Final Technical Report

Date of Final Report: August 31, 2006
EPA Grant Number: R827352C007
Center Name: Southern California Particle Center and Supersite (SCPCS)
Center Director: John R. Froines
Title: Exposure to Vehicular Pollutants and Respiratory Health
Investigators: Robert McConnell[1], Fred Lurmann[2], W. James Gauderman[1], Edward Avol[1]
Institution: [1]University of Southern California, [2]Sonoma Technology, Inc.
EPA Project Officer: Stacey Katz/Gail Robarge
Project Period: June 1, 1999–May 31, 2005 (no-cost extension to May 31, 2006)
Period Covered by the Report: June 1, 1999–May 31, 2006
RFA: Airborne Particulate Matter (PM) Centers (1999)
Research Category: Particulate Matter

Topic B: Studies of Emission Sources and Related Adverse Health Effects

Objective(s) of the Research Project:

Project Hypotheses

This project tested the hypothesis that exposure to vehicular pollutants is associated with respiratory health in children. We have examined this hypothesis using data from the Children's Health Study (CHS), a longitudinal evaluation originally designed to evaluate the effect of average community pollutant exposures on average community respiratory health outcomes. Pollution has been well characterized at central site monitors in 16 Southern California communities involving participants from two sets of school based cohorts. In the Southern California Particle Center and Supersite (SCPCS) we have characterized within-community variation in traffic related pollutant exposure. These new pollutant metrics have allowed us to examine the relationship of individual level exposures to the CHS outcomes:

1. We have developed modeled metrics of exposure and tested these against measured pollutants.

2. We have evaluated the association of these metrics and of measured indicator pollutants to:

    a. Prevalence and severity of asthma at entry
    b. Incidence of asthma during follow-up
    c. Lung function at study entry and its growth during follow-up
    d. School absence.

3. We have evaluated the association of chronic asthma exacerbation with chronic temporal variation from yearly variation in exposure to oxidant pollutants among asthmatics.

Project Objectives

Our approach has been to geo-code addresses of residences and schools and then to assign traffic exposures to these addresses, based on traffic estimates available from the California Department of Transportation (Caltrans). We have used ambient air quality measurements at 12 locations to evaluate these models. Using modifications of statistical modeling strategies developed for the CHS, we have examined the effect of exposure to traffic related pollutants on asthma prevalence, and we have found relationships not previously reported in this growing literature. In addition, we have examined outcomes for which there has been little previous study of the effects of traffic modeled pollutants: asthma severity, asthma incidence, lung function and lung function growth, and school absence.

Summary of Findings:

Aim 1: Characterization of Exposure to Traffic-Related Pollutants in CHS Communities

The overall objective of the exposure component of this study was to provide improved methods and databases to characterize seasonal and annual average population exposure to traffic-related pollutants with fine spatial resolution. Prior to this study, Sonoma Technology, Inc. had performed some initial traffic modeling for the CHS which pointed to the need for model refinements, database improvements, and model performance evaluation. Many potential improvements were explored; improvements in the spatial accuracy of roadway and receptor data, meteorological data, and emission rates proved most important. It is important to note that most assessments of traffic-related pollutant exposure focus on worst-case 1-hr or 8-hr maximum conditions. Our focus was on estimation of long-term exposures in order to evaluate relationships with chronic health effects.

Traffic Activity and Roadway Data. Annual average daily traffic counts data for Interstate freeways, other principal arterials, minor arterials, major collectors, and minor collectors in 2000 were obtained from Caltrans. The annual traffic counts are based on continuous measurement data for freeways and intermittent measurements (usually every three years) on other arterials and some collectors. Diurnal traffic volume variations and day-of-week variations for light-duty and heavy-duty vehicles on freeways were determined from Caltrans weigh-in-motion (WIM) data for Southern California. Diurnal variations for collectors were determined from more limited traffic measurements (Chinkin, et al., 2003).

ESRI ArcGIS software was used to process the Caltrans roadway link and traffic count data. The Caltrans roadway geometries were mostly based on TIGER files and were often inaccurate, based on comparisons with aerial photography images. Comparison to global positioning system (GPS)-accurate TeleAtlas Roadway Network data showed that the Caltrans' TIGER roadway links occasionally had 250-m discrepancies from actual roadway locations. The roadway geometry errors were random and affected roadways of all sizes in most communities. Zhu, et al. (2002) reported ten-fold differences in measured concentrations of traffic-related pollutants between 30 m and 200 m downwind of Southern California freeways. Errors of 100 m to 250 m in the location of major roadways relative to residence are not acceptable for neighborhood-scale assessments of traffic effects. Since the Caltrans roadway location data did not have sufficient accuracy for our intended use, methods (software) were developed to transfer the Caltrans annual traffic volumes to the GPS-accurate TeleAtlas roadway network. The TeleAtlas roadway database incorporated both more

accurate and more precise location information. For example, each direction of travel on moderate and large roadways is represented as a separate link in the TeleAtlas database.

Proximity Modeling. We wanted to examine the relationships of CHS participant health status with traffic indicators separately from the CALINE4 dispersion model estimates of concentrations from mobile source emissions. Dispersion modeling provides refinements but introduces additional uncertainties compared to analysis of traffic alone. A three-level hierarchical approach was adopted for traffic assessment that considered: (1) the distance of residences to nearest roadways of various types, (2) GIS-mapped traffic density assignments at residences, and (3) the CALINE4 dispersions model estimates of traffic-related pollutant concentrations at residences.

The first sets of traffic metrics were the distances from residences to the nearest roadways of different types. GIS tools were used to calculate the distance to the nearest: (1) interstate freeway, U.S. highway, or limited access highway; (2) other highways; (3) arterial roads; (4) collector roads; and (5) local roads.

The second approach for characterization of traffic exposures was to calculate traffic densities, which vary more smoothly in space than the distances to nearest roads. They also capture the effects of intersection and multiple roadway influences that are missed using only distance to the nearest roadways. The link-based traffic volumes are used to generate maps of traffic density using the ArcGIS Spatial Analyst software. The traffic density maps were created with a Gaussian decay function that has traffic densities decreasing by ~90% between the roadway and 150 m away (perpendicular) from the roadways, which is consistent with the characteristics observed by Zhu, et al. (2002).

Dispersion Modeling. The CALINE4 model, developed by Caltrans and the U.S. Federal Highway Administration (Benson 1989), is one of several Gaussian line source dispersion models that is designed to estimate local-scale pollutant concentrations from motor vehicle emissions. It has primarily been evaluated for inert traffic-related pollutants such as CO over short periods and was selected for the SCPCS analyses because it has a credible scientific formulation. The model was used to simulate ambient concentrations due to on-road motor vehicle emissions on all roads with traffic volume data located within a 20 km square centered in each CHS community.

A climatological approach was used to estimate long-term average concentrations. The model was applied for a wide range of meteorological cases in each community, and the seasonal or annual concentrations were calculated by weighting the results for individual cases by the frequency of occurrence of the conditions in the community. The results are post-processed to incorporate pollutant-specific emission factors, diurnal and day-of-week variations of traffic volumes, and chemical conversion (for NO to NO_2).

Vehicle emission factors were obtained from the California Air Resource Board's (CARB) EMFAC2002 vehicle emissions model. The elemental carbon (EC) and organic carbon (OC) fractions of exhaust particulate matter (PM) emissions were based on composite profiles from Gillies and Gertler (2000). Paved road-dust emission factors for $PM_{2.5}$ and PM_{10} were based on Southern California in-roadway measurements (Fitz and Bufalino, 2002). The EMFAC model also estimates the PM emissions from brake wear and tire debris.

Figure 1 shows comparisons of CALINE4 model estimates for the air monitoring station locations to the 4-year average observed ambient concentrations at the stations. There were correlations between the model estimates and the observations for all pollutants. The dispersion model estimates local motor vehicle emissions contributed 17% of the observed NO_2 concentrations, on average, and the coefficient of determination was 0.81. The model estimates for NO_x were more strongly correlated with observations ($r^2 = 0.97$) than those for NO_2. This result was consistent with the dispersion model being formulated for chemically non-reactive species and, therefore, the model was more accurate for NO_x than NO_2. The average estimated contribution of local motor vehicle emissions to the observed $PM_{2.5}$ EC concentrations was 16% and the coefficient of determination was 0.86. The relationships of the model results to the observed concentrations were very similar for $PM_{2.5}$ EC and NO_2. This was somewhat surprising given that EC emissions from vehicles are not well characterized and most emissions experts consider PM (and EC) emissions rates to be far more uncertain than those for NO_x. The model estimates for $PM_{2.5}$ OC were less well correlated with the observed concentrations ($r^2 = 0.71$). The model estimate for the Mira Loma station location was quite low compared to the very high OC concentration observed in the station. The comparison of estimated and observed $PM_{2.5}$ OC was confounded by not only the contributions of regional transport and other local sources, but also the contribution of secondary OC to the measured OC. The various estimates of the incremental traffic related pollutant contributions to the background concentrations were, in general, highly correlated. NO_2 and NO_x have been used in the CHS and other population based studies as indicators of within-community variation in exposure to traffic-related pollutants that can be measured at a reasonable price. For consistency we have examined exposure to these pollutants, but the associated health effects of modeled exposure from traffic might plausibly be attributed to particulate exposure or to other gaseous co-pollutants in tailpipe emissions.

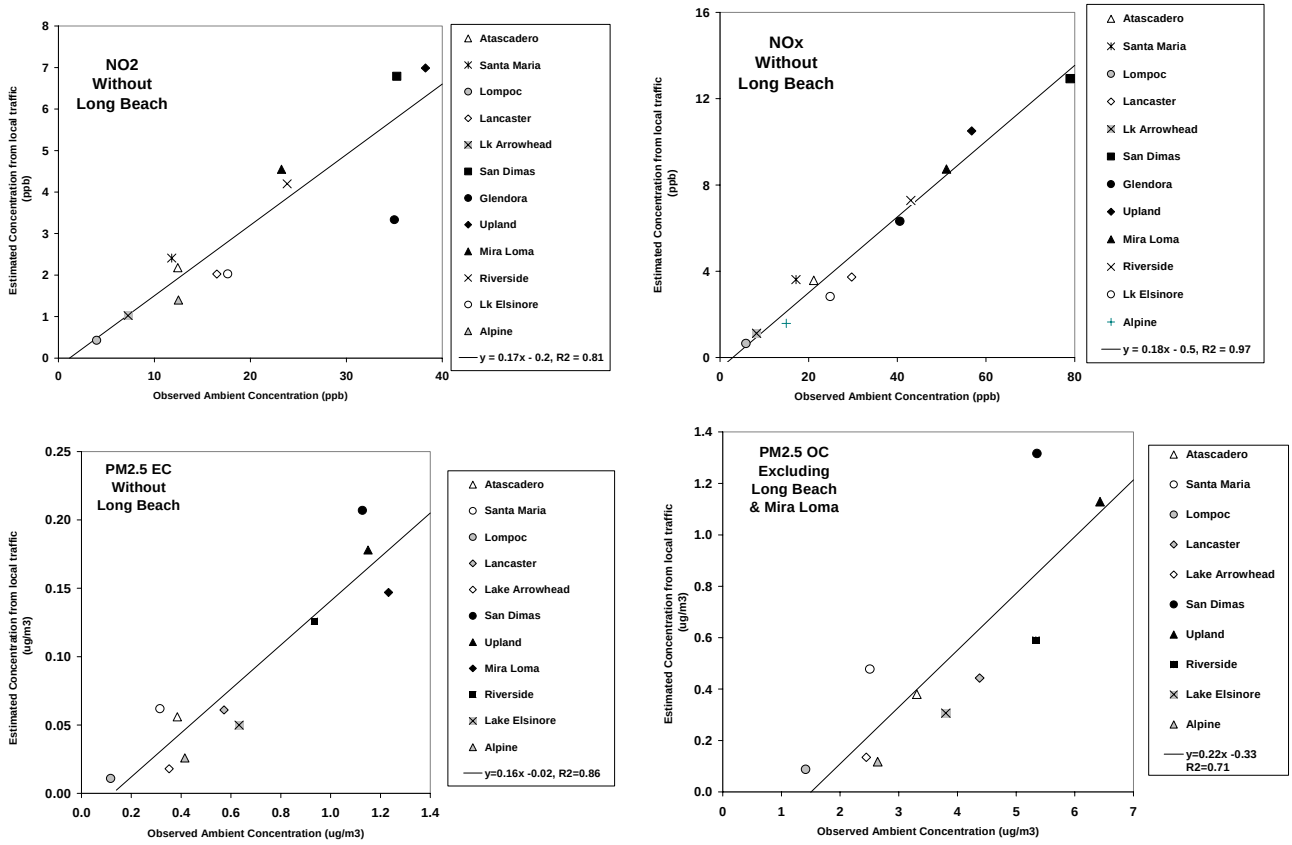

Figure 1. Comparison of Annual Average NO_2, NO_x, EC, and OC Concentrations Estimated by the CALINE4 Model for the Central Air Monitoring Station Locations and the Four-Year Average Observed Ambient Concentrations at the Stations. Note: the observations for San Dimas are based on only two years of data.

Simulations of concentrations from local roads in CHS communities confirmed the importance of regional-scale and urban-scale pollutant sources and meteorological transport in southern California. At the types of locations selected for community monitoring, the ambient pollutant concentrations due to transport from upwind and local non-mobile sources were always larger than the simulated concentrations from local on-road motor vehicle sources. This finding was consistent with regional modeling results completed for another SCPCS project led by Dr. Arthur Winer.

The CALINE4 dispersion model results were also used in the modeling of personal exposure of CHS participants. They were used as input to the Individual Exposure Model (IEM) developed under the SCPCS project led by Dr. Arthur Winer and described in Wu, et al., 2005b.

We have also examined the relationship between traffic modeled pollution and variation in ozone within communities (McConnell, et al. 2006b). In high ozone communities, ozone concentrations might be expected to vary inversely with fresh traffic exhaust, because NO in fresh exhaust scavenges ozone. To explore this hypothesis, we used a sample of homes for which ozone was measured simultaneously at the home and the central site monitor, as part of a previous study (Avol, et al. 1998). There was a highly significant inverse relationship observed

between measured ozone and our new traffic modeled exposure to oxides of nitrogen that varied spatially within communities. Although it is well known that ozone concentrations are low near major roadways, there has been little previous systematic study of the pattern of spatial variation within communities due to scavenging of ozone. Because ozone and traffic related particulate are both toxic to the lungs, and they are inversely correlated with respect to traffic, the risk of traffic exposure might be expected to be attenuated in high ozone communities. With separate funding we have embarked on a new study to measure ozone and NO_x at homes of children with and without incident asthma to explore the significance of these traffic-ozone relationships.

Aim 2a: Effect of Traffic on Asthma Prevalence and Severity

We evaluated the risk of lifetime asthma at study entry and traffic related pollutant exposure. We observed large risks associated with measured NO_2 at a sample of homes of children recruited into the original CHS study in 1993 and 1996 (Gauderman, et al., 2005). There were also high risks associated with residential distance to a freeway and with freeway modeled exposure to traffic related pollutants, using modeled exposures developed in Aim 1. The strength of this study is that we measured a traffic related pollutant at the homes, which has not been the case for most studies of traffic and asthma, and the consistency of results from measured and modeled exposures makes a causal relationship more plausible. Using modeled exposures, we observed a similar association in the entire cohort (unpublished data).

We then examined risks associated with early life asthma in younger children (5–6 years old) in a new cohort of CHS children recruited in 2003, using modeled exposures. The pattern of results largely replicated those observed in the earlier cohorts, and we found a strong relationship between prevalent and lifetime asthma and modeled exposure. In addition, there was a strong relationship with a relatively simple traffic metric, distance to a major road (McConnell, et al., 2006a) (see Table 1).

Table 1. Association of Asthma and Wheeze With Traffic Related Pollution Among Long Term Residents

		Lifetime Asthma	Prevalent Asthma	Current Wheeze
	N*	O.R.* (95% C.I.)	O.R.* (95% C.I.)	O.R.* (95% C.I.)
Major road distance				
>300 m	813	1.00	1.00	1.00
150 - 300 m	483	0.86 (0.59 , 1.24)	0.83 (0.56 , 1.21)	0.97 (0.69 , 1.38)
75-150 m	294	1.03 (0.68 , 1.56)	1.09 (0.71 , 1.66)	1.09 (0.73 , 1.62)
<75 m	266	1.46 (0.98 , 2.17)	1.64 (1.10 , 2.44) †	1.67 (1.14 , 2.43) §

*N is total exposed in each category of exposure; O.R. (95% C.I.) odds ratio (95% confidence interval), adjusted for age, sex, language of questionnaire, community, and race.
†p<.05; §p<.01

The effect varied by parental history of asthma and age of exposure, with much stronger associations observed in those with exposure before age 3 and with no family history. We believe this is an important finding, as some of the inconsistency in the literature may be explainable based on different proportions of susceptible children in different studies. Among children with a lifetime history of asthma in this cohort, we have also examined the relationship of traffic-related pollutants to various indicators of asthma severity, including wheeze and

bronchitic symptoms. Traffic related pollution was associated with bronchitic symptoms (unpublished results). This outcome is an indicator of chronic asthma exacerbation, which we have previously shown to be a sensitive endpoint for particulate effects (McConnell, et al., 1999).

During the past year we have undertaken a pilot study to evaluate the relationship of traffic modeled pollutants to measured particulate and NO_x pollution in Long Beach, a CHS community with heavy primary particulate pollution. We used nephelometers as indicators of $PM_{2.5}$ pollution that could be measured at reasonable cost in a relatively large number of sites simultaneously. NO, NO_2, and ozone were measured using integrated passive samplers. Residences of cases of lifetime asthma and controls from the 2003 cohort formed the sampling frame. The goal was to see if short term sampling at many locations would accurately indicate long term intra-community variation in exposure (and predict health effects), if the short term samples were co-located with a central site monitor for which continuous historical data are available. The nephelometers were co-located with the Ogawa ozone and NO_x samplers during 7 two week cycles at a total of 69 locations. There were 8 to 10 different residences per cycle. In addition, measurements were made at a central site and 4 other locations during all sampling periods. Traffic related exposure at all locations also was modeled. At selected sites, filters were also collected for PM_{10}, $PM_{2.5}$, and $PM_{0.25}$ EC and OC. Preliminary results show that measurements made with nephelometers were only weakly associated with NO, NO_2 and ozone. This indicates that NO_2, which is often measured as a cheap surrogate for traffic related pollutants, is not a good indicator of intra-community variation in $PM_{2.5}$. Traffic modeled exposure was associated with lifetime asthma, but measured pollutants were not. Likely explanations for this discrepancy include the short sampling period, which was unlikely to reflect annual or lifetime exposure. As expected, the measurements made at the community central site monitor varied by sampling period (due to varying meteorology). However, neither the residual of measurements at the central site and co-located locations in every cycle, nor the ratio of co-located measurements to the central site, were constant across cycles. A clear lesson for future epidemiologic studies is that short term sampling may not be adequate to characterize long term exposures, even if co-located with central site monitoring for which longer term exposure is well characterized. A manuscript is in preparation. These data have also been used for complementary analyses in SCPCS supported work conducted by Krudysz and Sioutas.

Aim 2b: Effect of Traffic on Incident Asthma

Although there is a growing literature on studies of prevalent asthma and traffic, few previous studies have examined the relationship of traffic related pollutants with incident asthma. We have examined the effect of traffic modeled exposures on incident asthma in both sets of CHS cohorts. In the older cohorts there was an association of incident asthma with traffic related pollutants at the sample of homes at which NO_2 was measured (Jerrett, et al., 2006). The effects of modeled exposure in the entire cohort were weaker and sensitive to the modeling strategy. Analyses of those data are ongoing. Among children recruited at age 5–7, early follow-up indicates that CALINE-modeled exposure is associated with new onset asthma. Results are stronger among children who had no history of wheeze at study entry (and who were therefore less likely to have pre-existing asthma that was merely exacerbated by exposure). The effects of traffic related exposure are independent of cross community effects of ozone that also were

observed and which are compatible with the previous associations of incident asthma with long term average ozone exposure we have observed in the CHS (McConnell, et al., 2002). A manuscript is in preparation.

Aim 2c: Effect of Traffic on Lung Function and Lung Function Growth

We have shown deficits in community average lung function in the CHS and particulate measurements made at central site monitors. Deficits in flow rates were observed at study entry and in replicated assessments of 4-year lung function growth rates in different cohorts of children (Gauderman, et al., 2002; Gauderman, et al., 2000; Peters, et al., 1999). Recently, we reported deficits in 8-year growth rates (Gauderman, et al., 2004). These findings are part of an emerging literature indicating that traffic related pollutants compromise lung function. However, there has been little study of within-community variability in traffic-related pollution and lung function and no previous study of its association with childhood lung function growth. We have examined the association of lung function deficits at study entry using a novel Bayesian estimator of NO_2 exposure at homes, using both measured and traffic modeled indicators of exposure (Molitor, et al., 2006). We found deficits in both lung flows and lung volumes associated with exposure to NO_2, suggesting a detrimental effect of traffic related pollutants.

We have now incorporated traffic modeled exposure into the evaluation of 8-year lung function growth in the CHS. Effects of traffic modeled exposure were independent of and similar in magnitude to the effects of previously reported regional background particulate pollution (Gauderman, et al., 2004) measured at the central site monitors. Thus, both local traffic and ambient background $PM_{2.5}$ and PM_{10} were associated with impaired lung function. These results are important because impaired lung function in childhood is associated with lung deficits in adulthood. In adults, impaired lung function is a strong predictor of respiratory morbidity and death. A manuscript is in review (Gauderman, et al., 2006).

Aim 2d: School Absence and Traffic

School absences are an outcome not initially proposed for this study, but we have observed strong associations with CALINE4 derived traffic metrics and distance to a freeway. The effects were observed exclusively among children with asthma. A manuscript is in preparation.

Aim 3: Chronic Asthma Exacerbation is Associated With Yearly Variation in Central Site Particulate Measurements

Most of our analyses have examined the effect of spatial variation within communities in pollutants modeled from traffic or measured at a sample of homes. Many other studies have examined acute effects of temporal variation in pollution over days to weeks, but little previous work has examined the effect of temporal variation over longer periods on chronic respiratory outcomes.

We developed novel statistical methods to evaluate the effect of year-to-year temporal variation in the average ambient pollutants measured at the central monitoring sites in each community. The relationship of bronchitic symptoms to ambient particulate matter and to particulate

elemental and organic carbon, nitrogen dioxide (NO_2), and other gaseous pollutants was examined in a cohort of the asthmatic children in the CHS (McConnell, et al., 2003a). Symptoms, assessed yearly by questionnaire from 1996–1999, were associated with the yearly variability of particulate matter with aerodynamic diameter less than 2.5 μm (odds ratio [O.R.] 1.09/μg/m^3; 95% confidence interval [C.I.] 1.01–1.17), OC (O.R. 1.41/μg/m^3; 95% C.I. 1.12–1.78), NO_2 (O.R. 1.07/part per billion (ppb); 95% C.I. 1.02–1.13) and ozone (O.R. 1.06/ppb; 95% C.I. 1.00–1.12). The odds ratios associated with yearly within-community variability in air pollution were larger than the effect of the between-community four-year average concentrations. In two pollutant models, the effects of yearly variation in OC and NO_2 were only modestly reduced by adjusting for other pollutants, the effects of all other pollutants were reduced after adjusting for OC or NO_2. We concluded that previous cross-sectional studies of bronchitic symptoms may have underestimated the risks associated with air pollution. In subsequent analyses, we have observed that the yearly variation in the effect of air pollution in these asthmatic children occurred exclusively among children with a dog (but not a cat) in the home (McConnell, et al., 2003b). We hypothesized that this interaction of pollution with dog ownership could be an indication of up-regulation of asthmatic response to air pollution by exposure to endotoxin, which has been shown in other studies to be present in higher concentration in homes with a dog. A manuscript is under review.

Conclusions

1. Proximity to high traffic corridors and exposure to intra-community variability in traffic-related pollutants was associated with lifetime asthma in cross sectional analyses and with incident asthma during follow-up of the CHS cohorts.

2. Lung function and lung function growth were associated with traffic related air pollution estimated both at the home and at the central site monitor.

3. School absence was associated with traffic related air pollution at home and school among children with asthma.

4. Yearly variation in organic carbon and other particulate pollutants were strongly associated with bronchitis among children with asthma; these effects were modified by a dog in the home, an indicator of endotoxin exposure.

References:

Avol EL, Navidi WC, Colome SD. Modeling ozone levels in and around Southern California homes. *Environmental Science & Technology* 1998;32:463-468.

Benson P. CALINE4--A dispersion model for predicting air pollutant concentrations near roadways. 1989.

Chinkin LR, Coe DL, Funk TH, Hafner HR, Roberts PT, Ryan PA, et al. Weekday versus weekend activity patterns for ozone precursor emissions in California's South Coast Air Basin. *Journal of the Air & Waste Management Association* 2003;53(STI-999679-2225):829-843.

Fitz DR, Bufalino C. Measurement of PM_{10} emission factors from paved roads using on-board particle sensors. In: U.S. Environmental Protection Agency's 11th Annual Emission Inventory Conference, Emission Inventories—Partnering for the Future, 2002.

Gauderman W, Vora H, McConnell R, Berhane K, Gilliland F, Thomas D, et al. The effect of exposure to traffic on lung development from 10 to 18 years of age (submitted, 2006).
.
Gauderman WJ, Avol E, Gilliland F, Vora H, Thomas D, Berhane K, et al. 2004. The effect of air pollution on lung development from 10 to 18 years of age. *The New England Journal of Medicine* 2004;351(11):1057-1067.

Gauderman WJ, Avol E, Lurmann F, Kuenzli N, Gilliland F, Peters J, et al. Childhood asthma and exposure to traffic and nitrogen dioxide. *Epidemiology* 2005;16(6):737-743.

Gauderman WJ, Gilliland GF, Vora H, Avol E, Stram D, McConnell R, et al. Association between air pollution and lung function growth in southern California children: results from a second cohort. *American Journal of Respiratory and Critical Care Medicine* 2002;166(1):76-84.

Gauderman WJ, McConnell R, Gilliland F, London S, Thomas D, Avol E, et al. 2000. Association between air pollution and lung function growth in southern california children. *American Journal of Respiratory and Critical Care Medicine* 2000;162(4 Pt 1):1383-1390.

Gillies JA, Gertler AW. Comparison and evaluation of chemically speciated mobile source $PM_{2.5}$ particulate matter profiles. *Journal of Air & Waste Management Association* 2000;50:1459-1480.

Jerrett M, Shankardass K, Gauderman W, Berhane K, Kunzli N, Lurmann F, et al. Air pollution and asthma incidence in children. *American Journal of Respiratory and Critical Care Medicine* 2006;173A23.

McConnell R, Berhane K, Gilliland F, London SJ, Islam T, Gauderman WJ, et al. 2002. Asthma in exercising children exposed to ozone: a cohort study. Lancet 359(9304):386-391.

McConnell R, Berhane K, Gilliland F, London SJ, Vora H, Avol E, et al. Air pollution and bronchitic symptoms in Southern California children with asthma. *Environmental Health Perspectives* 1999;107(9):757-760.

McConnell R, Berhane K, Gilliland F, Molitor J, Thomas D, Lurmann F, et al. Prospective study of air pollution and bronchitic symptoms in children with asthma. *American Journal of Respiratory and Critical Care Medicine* 2003a;168(7):790-797.

McConnell R, Berhane K, Molitor J, Gilliland FD. Air pollution and bronchitic symptoms in children: Effect modification by dog ownership. *American Journal of Respiratory and Critical Care Medicine* 2003b;167(7):A36.

McConnell R, Berhane K, Yao L, Jerrett M, Lurmann F, Gilliland F, et al. Traffic, susceptibility, and childhood asthma. *Environmental Health Perspectives* 2006a;114(5):766-772.

McConnell R, Berhane K, Yao L, Lurmann FW, Avol E, Peters JM. Predicting residential ozone deficits from nearby traffic. *Science of the Total Environment* 2006b;363(1-3):166-174.

Molitor J, Molitor NT, Jerrett M, McConnell R, Gauderman J, Berhane K, et al. Bayesian modeling of air pollution health effects with missing exposure data. *American Journal of Epidemiology* 2006;164(1):69-76.

Peters JM, Avol E, Gauderman WJ, Linn WS, Navidi W, London SJ, et al. 1999. A study of twelve Southern California communities with differing levels and types of air pollution. II. Effects on pulmonary function. *American Journal of Respiratory and Critical Care Medicine* 1999;159(3):768-775.

Wu J, Funk T, Lurmann F, Winer A. Improving spatial accuracy of roadway networks and geocoded addresses. *Transactions in GIS* 2005a;9(4):585-601.

Wu J, Lurmann F, Winer A, Lu R, Turco R, Funk T. Development of an individual exposure model for application to the Southern California Children's Health Study. *Atmospheric Environment* 2005b;39:259-273.

Zhu Y, Hinds WC, Kim S, Sioutas C. Concentration and size distribution of ultrafine particles near a major highway. *Journal of the Air & Waste Management Association* 2002;52(9):1032-1042.

Supplemental Keywords: NA

Relevant Web Sites: http://www.scpcs.ucla.edu