pollutant concentration (averaged across all study sites over the entire study period). This normalization process allowed for an aggregation of data for all sampling periods, including those with an incomplete data set. Only weeks with valid data from at least six schools were included in this process.

(2) Additional traffic metric: Schools were assigned a category for wind direction (upwind or downwind of a freeway) and a category for proximity to traffic (<300 m or >300 m from a major traffic source) as well as the combination of the two (i.e., downwind and close versus all others).

Data Analysis

To explore the extent to which additional covariates would have an effect on the pollutant estimate, we added pollutant values or school location indicators to the model, one at a time and then examined the effect of removing each covariate on the pollutant coefficient(s); if removal of a covariate from the model changed the magnitude of the pollutant coefficient by less than 10%, the covariate was dropped.

In a separate sensitivity analysis, we also initially included variables found to be predictive of respiratory symptoms in other studies even if they did not satisfy the p-value criterion. Other several sensitivity analyses included examining the model using other definitions of asthma and after stratifying the outcome of current bronchitis by asthma status. We also restricted the sample to the subgroup of children who had lived at the given address for more than one year (long-term residents) and performed stratified analysis by gender, both for the full sample and long-term residents. Additionally, we

re-ran the models after dropping one school that was an outlier in terms of race/ethnicity distribution.

Adjusted odds ratio(s) (ORs) were calculated for an interquartile range (IQR) of measured pollutant concentrations (i.e., the odds ratio for a given health outcome given a pollutant concentration at the 75 percentile of the distribution relative to that at the 25th percentile). All analyses were conducted using SAS version 8.2 for Windows and STATA version 8.

References, Online Supplement

E1.   Texas Transportation Insititute. 2002 Urban Mobility Report.   Available
        http://mobility.tamu.edu/ums/.

E2.   Rodes CE and Holland DM. Variations of NO, NO_2 and O_3 concentrations
        downwind of a Los Angeles freeway. Atmospheric Environment
        1981;15:243-50.

E3.   Zhu Y, Hinds WC, Kim S, Sioutas C. Concentration and size distribution of
        ultrafine particles near a major highway. J Air Waste Manag Assoc
        2002;52(9):1032-42.

E4.   Wjst M, Reitmeir P, Dold S, Wulff A, Nicolai T, von Loeffelholz-Colberg EF,
        and von Mutius E. Road traffic and adverse effects on respiratory health in
        children [see comments]. BMJ 1993;307(6904):596-600.

E5.   Edwards J, Walters S, and Griffiths RK. Hospital admissions for asthma in
        preschool children: relationship to major roads in Birmingham, United
        Kingdom. Arch Environ Health 1994;49(4):223-7.

E6.   Ware JH, Ferris BG Jr, Dockery DW, Spengler JD, Stram DO, Speizer FE.
        Effects of ambient sulfur oxides and suspended particles on respiratory
        health of preadolescent children. Am Rev Respir Dis 1986;133(5):834-
        42.

E7. Dockery DW, Cunningham J, Damokosh AI, Neas LM, Spengler JD, Koutrakis P, Ware JH, Raizenne M, Speizer FE. Health effects of acid aerosols on North American children: respiratory symptoms. Environ Health Perspect 1996;104(5):500-5.

E8. Peters JM, Avol E, Navidi W, London SJ, Gauderman WJ, Lurmann F, Linn WS, Margolis H, Rappaport E, Gong H, et al. A study of twelve Southern California communities with differing levels and types of air pollution. I. Prevalence of respiratory morbidity. Am J Respir Crit Care Med 1999;159(3):760-7.

E9. Gundel L, Dod R, Rosen H, and Novakov T. The relationship between optical attenuation and black carbon concentration for ambient and source particles. The Science of the Total Environment 1984;36:197-202.

E10. Edwards J, Ogren J, Weiss R, and Charlson R. Particulate air pollutants: a comparison of British "Smoke" with optical absorption coefficient and elemental carbon analysis. Atmospheric Environment 1983;17(11):2337-41.

E11. Singer BC, Hodgson AT, Hotchi T., and Kim JJ. Passive measurement of nitrogen oxides to assess traffic-related pollutant exposure for the East Bay Children's Respiratory Health Study. Atmos Environ 2004;38(3):393-403