# Traffic-Related Air Pollution and Asthma in Economically Disadvantaged and High Traffic Density Neighborhoods in Los Angeles County, California

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## ABSTRACT

A growing literature reports traffic exposure impacts on children's respiratory health, yet few U.S. studies have used advanced Geographic Information System modeling techniques to estimate exposures on a fine spatial scale. We developed a land use regression (LUR) model to estimate long-term exposure to traffic air pollution for 1,387 children who participated in the Los Angeles Family and Neighborhood Wave Two Survey (L.A. FANS-2). Using passive badges, we conducted two-week measurements of $NO_x$ and $NO_2$ at approximately 200 sites in two seasons in 65 neighborhoods and built LUR prediction surfaces on a 25 x 25 meter grid over the L.A. Basin that explained 81%, 86% and 85% of the variation in NO, $NO_2$ and $NO_x$ concentrations, respectively. Annual average concentrations at geocoded L.A. FANS-2 residential and school locations were extracted from the LUR surfaces and weighted by time spent at each location for various exposure periods (current home, 1-year, 2-years, 5-years prior to interview). Exposure surfaces for $O_3$ and $PM_{2.5}$ were generated by kriging available government monitoring data.

Multivariate logistic regression was used to estimate associations between these exposure metrics and doctor-diagnosed asthma (ever), wheeze in the past year ("current wheeze"), and medication use for asthma and wheeze in the past year ("current medication use"). Multivariate linear regression was used to estimate associations with cross-sectional measures of lung function assessed via EasyOne$^{TM}$ portable spirometers. Children more highly exposed to traffic pollution as estimated by LUR models for NO, $NO_2$ and $NO_x$ were approximately 30-40% more likely to report current wheeze regardless of adjustment for many family- and neighborhood-level socioeconomic factors. Smaller (15%) increases in odds were observed for current medication use and for doctor-diagnosed asthma (per interquartile (IQR) increase in NO, $NO_2$ and $NO_x$). In stratified analyses by median census tract-level economic disadvantage, odds for both asthma outcomes in higher SES areas only were found to increase by about 40% per IQR increase in traffic pollution. This may, in part, reflect differential access to health care and resulting differences in asthma diagnosis and reporting. In lower SES areas, we estimated 80-100% increases in odds of current wheeze and medication use per 30 ppb increase in peak daily $O_3$, while null or inverse associations emerged for peak $O_3$ in children from higher SES areas, potentially reflecting differences in children's time-activity patterns (e.g., outdoor physical activity) and resulting exposures during $O_3$ peak hours in higher versus lower SES areas. However, these findings are based on a relatively small sample size in each SES stratum.

We estimated 70-100 mL reductions in lung volume and 60-100 mL/s reductions in expiratory flow per IQR increase in NO, $NO_2$ and $NO_x$ in boys with one or more acceptable spirometry curves. Smaller associations were observed for $PM_{2.5}$ (40-50 mL reductions in volume and 60-90 mL/s reductions in flow per IQR increase). However, when restricting analyses to boys with three acceptable and reproducible curves, negative associations were less precisely estimated and did not reach statistical significance, except for $PM_{2.5}$ with $FEF_{75}$ and $FEF_{25-75}$. In girls, we estimated even greater associations between traffic pollution and expiratory flow (300-350 mL/s reductions in PEF and 200-300 mL/s reductions in $FEF_{25-75}$ per IQR increase in NO, $NO_2$ and $NO_x$), but results were not replicated in the group with three acceptable and reproducible curves. We also observed reductions in PEF in girls more highly exposed to peak daily $O_3$ (~100 mL/s decrement and ~400 mL/s decrement per 30 ppb increase in $O_3$ for girls with one or more acceptable curves and three acceptable and reproducible curves, respectively). Similar to previous literature, our results suggest important differences in the biological impact of air pollution on lung function in boys versus girls.

# I. EXECUTIVE SUMMARY

## Background

There is a growing literature reporting traffic exposure impacts on respiratory health in children. Very few studies in the U.S. have used advanced Geographic Information System (GIS) modeling techniques, such as land use regression (LUR), to estimate exposures on a fine spatial scale. LUR models based on intensive neighborhood monitoring of traffic pollutants have not been developed for the Los Angeles (LA) Basin in Southern California, one of most polluted regions in the U.S. There is currently a lack of neighborhood-level air pollution measurements for Californian children that live in high traffic density areas and who may be more susceptible to adverse health impacts from air pollution exposure due to economic disadvantage. Thus, the objectives of this research were to: (1) to conduct $NO_x$ and $NO_2$ monitoring at 200 locations within LA County neighborhoods with varying levels of economic disadvantage and varying exposures to air pollution originating from vehicular sources; (2) to use these monitoring data to help inform LUR models for predicting traffic pollutant exposures (i.e., $NO_x$, NO and $NO_2$); (3) to use geostatistical models to estimate regional background concentrations of $O_3$ and $PM_{2.5}$; (4) to evaluate associations between exposure to $NO_x$, NO and $NO_2$ and measures of respiratory health and lung function in children in conjunction with the Los Angeles Family and Neighborhood Survey (L.A. FANS) study; and (5) to evaluate whether concentrations of the more regionally distributed background pollutants ($O_3$ and $PM_{2.5}$) confound or modify the effects of exposure to the more heterogeneously distributed traffic-related pollutants ($NO_x$, NO and $NO_2$).

## Methods

We developed land use regression (LUR) models to estimate long-term exposure to traffic air pollution for 1,387 children who participated in the Los Angeles Family and Neighborhood Wave Two Survey (L.A. FANS-2) and examined associations with cross-sectional measures of respiratory symptoms and lung function while adjusting for many different family- and neighborhood-level socioeconomic characteristics assessed as part of L.A. FANS-2. First, a campaign of $NO_x$ and $NO_2$ monitoring using passive badges was conducted throughout 65 L.A. FANS neighborhoods (census tracts). Nitrogen oxides ($NO_x$), nitric oxide (NO) and nitrogen dioxide ($NO_2$) were selected as markers of motor vehicle exhaust exposure for this study since they are relatively easy to measure (both from a logistics and cost standpoint), which allowed us to conduct simultaneous measurements at a large number of locations throughout L.A. County. Existing data indicate these pollutants serve as a good marker for localized traffic pollution and are associated with asthma prevalence and symptoms.[1-5] Two-week measurements were collected during two time periods selected to best represent an annual average. These data were then used to build LUR prediction surfaces for NO, $NO_2$ and $NO_x$ on a 25 x 25 meter grid over the L.A. Basin. Geocoded L.A. FANS-2 residential and school locations were overlaid with the exposure surfaces and NO, $NO_2$ and $NO_x$ annual average estimates extracted for each location. Extracted annual averages were weighted by time spent at each home and school within various time periods to generate final exposure metrics (current home, 1-year, 2-years, 5-years prior to interview). Exposure surfaces for $O_3$ and $PM_{2.5}$ were also generated by kriging available government monitoring data for the years 2002 and 2000, respectively. Similar to the LUR metrics, final annual average $O_3$ and $PM_{2.5}$ exposure metrics were then created, weighting for time spent at home(s) and school(s). Multivariate logistic regression was used to evaluate associations between LUR and kriged air pollution estimates and odds of: doctor-diagnosed asthma (ever); wheeze, wheeze with any night waking, medication use for asthma or wheeze,

and sneezing or a runny or blocked nose apart from colds in the past 12 months, and more than 3 doctor-diagnosed ear infections in a year. Multivariate linear regression was used to estimate changes in lung function as assessed via EasyOne$^{TM}$ portable spirometers with increasing air pollution exposure. The specific lung function parameters evaluated were: peak expiratory flow rate (PEF), forced vital capacity (FVC), forced expiratory volume after 1 second ($FEV_1$), forced expiratory mean flow between 25% and 75% of FVC ($FEF_{25-75}$), and forced expiratory mean flow at 75% of FVC ($FEF_{75}$).

**Results**

Final LUR models for the L.A. Basin explained 81%, 86% and 85% of the variation in NO, $NO_2$ and $NO_x$ concentrations, respectively. Novel aspects of the LUR modeling effort include: (1) the use of a large number of sampling sites (~200) for simultaneous passive measurement of NO, $NO_2$ and $NO_x$ over a large and complex geographic region, (2) use of "A Distance Decay REgression Selection Strategy" (ADDRESS)[6] to explore importance of geographic features within many different size buffers and develop spatial models highly predictive of measured concentrations, and (3) use of remote sensing data to provide additional information on geographic distribution of traffic sources and improve LUR model predictions. LUR model results indicated traffic on highways and major roads as far away as 11 km from measurement sites still had important impacts on measured $NO_x$ concentrations, a much greater spatial extent than previously reported in the literature.[7] Although incorporating the influence of traffic at farther distances improved prediction ability for NO, $NO_2$ and $NO_x$, we generated a separate set of LUR models that excluded traffic at distances greater than 5000 m (resulting $R^2$ values were 0.64, 0.78 and 0.68, respectively), as concentrations of ultrafine particles have been shown to reach background concentration within closer proximity to roadways in LA.[8-13] Thus, LUR models excluding traffic at greater distances may provide better surrogate estimates of exposure for fresh vehicle exhaust and UF and associated toxics that are also of biologic interest for respiratory health (even though they were less predictive for NO, $NO_2$ and $NO_x$).

Children more highly exposed to traffic pollution as estimated by interquartile (IQR) increases in the "more local traffic" LUR model estimates for NO (11.8 ppb), $NO_2$ (6.1 ppb) and $NO_x$ (16.9 ppb) were approximately 30-40% more likely to report wheeze in the past 12 months (current wheeze). These estimates were robust to adjustment for many different family- and neighborhood-level socioeconomic factors. We observed weaker and more marginal 15% increases in odds of medication use for asthma and wheeze in the past year and doctor-diagnosed asthma per IQR increase in NO, $NO_2$ and $NO_x$. However, when we stratified analyses by median census tract-level economic disadvantage, odds for both asthma outcomes in higher but not lower SES areas were found to increase by approximately 40% per IQR increase in traffic pollution. This may, in part, reflect differential access to health care and resulting differences in asthma diagnosis and reporting in higher versus lower SES communities. However, in lower SES areas only, we estimated 80-100% increases in odds of current wheeze and medication use for asthma and wheeze per IQR increase in peak 8-hour $O_3$, while null or inverse associations between peak $O_3$ and these outcomes were observed for children living in higher SES areas. These results may in part reflect differences in children's behaviors (e.g., time spent outdoors in summer) and resulting exposures in lower versus higher SES communities during high $O_3$ pollution episodes. However, these findings are based on a relatively small sample size in each SES stratum.

Similar to previous cross-sectional studies in Europe and the U.S., we observed reductions in lung function with increasing exposure to traffic pollution, but our results differed

substantially between girls and boys and varied between children with lower and higher quality spirometry curves. In boys, we estimated 70-100 mL reductions in lung volume and 60-100 mL/s decrements in expiratory flow per IQR increases in LUR estimates of NO, $NO_2$, and $NO_x$. Slightly lower associations were observed for $PM_{2.5}$ exposures (40-50 mL reductions in volume and 60-90 mL/s reductions in flow per IQR increase). However, when restricting analyses to boys with three acceptable and reproducible curves, negative associations were more imprecisely estimated and in general did not reach statistical significance, except for those between $PM_{2.5}$ and $FEF_{75}$ and $FEF_{25-75}$. In girls, we estimated 40-80 mL reductions in $FEV_1$ with increasing exposure to LUR-estimates of NO, $NO_2$ and $NO_x$, but no associations with FVC. However, much greater associations between traffic pollution and measures of expiratory flow were observed in girls versus boys (300-350 mL/s reductions in PEF and 200-300 mL/s reductions in $FEF_{25-75}$ per IQR increase in NO, $NO_2$ and $NO_x$). However, these results were not replicated in the group of girls with three acceptable and reproducible curves. This may be due to the smaller sample size and/or the characteristics of the select group for which we had three reproducible curves available (i.e., girls of higher SES and with higher exposure to $O_3$ and lower exposure to traffic pollutants). Similar to previous U.S. cross-sectional studies,[14-16] we also observed reductions in PEF in girls more highly exposed to peak daily $O_3$ (~100 mL/s decrement and ~400 mL/s decrement per 30 ppb increase in $O_3$ for girls with one or more acceptable curves and three acceptable and reproducible curves, respectively).

**Conclusions**

L.A. FANS-2 children more highly exposed to traffic pollution were more likely reported as having current wheeze symptoms (66% of children with current wheeze were also reported as having a doctor-diagnosis of asthma, 34% did not). We also observed positive associations between LUR traffic exposure metrics and odds of doctor-diagnosed asthma and medication use for asthma and wheeze in the past year, although these associations were not as strong as those estimated for current wheeze. Differences in access to health care and physician practices for diagnosing asthma across communities may be factors affecting our results for these outcomes. This conclusion was supported by analyses in which we stratified on census-tract level economic disadvantage: in higher but not lower SES areas, we observed associations between LUR-estimated traffic exposures and odds of both asthma outcomes similar in magnitude to those observed for current wheeze. Relatively strong associations between exposure to peak daily $O_3$ and current wheeze and medication use for asthma and wheeze were observed in lower SES areas, while no or inverse associations were observed in higher SES areas, which may reflect differences in children's time-activity patterns and resulting exposures across communities during high $O_3$ pollution episodes. We observed reductions in lung function with increasing exposure to traffic pollution, but our results differed substantially between girls and boys and were not consistent for children with poorer versus better quality spirometry curves. Decrements in lung volumes and flows with increasing exposure to traffic pollution were observed in boys, but when restricting to subjects with higher quality spirometry curves (three acceptable and reproducible curves), results were imprecise and associations did not reach statistical significance, except for those between $PM_{2.5}$ and $FEF_{75}$ and $FEF_{25-75}$. For girls, much stronger associations were observed between LUR-estimates of traffic exposure and expiratory flows (PEF and $FEF_{25-75}$) than for boys, however these traffic effects were not replicated in the smaller and select sub-group of girls with three acceptable and reproducible curves. Girls who were more highly exposed to peak daily $O_3$ had substantially lower measures of PEF. Similar to previous

studies, our results suggest important differences in the biological impact of air pollution on lung health in boys versus girls.

## II. INTRODUCTION

### Scope and Purpose

A large literature links outdoor air pollution exposure to adverse respiratory health effects in children and adults.[17-21] Recently, air pollution research has focused on the contributions of specific motor vehicle exhaust components such as polycyclic aromatic hydrocarbons (PAHs) adsorbed to particles from diesel engines and ultrafine particles (less than 0.1 μm in aerodynamic diameter), which are more able to penetrate cellular targets in the lung and enter systemic circulation.[1,22-24] Various measures of traffic exhaust exposure have been associated with adverse respiratory outcomes including reduced lung function and growth, asthma hospitalizations, and prevalence of asthma, wheeze, bronchitis, and allergic rhinitis.[4,25-27] Many of the studies linking pollutants originating from traffic to poorer respiratory health have relied on surrogate exposure measures such as proximity to and extent of traffic on roadways near residences and schools. A relatively new approach for predicting outdoor traffic pollutant concentrations is land use regression (LUR) modeling. Land-use regression utilizes measured levels of the pollutant of interest as the dependent variable and traffic, topographic, and other geographic variables as independent predictor variables in a multivariate regression model.[28,29] The incorporation of site-specific variables in this method detects small area variations of traffic related pollution more effectively than other methods of geostatistical interpolation.[28-30]

European studies associated levels of $PM_{2.5,}$ soot, and $NO_2$ assessed via LUR modeling with the development of adverse respiratory symptoms such as wheezing early in life.[26,31,32] LUR-based estimates of $PM_{2.5}$ were also associated with an increase in exhaled NO (a marker of lung inflammation) and reduced forced vital capacity in Canadian schoolchildren.[33] Overall, there have been very few studies in the U.S. using LUR exposure metrics to examine traffic impacts on respiratory health in children, and no such study has been conducted in the Los Angeles (LA) Basin in Southern California, one of most polluted regions in the U.S. where traffic is a major source of air pollution. Currently, neighborhood level air pollution measurements for Californian children that live in high traffic density areas are lacking and these children may also be more susceptible to adverse health impacts from such exposures due to economic disadvantage. Gunier et al.[34] recently reported that low-income children and children of color in California are more likely to live in census block groups with high traffic density and concluded that future studies should target these high density traffic areas and evaluate differences in health risks by income and race/ethnicity.

Thus, the objectives of this research were to: (1) to conduct $NO_x$ and $NO_2$ monitoring at 200 locations within LA County neighborhoods with varying levels of economic disadvantage and varying exposures to air pollution originating from vehicular sources; (2) to use these monitoring data to help inform land use-based regression (LUR) models developed to predict traffic pollutant – i.e., $NO_x$, NO and $NO_2$ – exposures; (3) to use geostatistical models to estimate regional background concentrations of $O_3$ and $PM_{2.5}$; (4) to evaluate associations between exposure to $NO_x$, NO and $NO_2$ (as estimated by the developed LUR models) and and measures of respiratory health and lung function in children in conjunction with the Los Angeles Family and Neighborhood Survey (L.A. FANS) study;[35] and (5) to evaluate whether concentrations of the more regionally distributed background pollutants ($O_3$ and $PM_{2.5}$) confound or modify the effects of exposure to the more heterogeneously distributed traffic-related pollutants ($NO_x$, NO and $NO_2$).

## Traffic-Related Air Pollution Impacts on Children's Respiratory Health

### *Traffic Impacts on Childhood Lung Function*

Most existing studies examining effects of traffic-related air pollution on lung function in children were cross-sectional, conducted in Europe, and used traffic exposure metrics based on residential and/or school proximity to high traffic roadways. Most of the work done in the U.S. has relied on ambient monitoring data to estimate air pollution exposure. A study in Canada[33] examined associations between LUR exposure metrics and lung function; no similar study has been conducted in the Los Angeles Basin in Southern California. Here we summarize results from both cross-sectional and longitudinal studies of air pollution impacts on lung function in childhood.

*Cross-Sectional Studies*

Most studies examining traffic air pollution impacts on cross-sectional measures of lung function in children were conducted in Europe. Wjst et al.[36] reported significant associations between traffic density in school districts and expiratory flow measures in 4,320 German schoolchildren ages 9-11 years. Fritz and Herbath[37] similarly reported lower lung function in German preschoolers (5 years of age) living in areas with traffic-related pollution profiles. Studying the impact of German reunification using serial cross-sectional assessments of respiratory health, Sugiri et al.[38] reported that improvement in lung function with decreasing TSP and $SO_2$ levels among 2,574 east German 6 year olds was weaker in children living within 50 m of a busy street and this finding was attributed to the 50-75% increase in traffic during this period in eastern Germany. In the Netherlands, Brunekreef et al.[39] reported negative impacts of truck traffic density on several lung function indicators ($FEV_1$, PEF, $FEF_{25-75}$) ranging between 2.5% and 8% reductions in function per 10,000 trucks/day for children residing within 1000 m of a motorway. Black smoke, $NO_2$ and car traffic density tended to show similarly negative associations with $FEF_{25-75}$. A second, larger follow-up study did not find similar associations between truck or car traffic density, or school or residential proximity to traffic and lung function, although associations were still observed for respiratory symptoms.[40] In a novel approach, Hogervorst et al.[41] measured oxygen-radical formation by particles as a marker for potential to cause oxidative stress, which is one of the hypothesized pathways by which traffic particles may induce lung inflammation. Exposure to air pollution exhibiting higher radical formation per particle mass – and to a lesser extent radical formation per volume of air – reduced lung function among Dutch children attending schools located at varying distances from traffic. However, only 4 days of measurements at schools during the study period were collected and assumed to represent long-term exposure. Also, contrary to expectation, increasing $PM_{10}$ levels were found to be positively associated with $FEV_1$ and FVC. Other European studies reported no associations between various measures of traffic near homes and schools and lung function measures.[42,43]

In a recent Canadian study, Dales et al.[33] reported reductions in $FEV_1$ (expressed as a percentage of predicted) with increases in neighborhood-level $SO_2$, $PM_{2.5}$ and black smoke concentrations modeled via LUR as well as reductions in FVC for $SO_2$ and $PM_{2.5}$ in 2,328 children ages 9-11 years living in Windsor, Ontario (although none of these estimates reached traditional statistical significance). Non-significant reductions in lung function were also observed with increasing length of roadways within 200 m of the home. Rosenlund et al.[44] reported deficits in $FEV_1$ as a percentage of FVC, $FEF_{25-75}$ and PEF with increasing exposure to LUR-modeled $NO_2$ at residences in a cross-sectional, school-based study of 2,107 children ages

9-14 years living in Rome, Italy. Associations appeared to be stronger in girls, older children, children of higher SES and those exposed to parental smoking.

Most of the U.S. studies used ambient air monitoring data to estimate exposures to routinely measured, criteria air pollutants. A number of these studies reported reductions in lung function with increases in exposure to pollutants that could potentially be acting as (imperfect) markers of motor vehicle exhaust exposures, specifically TSP and $NO_2$,[45] $NO_2$ and particle acidity (a marker of very small particles),[46] and $PM_{10}$, $PM_{2.5}$, $NO_2$ and acid vapor.[14] Early results from the Harvard Six Cities Study[47] showed no associations between exposure to TSP, $PM_{10}$ and $PM_{2.5}$ and $FEV_1$, FVC, and MMEF for a sample of 5,422 10-12 year olds. Yet, a second follow-up study of 24 cities and over 13,000 children ages 8-12 years found that as $PM_{2.1}$ and sulfate particle concentrations increased, so did the percentage of children with abnormal lung function.[46] More recently, Mortimer et al.[48] reported that asthmatic children living in the San Joaquin Valley of California, who had been exposed to higher levels of CO, $NO_2$ and $PM_{10}$ – interpreted as markers of motor vehicle exhaust – *in utero* during early pregnancy (1st-2nd trimesters) as well as in the first 6 years of life had lower lung function at ages 6-11 years. These effects were, however, limited to African-Americans, children diagnosed with asthma before age 2 yrs and children exposed to maternal smoking during pregnancy. Using dispersion modeling to predict outdoor residential concentrations, Oftedal et al.[49] reported that early and lifetime exposures to $PM_{10}$, $PM_{2.5}$ and $NO_2$ were associated with reduced forced expiratory flows, but not forced expiratory volumes in 2,307 9-10 year old children that had lived in Oslo, Norway since birth.

Apart from traffic-related air pollution, a number of studies also linked $O_3$, a more regionally distributed secondary pollutant, to worse lung health in children. Kuenzli et al.[15] reported that higher lifetime exposure to $O_3$ negatively affected flow measures but not $FEV_1$ and FVC in U.S. college freshman ages 17 to 21; there were no associations found for $PM_{10}$ and $NO_2$. In a follow-up study, Tager et al.[16] reported negative associations between flows and lifetime $O_3$ exposure in subjects with a low $FEF_{25-75}$/FVC ratio, a marker of narrower small airways. Galizia and Kinney[50] observed lower lung function among U.S. male college freshman who grew up in counties with high long-term $O_3$ levels compared to those who grew up in low $O_3$ counties. Also, some of the studies cited above for showing traffic-related associations, also reported associations with $O_3$.[14,45,46] It should be noted that $PM_{10}$ and $PM_{2.5}$ are complex mixtures of particles in different size ranges and with different chemical characteristics. The contribution of direct traffic particle emissions to these mixtures is highly complex and depends on source profiles, location and season.[51,52] Thus, the existing evidence based on these cross-sectional studies does not allow conclusions as to whether mainly traffic-related air pollutants or regionally distributed pollutants (i.e., $O_3$ and secondarily formed particles), or both types of pollutants together impact lung function in children.

*Longitudinal Studies*

The Children's Health Study (CHS) – which focused on asthma and lung development in 4th through 10th graders living in 12 Southern Californian communities – is one of the largest and most comprehensive studies of the long-term consequences of air pollution exposure on children's respiratory health.[1] Based on a follow-up of 1,759 children from age 10 to 18 years (n=747 at last follow-up), children living in the most polluted community (i.e., highest levels of $NO_2$, $PM_{10}$, $PM_{2.5}$, acid vapor and elemental carbon) had a growth deficit in $FEV_1$ of approximately 100 mL (~7% for girls and ~4% for boys) as compared to children living in the

cleanest community.[53-55] Concentrations of these pollutants were highly correlated in all communities (r values ranging from 0.64-0.97) and considered to represent a mixture of traffic-origin pollutants. The proportion of children with clinically low lung function at age 18 ($FEV_1$<80% of predicted) was estimated to be 5 times larger in the most polluted community compared with the cleanest community (29 $\mu g/m^3$ versus 6 $\mu g/m^3$ $PM_{2.5}$.[56] No associations were observed between $O_3$ and changes in lung function over the 8-year period. Based on an analysis of a sub-sample of 110 children who moved after the initial CHS examination, Avol et al.[57] reported an improvement in lung function growth among children who moved to lower $PM_{10}$ areas, and slower lung function growth in children who moved to higher $PM_{10}$ areas. More recently, Gauderman et al.[58] reported significant deficits in 8-year growth of $FEV_1$ (-81 mL) and MMEF (-127 mL/s) in children living within 500 m of a freeway compared those living at least 1500 m away from a freeway. Joint models showed that both residential proximity to freeways as well as regional air pollution (i.e., central site measures of $NO_2$, $PM_{10}$, $PM_{2.5}$ EC, and acid vapors) had detrimental and independent estimated effects on lung function growth.

Outside of the U.S., in another major prospective study, Rojas-Martinez et al.[59] examined lung function growth in Mexico City schoolchildren over a 3 year period. Long-term exposures to $O_3$, $PM_{10}$ and $NO_2$, as assessed by monitors located <2 km from schools, were associated with deficits in lung function growth, even when adjusting for other pollutants. Studies in Austrian school children reported negative impacts of $O_3$ and to a lesser degree $PM_{10}$ on lung function growth during summer, however these deficits were compensated for during the winter seasons and over the 3.5 years of study, no overall deficits in lung function growth among children living in more polluted areas were observed.[60-63] Based on a follow-up study of 200 Austrian children, Neuberger et al.[64] reported small improvements in lung function which they attributed to a decrease in ambient $NO_2$ levels over 5 years.

*Summary of Studies on Childhood Lung Function*

Existing studies provide evidence for air pollution from traffic related sources negatively impacting lung function in children. Findings from the CHS study indicate both regional and local traffic-related pollutants contribute to lung function deficits in Southern Californian children, a finding further corroborated by a study conducted in Mexico City. However, there is very limited research examining potential interactions between socioeconomic and other contextual factors and traffic-related air pollution on lung function in children.

***Traffic Impacts on Respiratory Symptoms in Childhood***

Throughout the 1990's, a growing number of studies reported associations between various traffic exposure metrics and poorer childhood respiratory health. Although these mostly European studies differed in which respiratory endpoints and traffic exposure measures they evaluated, in general, they reported positive associations between residential and/or school proximity to heavy traffic roadways and adverse respiratory outcomes including asthma hospitalizations, and prevalence of asthma, wheeze, bronchitis, and allergic rhinitis.[36,42,65-73] Furthermore, traffic pollutant concentrations at homes and schools derived from sophisticated emissions and air dispersion models were linked to a greater number of hospitalizations for wheezing bronchitis in girls,[74] prevalence of asthma, wheeze and cough,[43] and respiratory medication use.[75]

The Dutch researchers cited above[39] also reported associations between residential proximity to major freeways and chronic respiratory symptoms assessed cross-sectionally via

questionnaires in 1,068 Dutch schoolchildren.[71] Cough, wheeze, runny nose, and doctor-diagnosed asthma were more often reported for children living within 100 meters of a freeway. Truck traffic intensity and the concentration of black smoke measured at schools were also found to be associated with these outcomes, with relationships more pronounced in girls than boys. In a larger follow-up study, respiratory symptoms including current wheeze, ever asthma, current conjunctivitis, current itchy rash, eczema ever, current phlegm (with no cold) and current bronchitis were found to also be positively associated with the level of truck traffic, but not with car traffic on major freeways within 400 meters of schools and 1000 meters of homes.[40] Positive associations were almost entirely restricted to children with bronchial hyper-responsiveness and/or sensitization to common allergens. Symptoms also increased with increasing levels of traffic-related air pollutants ($PM_{2.5}$, soot and $NO_2$) measured at schools; levels of these pollutants also increased near freeways with high truck traffic counts.

Several studies assessed exposure to traffic exhaust pollutants using residence-based and/or personal $NO_2$ measurements or ambient measures in non-urban areas where traffic is the main source of air pollution. In a Swiss study, the incidence of upper respiratory symptoms increased with increasing residential levels of outdoor but not indoor $NO_2$ (measured by Palmes tubes) in preschoolers.[76] Additionally, annual average $NO_2$ was associated with the duration of all respiratory episodes and with upper respiratory episodes. The investigators suggested that the lack of association between indoor $NO_2$ and some of the outcomes might indicate that outdoor but not indoor sources of $NO_2$ represent the relevant exposures originating from outdoor sources, i.e. motor vehicle exhaust. Studnicka et al.[77] reported associations between prevalence of "ever asthma", wheeze, and cough apart from colds and three-year mean concentrations of $NO_2$ in eight non-urban communities where traffic was the only source of air pollution. In a study of nine year olds living near major roads in two urban areas and one suburban area of a West German city, Kramer et al.[78] reported associations between outdoor $NO_2$ (measured by Palmes tubes at children's residences) and atopy (assessed by respiratory symptom diaries, skin-prick tests and allergen-specific serum IgE). Outdoor $NO_2$, but not personal $NO_2$, was related to hay fever, symptoms of allergic rhinitis, wheezing, and sensitization against pollen, house dust mites or cats, and milk or eggs in urban areas. Outdoor $NO_2$ concentrations correlated fairly well with traffic levels outside the children's homes ($r=0.70$), again suggesting the relevance of traffic-related air pollutants for these outcomes and the usefulness of $NO_2$ as a marker of vehicle exhaust exposure.

In addition to their landmark studies that associated air pollution and development of lung function, the Children's Health Study (CHS) also examined the influence of air pollution on respiratory symptoms including asthma and bronchitis in Southern California schoolchildren.[1] In the first cross-sectional examination of 3,676 $4^{th}$, $7^{th}$ and $10^{th}$ graders, higher exposure to acid vapor ($HNO_3$+HCl) and $NO_2$ (as measured by central site monitors in each community) were associated with greater odds of wheeze in boys. A subsequent cross-sectional study reported greater prevalence of chronic phlegm production and bronchitis in asthmatic children living in communities with higher levels of ambient $PM_{10}$; $NO_2$ and acid were also associated with increased phlegm prevalence in asthmatics.[79] Based on analyses of data collected prospectively from 1996 to 1999, bronchitic symptoms in asthmatics were associated with yearly variability in $PM_{2.5}$, organic carbon (OC), $NO_2$, and $O_3$.[80] Organic carbon was of interest due to its potential to elicit oxidative stress responses that potentially could be important for asthma exacerbation.[22] Odds ratios for yearly within-community variability in air pollution were larger than those for between-community 4-year average concentrations and the most stable effect estimates in multi-

pollutant models were for OC and $NO_2$. In the CHS communities, $NO_2$, acid vapor, and the particulate matter pollutants ($PM_{10}$, $PM_{2.5}$, OC, elemental carbon (EC)), are correlated with each other and considered markers of vehicular sources.[55]

Following up on the potential importance of assessing within-community variability in traffic pollution levels, Gauderman et al.[2] measured outdoor $NO_2$ during summer and winter outside the homes of 208 CHS children. They also determined residential distance to the nearest freeway, traffic volumes on roadways within 150 m of the home, and modeled pollution from nearby roadways using the CALINE air dispersion model. Lifetime history of doctor-diagnosed asthma, wheezing and asthma medication use were associated with outdoor $NO_2$, closer residential proximity to a freeway, and CALINE model-based estimates of pollution from freeways. However, associations for these outcomes were not observed with modeled pollution for non-freeway roads and traffic volumes within 150 meters of homes. The authors suggested that this might be due to the low relative importance of emissions from smaller roadways compared to large freeway arterials in LA and/or more accurate measures of traffic on freeways versus local roads. McConnell et al.[3] later examined associations between asthma and wheeze and traffic pollution in a new cohort of children ages 5-7 years attending 13 schools in Southern California (9 communities were the same as in the original CHS, 4 were new). Children residing within 75 m of a major road (defined as freeways, other highways, and arterial roads) had higher odds of lifetime asthma, prevalent asthma, and wheeze. These effects appeared to be greater for long-term residents (i.e., living at same residence since at least 2 years of age) with no parental history of asthma. The higher risk of asthma near major roadways decreased to background rates at 150-200 m. Similar associations were observed for model-based traffic exposure metrics. Since very few children in the study lived within 75 m of a freeway, associations with asthma and wheeze were due to proximity to non-freeway roads. This discrepancy with the previous study[2] that had found associations only for proximity to freeway traffic, may reflect differences in the distribution of freeways and major roads around homes in these different cohorts. More recently, Jerrett et al.[81] extended the work by Gauderman et al.[2] by assessing associations between incident asthma and $NO_2$ measured outside children's homes for 217 CHS participants. They excluded children with a lifetime history of asthma at study entry, included an $11^{th}$ community (Lompoc) with local traffic but no major freeways, and excluded a $12^{th}$ community (Lake Arrowhead) with little local traffic. Incident asthma cases were defined as children who answered yes to doctor-diagnosed asthma on any annual interview during up to 8 years of follow-up. Incident asthma was positively associated with annual residential $NO_2$. In a multilevel model, the within-community effects indicative of long-term local traffic sources were similar in magnitude to effects of community-average $NO_2$ across communities, suggesting that both regional and local pollution contributed to the associations seen with new onset asthma.

In another California school-based study, Kim et al.[4] evaluated associations between asthma and bronchitis and exposure to traffic pollutants (black carbon (BC), $NO_x$, $NO_2$, $PM_{2.5}$ and $PM_{10}$) in $3^{rd}$ to $5^{th}$ graders at 10 schools in the San Francisco Bay Area, a region with high traffic congestion, but generally good regional air quality due to coastal breezes. Concentrations of BC, $NO_x$, NO and to a lesser extent $NO_2$ were higher at schools located within 300m downwind of a freeway compared to those measured at schools upwind or further removed from major traffic sources. There was less variation in $PM_{2.5}$ and $PM_{10}$ concentrations across schools and the study average $PM_{2.5}$ was similar to the annual average $PM_{2.5}$ concentration measured at the government monitoring station located about 15 miles south of the study area. $NO_x$ and $NO_2$ concentrations at schools far from traffic (>1,000 m) were also similar to those measured at the

government site. Odds of a doctor-diagnosis of asthma in the preceding 12 months increased with interquartile increases in school levels of $NO_x$, NO and BC for children who lived in the same home for at least one year. School-based pollutant concentrations were assumed more reliable estimates of overall exposure for these children since most children in the study lived within walking distance of the schools and did not use buses. In a follow-up study, Kim et al.[5] sought to refine exposure estimates using GIS-derived traffic measures at the children's residences. Positive associations were observed between several different traffic exposure metrics and odds of current asthma (i.e., asthma episode in the previous 12 months). The most consistent exposure-response relation was observed for the metric traffic density within 150 m of the home with an approximately 2-fold increase in odds in the highest quintile of exposure. The highest odds were observed among those living within 75 m of a freeway/highway. Distance to freeway/highway of 150 m or less was most strongly correlated with measurements of NO, $NO_2$ and $NO_x$ taken at 52 sites (schools and homes) in the study area.

While observing associations between residential LUR estimates for $NO_2$ and reduced lung function (particularly expiratory flows) in Italian schoolchildren, Rosenlund et al.[44] did not find similar associations between LUR exposure estimates and prevalence of respiratory symptoms or allergic sensitization assessed by skin prick tests.

There is also a growing literature reporting traffic impacts on respiratory health during the first years of life. As part of the "Traffic-Related Air Pollution and Childhood Asthma" (TRAPCA) study in Europe, exposure to $NO_2$ assessed via LUR modeling was associated with dry cough at night and bronchitis in the first year of life, and LUR-derived measures of $PM_{2.5}$ and soot (determined as the reflectance of the $PM_{2.5}$ filters) with sneezing, runny/stuffed nose in the first two years of life in infants residing in Munich, Germany.[31,32] In the Netherlands, LUR measures of $PM_{2.5}$, soot and $NO_2$ were linked to wheezing, doctor-diagnosed asthma, ear/nose/throat infections and flu/serious colds at age four.[26] In Sweden, higher exposure to traffic-origin $NO_x$ and $PM_{2.5}$ during the first year of life (assessed via emission inventories and air dispersion modeling) was associated with persistent wheeze, lower peak expiratory flow and sensitization to pollen at four years of age.[82] As part of the on-going Cincinnati Childhood Allergy and Air Pollution prospective birth cohort study, Ryan et al.[83] created a LUR model of elemental carbon (EC) attributable to traffic sources (ECAT). ECAT levels at monitoring sites throughout the Cincinnati airshed were estimated based on speciation of $PM_{2.5}$ samples and multivariate receptor and chemical mass balance models. ECAT was considered a marker of diesel exhaust particulate (DEP). Infants residing at homes with higher LUR estimates for ECAT were more likely to be reported as suffering from recurrent wheeze during the first year of life (i.e., parent reported wheezing without a cold at approximately 6-7 months of age and at least one other occasion before the first birthday).

A number of European studies have reported associations between exposure to traffic air pollution children and allergic sensitization. Duhme et al.[68,69] reported associations between self-reported traffic near residences and odds of allergy symptoms in German children 5-15 years of age as assessed by the ISAAC definition "sneezing, or runny/blocked nose apart from colds in the previous 12 months". Weiland et al.[67] also reported positive associations between self-reported traffic near homes and ever having hay fever or allergic rhinitis in German seventh- and eighth- graders based on questionnaire reports. More recently, Morgenstern et al.[32] reported associations between sneezing, runny/blocked nose apart from colds and exposure to LUR estimates of $PM_{2.5}$ in Munich children during the first and second year of life. In a follow-up of the same children at age six years, the positive associations between LUR estimates of $PM_{2.5}$ and

soot and sensitization to inhalant allergens and pollen as assessed by IgE antibody levels persisted.[84] For the variable ‘‘living close to main roads,’’ a clear dose–response was identified with the highest effect estimates for sensitization in children living less than 50 m from busy streets. Similarly, Nordling et al.[82] also reported greater odds of allergic sensitization to pollen at age 4 as assessed by IgE levels in Swedish children more highly exposed to traffic pollution during the first year of life. However, a similar study of Dutch children found increased odds of sensitization to food allergens but not total IgE at age four for children more highly exposed to traffic pollution as assessed by LUR models.[26] Rosenlund et al.[44] reported no associations between LUR estimates of $NO_2$ exposure and allergic sensitization based on skin prick tests in Italian children at the ages of 9-14 years.

Although exposure to environmental tobacco smoke is a known risk factor for otitis media, little information is available regarding potential associations with air pollution.[85,86] As part of the TRAPCA study in Europe, Brauer et al.[85] examined associations between exposure to traffic pollutants and physician diagnosis of otitis media (ear infections) in the first two years of life for children residing in the Netherlands (n=3,700) and Munich (n=650). Children more highly exposed to traffic, as assessed by LUR model estimates of $NO_2$, $PM_{2.5}$ and soot, were 10-24% more likely to report physician diagnosed ear infections in the first two years of life.

*Summary of Studies on Childhood Respiratory Health*

Research conducted over the last 10 years provides compelling evidence of traffic-related air pollution impacts on respiratory health in Californian children. Results from the CHS suggest traffic pollutants can not only exacerbate symptoms in asthmatics, but that traffic pollution also causes new onset asthma and that, similar to lung function, both local and regional pollution are important (at least in Southern California). Studies associating early life exposures to reports of wheeze and allergic sensitization in children as young as one year old provide further evidence of traffic pollutant’s possible early life impact on the development of respiratory disease and asthma.

**Potential Confounding and/or Modifying Effects of Psychosocial Stress and Neighborhood Environment on Associations between Air Pollution and Respiratory Health**

Although there is growing evidence indicating traffic pollution increases incidence of wheeze and asthma in addition to exacerbating existing disease, the etiology is complex with multiple contributing factors.[87] In addition to genetic propensity, aspects of both the social and physical environment are likely important in asthma causation and progression. Reports of higher asthma morbidity in low socioeconomic status (SES) neighborhoods might reflect independent main effects as well as the interplay between social and physical aspects of the community.[88,89] Outdoor air pollution is one physical neighborhood factor that can impact asthma, and there is evidence that economically disadvantaged neighborhoods are often more exposed to air pollution.[34,90,91] Low SES neighborhoods’ influence on asthma morbidity may also reflect differences in access to health care, health behaviors such as diet and smoking, and other aspects of the social environment. For example, neighborhood factors, such as economic disadvantage, violence, low social cohesion, and low social capital may act through stress pathways to worsen asthma outcomes.[88] Higher levels of psychosocial stress have been linked to greater morbidity in asthmatic children,[92-95] and there is growing evidence from prospective studies that psychosocial stress may contribute to the development of wheezing illnesses and asthma, especially in early life.[88,96-98] Recently, Suglia et al.[99] reported reductions in $FEV_1$ and

FVC among girls ages 6-7 years with high exposure to parental verbal aggression, independent of SES, tobacco smoke exposure, birth weight, and a history of respiratory illness. In boys, high exposure to community violence was found to be associated with reduced lung function.

A postulated biologic mechanism by which psychological stress may cause or worsen asthma is through chronic inflammation pathways. Imbalance of physiologic processes that lead to chronic inflammation (e.g., hypothalamic-pituitary-adrenal (HPA) axis function, the proteases-antiproteases, the oxidants-antioxidants) can occur under chronic stress.[93,99] Stress is associated with increased secretion of adrenocorticotrophic hormone and cortisol, which have been observed to enhance the production of inflammatory cytokines;[98,100] altered cortisol expression has been negatively associated with lung function in humans.[101] Although high levels of stress-related hormones can acutely suppress inflammation in the airways, receptors for these molecules can become down-regulated under chronic exposure, leading to diminished regulation of inflammatory responses to asthma triggers.[98,102]

Two studies examined potential interactive effects between chronic stressors and air pollution on asthma. Using a LUR model of $NO_2$ to estimate traffic air pollution exposure among 413 children living in East Boston, Massachusetts, Clougherty et al.[103] reported an elevated risk of asthma with increasing levels of $NO_2$ only among children with above-median lifetime exposure to violence. Among children always living in the same community, this association was magnified, furthermore $NO_2$ levels during the year of diagnosis were most predictive of asthma outcomes. Chen et al.[104] examined interactions between chronic exposure to traffic air pollution and family stress in predicting biologic and clinical outcomes in 73 asthmatic children ages 9-18 years residing in Vancouver, Canada. Exposure was assessed via a LUR model of $NO_2$ concentrations. Children were interviewed about life stress and asthma-relevant inflammatory markers (cytokine production, immunoglobulin E (IgE), eosinophil counts) were measured. They also completed daily symptom diaries and performed PEF measures at baseline and at six months of follow-up. Parents also reported on their children's symptoms. Contrary to Clougherty et al.[103] who reported greatest risk of asthma among children exposed to both high pollution and violence, in this study, higher chronic stress was associated with heightened inflammatory profiles (i.e., higher interleukin-5, IgE, and eosinophil counts) as traffic pollution decreased. In other words, the detrimental effects of chronic psychosocial stress were more evident among children living in lower pollution areas.[104] To explain these findings, the investigators hypothesized a threshold above which chronic physical exposures such as air pollution begins to have effects on health outcomes, and that one role of chronic stress may be to lower the threshold at which physical exposures affect biologic and clinical outcomes. One reason why this may occur is that when chronic exposure to traffic-related air pollutants is more modest, there may be greater room for social factors to increase or decrease vulnerability biologically. Longitudinally, higher chronic stress was also associated with increases over time in symptoms (as recorded in diaries and by parent report) and decreases in PEF, but again only in lower pollution areas.

Thus, it has been argued that in order to adequately evaluate the contributions of the physical environment to health outcomes such as asthma, it is important to consider social aspects not only as potential confounders but also as effect measure modifiers.[90] Similar to L.A. FANS-1,[35] the L.A. FANS-2 survey collected extensive data on individual, family, and neighborhood characteristics of study participants, allowing us to evaluate associations between traffic air pollution and respiratory health taking into account both physical and social aspects of neighborhoods. While most previous studies examining this issue relied solely on SES measures mainly derived from administrative data sources (such as census data) to assess adverse social

conditions, the L.A. FANS study collected information directly from participants such as ratings of neighborhood safety, cohesion and social support, thus providing us with additional and detailed measures of the neighborhood social environment to be considered in our analyses of air pollution's impact on respiratory health.

**L.A. FANS Background**

The Los Angeles Family and Neighborhood Survey (L.A. FANS) is a longitudinal study of families in Los Angeles County and of the neighborhoods in which they live. The study is specifically designed to answer key research and policy questions in several areas, with a focus on understanding neighborhood, family, and peer effects on children's development and well-being.[35] The first wave of data collection (L.A. FANS-1) was a field survey of 3,090 households conducted from April 2000 to January 2002. L.A. FANS-2 is a continuation of this study and is funded by grants from NICHD, NIA and NIEHS. It is a collaboration of three institutions: RAND, UCLA, and Research Triangle Institute (RTI). The design of L.A. FANS-2 included re-interviewing all respondents from L.A. FANS-1 and adding a new sample of residents who moved into each neighborhood between the two waves. L.A. FANS-2 was also expanded to collect biomarkers of stress and health for children and adults of all ages.

Since one of the main goals of L.A. FANS is to provide a rich dataset for examining the effects of neighborhoods and families on children's development and well-being, a multistage method was used to select subjects for L.A. FANS-1, with stratified random sampling first of census tracts (which is the definition of neighborhoods for the study), followed by census blocks, families, and finally adults and children within selected families. Census tracts in LA County were broken down into three strata based on the percent of the tract population living below the poverty line. Very poor tracts were defined as those in the top 10% of the poverty distribution, poor tracts were those in the 60-89$^{th}$ percentile of the poverty distribution and non-poor tracts were those at or below the 59$^{th}$ percentile of the poverty distribution.[35] Figure 1 shows LA County census tracts by poverty ranking. Since an approximately equal number of tracts in each stratum were selected, the very poor and poor tracts were over sampled. Also, households with children under age of 18 years were over sampled since a key focus of the study is on children's health. Among those L.A. FANS-1 children who lived in the LA Basin (n=3,017), 32.6% lived in "very poor" tracts, 33.9% lived in "poor" tracts", and 33.5% lived in "non poor" tracts. Within each household, one adult (18 years or older) was randomly selected for interview, as was one child (17 years or younger). Only children ages 9 years or older were directly interviewed. The primary caregiver of the randomly selected child (usually the child's mother) was also interviewed about the child (regardless of the child's age) and if the randomly selected child had one or more siblings with the same biological or adoptive mother and the same primary caregiver, one of the siblings was also randomly selected for interview. L.A. FANS-1 included 3,090 households in 65 census tracts (with 30%, 31% and 39% of the households in the very poor, poor and nonpoor strata, respectively). The children surveyed as part of L.A. FANS-1 were predominately Latino (59.1%), but also included African Americans (8.3%), Asians (6.1%), Pacific Islanders (0.7%), Native Americans (1.4%) and children reporting mixed races/ethnicities (4.1%); 20.2% of the children were identified as White.

The L.A. FANS-2 survey was conducted between August 2006 and November 2008. The design of L.A. FANS-2 included: (1) re-interviewing all L.A. FANS-1 respondents, both adults and children, even if they moved out of the neighborhood, (2) re-interviewing all L.A. FANS-1

respondents who remained in the neighborhood, and (3) interviewing a sample of new entrants into the neighborhood. Those individuals who participated in both L.A. FANS-1 and L.A. FANS-2 are called "panel" respondents. Personal interviews were done with L.A. FANS-1 respondents still living somewhere in Los Angeles County, while new entrants had to reside in the 65 original L.A. FANS-1 census tracts in LA county. If the panel respondent had moved out of LA County, an abbreviated telephone interview was performed (and no health measures were collected). Our analyses include respondents residing in LA County. In L.A. FANS-1, Primary Care Givers (PCGs) were asked whether child respondents were ever diagnosed with asthma by a doctor or other health professional, and if yes, whether they had one or more asthma attacks in the previous year. In L.A. FANS-2, the assessment of asthma was modified to ask about wheeze and medication use for asthma and wheeze, regardless of doctor-diagnosis of asthma (based on the International Study of Asthma and Allergies in Childhood (ISAAC) questionnaire).

In addition, a battery of physiological measurements were added to L.A. FANS-2: anthropometry (height and weight) in children ages 2 years and older, glycosylated hemoglobin, and total and HDL cholesterol via dried blood spots and cortisol in saliva samples collected on the day after the interview upon waking, 30 minutes after waking, and at bedtime in children ages 3 years and older; and lung function using portable spirometers and blood pressure in children ages 5 years and older.

A total of 939 families with children were interviewed as part of L.A. FANS-2; parents in these households completed questionnaires regarding 1,387 children. Of these children, 1,091 were panel participants (i.e., participated in both surveys) while 296 were new entrants to the study. Of the 1,387 interviewed children, 1,225 also completed some part of the health measures module and 1,070 participated in spirometry. Originally, the L.A. FANS-2 study planned to enroll approximately 4,000 children (all of the L.A. FANS-1 children plus a sample of new entrants into each neighborhood). Despite extending data collection by approximately 1.5 years, the total number of L.A. FANS-2 child participants was approximately 35% of the originally planned enrollment number. This limitation is discussed further in the Discussion section.

## III. Materials and Methods

### GIS Exposure Model Development

We estimated long-term exposure to traffic-related air pollution for L.A. FANS-2 participants using land use based regression (LUR) modeling. First, a campaign of $NO_x$ and $NO_2$ monitoring using passive badges was conducted throughout the 65 LA FANS Wave One neighborhoods (census tracts). Nitrogen oxides ($NO_x$), NO (which can be assessed as the difference in measured $NO_x$ and $NO_2$ concentrations, and $NO_2$ were selected as markers of motor vehicle exhaust exposure for this study since they are relatively easy to measure (both from a logistics and cost standpoint) and allowed us to conduct simultaneous measurements at a large number of locations throughout L.A. County. Existing data indicate these pollutants serves as a good marker for localized traffic pollution and are associated with asthma prevalence and symptoms.[1-5] Two-week measurements were collected during two time periods selected to best represent an annual average. These data were then used to build LUR prediction surfaces for NO, $NO_2$ and $NO_x$ on a 25 x 25 meter grid over the LA Basin. Geocoded L.A. FANS-2 residential locations were overlaid with the exposure surfaces and NO, $NO_2$ and $NO_x$ annual average estimates extracted for each location. Extracted annual averages were weighted by time spent at each home within various time periods to generate final exposure metrics (1-year, 2-years, 5-years prior to interview). Exposure surfaces for $O_3$ and $PM_{2.5}$ were also generated by kriging

available government monitoring data for the years 2002 and 2000, respectively. Similar to the LUR metrics, final estimates for annual average $O_3$ and $PM_{2.5}$ exposure were then created, weighting for time spent at home(s).

### *$NO_X$ Neighborhood Monitoring*

*Sampling location determination*

We selected neighborhood $NO_x$ and $NO_2$ monitoring locations (n = 201) using a location-allocation algorithm that took into account variability in traffic pollution and the spatial distribution of the Los Angeles Family and Neighborhood Study (L.A. FANS) participants. Kanaroglou et al.[105] provides a detailed discussion of the location-allocation methodology. The estimation domain for locating optimal sampling sites for this project covered more than 10,000 $km^2$ of the L.A. Basin (Figure 2). Briefly, the location-allocation algorithm involves a two-step algorithm that: (1) builds a demand surface of spatial variation (i.e., semi-variances) and (2) solves a constrained spatial optimization problem to determine locations for a pre-specified number of samplers. The demand surface was created using two criteria: first, samplers should be placed where the pollution surface is expected to exhibit high spatial variability and second, population density should be relatively high. To create an initial pollution surface across the L.A. metropolitan area, a LUR model was applied to predict $NO_2$ concentrations by adapting L.A. land use and transportation data to the regression coefficients previously derived for the San Diego area.[106] Specifically, traffic volumes (within 40-300m and 300-1000m), road lengths (within 40m) and distance to coast were used to estimate an initial surface for calculation of the semi-variance surface.

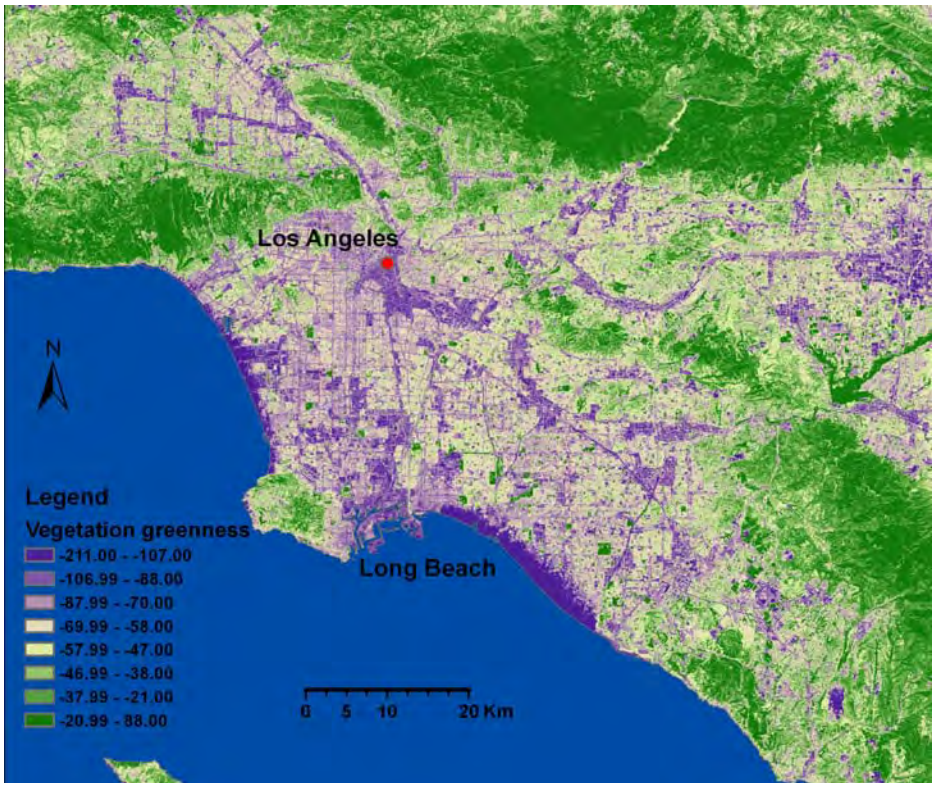


**Figure 2. Spatial analysis domain for sampling design**

Given a first estimate of the pollution surface, spatial variability of pollution *Z(x, h)* at location *x* with a distance *h* is determined by the following semivariogram equation:[107]

$$\gamma(x,h)=\frac{1}{2N}\sum_{i=1}^{n}(Z(x)-Z(x+h_i))^2 \quad (1)$$

This creates the demand surface that satisfies the first criterion noted above. To satisfy the second criterion, we appropriately modified the demand surface achieved through Eq. (1) by intensifying the demand for pollution monitors in areas with high densities of populations. To achieve this effect, a weighting scheme is implemented according to the Eq. (2):

$$W_R=\frac{P_R/P_T}{\hat{\gamma}_R/\hat{\gamma}_T} \quad (2)$$

$P_R$ is the population of interest in region $R$ within the study area and $P_T$ is the population for the entire study area. Thus, a semivariance surface reflects the spatial variability of a pollutant and is a key to apply the location-allocation algorithm.[105] The sampling network based on the location-allocation algorithm places samplers in areas of high, middle and low concentrations in a systematic way.

The task at hand was to place all monitors within 500 m of the Census Tracts (CT) of residences included in L.A. FANS and, at the same time, in areas with the most spatial variation in traffic air pollution. Thus, CTs that included L.A. FANS residences were buffered to a distance of 500 m and the eligible census blocks were assigned a population weighted semi-variance value based on the specific population counts from L.A. FANS. The original 65 CTs sampled for L.A. FANS-1 and resulting respondent population counts were utilized, as L.A. FANS-2 was on-going at the time of this work. Thus, the monitoring sites were optimized to residential locations of LA FANS respondents at the beginning of the follow-up period between Wave One (April 2000-January 2002) and Wave Two (October 2005-November 2008). The CTs were widely dispersed throughout the urbanized area of LA, including coastal areas, neighborhoods in the downtown and south central urban areas, and more suburban areas on the eastern side of the basin and in the San Fernando Valley. Irregular lattice points were created using street block centroids for locating sampling sites. Finally, monitoring locations were selected using a maximum attendance location-allocation algorithm Eq. (3) based on the population-weighted semi-variance.

$$\sum_{i=1}^{k}\sum_{j=1}^{m}w_i(1-bd_{ij})x_{ij} \quad (3)$$

where $k$ is the number of demand locations and $m$ is the number of candidate locations. In our case, $k$ = 201, including 15 co-located sites with the governmental monitoring stations. The weight $w_i$ at location $i$ represents demand, while $d_{ij}$ is the distance between locations $i$ and $j$. $x_{ij}$ is the allocation decision variable attaining the value of 1 if demand location $i$ is served by a station

in *j* and 0 otherwise. Attendance linearly decreases with distance at the rate of parameter *b*, a value determined by the maximum distance of influence.

*Selection of monitoring periods*

The goal of our geostatistical modeling was to estimate long-term (annual average) air pollution exposure to traffic-related pollutants and thus required selecting two-week monitoring periods that would most closely approximate such averages. Therefore, we obtained existing hourly measurement data for CO and $NO_2$ from existing, government monitoring stations in LA County and generated time series plots of moving two-week averages for the time period 1998-2003. The time series trends were compared to the average of all two-week averages during this time period and the months of February-March and September-October were selected as having two-week averages most closely approximating the long-term trend. The exact two-weeks monitored during each of these general time periods were selected based on logistical considerations (e.g., installation and deployment of monitors was conducted on weekends to minimize the impact of Los Angeles commuter traffic on our ability to hang all monitors within a 48-hour period).

*Neighborhood Monitoring Using Ogawa Samplers*

We used passive air samplers (part number PS-100) from Ogawa & Company USA, Inc. (Pompano Beach, FL) to conduct two weeks of monitoring in two seasons from September 16 to October 1, 2006 and from February 10 to February 25, 2007. Each air sampler was loaded with two cellulose collection pads – one pad to measure $NO_2$ (part number PS-134, pre-coated by the manufacturer with triethanolamine (TEA)) and the other for $NO_x$ (part number PS-124, pre-coated by the manufacturer with triethenolamine and PTIO). PTIO is an oxidation reagent which oxidizes NO to $NO_2$, which is then collected on the $NO_x$ filter together with $NO_2$.[108] Thus, NO concentrations were derived as the difference between $NO_2$ and $NO_x$ concentrations. To protect against rain damage, each sampler was placed inside a plastic shelter that shielded the sampler from the top but allowed air to flow freely through the bottom (Figure 3). The shelter was constructed from a 4-inch diameter PVC plumbing cap, with eye bolts on the sides and top of the shelter, which were used to secure the shelter to the mounting locations. This was the same shelter methodology utilized in the East Bay Children's Respiratory Health Study.[109]

In each season, samplers were placed in 186 neighborhood locations, which were selected using the location-allocation algorithm described above. Detailed maps with latitude and longitude coordinates and 50 and 200 meter buffers were made of each site selected by the location-allocation algorithm. Based on these maps, field teams visited each site and selected the closest possible installation location, completed a field log sheet detailing this location, and took digital photographs as well as GPS readings. Appendix A provides an example field log sheet. We used Magellan Explorist 200 GPS devices to record the latitude, longitude, and accuracy of each coordinate, using the North American 1983 datum. The rated accuracy of the Magellan Explorist 200 is within 3 meters. These GPS readings were then plotted in ArcView GIS software to ensure all selected locations were within 200 m of the original algorithm locations. The majority of neighborhood sites where the samplers were installed were within 50 m of the exact location selected; all sites were within 200 m of the selected location. In addition to the neighborhood monitors, we co-located a sampler at each of 15 South Coast Air Quality Management District (SCAQMD) air monitoring stations in the Los Angeles County area (Table 1). We also deployed 50 duplicate samplers and collected data from 30 field blanks during each

monitoring session. Sites with duplicate monitors were randomly selected. Similarly, 30 sites were randomly selected for a field blank, i.e., the blank traveled with the sampler for the selected site, but remained inside the Ogawa re-sealable pouch and storage vial while the other samplers were being installed or removed, and was returned to the refrigerator at the end of each installation or removal day.

Samplers were washed and loaded by the Ogawa & Co. USA contract laboratory at the Research Triangle Institute (RTI International, Research Triangle Park, North Carolina). Once the samplers were washed in de-ionized water and loaded with the $NO_x$ and $NO_2$ sample collection pads, each sampler was sealed inside an Ogawa plastic re-sealable pouch (part number PS-111), and then placed within an Ogawa airtight storage vial (part number PS-155). The samplers were shipped overnight to arrive 2-3 days prior to the start of monitoring. Samplers were placed in a refrigerator until the day of deployment, when they were placed in coolers (without ice) and brought out to the field locations by car. During each monitoring session, all samplers were installed over a 48-hour period. When each sampler was installed, the field staff would record the exact time of installation; the time of removal two weeks later was required to be no more than +/- 2 hours of the installation time. Therefore, each sampler remained in the field for two weeks +/- 2 hours. When the samplers were collected, the field staff also recorded the exact collection time, sealed the sampler into the re-sealable pouch and then into the storage vial, placed the sampler into the cooler (without ice), returned to our offices and placed the samplers into the refrigerator. During both the installation and collection efforts, field blanks were placed in the coolers along with the other samplers, traveled in the car with the batch of samplers, and were returned to the refrigerator at the end of each day. Once all the samplers were collected, they were shipped overnight to the Ogawa laboratory for analysis.

Field staff followed a strict protocol for installing and removing the samplers in the field. We identified the exact location of installation for each sampler, using text descriptions and photographs of the exact pole, fence, or tree on which the sampler was to be hung. Samplers were installed approximately 8 to 10 feet above the ground level (the height of each sampler was recorded on the log sheet). Duplicate samplers were installed either side-by-side or on opposite sides of the pole ("back-to-back"), ensuring that duplicates were hung at the same height.

During installation and collection efforts, two additional GPS measurements were taken at each location (total of 4 measurements per site, per season). Appendix B includes a sample log sheet where field staff recorded the sampler installation times, installation height, and GPS coordinates with accuracies.

In total, of the 186 samplers deployed in each season to neighborhood sites, measurements were obtained for 183 sites in September 2006 and for 181 sites in February 2007 (some monitors were stolen or vandalized). Eight neighborhood sites and one SCAQMD monitoring site were relocated slightly in February 2007 because access to the exact location where the sampler was installed in September 2006 was unavailable. Thus, in total, we used 181 sites for the LUR analysis, modeling the average of the two, two-week readings. Duplicate measurements were available for 49 sites in September 2006 and 47 sites in February 2007.

The samplers were analyzed by the Ogawa & Co. USA contract laboratory at the Research Triangle Institute (RTI International, Research Triangle Park, North Carolina). The sampler pads were analyzed for $NO_2$ and $NO_x$ according to the manufacturer's protocol.[110] The sample absorbance for each pollutant was used to calculate the collected weight of the pollutant on each collection pad. The average collected weight of all the field blank samplers was subtracted from the corresponding collected weight for the samplers exposed in the field,

separately for $NO_2$ and $NO_x$. The nitric oxide (NO) blank filter weights were calculated as the difference in the blank-adjusted $NO_x$ and $NO_2$ collected weights. The blank-adjusted weights in nanograms for $NO_2$ and NO were converted into concentrations (ppb) by multiplying by the corresponding concentration conversion coefficient ($\alpha_{NO2} = 56$ or $\alpha_{NO} = 60$) and dividing by the exposure time of the sampler, in minutes. Then, the concentration of $NO_x$ was calculated as the sum of the concentrations of $NO_2$ and NO.

Finally, GPS coordinate readings for each site needed to be combined into one single coordinate location for the LUR modeling, factoring in information about the accuracy of each reading. Latitude and longitude information was converted into UTM coordinates and the accuracy recorded in feet was converted into meters. For each site, we compared each GPS reading with each of the other readings for that site by calculating the distance in meters between the two readings and comparing this to two times the sum of the accuracies of these readings.

$$\text{Distance between two readings} = \text{SQRT}\ [\ (\text{lat1} - \text{lat2})^2 + (\text{long1} + \text{long2})^2\ ]$$

If this calculated distance was greater than two times the sum of the accuracies of these readings, then the reading with the inferior accuracy was removed. The remaining GPS readings were averaged to generate the best possible GPS measure of that site. With the exception of 5 sites where obtaining reliable GPS coordinates were difficult (mostly due to natural canyons or tall buildings obstructing GPS satellite signals), the average accuracy was approximately 5 meters; 25% of readings had accuracies of 3 meters or better, 50% had accuracies of 4 meters or better and 75% had accuracies of 6 meters or better.

***Land Use Regression (LUR) Modeling***

The neighborhood $NO_x$ and $NO_2$ measurement data were then used to develop a land use based regression model for the L.A. Basin. LUR treats the pollutant of interest as the dependent variable and proximate land use, traffic, and physical environmental variables as independent predictors.[29] Thus, the methodology seeks to predict pollution concentrations at a given site based on surrounding land use and traffic characteristics. Typically, land use regression models use information on land use classification (e.g., commercial, industrial, institutional), road networks, traffic, population distribution and physical properties (Jerrett et al., 2005). These variables were applied in our modeling process. In addition, we used new sources of land use information such as remote sensing-derived greenness and soil brightness. The overall steps were as follows: (1) neighborhood monitoring locations were mapped using ESRI ArcGIS software based on the average GPS coordinates described above; (2) buffers ranging in size from 50 to 15,000 meters were constructed around each site and various land use, traffic and physical environmental characteristics within these buffers calculated; (3) an ADDRESS selection strategy[6] was used to determine multivariate linear models that best predicted measured NO, $NO_x$ and $NO_2$ concentrations; (4) model diagnostics were used to examine the efficacy of the model predictions; (5) the resulting models were used to generate NO, $NO_x$ and $NO_2$ annual average exposure surfaces over the entire L.A. Basin on a 25 X 25 m grid. The following sections provide detailed methods for each of these steps.

*Calculation of Model Input Parameters*

Traffic data

Three types of roadway configuration and traffic volume data were analyzed for their

ability to predict traffic related pollution. They included Dynamap data from TeleAtlas (Global Crossroads, Boston, MA), Highway Performance Monitoring System (HPMS) data from the National Transportation Atlas Database, and Metropolitan Planning Organization (MPO) data from the Southern California Association of Governments (SCAG). Since each of these data sources has some limitations and because traffic was of great importance for the exposure contrasts, extensive efforts were made to derive the most comprehensive traffic data available.

*TeleAtlas Dynamap*

We used Teleatlas' Dynamap 2000 (TeleAtlas Global Crossroads, Boston, MA) as our base roadway configuration and traffic volume data because the underlying road network had the most accurate spatial representation when compared to digital orthophotos. The Dynamap data were combined into a mosaic from individual county files with repeated road segments removed before the analysis. The complete Dynamap physical coverage provided traffic volumes (i.e., 24-hour Annual Average Daily Traffic (AADT) traffic counts) for 2.5% of the road network in LA (18504 out of 740047 roadway segments) during the period from 1987 to 2005 (Table 2). The median AADT value projected to year 2005 from measured road segments within a road category (e.g., highway with or without limited access) was assigned to road segments of the same category; i.e. to impute traffic data to road segments without measurements. The circular area distances (buffers) we chose for LUR model development ran from 50 m to 5000 m at an interval of 100 m. Such large buffer sizes were selected because previous studies in L.A. indicated influence from land use over this extended spatial range.[111] Buffer statistics included total vehicle miles traveled (count * km) for: (1) highways (including primary roads with limited access or interstate highways (A1) and primary roads without limited access or state highways (A2)); (2) major roads (i.e., secondary and connecting roads (A3)); (3) highways and major roads (A1 + A2 + A3); and (4) all roads (A1 + A2 + ... + A7, A4 = local roads, A5 = one way vehicle dirt trails, A6 = road ramps, A7 = road as other thoroughfare) (Table X). Within a circular distance of *j* of sampler *i*, total vehicle miles traveled (VMT) $T^{v}_{i,j}$ was estimated by summing all (*k*) traffic volumes ($V_{i,j,k}$) of a road segment (*l*) within that search distance.

$$T^{v}_{i,j} = \sum_{k=1}^{m} (V_{i,j,k} * l_k) \qquad (1)$$

VMT estimated in equation (1) thus include statistics for highways, major roads, highways + major roads, and all road traffic categories.

*Highway Performance Monitoring System (HPMS) Data*

HPMS data cover highways and major roadways in LA and include modeled traffic for each road segment (http://www.fhwa.dot.gov/policy/ohpi/hpms/; accessed July 1, 2008). The HPMS network covers 92% of all highways and 54% of all major roads, a much greater coverage than TeleAtlas' Dynamap. Although more complete than the TeleAtlas Dynamap in terms of traffic counts, the HPMS physical road network was found to be simplified (e.g., the curvature of some road segments were straightened) compared to the TeleAtlas road network and digital orthophotos and locations of some roads were shifted by up to 100 m. Thus, these data offered the advantage of greater completeness, but with the disadvantage of lower spatial accuracy. Similar to the TeleAtlas' Dynamap data, we generated buffer statistics for total vehicle miles traveled (count * km) for highways and major roadways based on the HPMS data.

*Combined HPMS and TeleAtlas Dynamap Data*

To overcome the weakness in Dynamap traffic count data, we conflated the HPMS data to the corresponding Dynamap road segments that did not have traffic measurements. To minimize the rate of mis-assignment of HPMS traffic count data to Dynamap roadway segments, the conflation was performed for highway and major roadways separately. We found that none of the parallel highways were less than 200 m apart in the two maps and major roads were less than 100 m apart in the L.A. Basin. When a highway in the TeleAtlas' Dynamap was within 100 m of a highway in the HPMS roadway map, they were treated as the same highway and the highway traffic count from the HPMS data was assigned to the corresponding highway in the Dynamap data. At distances greater than 100 m, however, we considered a Dynamap highway as not corresponding to the HPMS highway and instead assigned a traffic count based on imputation of the Dynamap counts as described above. Overall, 92% of the highways in the Dynamap were conflated with traffic data from HPMS and for 8% we employed imputation. The conflation and imputation process for the Dynamap major roadways were completed in a similar fashion, except that roadways had to be within 50 m of each other for a HPMS value to be assigned to a Dynamap roadway segment. Otherwise, the median traffic data from a corresponding road category (i.e., A3) in Dynamap were imputed. Overall, 54% of the major roadways in Dynamap were conflated for traffic data with the HPMS data and for 46% we employed imputation. To avoid mis-assignment of traffic counts at road intersections, all the HPMS roads were split into polyline segments of a maximum length of 10 m. This was necessary because the conflation was done through a spatial join process that used the mid-point of a roadway as the location for the minimum distance calculation and assignment, i.e., a Dynamap road segment used its midpoint location on the roadway to locate the closest midpoint of a road segment in HPMS.

Similar to the imputed Dynamap data, the buffer statistics for the combined HPMS and Dynamap data included total vehicle miles traveled (count * km) for highways (including primary (A1) and secondary (A2) highways), major roads (A3) and both (A1 + A2 + A3). In conflating HPMS traffic data to TeleAtlas Dynamap data, possible errors will occur at the intersections of highways or major roads. Specifically, one direction of traffic might be mistakenly assigned to another direction of the same road category (highway or major road) at intersections. We found that imputation of TeleAtlas Dynamap data using road network FCC (Feature Class Classification) was a better predictor in LUR models than data from the conflation process, and thus only imputation data were used in final models. The limitation for imputation of traffic based on FCC is that even though some of the road network has the same FCC classification, they might have different traffic volumes.

*SCAG Metropolitan Planning Organization (MPO) data*

The SCAG MPO traffic data are mainly used for planning purposes (http://www.scag.ca.gov/modeling/index.htm). The data included not only physical roadway traffic volumes but also traffic volumes for "connectors" which carried the unattributed traffic load from one region to another. The connectors were represented in the road network but do not exist in the real world. Similar to the two methods described above, the buffer statistics for the combined MPO data included all roadways.

Employing the three methods described above, the estimated total VMT for various buffer sizes were then added separately and in addition to other spatial covariates in ADDRESS to model $NO_x$ concentrations.

***Road network and slope gradient***

We also used the road network configuration (including highway, major and local roads) from Dynamap as surrogates for traffic related pollution. The total length $L^c_{i,j}$ (m) of all road segments ($k$) of road category $c$ within a circular search distance $j$ of sampler $i$ was estimated by:

$$L^c_{i,j} = \sum_{k=1}^{n} L^c_{i,j,k} \quad (2)$$

As a separate roadway factor potentially predictive of pollution, the slope of a truck route was defined as an angle in degrees. We used truck route slope gradient in our analyses because we found that truck routes alone explained more than 40% of variability in pollutant concentrations. Furthermore, trucks routes correspond well with highways in Los Angeles and trucks have a much higher rate of pollutant emissions when accelerating on hills.

We first converted the Dynamap roadway network into raster cells and assigned each raster cell a slope derived from a digital elevation model (DEM) produced by the US Geological Survey (USGS, 1999). The average slope $M_{i,j}$ of all the truck route segments ($k$) within a circular search distance $j$ of sampler $i$ was estimated by:

$$M_{i,j} = \frac{1}{n} \sum_{k=1}^{n} m_{i,j,k} \quad (3)$$

We also included distance to truck routes as a potential explanatory variable during model selection. Truck routes were extracted from HPMS data for 2007 and the perpendicular distance to the closest truck route for each monitoring site was created for the study region. The HPMS data include nationally designated truck routes designated for use by dimensioned commercial vehicles under the Surface Transportation Assistance Act (STAA) of 1982. Nationally designated truck routes include the Interstate System, non-Interstate routes specifically listed in 23CFR658 and the other existing Federal-aid Primary (FAP) routes as defined in 1991. These routes are to be coded as "1" in HPMS Item 28.[112]

Also, the number of major road intersections inside each circular buffer area was calculated to identify whether areas with more intersections have higher $NO_x$ concentrations.

***Tasseled-cap transformation***

Current LUR models use road network information as a surrogate for levels of traffic- related pollution; however, effects from some land use types such as parking lots, which have similar spectral reflectance as roads, are usually unavailable in road network data and thus, unaccounted for in LUR models. In addition, most land use variables such as industrial, commercial and open land use applied in LUR models to date were in originally classified from remote sensed data. The most comprehensive, high resolution (finer than 30 m) and freely available global coverage remote sensing data are Landsat Enhanced Thematic Mapper Plus (ETM+) data. Because of the complexities involved with display and extraction of information contained in the Landsat ETM+ data (7 bands), a tasseled-cap transformation[113] was used to reduce the number of channels to be considered, and to provide a more direct association between signal response and physical processes on the ground. Tasseled-cap indices for LA were derived from the ETM+ data collected from a nominal altitude of 705 kilometers in a near-polar, near-circular, sun-

synchronous orbit at an inclination of 98.2 degrees, imaging the same 183-km swath of the Earth's surface every 16 days (http://landsat.gsfc.nasa.gov/). The ETM+ imagery we acquired included three visible (resolution 30 m), three infrared (30 m), two thermal (60 m), and a panchromatic (15 m) band. The scenes for LA were at path 41/row 36, 41/37 and 40/37, all captured on June 21, 2001. These images were orthorectified by the United States Geological Survey (USGS) and projected to UTM (Universal Transverse Mercator) zone 10N coordinate system with a WGS84 datum (World Geodetic System of 1984). Orthorectification is the process by which the geometric distortions of the image are modeled and accounted for, resulting in a planimetricly correct image. Because the Earth is in 3D while most sensors are in 2D, orthorectification corrects for many of the anomalies resulting from this conversion. The success of the orthorectification process depends on the accuracy of the digital elevation map and the correction formulae. Because the root mean square error is less than 30 m, the EMT+ data were not atmospherically and topographically corrected.

The tasseled cap transformation is an orthogonal transformation of the reflective bands of the TM data,[113] in which the first component, brightness, is related to the amplitude of reflectance associated with soils and impervious surfaces such as concrete and asphalt. Current LUR models use road network information as a surrogate for traffic related pollution; however, effects from some land use such as parking lots, which have similar spectral reflectance to roads, are usually unavailable. The second component, greenness, is orthogonal to brightness and is strongly related to the amount of green vegetation and, therefore, inversely related to the amount of impervious area. Unlike open space defined for LUR, greenness is independent of brightness and increases with increasing proportions of green vegetation. Thus, greenness might be a better, though inversely related, surrogate for the degree of influence of traffic or lack of stationary sources.

Here, we utilized average tasseled cap greenness and brightness in buffer sizes ranging from 100-5000 meters at 100 meter increments as potential predictor variables in the LUR models.

***Land use characteristics***

Land use data for L.A. were acquired from the Southern California Association of Governments (SCAG) for the year 2000. Major land use types included commercial, residential, industrial, and open land use. The total area $A_{i,j}$ of a land use type within a circular buffer search distance $j$ of sampler $i$ was estimated by summing over all ($k$) areas of a land use type inside the buffer:

$$A_{i,j} = \sum_{k=1}^{m} (S_{i,j,k}) \qquad (4)$$

Additionally, physical geographic variables like distance to coast, elevation, and coordinates of latitude and longitude were calculated for each sampler location and used as covariates for our ADDRESS modeling process.

***Model selection and diagnostics***

For the model selection process we used ADDRESS (A Distance Decay REgression Selection Strategy).[6] The selection process includes multiple steps and, at each step, a full spectrum of correlation coefficients and buffer distance decay curves are used to select a spatial covariate of the highest correlation (compared to other variables) at its optimized buffer distance.

At the first step, the series of distance decay curves are constructed using the measured concentrations against the chosen spatial covariates. A variable with the highest correlation to pollutant levels at its optimized buffer distance is chosen as the first predictor of the LUR model from all the distance decay curves. Starting from the second step, the prediction residuals are used to construct new series of distance decay curves and the variable of the highest correlation at its optimized buffer distance is chosen to be added to the model. This process continues until a variable being added does not contribute significantly ($p>0.10$) to the model performance. The distance decay curve yields a visualization of change and trend of correlation between the spatial covariates and air pollution concentrations or their prediction residuals, providing a transparent and efficient means of selecting optimized buffer distances. The spatial variables used in this research include various traffic data derived as described above, road network and truck slope gradient, remote sensing vegetation greenness and soil brightness, land use characteristics and population density. However, not all the variables were included in the final LUR model. The buffer distances ran from 0-100 m to 0-200 m and to the maximum buffer distance 5000 m (interval 100 m). Because primary and secondary highways and major roads are densely distributed throughout LA, any subject's exposure at their residence is influenced not only by the nearest roadways and traffic but likely also by urban-scale traffic patterns that vary over ranges of 5 km or more. Thus, the maximum distance of a buffer was set to 5000 m. However, if a distance decay curve still showed an upward trend at 5000 m, the maximum buffer distance was further extended until a downward trend was identified. In an urban environment, correlations might not be zero even at very high buffer distances because of the influence of background pollutant concentrations; however, we expected to see a decrease in the influence of certain emission sources, such as emissions from a roadway, after a large enough distance.

To test the efficacy of the prediction models, model diagnostics included (1) evaluating whether selected variables were collinear based on variance inflation factors (VIFs); (2) outlier assessment, i.e. determining Cook's distances[114] to assess whether a single observation changed regression estimates; (3) examining whether spatial autocorrelations of the prediction residuals in our final optimized models existed based on the Moran's *I* statistic;[115] (4) assessing whether predictions satisfied the U.S. EPA (Environmental Protection Agency) requirements of a prediction model[116] by adopting the normalized mean bias (NMB) (see eq. 5) and normalized mean error (MME) (see eq. 6) tests below; (5) conducting a Chow test[117] to assess whether large sample sites would benefit LUR modeling results; and (6) applying cross-validation techniques to 16 random samples for model reliability tests.

$$NMB = \frac{1}{N}\sum_{i=1}^{N}\left(\frac{C_i^* - C_i}{C_i}\right) * 100\% \quad (5)$$

$$NME = \frac{1}{N}\sum_{i=1}^{N}\left(\frac{|C_i^* - C_i|}{C_i}\right) * 100\% \quad (6)$$

$C_i^*$ and $C_i$ refer to the predicted and observed pollutant concentrations at sampler *i*.

The Moran's *I* statistics were conducted on a first and second order Queen's contiguity matrix[118] to the Thiessen polygons[119] created from the sampler sites. Statistical significance was tested using a permutation test with 999 iterations. Although a location-allocation algorithm was applied to locate samplers, the samplers were also restricted to residential census tracts of subjects in the LA FANS health study. If samplers were clustered within census tracts of residence, it would be impossible to remove the near range autocorrelation with fixed effects or

with standard autoregressive techniques. Therefore all models were refit in STATA 8.0 adding a cluster parameter (based on census tract ID) to our ADDRESS models and also using Generalized Estimating Equations (GEE)[120] clustered on the census tracts where the monitors were located. An "exchangeable" correlation structure was used for the GEE model, assuming all the measurements inside a census tract were equally correlated. Sensitivity analyses were also done with robust standard error estimation using census tract as the cluster unit.

The EPA's suggested performance criterion for NMB is ±5% to ±15%, and for NME 30–35% of pollutant levels greater than 60 ppb ($C_i \geq 60$ ppb).[116] For the Chow test,[117] half of the total sites available were randomly selected from each of four quartile groups of $NO_x$ concentrations and the remaining measurements were used separately to model NO, $NO_2$ and $NO_x$ using the same spatial covariates from the full dataset. The Chow test identifies structural stability of regression models of the two subsets compared to the pooled full dataset.[121]

The ADDRESS model was developed using the measured monitoring station concentrations as dependent variables and the above 5 categories of spatial covariates as predictors. The final model was then used to predict pollutant concentrations on a 25 X 25 meter grid using the selected spatial covariates and corresponding optimized buffer distances.

**$O_3$ and $PM_{2.5}$ Kriging**

We used geostatistical interpolation to estimate long-term (annual average) exposure to the more regionally distributed pollutants $O_3$ and $PM_{2.5}$. These models originate from spatial interpolation techniques and result in the creation of a pollution surface, whereby pollution levels at sites other than monitoring stations are estimated. The most advanced form of spatial interpolation is kriging, because it produces the best linear unbiased estimate (BLUE) and allows for mapping of error variances. These variances can be used to view the location of errors for the predicted pollution surface[122]and may be incorporated into subsequent uncertainty models. Data required for this model include a network of sampling sites chosen based on factors such as the extent of analysis, topography of local area, local emissions, and the scale of variability of the measured pollutant. Kriging models exploit spatial dependence in the data to develop continuous surfaces of pollution.[29] Beyond random error or noise in the data, spatial dependence embodies two types of effects. First-order effects, otherwise known as global trends, measure broad trends in the data over the entire study area. In contrast, second-order effects measure local variations that are a function of distance between the points.[29,115]

To derive the annual average $PM_{2.5}$ kriged exposure surface, we interpolated annual $PM_{2.5}$ measurement data from 23 state and local district monitoring stations in the LA basin for the year 2000 using 5 interpolation methods: bicubic splines, two ordinary kriging models, universal kriging with a quadratic drift, and a radial basis function multiquadric interpolator.[123-125] The estimates were based on year 2000 monitoring data as it contains the first complete run of $PM_{2.5}$ samples for the LA Basin. Figure 4 shows the modeling domain and location of $PM_{2.5}$ monitors. Because of the limited number of monitoring sites, a leave-one-out cross-validation was conducted to select a kriging method which created the highest prediction power. After cross-validation, we used a combination of universal kriging and multiquadric models. This approach takes advantage of the local detail in the multiquadric surface and the ability to handle trends in the universal surface. We averaged estimated surfaces based on 25-m grid cells.

For ozone, the average of the four highest 8-hr maximum averages for 2002 at 42 sites (Figure 5) were modeled using ordinary kriging; an additional 42 sites surrounding the study area were used solely to better estimate the correlation structure in the data. The correlation structure

of the data was modeled using a spherical model estimation of the semivariance. Similar to $PM_{2.5}$, we averaged estimated surfaces based on 25-m grid cells.

**Exposure Assessment for L.A. FANS participants**

As part of the L.A. FANS-2 survey, residential history information (i.e., addresses and dates in each home) were asked of all adult participants as part of an Event History Calendar (EHC). The EHC calendar collected data for residences occupied within the last six years for new entrants to the study. Since respondents were asked when they moved to a given residence, the start date for the earliest residence lived in during the 6-year window varies. Panel respondents were asked to report all residences they lived in since the L.A. FANS-1 interview, a time span that could have included more than six years depending on the timing of the two interviews. Here we utilized residential history information collected as part of L.A. FANS-2. Thus, for new entrants all homes which fell within a six-year window prior to the L.A. FANS-2 interview are included. For panel respondents, the histories include the residence location at L.A. FANS-1 (which may extend some period back from the L.A. FANS-1 interview date) as well as any moves up until the time of the L.A. FANS-2 interview. ArcGIS Geographic Information System (GIS) software (ESRI, Redlands, California, version 9.3) was used to map these residential addresses and derive latitude and longitude locations. The underlying street network used to geocode residential addresses was the ESRI StreetMap$^{TM}$ (series issue 2008, North America). In addition, Primary Caregivers were asked about each child's school attendance, i.e., names, addresses and dates for all schools attended by the child. Reported school locations were geocoded using the same method as for residence locations.

Home and school location coordinates (latitude and longitude) were overlaid with the LUR and kriged $O_3$ and $PM_{2.5}$ surfaces created as described above and the following concentration values extracted for each location: NO, $NO_2$, $NO_x$ in ppb, $O_3$ in ppm and $PM_{2.5}$ in $\mu g/m^3$. (Note that $O_3$ values in ppm were converted to ppb the for health analyses). Information on time spent in each home and school was then used to create the following weighted annual averages: current home, homes during the previous 12 months (i.e., weighting annual averages extracted for each home by the time spent in each home during this period), homes during the previous 24 months, and homes during the previous 5 years. For schools, it was assumed that a child, on average, spents 1,080 hours at school per year (180 days per year and 6 hours per day).

**L.A. FANS Health Outcome Assessment**

Respiratory health in children was assessed in L.A. FANS Wave-2 via in-person interviews with Primary Caregivers (PCGs) and also using portable spirometers to measure lung function for respondents at least five years of age. Table 3 provides a list of the L.A. FANS Wave-2 respiratory health questions; these questions were based on the International Study of Asthma and Allergies in Childhood (ISAAC) questionnaire. Based on the responses to these questions, we performed logistic regressions to examine associations between the LUR air pollution metrics and the following dichotomous health outcomes: (1) doctor-diagnosed asthma (ever); (2) wheeze in the past 12 months; (3) wheeze with any night waking in the past 12 months; (3) medication use for asthma or wheeze in the past 12 months; (4) sneezing or a runny or blocked nose apart from colds in the past 12 months; (5) more than 3 doctor-diagnosed ear infections in a year. There were too few subjects reporting wheeze severe enough to limit speech in the past 12 months for meaningful statistical analyses (n=18).

As a part of the L.A. FANS-2 survey, child participants at least 5 years of age were given

lung function tests using portable spirometers in conjunction with the field interviews. The EasyOne Diagnostic Spirometer from ndd Medical Technologies (http://www.nddmed.com, Andover, MA) was selected for the L.A. FANS Wave-2 field work. The key features of this instrument that made it well suited for the study were: (1) it is small, portable, and requires minimal power (approximately 400 measurements can be completed with two AA alkaline batteries), (2) has the ability to record and store approximately 700 sessions of spirometric data in memory including full flow-volume curves, (3) includes quality control software and prompts to obtain acceptable and repeatable efforts, (4) has time and date stamping of all records, (5) allows easy transfer of specific flows and volumes to a personal computer database, (6) can be re-used to test multiple subjects with minimal cleaning, (6) allows easy calibration, and (7) complies with American Thoracic Society (ATS) criteria for spirometer performance. A recent evaluation by the Fresno Asthmatic Children's Environment Study (FACES) study team indicated this spirometer accurately and reliably measures pulmonary function in children, relative to a "gold-standard" laboratory-style instrument.[126]

Portable spirometers can measure a wide range of pulmonary function parameters, including peak expiratory flow rate (PEF), forced vital capacity (FVC), forced expiratory volume after 1 second ($FEV_1$), forced expiratory mean flow between 25% and 75% of FVC ($FEF_{25-75}$), and forced expiratory mean flow at 75% of FVC ($FEF_{75}$). This set of lung function parameters reflect conditions in both small and large airways and are more sensitive to changes in functional status in asthma. Table 4 describes these spirometry measurements in greater detail. FVC is the maximum volume of air expelled during an expiration made as forcefully and completely as possible starting from full inspiration, while $FEV_1$ is the volume of air delivered during the first second of the FVC maneuver (ATS, 1994). PEF, $FEF_{25-75}$ and $FEF_{75}$ are all flow measures and are considered markers of small airway function, converse to the volume measures that provide information on the larger, central airways.[49,56]

As a part of the L.A. FANS-2 survey, trained field technicians collected spirometry measurements at the subjects' homes. Based on discussions with our collaborators from the Fresno Asthmatic Children's Environment Study (FACES) and recommendations from CARB internal and external reviewers, additional training was conducted for L.A. FANS field interviewers to help increase the quality of lung function data collected for children (some of which were as young as 5 years). Specifically, a separate contract was established for Dr. Kathleen Mortimer and Mr. Lucas Carlton (UC Berkeley) to provide training workshops to the L.A. FANS interviewers. Dr. Mortimer and Mr. Carlton have extensive experience in using the EasyOne spirometer to assess lung function, especially in asthmatic children where these manuevers can be most difficult, based on their work on FACES. All field interviewers were required to attend at least one 6-hour training session. Appendix C provides a detailed summary of the L.A. FANS spirometry training. Appendix D provides the L.A. FANS-2 spirometry protocol used by the field interviewers. People with specific health conditions (mostly serious respiratory problems, or conditions that made completing the spirometry maneuvers physically difficult for the subject) were not asked to complete the spirometry procedure. Out of 1,287 total L.A. FANS-2 child respondents at least 5 years of age, a total of 1,070 (83%) subjects attempted the spirometry procedure.

A detailed description of procedures used to collect spirometry measures are provided in the protocol in Appendix D. Briefly, the field technician demonstrated a sample spirometry maneuver by taking a deep breath and blowing through the tube until the EasyOne device indicated the end of test, or as long as possible, whichever occurred first. Participants completed

the spirometry maneuvers by placing nose clips on his/her nose, standing as straight as possible, taking a very deep breath, and blowing hard and fast through the mouth into the spirometry device. They were instructed to keep blowing until the device beeped to indicate the end of the test, or until they could no longer keep blowing. For each subject, the goal was to complete 3 maneuvers that were considered acceptable by the EasyOne device, with a maximum of 8 attempts. Specifically, the EasyOne device evaluated each maneuver for acceptability based on the following 1994 ATS criteria:[127]

- Back-extrapolated volume ≤150 ml or 5% of FVC, whichever is greater;
- Time to peak flow ≤120 milliseconds; and
- Expiration time ≥2 seconds or volume accumulation dropped below 100 ml per 0.5 seconds.

Subjects were prompted to continue to perform maneuvers until 3 acceptable and reproducible curves were achieved; reproducibility was defined per 1994 ATS criteria as:

- Two largest $FEV_1$ values within 200 ml; and
- Two largest FVC values within 200 ml.

The EasyOne device signaled completion of the test when: (1) there were 3 acceptable blows and the best 2 were reproducible within 200 ml if the person did 4 or fewer blows; or (2) there were 3 acceptable blows and 2 were reproducible within 250 ml if the person did 5 or more blows; or (3) a maximum of 8 attempts was completed. The EasyOne device automatically stored the 3 best curves for each participant, i.e., the 3 curves with the highest sum of $FEV_1$ and FVC, per ATS criteria.

The spirometry data collected during the field interviews were then downloaded into computer databases for review and analysis. We performed a review of the acceptability of each spirometry curve assigned by the EasyOne device by visual inspection of each flow-volume curve. The EasyOne's pre-programmed criteria are sometimes too strict for very young children since they cannot expel air long enough for the EasyOne device to register a manuever as a valid test. Additionally, the EasyOne software may not detect all faulty curves that can be identified only through visual inspection of the hard copy tracings of the curves. This review and re-grading of all the spirometry curves was completed by Mr. Lucas Carlton at UC Berkeley, under a separate subcontract (Number 05-311). As mentioned previously, Mr. Carlton has specific experience evaluating spirometry data from children as part of the UC Berkeley Fresno Asthmatic Children's Environment Study. A summary of Mr. Carlton's review activities is provided in Appendix C. Briefly, the data from the EasyOne portable spirometers were downloaded into an MS Access database, and an electronic form was added so that the grades could be entered directly into the database. Mr. Carlton graded each curve to determine acceptability using the following criteria (some of which overlap with the 1994 ATS criteria):

(1) The Back Extrapolated Volume must be ≤5% or 150mL, whichever is greater;
(2) Time to Peak Flow must be ≤120 milliseconds;
(3) No abrupt end to test;
(4) FET must be ≥ 2 seconds;
(5) Time/Volume curve must begin at origin (to ensure proper start of test);
(6) Curve must show that subject exhaled using only one continuous blast of air; and
(7) Curve must show no leaks or negative flow throughout test (i.e. no inhalation).

In our analyses, we examined all children with one or more acceptable curves per Mr. Carlton's review. If the EasyOne software deemed a curve acceptable, but Mr. Carlton did not based on his curve review (mostly having to do with start of test problems), then the curve was not included. For children with two acceptable curves, we used the highest values to analyze $FEV_1$ and FVC, while for PEF, $FEF_{25-75}$, and $FEF_{75}$ we used the value from the curve with the highest sum of $FEV_1$+FVC per ATS guidelines.[127] We also performed additional analyses only including subjects having 3 acceptable curves and 2 reproducible curves for $FEV_1$ and FVC per 1994 ATS criteria; i.e., the 2 largest $FEV_1$ and FVC values were within 200 ml, similar to other cross-sectional studies of air pollution impacts on lung function in children.[33,44] To analyze $FEV_1$ and FVC, we used the highest values from the 3 best curves; for all other parameters (PEF, $FEF_{25-75}$, $FEF_{75}$) we used values from the curve with the highest sum of $FEV_1$+FVC, again based on ATS criteria. Overall, Mr. Carlton graded spirometry curves for over 3,000 participants, including both children and adults. The large majority of these subjects (over 75%) were able to achieve 2 or more acceptable spirometry curves, and over 78% of these met the ATS criteria for reproducibility. Adults were most likely to achieve reproducible curves (over 63% were reproducible), and results were similar among teenagers (59%) and preteens (56%). However, children <8 years of age were far less likely to achieve reproducible curves (42%), partly due to many of the children being unable to obtain a minimum of 2 acceptable curves.

**Confounder/Effect Measure Modifier Assessment**

*Individual Level*

A number of individual, family and neighborhood-level characteristics were considered for inclusion in our models. Individual-level risk factors considered were child's race/ethnicity, age, gender, health insurance status, and whether the child had a usual source of sick care. Information on these variables was collected as part of the L.A. FANS-2 survey. Objective physiological health measures taken as part of L.A. FANS-2 included height and weight for children age two years and older (children two years of age had to be able to stand unassisted). These data were used to estimate body mass index (BMI) for each child. We defined as overweight children with BMI-for-age values at or above the 85$^{th}$ percentile based on 2000 U.S. Centers for Disease Control BMI-for-age charts for boys and girls (www.cdc.gov/growthcharts/).

*Family Level*

At the family level, we examined the following measures of socioeconomic status (SES): family income, homeowner status (yes/no), and primary caregiver (PCG) education (years). We also considered PCG birthplace (U.S. versus outside the U.S.) and current marital status as indirect markers of SES and access to health care. Information on these variables was collected via in-person interviews with Primary Caregivers (PCGs). If the PCG did not provide information on family income or homeowner status, but a separate Randomly Selected Adult (RSA) did provide this information, then the RSA's response was used in our analyses.

We also evaluated a number of family-level neighborhood perception variables, where the Randomly Selected Adult participant from each family was asked to rate their overall neighborhood satisfaction and opinion of neighborhood safety, cohesion and support (see Table 12 in Results section for a listing of these variables). The overall neighborhood cohesion score was based on the average of responses to a series of questions asking whether the neighborhood was close-knit, whether neighbors get along, are willing to help each other, share the same

values, can be trusted, and whether adults look out for and discipline children in the neighborhood as needed (see Table 12 for more details). Participants were also asked about the number of other adults they recognize in their neighborhood. The neighborhood support score was based on the average of responses (1=often, 2=sometimes, 3=rarely, 4=never) to the following questions: (a) How often do neighbors do favors for each other; (b) How often do neighbors watch each others' property; and (c) How often do neighbors ask for advice. Adults were also asked about the number of friends and relatives living in the neighborhood, group participation in the previous 12 months, and number of conversations with neighbors. For these questions, "neighborhood" was defined to respondents as "both the block or street you live on and several blocks or streets in each direction". Based on the design of the L.A. FANS survey, these perception questions were answered by the PCG for 53% of the families, by the PCG's spouse or partner for 24% of the families and by another adult in the household for 7% of the families. Approximately 16% of the children are missing data for these neighborhood perception questions. In L.A. FANS-2 a household may not have an RSA interviewed (and thus have missing data on neighborhood perception) if the panel RSA (i.e., the adult interviewed during Wave 1) moved away from the panel Randomly Selected Child (RSC) and the RSC's sibling (SIB) and thus is no longer in the household. A new RSA was selected only in a random subset of panel households. Only those adults who did not live in the L.A. FANS-1 tract of the panel household were eligible to be selected as new adult respondents.

As part of L.A. FANS-2, a series of questions was added to assess potential exposures to indoor allergens and indoor air pollution sources. PCGs were asked whether anyone currently living in the house smoked cigarettes, cigars or pipes, and if yes, the number of people who smoke everyday inside the home. The PCG was also asked if she currently smoked. Since based on the examination of the data, it was apparent that sometimes the PCG did not include herself when reporting current smokers in the home (only other smokers), we constructed a separate household smoking variable which indicated whether the PCG reported current smokers in the home or that s/he was a current smoker. Respondents were asked whether they currently had a gas appliance in their home (stove, range or oven), and if yes, how these devices were lit (i.e., electronic ignition with no pilot light, match lighting with no pilot light and continuous pilot light). Respondents were asked whether they had pests in their home in the last 12 months (rats, mice, cockroaches, ants, spiders or termites) or no problems with pests during this time. We also evaluated whether respondents reported cockroaches in the home separately, as a surrogate measure of potential exposure to these specific allergens. To assess potential exposure to molds, respondents were asked whether there was mold or mildew on the walls, ceilings or floors of the home or a moldy or musty smell in the home during the past 12 months. Respondents were also asked to identify furry pets (dog, cat, etc.) that come inside the house or apartment at least part of the time.

Finally, PCGs were asked whether each child's biological mother and father had asthma.

*Neighborhood Level*

At the neighborhood level, we constructed a census tract-level disadvantage score based on US Census 2000 data similar to Cohen et al. (2006). This continuous index of neighborhood socioeconomic conditions represents the average of four measures for each tract: percent poor families, percent households on public assistance, percent female-headed families with children under 18 years of age, and percent male unemployment. Other census-based measures included the percent of the tract population that lived in the same house 5 years ago (as a measure of

neighborhood stability) and whether there was a dominant racial/ethnic group in the tract (as a measure of neighborhood homogeneity).[128] Finally, we averaged the neighborhood cohesion scores and opinions of neighborhood safety across all L.A. FANS adults living in the same census tract as additional measures of neighborhood quality.

**Statistical Analyses**

*Respiratory Health Endpoints*

We used logistic regression to evaluate associations between the LUR and kriged air pollution estimates and odds of the following outcomes: (1) doctor-diagnosed asthma (ever); (2) wheeze in the past 12 months; (3) wheeze with any night waking in the past 12 months; (3) medication use for asthma or wheeze in the past 12 months; (4) sneezing or a runny or blocked nose apart from colds in the past 12 months; (5) more than 3 doctor-diagnosed ear infections in a year. There were too few subjects reporting wheeze severe enough to limit speech in the past 12 months for meaningful statistical analyses (n=18). For the outcomes wheeze in the past 12 months, wheeze with any night waking in the past 12 months, medication use for asthma or wheeze in the past 12 months, and sneezing or runny or blocked nose apart from colds in the past 12 months, our main exposure of interest was annual average NO, $NO_2$ and $NO_x$ as estimated by the LUR model, accounting for all homes lived in during this time period. However, as a sensitivity analysis, we also examined associations between averages based on the current home (since this allowed for the largest sample size), 2 years prior to interview date and 5 years prior to interview date (the latter two metrics to examine potential importance of longer-term exposures on the outcomes). We also examined associations with air pollution metrics taking estimated concentrations at school locations into account. For doctor-diagnosed asthma and ear infections, our main exposure of interest was 5 years prior to the interview date, as this was the longest weighted average we could generate for the majority of subjects. Unfortunately, the number of children for whom lifetime residential histories became available was too small for meaningful statistical analyses of associations between lifetime air pollution exposures and these health outcomes. We examined crude associations between each outcome and each pollutant (NO, $NO_2$, and $NO_x$) separately, as these exposure metrics were highly correlated (r~0.8-0.9). We examined changes in the odds of each outcome per interquartile increase in each pollutant. For example, we fit the following logit-linear model for the binary outcome "wheeze in the past 12 months" (noted here as A, where A=1 if a child was reported to have wheezing):

$$\text{logit}(A=1|\ NO) = \beta_0 + \beta_1\ (NO)$$

Here $\exp(\beta_1)$ represents the odds ratio for wheeze in the past 12 months corresponding to an IQR change in average NO 12 months prior to the interview date (in ppb). Logistic regressions were conducted using SAS statistical software (version 9.1).

Second, we used multivariate logistic regression analyses to quantify associations between LUR exposure metrics and outcomes while controlling for potential confounders. For example, we fit the following logit-linear model again for "wheeze in the past 12 months" as a function of average NO 12 months prior to the interview, considering age and gender (gender=1 if the child is a boy, 0 if the child is a girl) as potential confounders:

$$\text{logit}(A=1|\ NO) = \beta_0 + \beta_1\ (NO) + \beta_4\ (age)_i + \beta_5\ (gender)_i$$

We evaluated changes in point estimates and 95% confidence intervals (CIs) for air pollution association measures (odds ratios) when entering each individual, family and neighborhood level risk factor discussed above into the models. Variables included in final

models were determined based on subject-matter criteria, correlations between variables, and impact on estimates (i.e., whether their inclusion changed associations estimates by at least 5%).[129] Each final model included child's age, sex, race/ethnicity, and family income. Additional variables were included depending on health endpoint (see Tables 20-25 in the Results Section). We also examined changes in effect estimates for the LUR exposure metrics when entering the peak $O_3$ exposure metrics into models. Finally, a two-level model with a random intercept for family was used to account for non-independence of siblings (i.e., clustering at the family-level).

We explored differences in effect estimates when stratifying on the neighborhood quality variables, specifically census tract-level measures of neighborhood cohesion, safety, and economic disadvantage, as well as percentages of residents living in the same home as five years ago. Not enough subjects resided in tracts that were predominately Latino or White for meaningful statistical analyses stratified on this variable. Overall statistical power for all stratified analyses was limited due to the relatively small overall sample size available to us. Originally, the L.A. FANS-2 study planned to enroll approximately 4,000 children (all of the L.A. FANS-1 children plus a sample of new entrants into each neighborhood). Despite extending data collection by approximately 1.5 years after the originally scheduled enrollment period, the total number of L.A. FANS-2 child participants reached only about 35% of the originally planned enrollment number. This issue is addressed further in the Discussion section.

*Lung Function*

Linear regression was used to estimate associations between lung function measures and annual average levels of NO, $NO_2$, and $NO_x$ estimated by LUR models and $O_3$ and $PM_{2.5}$ estimated by kriging. The specific lung function measures evaluated were: peak expiratory flow rate (PEF), forced vital capacity (FVC), forced expiratory volume after 1 second ($FEV_1$), forced expiratory mean flow between 25% and 75% of FVC ($FEF_{25-75}$), and forced expiratory mean flow at 75% of FVC ($FEF_{75}$). When more than one acceptable curve was available, the largest values for FVC and $FEV_1$ were selected for analysis, as explained previously. For the flow measures, values were obtained from the curve with the highest sum of FVC and $FEV_1$. We examined several different pollutant averaging periods in our models: annual average pollutant values based on the current home only, and considering (i.e., weighting for) all homes lived in for the past 12 months, 24 months and 5 years prior to the interview date. We also examined effects taking concentrations at school locations into account. We examined crude associations between each outcome and each pollutant (NO, $NO_2$, and $NO_x$) separately, as these exposure metrics were highly correlated. For example, we fit the following model for the continuous outcome PEF as a function of average NO 12 months prior to the interview:

$$PEF_i = \beta_0 + \beta_1 (NO)_i + \varepsilon_i \quad \varepsilon_i \sim N(0,\sigma^2)$$

where i is the index for the individual and $\varepsilon_i$ represents individual-level variation.

Similar to the multivariate logistic regression analyses, multivariate linear regression was used to quantify associations between LUR exposure metrics and outcomes while controlling for potential confounders. To illustrate, we fit the following model for the continuous outcome PEF as a function of average NO over the 12 months prior to interview, considering age and gender (gender=1 if the child is a boy, 0 if the child is a girl) as potential confounders:

$$PEF_i = \beta_0 + \beta_1 (NO)_i + \beta_2 (age)_i + \beta_3 (gender)_i + \varepsilon_i \quad \varepsilon_i \sim N(0,\sigma^2)$$

We evaluated changes in betas and 95% confidence intervals (CIs) for air pollution metrics as well as changes in model fit (based on adjusted $R^2$, Mallow's Cp and Akaike Information

Criterion (AIC) values[130] when entering each individual, family and neighborhood level risk factor discussed above into the models. Since initial results indicated differences in associations between air pollution and lung function by gender, we conducted analyses for boys and girls separately. We examined models including height, weight, and BMI using several different parameterizations, and selected height, height$^2$, and being overweight (yes/no) for our final models for boys and height, height$^2$, weight, weight$^2$, and being overweight (yes/no) for our final models for girls. Other variables included in final models were selected based on subject-matter criteria, correlations between variables, changes in air pollution parameter estimates, and model fit (see Tables 28-30 in Results sections for listing of final adjustment variables). Cook's distance[114] as well as DFFITS and leverage statistics[130] were used to assess the impact of each observation on the estimated effects and some outliers were excluded from analyses. Finally, a two-level model with a random intercept for family was used to account for non-independence of siblings (i.e., clustering at the family-level).

We explored differences in effect estimates when stratifying on the neighborhood quality variables, specifically census tract-level measures of neighborhood cohesion, safety, and economic disadvantage, as well as percentages of residents in neighborhood living in the same home as five years ago. Not enough subjects resided in tracts that were predominately Latino or White for meaningful statistical analyses stratified on this variable. Overall statistical power for these stratified analyses was limited due to the small sample size available to us.

## IV. RESULTS

### LUR and Kriging Modeling

#### *$NO_x$ measurements*

The sampler detection limits, calculated as three times the standard deviation of the field blank measures during each season, were <0.14 μg for $NO_2$ and <0.76 μg for $NO_x$. Our duplicate measurements indicated that the average coefficient of variation was low (3.3% for $NO_2$ and 2.1% for $NO_x$). Measured pollutant levels ranged from 5.3 ppb to 42.7 ppb for $NO_2$ (median = 27.3 ppb) and from 8.1 ppb to 157.0 ppb for $NO_x$ (median = 60.9 ppb), after correcting for blank concentrations (Table 5). Annual means measured by 14 monitors in the Southern California Air Quality Management District regulatory network showed strong relationships with the campaign-specific means. We calculated the intraclass correlation coefficient (ICC) using a generalized linear model with compound symmetry covariance, clustered by site, and found high correlations between our 2, 2-week average measurements and annual averages based on measurements at the government monitoring stations (ICC = 0.93 for NO, 0.87 for $NO_2$, and 0.96 for $NO_x$). Comparison of February 2007 measurements made by Ogawa samplers collocated at government sites (which are chemiluminescence samplers) produced slopes of 0.82 ($R^2$ = 0.91), 0.72 ($R^2$ = 0.64) and 0.81 ($R^2$ = 0.92) for NO and $NO_2$ and $NO_x$, respectively. The relationships between collocated measurements in September 2006 were also strong, with slopes of 1.16 ($R^2$ = 0.82), 0.82 ($R^2$ = 0.84) and 1.01 ($R^2$ = 0.89) for NO, $NO_2$ and $NO_x$, respectively. To compare the duplicate measurements for both seasons combined, we calculated the ICC as above, clustering by site and season, and found high correlations for all three pollutants of interest (ICC = 0.97 for NO, 0.92 for $NO_2$, and 0.98 for $NO_x$). These results suggest that our short-term monitoring captured the longer term spatial patterns of exposure well in the Los Angeles area.

#### *LUR Distance decay curves*

Because distance decay curves for NO, $NO_2$ and $NO_x$ showed an upward trend for all

traffic estimates at the pre-specified 5000 m buffer distance, we extended the maximum buffer distance to 15 km to identify the optimal distance of influence from traffic sources. Among the four types of traffic density estimates we considered, traffic measures imputed from TeleAtlas Dynamap data were found to have the highest correlations with measured NO, $NO_2$ and $NO_x$ concentrations and were therefore used in the ADDRESS model selection process. As illustrated in the distance decay curves presented in Figure 6a-d, correlations between VMT and traffic-related air pollution concentrations increased steeply out to 10 km and reached a peak around 11 km, especially for $NO_x$. Highway vehicle density within 11 km alone explained 46.6% of the model variance for $NO_x$. After a buffer distance of 11 km, correlations trended to slope downward. The explanatory power of highway vehicle density within 11 km suggests the increasing contribution from background $NO_x$ transport within that distance, a finding consistent with earlier integrated meteorological models calibrated for L.A.[131] Since previous studies identified the maximum distance of influence from a roadway to be 1500 m,[29] we created semivariograms of NO and $NO_2$ concentrations (Figure 7) using the 201 monitoring site measurements to identify distances of spatial dependency for the two pollutants. Based on these analyses, the distance of spatial dependency for NO was about 11000 m, while $NO_2$ concentrations experienced an even slower decrease, reaching background levels at about 20000 m. For local effects, we saw a sharp drop of influence of major road from near source (100 m) to a distance of 500-700 m; however, for highways, highways and major roads combined, and all road categories, we saw a sharp increase of influence from near source to a 500 m buffer distance (not shown on figures).

Compared to all other variables we explored, distance to truck routes (not shown on figures) correlated most strongly with NO, $NO_2$ and $NO_x$ measures (correlation coefficient = 0.57-0.67), and explained 44.2% of the model variance for $NO_2$. Remote sensing derived greenness (Figures 6b-6d) also correlated highly with the three pollutant concentrations and was seen as a much better predictor than open space, even though soil brightness correlations (Figures 6b-6d) were lower than those for greenness. The greenness and soil brightness surfaces in Figures 8a and 8b also demonstrate that greenness was a better predictor of road networks and other off-road traffic-related land uses (e.g., parking lots). Truck route slope gradient explained up to 9% of $NO_x$ concentration variance.

Based on the distance decay correlation curves, we found that influence from the majority of the variables analyzed had a spatial extent greater than 3000 m, especially for traffic related variables. This is inconsistent with previous research findings from medium- or smaller-size urban areas (e.g., traffic influence < 1500 m). Using the highway network in L.A. as an example, Figures 9 and 10 show the distance decay of NO and $NO_2$ concentrations with increasing distance from highways (the bar charts show average measured pollutant concentrations within a certain distance interval, i.e., 100, 200, 500 and 1000 m). If we use a 50% reduction in concentration as a standard for spatial extent identification, then the spatial extents for NO and $NO_2$ would be 3000 and 5000 m from highways, respectively. If 10% is used as a threshold, then the spatial extent for both pollutants would be greater than 10000 m.

***ADDRESS modeling results***

Before we modeled the NO, $NO_2$ and $NO_x$ concentrations, we chose 16 monitoring sites randomly for cross validation, four sites from each of four quartile groups of $NO_x$ concentrations. The remaining 167 sites were used in ADDRESS to model annual concentrations of NO, $NO_2$ and $NO_x$, based on the mean concentrations from the two measurement periods. The final

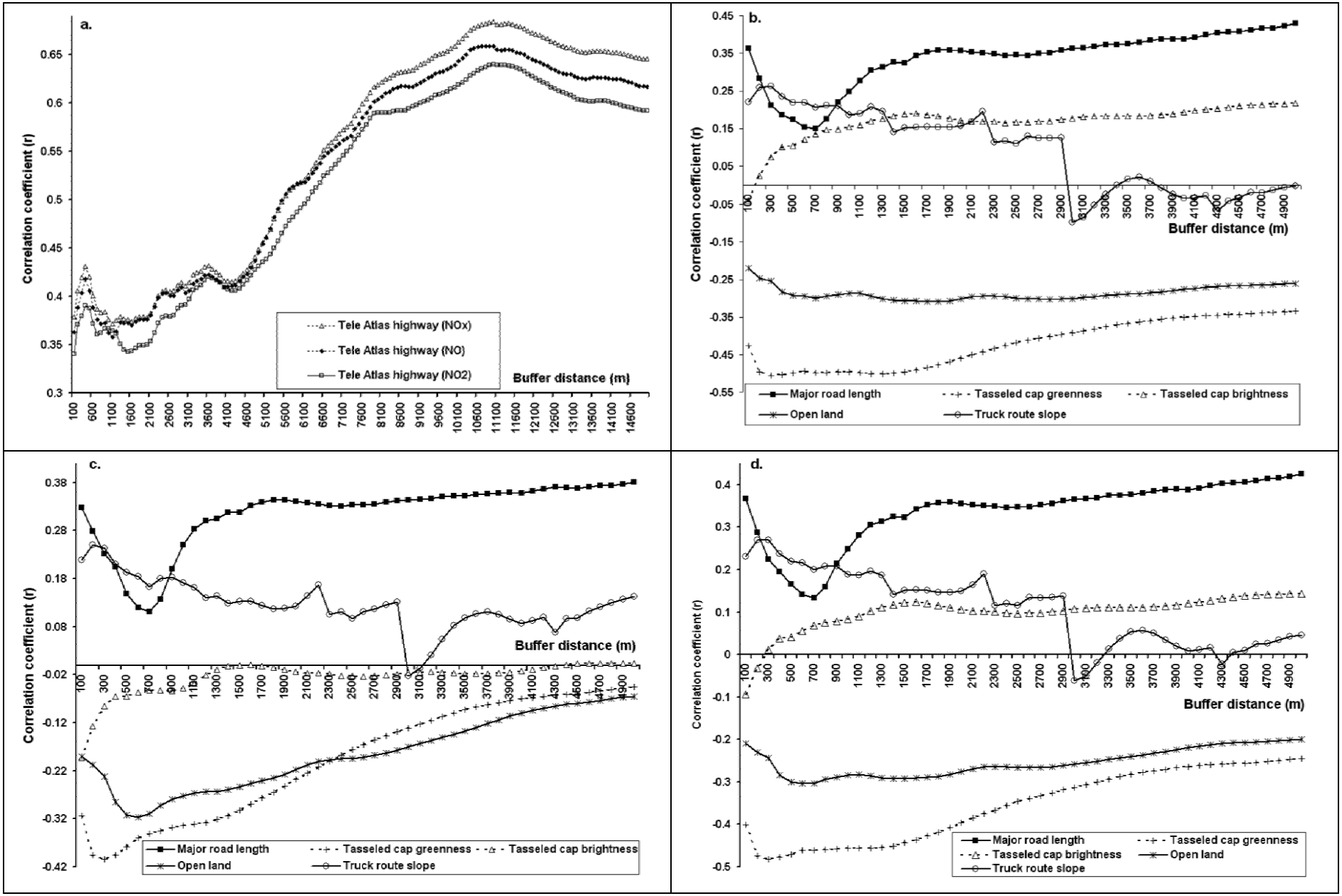


**Figure 6a-6d. Distance decay curves of correlations between selected spatial covariates and measured air pollution concentrations (6a for traffic volumes - total vehicle miles traveled, 6b for NO, 6c for $NO_2$ and 6d for $NO_x$**

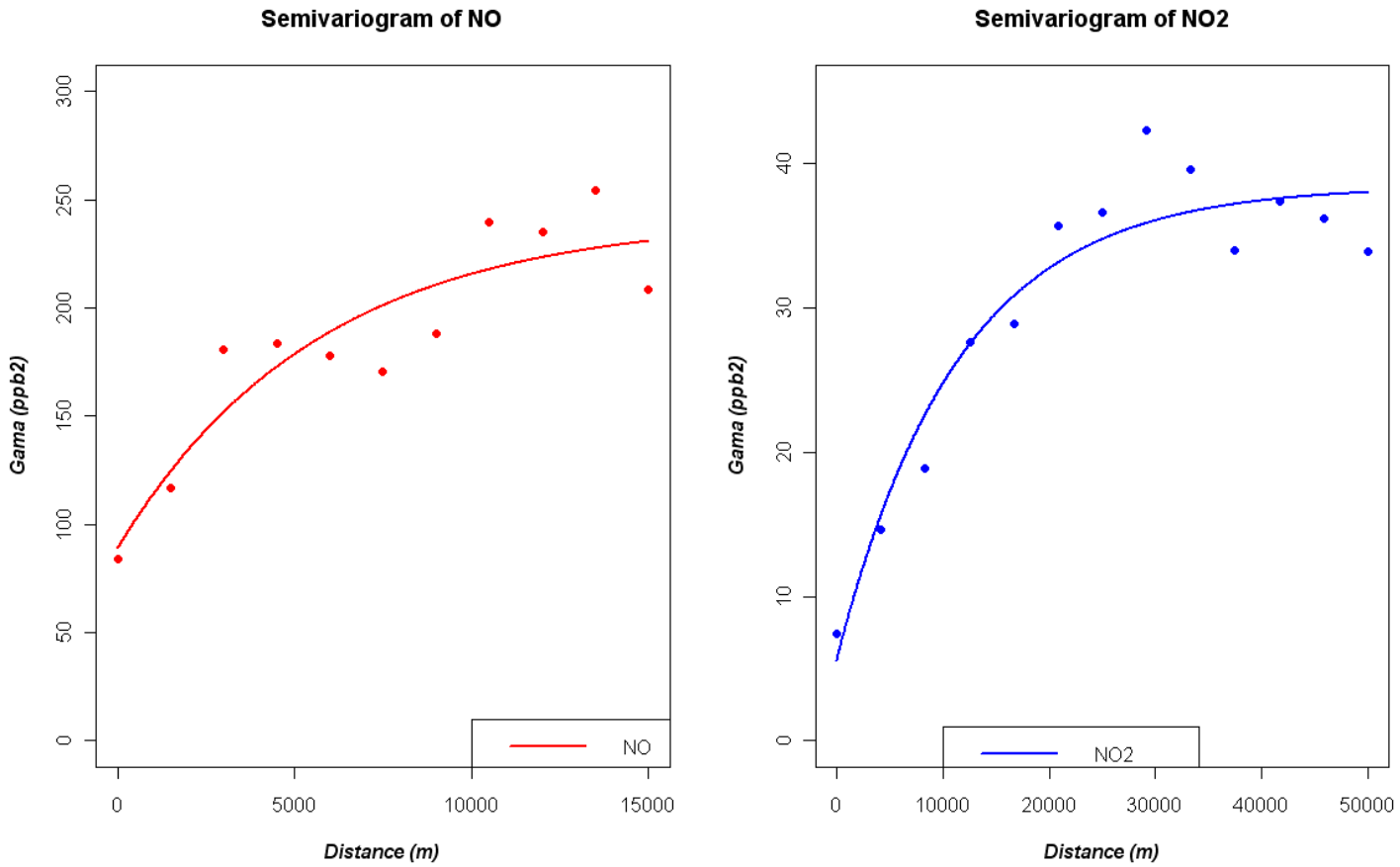


**Figure 7. Semivariograms of NO and $NO_2$ based on measurements from the 201 monitoring sites**

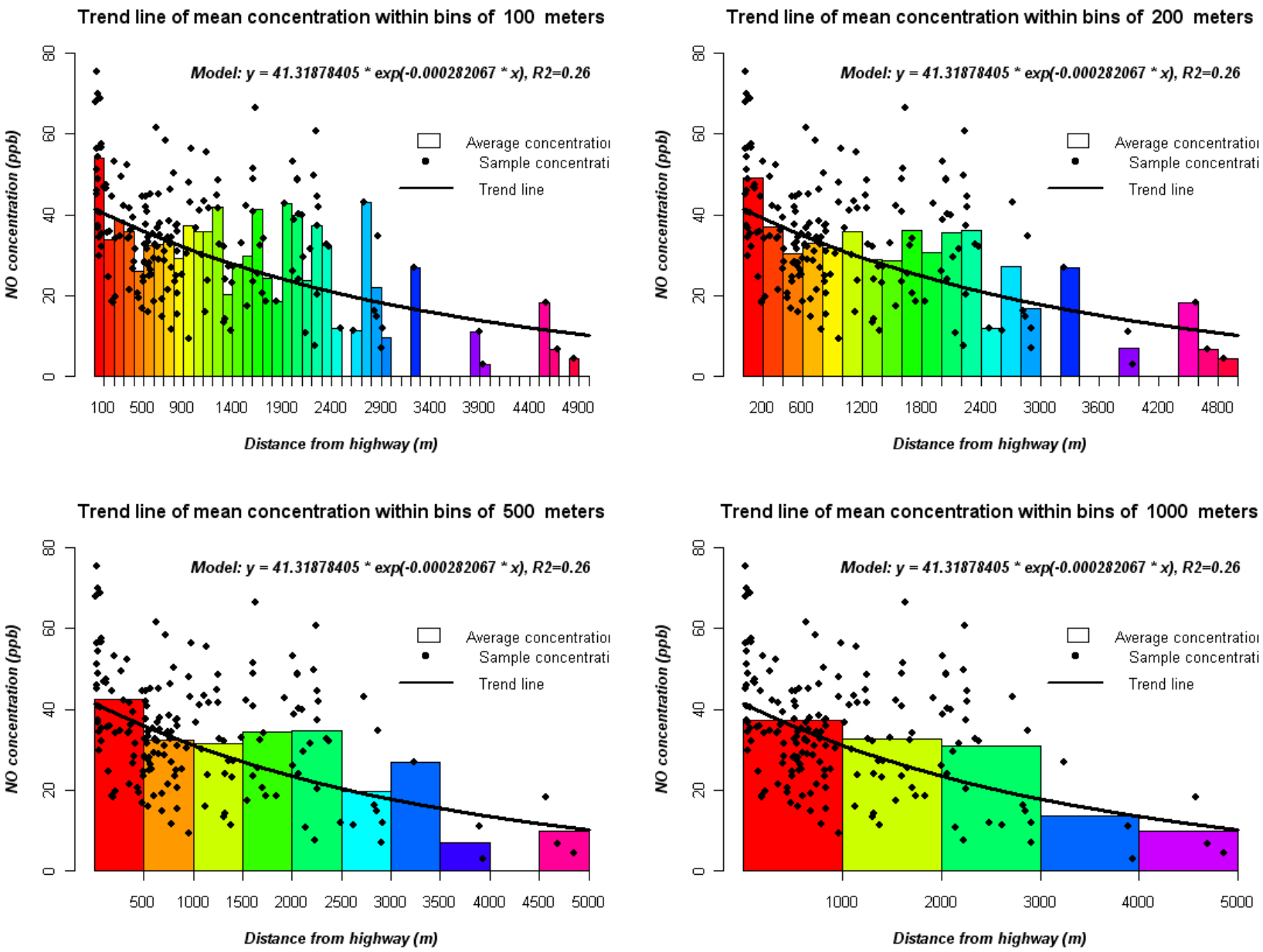


**Figure 9. Distance decay of NO concentrations further away from highways (A1 and A2) based on 201 monitoring sites in the LA metropolitan area**

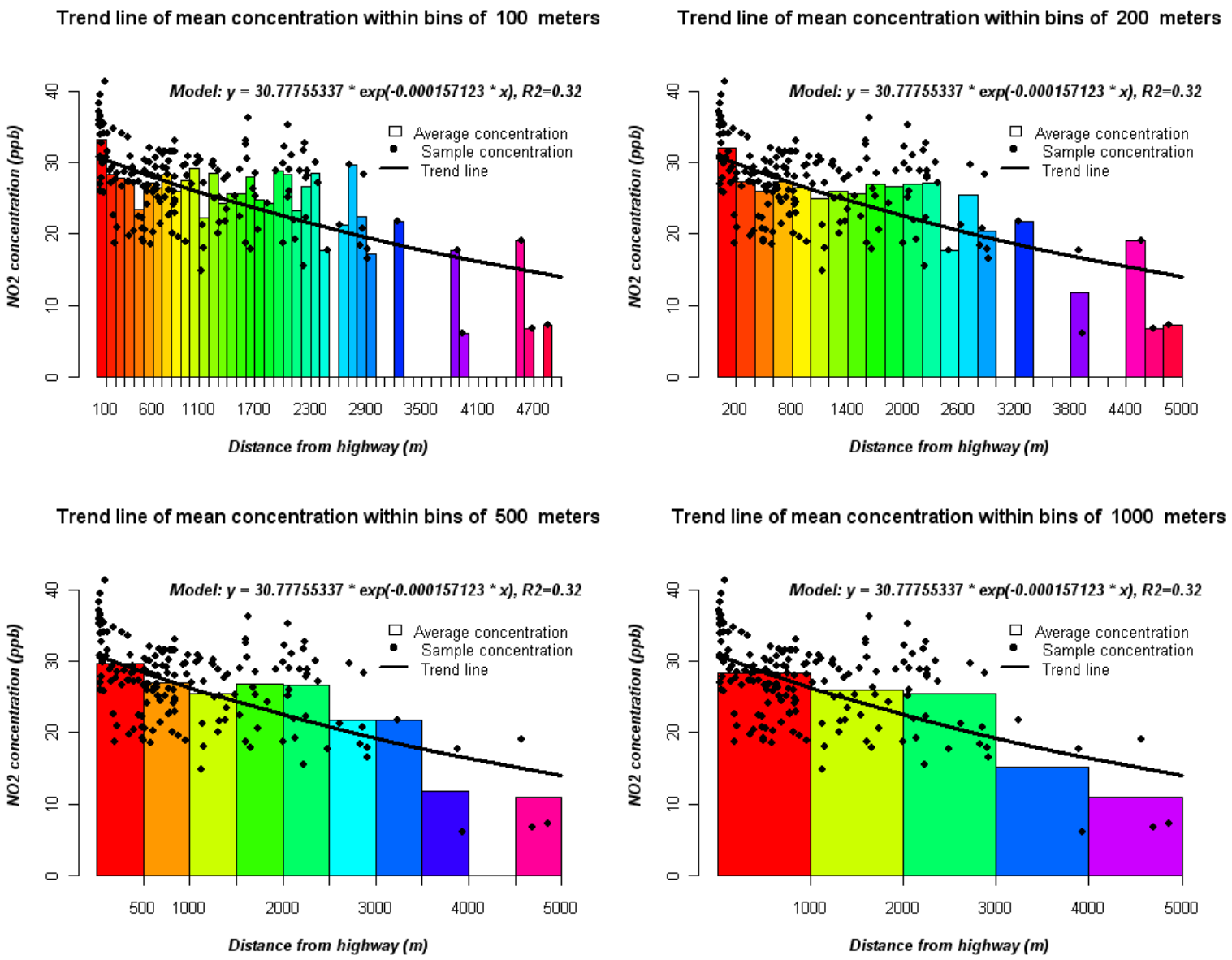


**Figure 10. Distance decay of $NO_2$ concentrations further away from highways (A1 and A2) based on 201 monitoring sites in the LA metropolitan area**

optimized models explained 81, 86 and 85% model variance for NO, $NO_2$ and $NO_x$, respectively. All the chosen spatial covariates were statistically significant at a 0.05 level with accepted signs of correlation. The average Variance Inflation Factor (VIF) for NO, $NO_2$ and $NO_x$ were 1.25, 1.31 and 1.22, respectively, with the maximum VIF being 1.56, demonstrating a lack of significant collinearity between the chosen spatial covariates. The prediction scatter plots displayed in Figure 11a (for NO), 11b ($NO_2$) and 11c ($NO_x$) further demonstrate that the prediction models were not influenced by significant outliers and the model prediction residuals were normally distributed.

The maximum Cook's distance for NO, $NO_2$ and $NO_x$ was 0.10, 0.36 and 0.23, respectively, also confirming the absence of influential outliers in each model. After selecting these parsimonious models, we tested for spatial autocorrelation based on first and second order Thiessen polygon connectivity matrices with the Moran's *I*. There was significant autocorrelation with the first order matrix in all models, but not in the second order tests (with *I* being equal to -0.008 ($p = 0.48$), -0.023 ($p = 0.35$) and 0.031 ($p = 0.18$) for NO, $NO_2$ and $NO_x$ model residuals). The ADDRESS models with an extra cluster parameter and the GEE models (Table 6 second and third columns) showed similar results to corresponding ADDRESS models that did not incorporate adjustment for clustering, except some small changes to the standard errors.

In our cross-validation models (Figure 11d, 11e and 11f), the 16 randomly picked samples explained 91, 87 and 92% of the model variances for NO, $NO_2$ and $NO_x$, respectively (Table 6). The Chow test showed that there was no significant difference between the models with the full and half dataset for NO, $NO_2$ and $NO_x$, indicating model stability to subset selection.
The final prediction surfaces for NO, $NO_2$ and $NO_x$ are displayed in Figures 12a-12d. Figure 12a (for NO) and 12d (for $NO_x$) show similar concentration patterns, i.e., higher NO and $NO_x$ levels near to emission sources such as highways and industrial land use, while the predicted $NO_2$ concentration surface (Figure 12b) is smoother overall, with highest concentrations in the northern and eastern parts of the city, and lower concentrations in the western areas. The $NO_2$ gradients reflect importance of the onshore sea breeze and secondary formation of $NO_2$ associated with inland air transportation processes in the LA Basin.

To test whether removing the influence from the extended 11 km buffer distance would maintain similar prediction powers, we developed another set of models. Specifically, these models were re-run with all the spatial variables as the final "optimized" models with corresponding buffer distances except the TeleAtlas traffic counts on highway and major roads within 11000 m (Figure 12c). The prediction powers for NO, $NO_2$ and $NO_x$ in these new models that ignored traffic within a buffer distance greater than 5000 m were 0.64, 0.78 and 0.68, respectively, i.e. comparable to previously published LUR models but much lower than the optimized models developed here. Thus, while previous LUR models successfully predicted local sources of air pollution, they paid little attention to the influence of background sources (e.g., at buffer distances >5 km), that we found important in a mega city like L.A. However, we also extracted exposure estimates from the LUR models excluding traffic at 11 km for LA FANS participants as a sensitivity analysis. Although incorporating the influence of traffic from roadways at distances up to 11 km away improved the prediction ability for models of NO, $NO_x$ and $NO_2$, particle measurement data collected in L.A. suggest concentrations of ultrafine particles (UF, <0.1 microns in aerodynamic diameter) drop off rapidly at approximately 150 meters from major roadways reaching background concentrations at approximately 300 meters.[8,9] Thus, utilizing models which only incorporate the influence of traffic within smaller

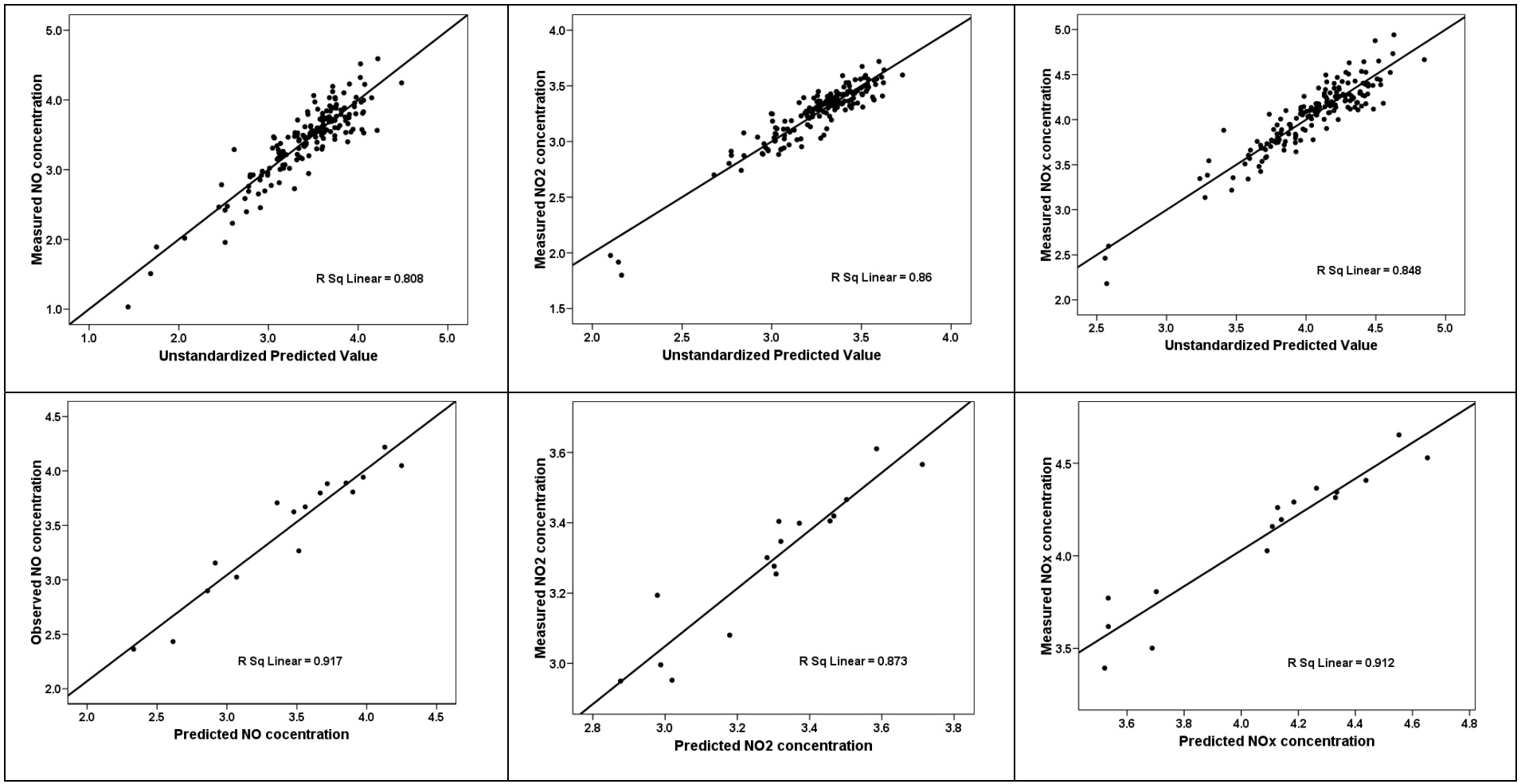


**Figure 11. Model predictions of natural log-transformed NO, $NO_2$ and $NO_x$ (11a,11b and 11c) and corresponding cross-validation results (11d, 11e and 11f)**

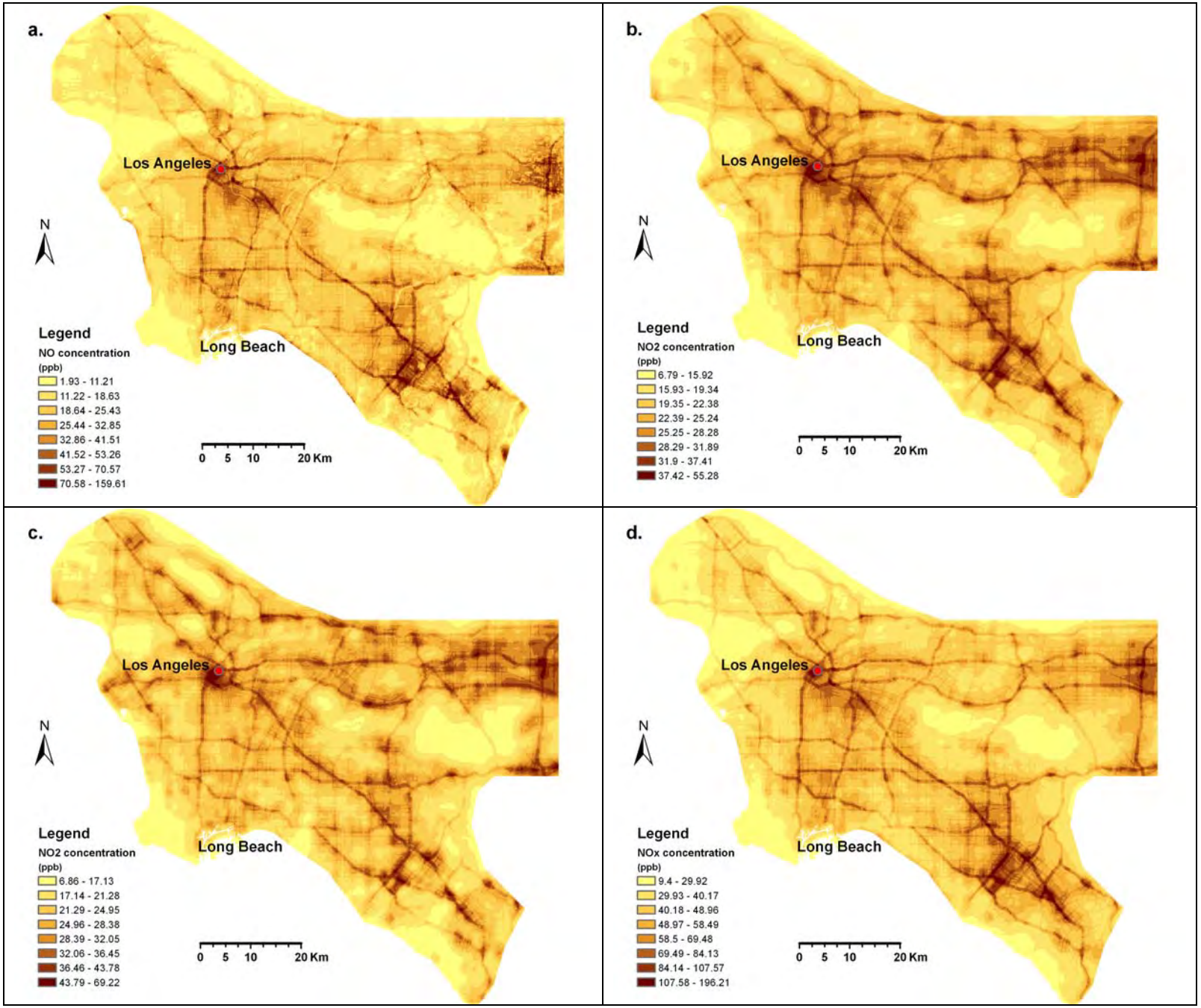


**Figure 12. Model prediction surfaces of NO (12a), $NO_2$ (12b, 12c) and $NO_x$ (12d) through an ADDRESS selection process (12b with and 12c without buffer distance within 11 km)**

**Table 6. Model prediction results using ADDRESS model, ADDRESS model with clustering considered, and GEE model for NO, $NO_2$ and $NO_x$.**

| Pollutant | Variable | ADDRESS model* | | | | | ADDRESS model with clustering** | | | | GEE model*** | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | Coef. | Std. Err. | t | P>\|t\| | VIF* | Coef. | Std. Err. | t | P>\|t\| | Coef. | Std. Err. | t | P>\|t\| |
| 6a. NO | Intercept | -2.6407240 | 0.654861 | -4.03 | 0.000 | | -2.6407240 | 0.792661 | -3.33 | 0.001 | -2.6407240 | 0.634952 | -4.16 | 0.000 |
| | TeleAtlas traffic highway and major roads (11000 m) | 0.00000003 | 0.000000 | 11.93 | 0.000 | 1.35 | 0.00000003 | 0.000000 | 10.58 | 0.000 | 0.00000003 | 0.000000 | 12.30 | 0.000 |
| | TeleAtlas traffic all roads (400 m) | 0.00000210 | 0.000000 | 6.75 | 0.000 | 1.27 | 0.00000210 | 0.000000 | 5.91 | 0.000 | 0.00000210 | 0.000000 | 6.96 | 0.000 |
| | Distance to truck routes (m) | -0.00003880 | 0.000013 | -3.06 | 0.003 | 1.49 | -0.00003880 | 0.000014 | -2.87 | 0.005 | -0.00003880 | 0.000012 | -3.15 | 0.002 |
| | Major road (100 m) | 0.00053750 | 0.000143 | 3.77 | 0.000 | 1.13 | 0.00053750 | 0.000178 | 3.01 | 0.004 | 0.00053750 | 0.000138 | 3.89 | 0.000 |
| | Industrial (2700 m) | 0.00036130 | 0.000084 | 4.29 | 0.000 | 1.20 | 0.00036130 | 0.000089 | 4.08 | 0.000 | 0.00036130 | 0.000082 | 4.42 | 0.000 |
| | Commercial (1200 m) | 0.00277730 | 0.000533 | 5.21 | 0.000 | 1.19 | 0.00277730 | 0.000514 | 5.40 | 0.000 | 0.00277730 | 0.000517 | 5.37 | 0.000 |
| | Soil brightness (700 m) | 0.01005310 | 0.001727 | 5.82 | 0.000 | 1.32 | 0.01005310 | 0.001935 | 5.20 | 0.000 | 0.01005310 | 0.001674 | 6.00 | 0.000 |
| | X coordinate | 0.00000660 | 0.000001 | 5.76 | 0.000 | 1.09 | 0.00000660 | 0.000001 | 4.80 | 0.000 | 0.00000660 | 0.000001 | 5.94 | 0.000 |
| | Open (100 m) | -0.1542625 | 0.049998 | -3.09 | 0.002 | 1.20 | -0.1542625 | 0.047074 | -3.28 | 0.002 | -0.1542625 | 0.048478 | -3.18 | 0.001 |
| | $R^2$ (p) \| $R^2$ (p)**** | 0.81 (< 0.0001) \| 0.92 (< 0.0001) | | | | | 0.81 (< 0.0001) | | | | | | | |
| 6b. $NO_2$ | Intercept | -11.282530 | 2.443303 | -4.62 | 0.000 | | -11.282530 | 3.725334 | -3.03 | 0.003 | -11.2825300 | 2.369021 | -4.76 | 0.000 |
| | TeleAtlas traffic highway and major roads (11000 m) | 0.00000001 | 0.000000 | 9.72 | 0.000 | 1.44 | 0.00000001 | 0.000000 | 7.23 | 0.000 | 0.00000001 | 0.000000 | 10.03 | 0.000 |
| | TeleAtlas traffic all roads (400 m) | 0.00000072 | 0.000000 | 5.26 | 0.000 | 1.28 | 0.00000072 | 0.000000 | 4.04 | 0.000 | 0.00000072 | 0.000000 | 5.43 | 0.000 |
| | Distance to truck routes (m) | -0.0000439 | 0.000006 | -7.90 | 0.000 | 1.49 | -0.00004390 | 0.000014 | -3.04 | 0.003 | -0.00004390 | 0.000005 | -8.15 | 0.000 |
| | Major road (100) | 0.00018990 | 0.000063 | 3.01 | 0.003 | 1.15 | 0.00018990 | 0.000070 | 2.72 | 0.008 | 0.00018990 | 0.000061 | 3.11 | 0.002 |
| | Local road (1400) | 0.00000234 | 0.000001 | 2.82 | 0.005 | 1.56 | 0.00000234 | 0.000001 | 2.73 | 0.008 | 0.00000234 | 0.000001 | 2.91 | 0.004 |
| | Industrial (1700 m) | 0.00059240 | 0.000096 | 6.16 | 0.000 | 1.36 | 0.00059240 | 0.000145 | 4.10 | 0.000 | 0.00059240 | 0.000093 | 6.35 | 0.000 |
| | Commercial (1000 m) | 0.00261960 | 0.000308 | 8.50 | 0.000 | 1.16 | 0.00261960 | 0.000376 | 6.96 | 0.000 | 0.00261960 | 0.000299 | 8.77 | 0.000 |
| | X coordinate (m) | 0.00000515 | 0.000001 | 10.11 | 0.000 | 1.12 | 0.00000515 | 0.000001 | 7.38 | 0.000 | 0.00000515 | 0.000000 | 10.43 | 0.000 |
| | Y coordinate (m) | 0.00000316 | 0.000001 | 4.98 | 0.000 | 1.20 | 0.00000316 | 0.000001 | 3.26 | 0.002 | 0.00000316 | 0.000001 | 5.14 | 0.000 |
| | $R^2$ (p) \| $R^2$ (p) | 0.86 (< 0.0001) \| 0.87 (< 0.0001) | | | | | 0.86 (< 0.0001) | | | | | | | |
| 6c. $NO_x$ | Intercept | -0.0590325 | 0.429163 | -0.14 | 0.891 | | -0.0590325 | 0.543035 | -0.11 | 0.914 | -0.05903250 | 0.417438 | -0.14 | 0.888 |
| | TeleAtlas traffic highway and major roads (11000 m) | 0.00000002 | 0.000000 | 13.13 | 0.000 | 1.30 | 0.00000002 | 0.000000 | 10.83 | 0.000 | 0.00000002 | 0.000000 | 13.5 | 0.000 |
| | TeleAtlas traffic all roads (400 m) | 0.00000144 | 0.000000 | 7.11 | 0.000 | 1.27 | 0.00000144 | 0.000000 | 5.20 | 0.000 | 0.00000144 | 0.000000 | 7.31 | 0.000 |
| | TeleAtlas traffic major road (100 m) | 0.00002710 | 0.000007 | 3.92 | 0.000 | 1.14 | 0.00002710 | 0.000008 | 3.22 | 0.002 | 0.00002710 | 0.000007 | 4.03 | 0.000 |
| | Distance to truck routes (m) | -0.0000450 | 0.000008 | -5.47 | 0.000 | 1.47 | -0.0000450 | 0.000011 | -3.94 | 0.000 | -0.0000450 | 0.000008 | -5.63 | 0.000 |
| | Industrial (2700 m) | 0.00029010 | 0.000054 | 5.38 | 0.000 | 1.16 | 0.00029010 | 0.000070 | 4.15 | 0.000 | 0.00029010 | 0.000053 | 5.53 | 0.000 |
| | Commercial (1000 m) | 0.00328070 | 0.000450 | 7.29 | 0.000 | 1.12 | 0.00328070 | 0.000488 | 6.72 | 0.000 | 0.00328070 | 0.000438 | 7.49 | 0.000 |
| | Soil brightness (1700 m) | 0.00442720 | 0.001104 | 4.01 | 0.000 | 1.19 | 0.00442720 | 0.001500 | 2.95 | 0.004 | 0.00442720 | 0.001074 | 4.12 | 0.000 |
| | X coordinate (m) | 0.00000572 | 0.000001 | 7.62 | 0.000 | 1.10 | 0.00000572 | 0.000001 | 6.67 | 0.000 | 0.00000572 | 0.000001 | 7.83 | 0.000 |
| | $R^2$ (p) \| $R^2$ (p) | 0.85 (< 0.0001) \| 0.92 (< 0.0001) | | | | | 0.85 (< 0.0001) | | | | | | | |

* ADDRESS model: An optimized distance decay model selection strategy for our land use regression models. VIF = variance inflation factor. ** For clustering analysis, observations were grouped using census tract. We assumed that measurements from multiple sites within a census tract might be correlated but, across census tracts, they were uncorrelated.*** GEE model: Generalized estimation equation model to analyze correlated data within census tracts.**** R2 (p) | R2 (p): the left side part is for model prediction power and right side for cross-validation result.

buffer distances may provide a better marker of exposure to ultrafine particles, even though such models are less predictive of NO, $NO_2$ and $NO_x$ specifically. Use of exposure predictions from models including long-range and short-range traffic impacts (i.e., the "optimized" models) and including only shorter-range traffic impacts (i.e., the "more local traffic" models) may therefore provide further clues about the importance of different traffic components to respiratory health.

Most previous land use regression models[29,132] have included population density as a predictor. However, in our modeling process, population density was not included for all three prediction models because of the high VIFs (Variance Inflation Factor >2). Our sensitivity analyses demonstrated that if population density was added, the model prediction power increased only by 0.50%, 1.28% and 0.71%, respectively, for NO, $NO_2$ and $NO_x$; thus, omitting population density from our LUR did not substantially decrease the predictive power of our models.

**$O_3$ and $PM_{2.5}$ Kriging Results**

Figures 13-15 illustrate the kriged $PM_{2.5}$ and $O_3$ surfaces for the L.A. region. About 50% of the modeled $PM_{2.5}$ surface has errors that are less than 15% of the monitored values, while 67% of the surface lies within 20% of the monitored values. For the most part, absolute standard errors in $PM_{2.5}$ estimates for the densely populated areas of the L.A. FANS study are less than 3 $\mu g/m^3$. For $O_3$, actual measured values within the modeled study area range from 0.050 ppm to 0.138 ppm with a mean of 0.095 ppm. The average standard error of the modeled values compared to actual values is 0.012 ppm.

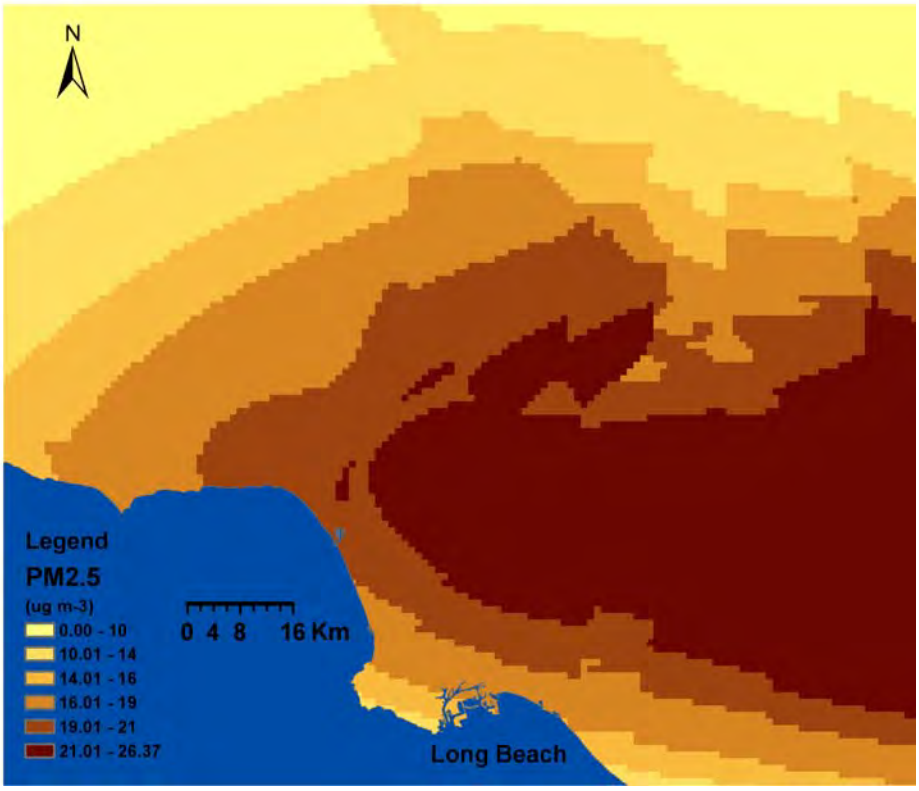


**Figure 13. $PM_{2.5}$ surface through kriging for the LA Basin.**

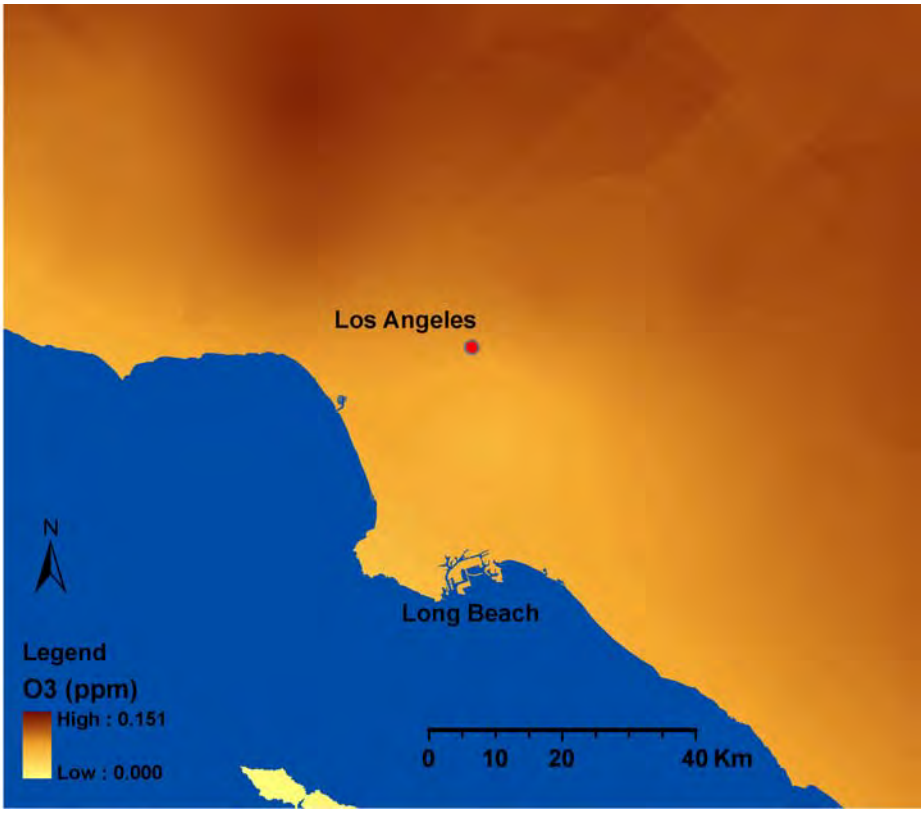


**Figure 14. $O_3$ ordinary kriging surface for the LA Basin**

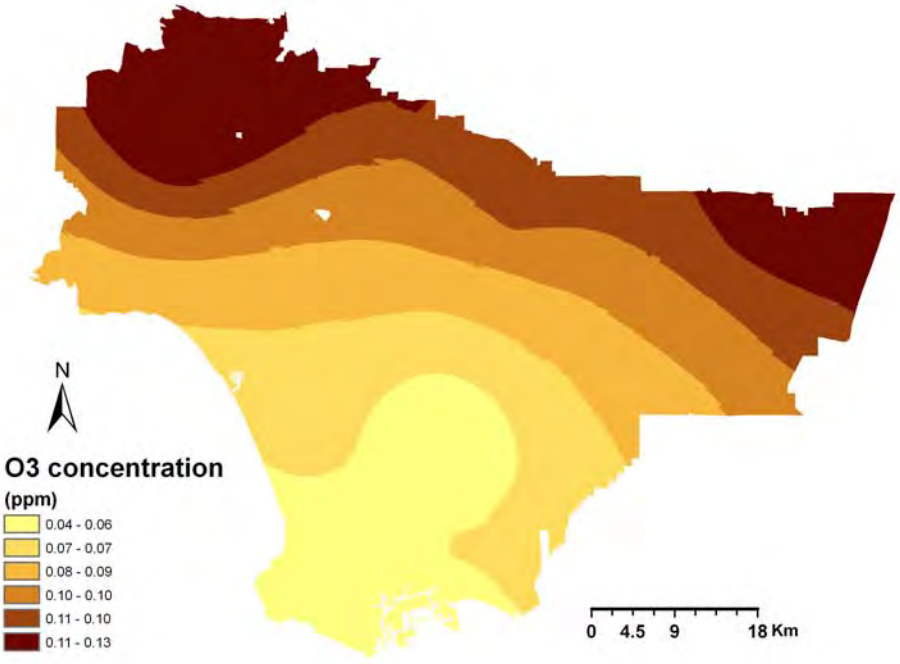


**Figure 15. $O_3$ ordinary kriging surface for the LA Basin (close up of urban core area)**

**Exposure Estimates for L.A. FANS Participants**

For the 1,387 children for whom health and covariate data were collected as part of L.A. FANS-2, residential information (addresses and start and end dates) was obtained for 1,685 homes. Of these homes, 1,569 (93.1%) were successfully geocoded. Reasons for unsuccessful geocoding of homes include errors in or missing address data, inability to match reported address to the street map used for geocoding, or residence locations outside of California. Of 1,266 children who had started school, for 1,253 (99%) we were able to successfully geocode current school locations, however, the percent successfully geocoded declined to approximately 95% when considering schools attend 1 and 2 years prior to interview and by approximately 75% when considering schools attended 5 years prior to interview. Tables 7 and 8 provide a breakdown of the number of child respondents with available residential and school air pollution exposure metrics by exposure period, respectively. Table 9 provides information on the distribution of and correlations between residential air pollution metrics for all child respondents for whom a questionnaire was completed (n=1,387). We present values for the 12-month prior to interview averages; distributions and correlations for the other time periods considered were very similar. The final, optimized model NO, $NO_2$ and $NO_x$ exposure estimates were strongly positively correlated (r~0.9 or greater). The NO, $NO_2$ and $NO_x$ values from the "more local traffic" model were also strongly positively correlated with each other and with the full model values, with the lowest correlations being estimated between the local traffic $NO_2$ and full model NO (r=0.75) and $NO_x$ (r=0.83) values. Thus, we consider these three pollutants as markers of the correlated suite of pollutants in traffic exhaust. Ozone levels were moderately negatively correlated with LUR values for NO and $NO_x$ (r~-0.3-0.4) and only marginally negatively associated with LUR $NO_2$ values (r~-0.18 and -0.08 for the full and local traffic models, respectively). Exposure estimates for $PM_{2.5}$ were positively correlated with the full model estimates for NO (r=0.51), $NO_2$ (r=0.68) and $NO_x$ (r=0.59), but less correlated with the local traffic model NO (r=0.37) and $NO_x$ (0.44) values. The stronger correlations between $PM_{2.5}$ and full model estimates for NO and $NO_x$ (versus the more local traffic values) and with $NO_2$ overall, suggest the importance of both local and background contributions to $PM_{2.5}$ levels in the basin. Ozone and $PM_{2.5}$ exposure values were moderately negatively correlated (r~-0.4). Distributions and correlations between air pollution metrics for the 890 children with at least one acceptable spirometry curve were very similar to those for all child respondents (Table 10). Ozone was slightly more negatively correlated with the other exposure metrics in this group. The 395 children with 3 acceptable and reproducible spirometry curves had slightly lower interquartile ranges for the LUR pollution metrics (Table 11), but a greater interquartile range in $O_3$ exposure estimates, indicating a greater spread in the distribution of values for this pollutant for these children. Positive correlations between LUR and $PM_{2.5}$ metrics were slightly lower, while negative correlations between $O_3$ and these metrics were similar to those for the 890 children with 1 acceptable curve and slightly stronger compared to all children. Correlations between residential pollution exposure metrics and air pollution exposure metrics taking into account school locations (home and school locations taken together and weighting for the time spent at school and at home) were greater than 0.9 across all pollutants and time periods (results not shown).

**Characteristics of L.A. FAN-2 Study Population**

A total of 939 families with children were interviewed as part of L.A. FANS-2; parents in these households completed questionnaires regarding 1,387 children. Of these children, 1,091

**Table 7. Description of Available Residential Air Pollution Estimates by Exposure Period for n=1,387 Children included in L.A. FANS-2**

| Air Pollution Averaging period | No. (%) of subjects with NO, $NO_2$, $NO_x$ and $O_3$ values | No. (%) of subjects with NO, $NO_2$, $NO_x$, $O_3$ and $PM_{2.5}$ values | No. of subjects with LUR or $O_3$ values missing for one or more homes[1] | No. of additional subjects missing $PM_{2.5}$ values for one or more homes[2] |
|---|---|---|---|---|
| Current home | 1378 (99%) | 1364 (98%) | 9 | 14 |
| 12 months prior to interview | 1311 (95%) | 1301 (94%) | 76 | 10 |
| 24 months prior to interview | 1288 (93%) | 1278 (92%) | 99 | 10 |
| 5 years prior to interview | 1223 (88%) | 1213 (88%) | 164 | 10 |

(1) Subjects are missing data because: (a) one or more homes not geocoded and/or (b) homes dates (i.e., dates in each home) do not span this period or are missing data and/or (c) one or more homes fell outside the modeling domain.
(2) These are homes were successfully geocoded, but fell outside the modeling domain for the $PM_{2.5}$ exposure surface which covered a slightly different area than the LUR and $O_3$ modeling domains.

**Table 8. Description of Available School Air Pollution Estimates by Exposure Period for n=1,387 Children included in L.A. FANS-2**

| Air Pollution Averaging period | No. (%) of subjects with NO, $NO_2$, $NO_x$ and $O_3$ values[1] | No. (%) of subjects with NO, $NO_2$, $NO_x$, $O_3$ and $PM_{2.5}$ values[1] | No. of subjects with LUR or $O_3$ values missing for one or more schools[1] | No. of additional subjects missing $PM_{2.5}$ values for one or more schools[2] |
|---|---|---|---|---|
| Current school | 1253 (99%) | 1237 (98%) | 13 | 16 |
| 12 months prior to interview | 1220 (96%) | 1206 (95%) | 46 | 14 |
| 24 months prior to interview | 1180 (93%) | 1170 (92%) | 86 | 10 |
| 5 years prior to interview | 912 (72%) | 903 (71%) | 354 | 9 |

(1) Percentages are based on a total of 1,266 children reported as having started school. Subjects are missing data because: (a) one or more schools not geocoded and/or (b) school dates (i.e., dates in each school) do not span this period or are missing data and/or (c) one or more schools fell outside the modeling domain.
(2) These schools were successfully geocoded, but fell outside the modeling domain for the $PM_{2.5}$ exposure surface which covered a slightly different area than the LUR and $O_3$ modeling domains.

**Table 9. Pollutant Disributions and Pearson Correlation Coefficients for LUR, $O_3$ and $PM_{2.5}$ Annual Averages (12 months prior to interview) for n=1,387 Children with Questionnaire Data**

| Pollutant | Median (range) | IQR | Pearson Correlation Coefficients | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | | | NO | $NO_2$ | $NO_x$ | NO-LT[2] | $NO_2$-LT | $NO_x$-LT | $O_3$ |
| NO (ppb)[1] | 23.5 (2.5-69.0) | 10.7 | 1.0 | | | | | | |
| $NO_2$ (ppb)[1] | 23.7 (6.2-36.9) | 6.1 | 0.86 | 1.0 | | | | | |
| $NO_x$ (ppb)[1] | 47.9 (11.3-97.5) | 16.9 | 0.97 | 0.93 | 1.0 | | | | |
| NO-LT (ppb)[2] | 28.7 (2.8-73.5) | 11.8 | 0.86 | 0.82 | 0.88 | 1.0 | | | |
| $NO_2$-LT (ppb) | 25.6 (6.0-43.0) | 6.1 | 0.75 | 0.95 | 0.83 | 0.85 | 1.0 | | |
| $NO_x$-LT (ppb) | 55.3 (6.3-126.2) | 16.9 | 0.83 | 0.87 | 0.89 | 0.98 | 0.89 | 1.0 | |
| $O_3$ (ppb)[3] | 71.1 (46.2-129.8) | 29.1 | -0.35 | -0.18 | -0.36 | -0.27 | -0.08 | -0.27 | 1.0 |
| $PM_{2.5}$ ($\mu m/m^3$) | 21.5 (8.5-23.7) | 2.4 | 0.51 | 0.68 | 0.59 | 0.37 | 0.56 | 0.44 | -0.36 |

(1) Estimates are from the final, optimized LUR model.
(2) "LT" stands for "more local traffic impact"; these estimates are from the LUR model excluding traffic within 11 km buffers.
(3) Kriged $O_3$ estimates based on 8-hour maximum concentrations.

were panel participants (i.e., participated in both surveys) while 296 were new entrants to the study. Of the 1,387 interviewed children, 1,225 also completed some part of the health measures module and 1,070 participated in spirometry. Table 12 provides demographic information for all child respondents (n=1,387) and for those children with one or more acceptable spirometry curves (n=890) and for children with three acceptable and reproducible spirometry curves (n=395). The majority of children were 10 years of age or older (65%) and of Hispanic race/ethnicity (66%). Approximately 45% of children were overweight based on 2000 U.S. CDC BMI-for-age charts. Most children were reported to have health insurance in the previous month and a usual source of sick care. One-quarter of the families reported an income less than $20,000, which is approximately below the Federal Poverty Limit for a family of four for the period 2006-2008. More than half of the families rented their home (58%) and 58% of PCG's had a high school education or less. The majority of PCG's were born outside of the US (62%). Percentagebreakdowns for other family and neighborhood characteristics considered in analyses are presented in Table 12.

A greater percentage of boys and children 10 years of age or older had one or more acceptable spirometry curves (Table 12). Children with three acceptable and reproducible curves were less likely to be Hispanic compared to those who had one or more acceptable curves and to all children who completed the survey. Children with one or more acceptable curves were also more likely to have respiratory symptoms based on parental report (i.e., doctor-diagnosed asthma, wheeze in the past 12 months, and asthma medication use for asthma or wheeze in the past 12 months), and a greater percentage came from families with incomes >$65,000, from families that were homeowners, and from families where the PCGs were born in the US compared to all children who completed the survey. A greater percentage of children with three acceptable and reproducible curves had PCGs with more than a high school education compared to all children and those with one or more acceptable curves. A greater percentage of adults in these families reported they were satisfied with their neighborhood, and reported higher levels of neighborhood cohesion and support. Higher percentages lived in neighborhoods (census tracts) with higher ratings of neighborhood cohesion and safety (based on averages of adult responses) and with less economic disadvantage. Overall, these differences were more pronounced for children with three acceptable and reproducible curves than for those with one or more acceptable curves.

**Table 12. Demographic Characteristics (Number, Percent) of L.A. FANS-2 Child Participants**

| Parameter | All Subjects with Questionnaire Data (n=1,387) | Subjects with One or More Acceptable Spirometry Curve (n=890) | Subjects with Three Acceptable and Reproducible Spirometry Curves (n=395) |
|---|---|---|---|
| *Individual Level* | | | |
| Gender | | | |
| Female | 716 (51.6) | 404 (45.4) | 174 (44.1) |
| Male | 671 (48.4) | 486 (54.6) | 221 (55.9) |
| Age (years) | | | |
| <5 | 100 (7.2) | -- | -- |
| 5-<10 | 379 (27.3) | 222 (25.0) | 78 (19.8) |
| 10-<15 | 551 (39.7) | 415 (46.6) | 190 (48.1) |
| ≥15 | 357 (25.7) | 253 (28.4) | 127 (32.1) |
| Race/ethnicity | | | |
| Non-Hispanic White | 262 (18.9) | 165 (18.6) | 73 (18.5) |
| Hispanic | 921 (66.4) | 594 (66.7) | 246 (62.3) |
| African American | 105 (7.6) | 65 (7.3) | 36 (9.1) |
| Asian/Other | 99 (7.1) | 66 (7.4) | 40 (10.1) |
| Health insurance during past month | | | |
| Yes | 1187 (85.8) | 758 (85.3) | 335 (84.8) |
| No | 196 (14.2) | 131 (14.7) | 60 (15.2) |
| Missing | 4 | 1 | |
| Usual source of sick care | | | |
| Yes | 1298 (93.7) | 833 (93.7) | 370 (93.9) |
| No | 88 (6.4) | 56 (6.3) | 24 (6.1) |
| Missing | 1 | 1 | 1 |
| Overweight[1] | | | |
| Yes | 531 (44.7) | 411 (46.6) | 179 (45.8) |
| No | 658 (55.3) | 472 (53.4) | 212 (54.2) |
| Missing | 198 | 7 | 4 |
| Doctor-diagnosed asthma (ever) | | | |
| Yes | 191 (13.8) | 131 (14.7) | 67 (17.0) |
| No | 1196 (86.2) | 759 (85.3) | 328 (83.0) |
| Age of asthma diagnosis (years) – only for diagnosed asthmatics | | | |
| ≤5 | 125 (67.6) | 84 (66.1) | 38 (58.5) |
| 6-<10 | 33 (17.8) | 25 (19.7) | 17 (26.2) |
| 10-<15 | 22 (11.9) | 15 (11.8) | 9 (13.8) |
| ≥15 | 5 (2.7) | 3 (2.4) | 1 (1.5) |
| Missing | 6 | 4 | 2 |
| Wheeze in past 12 months | | | |
| Yes | 145 (10.5) | 101 (11.4) | 54 (13.7) |
| No | 1242 (89.6) | 789 (88.6) | 341 (86.3) |
| Wheeze with any night waking in past 12 months | | | |
| Yes | 73 (5.3) | 52 (5.8) | 29 (7.3) |
| No | 1314 (94.7) | 838 (94.2) | 366 (92.7) |
| Medication use for asthma or wheeze in past 12 months | | | |
| Yes | 162 (11.7) | 106 (11.9) | 57 (14.4) |
| No | 1225 (88.3) | 784 (88.1) | 338 (85.6) |
| Sneezing or runny/block nose apart from cold in past 12 months | | | |
| Yes | 270 (19.5) | 179 (20.1) | 86 (21.8) |
| No | 1116 (80.5) | 710 (79.9) | 308 (78.2) |
| Missing | 1 | 1 | 1 |

| Parameter | All Subjects with Questionnaire Data (n=1,387) | Subjects with One or More Acceptable Spirometry Curve (n=890) | Subjects with Three Acceptable and Reproducible Spirometry Curves (n=395) |
|---|---|---|---|
| More than 3 doctor-diagnosed ear infections in 1 year | | | |
| Yes | 140 (10.1) | 94 (10.6) | 50 (12.7) |
| No | 1246 (89.9) | 795 (89.4) | 344 (87.3) |
| Missing | 1 | | 1 |
| ***Family Level*** | | | |
| Family Income (dollars) | | | |
| <20,000 | 310 (24.5) | 208 (24.9) | 91 (25.0) |
| 20,000-<35,000 | 321 (25.4) | 200 (24.0) | 70 (19.2) |
| 35,000-<65,000 | 318 (25.2) | 209 (25.0) | 95 (26.1) |
| ≥65,000 | 315 (24.9) | 218 (26.1) | 108 (29.7) |
| Missing | 123 | 55 | 31 |
| Homeowner | | | |
| Yes | 548 (41.6) | 359 (41.9) | 178 (46.8) |
| No | 768 (58.4) | 497 (58.1) | 202 (53.2) |
| Missing | 71 | 34 | 15 |
| PCG's education (years) | | | |
| <12 | 563 (40.8) | 362 (40.9) | 148 (37.6) |
| 12 | 242 (17.5) | 154 (17.4) | 67 (17.0) |
| >12 | 576 (41.7) | 369 (41.7) | 179 (45.4) |
| Missing | 6 | 5 | 1 |
| Foreign born status of PCG | | | |
| US born | 529 (38.1) | 336 (37.8) | 166 (42.0) |
| Foreign born | 858 (61.9) | 554 (62.3) | 229 (58.0) |
| PCG current marital status | | | |
| Married or living with partner | 1071 (77.2) | 689 (77.4) | 294 (74.4) |
| Unmarried/not living with partner | 316 (22.8) | 201 (22.6) | 101 (25.6) |
| Current household smokers | | | |
| Yes | 297 (21.4) | 190 (21.4) | 84 (21.3) |
| No | 1088 (78.6) | 698 (78.6) | 311 (78.7) |
| Missing | 2 | 2 | |
| PCG current smoking status | | | |
| Yes | 126 (9.1) | 85 (9.6) | 52 (13.2) |
| No | 1260 (90.9) | 804 (90.4) | 342 (86.8) |
| Missing | 1 | 1 | 1 |
| Current smoker in home or PCG smoker | | | |
| Yes | 325 (23.5) | 207 (23.3) | 97 (24.6) |
| No | 1059 (76.5) | 680 (76.7) | 297 (75.4) |
| Missing | 3 | 3 | 1 |
| Either parent has asthma | | | |
| Yes | 144 (10.7) | 98 (11.3) | 49 (12.7) |
| No | 1204 (89.3) | 770 (88.7) | 336 (87.3) |
| Missing | 39 | 22 | 10 |
| Mother has asthma | | | |
| Yes | 96 (7.0) | 62 (7.0) | 34 (8.7) |
| No | 1277 (93.0) | 823 (93.0) | 357 (91.3) |
| Missing | 14 | 5 | 4 |
| Gas appliance in home (stove, range or oven) | | | |
| Yes | 1287 (92.8) | 821 (92.2) | 370 (93.7) |
| No | 100 (7.2) | 69 (7.8) | 25 (6.3) |
| Gas appliance with pilot light (stove, range or oven) | | | |
| Yes | 579 (42.5) | 384 (43.7) | 172 (44.3) |
| No | 782 (57.5) | 494 (56.3) | 216 (55.7) |
| Missing | 26 | 12 | 7 |
| Pests in home in past 12 months | | | |
| Yes | 1047 (75.5) | 677 (76.1) | 301 (76.2) |
| No | 340 (24.5) | 213 (23.9) | 94 (23.8) |

| Parameter | All Subjects with Questionnaire Data (n=1,387) | Subjects with One or More Acceptable Spirometry Curve (n=890) | Subjects with Three Acceptable and Reproducible Spirometry Curves (n=395) |
|---|---|---|---|
| Cockroaches in home in past 12 months | | | |
| Yes | 404 (29.1) | 253 (28.4) | 100 (25.3) |
| No | 983 (70.9) | 637 (71.6) | 295 (74.7) |
| Mold in home in past 12 months | | | |
| Yes | 296 (21.4) | 185 (20.9) | 79 (20.1) |
| No | 1087 (78.6) | 702 (79.1) | 314 (79.9) |
| Missing | 4 | 3 | 2 |
| Furry pets in home | | | |
| Yes | 467 (34.7) | 311 (36.0) | 144 (37.4) |
| No | 879 (65.3) | 554 (64.0) | 241 (62.6) |
| Missing | 41 | 25 | 10 |
| Neighborhood satisfaction | | | |
| Very satisfied | 283 (24.3) | 179 (23.5) | 90 (26.6) |
| Satisfied or Neutral (if volunteered) | 705 (60.5) | 475 (62.2) | 201 (59.5) |
| Dissatisfied or Very dissatisfied | 178 (15.3) | 109 (14.3) | 47 (13.9) |
| Missing | 221 | 127 | 57 |
| How safe to walk alone after dark in this neighborhood | | | |
| Completely safe | 181 (15.6) | 121 (15.9) | 54 (16.0) |
| Fairly safe or Somewhat dangerous | 921 (79.5) | 600 (79.1) | 267 (79.0) |
| Extremely dangerous | 57 (4.9) | 38 (5.0) | 17 (5.0) |
| Missing | 228 | 131 | 57 |
| No. adults you recognize in neighborhood | | | |
| Many adults or most or all adults | 595 (51.0) | 396 (51.9) | 179 (53.0) |
| A few adults or no adults | 571 (49.0) | 367 (48.1) | 159 (47.0) |
| Missing | 221 | 127 | 57 |
| Neighborhood cohesion score[2] | | | |
| <2.52 (median) (higher) | 631 (54.7) | 421 (55.6) | 196 (58.2) |
| ≥2.52 | 523 (45.3) | 336 (44.4) | 141 (41.8) |
| Missing | 233 | 133 | 58 |
| No. relatives living in neighborhood | | | |
| Any | 468 (40.3) | 295 (38.8) | 125 (37.1) |
| None | 694 (59.7) | 465 (61.1) | 212 (62.9) |
| Missing | 233 | 130 | 58 |
| No. friends living in neighborhood | | | |
| Any | 804 (67.0) | 532 (69.7) | 232 (68.6) |
| None | 362 (31.1) | 231 (30.3) | 106 (31.4) |
| Missing | 221 | 127 | 57 |
| No. of neighbors talked to for 10 min in past 30 days | | | |
| Any | 1021 (87.6) | 671 (88.1) | 294 (87.2) |
| None | 144 (12.4) | 91 (11.9) | 43 (12.8) |
| Missing | 222 | 128 | 58 |
| No. groups participated in past 12 months | | | |
| Any | 420 (36.1) | 279 (36.6) | 123 (36.4) |
| None | 745 (63.9) | 483 (63.4) | 215 (63.6) |
| Missing | 222 | 128 | 57 |
| Neighborhood support score[3] | | | |
| 1-<2 (higher) | 326 (28.3) | 222 (29.4) | 109 (32.6) |
| 2-<4 | 737 (63.9) | 475 (63.0) | 197 (59.0) |
| ≥4 | 90 (7.8) | 57 (7.6) | 28 (8.4) |
| Missing | 234 | 136 | 61 |
| *Neighborhood Level* | | | |
| Census tract level rating of neighborhood cohesion[4] | | | |
| <2.53 (median) (higher) | 675 (51.1) | 446 (51.8) | 202 (52.7) |
| ≥2.53 | 646 (48.9) | 415 (48.2) | 181 (47.3) |
| Missing | 66 | 29 | 12 |

| Parameter | All Subjects with Questionnaire Data (n=1,387) | Subjects with One or More Acceptable Spirometry Curve (n=890) | Subjects with Three Acceptable and Reproducible Spirometry Curves (n=395) |
|---|---|---|---|
| Census tract level rating of neighborhood safety[5] | | | |
| <2.13 (median) (higher) | 658 (49.8) | 433 (50.2) | 194 (50.7) |
| ≥2.13 | 663 (50.2) | 429 (49.8) | 189 (49.4) |
| Missing | 66 | 28 | 12 |
| Tract-level disadvantage[6] | | | |
| <0.13 (median) (lower) | 685 (49.7) | 433 (48.8) | 213 (54.1) |
| ≥0.13 | 694 (50.3) | 454 (51.2) | 181 (45.9) |
| Missing | 8 | 3 | 1 |
| Percent of tract in same home 5 years ago[7] | | | |
| <0.52 (median) | 684 (49.6) | 451 (50.8) | 189 (48.0) |
| ≥0.52 | 695 (50.4) | 436 (49.2) | 205 (52.0) |
| Missing | 8 | 3 | 1 |
| Census tract predominately Latino or White[7] | | | |
| Yes | 1072 (77.7) | 688 (77.6) | 294 (74.6) |
| No | 307 (22.3) | 199 (22.4) | 100 (25.4) |
| Missing | 8 | 3 | 1 |

(1) Overweight is based on 2000 U.S. Centers for Disease Control BMI-for-age charts; separate charts are used for boys and girls. Children with BMI for age values at 85-<95$^{th}$ percentile are considered at risk for overweight; children with BMI for age values ≥95$^{th}$ percentile are considered overweight. Here the at risk of overweight and overweight groups were combined. Only children age 2 years and older that could stand on their own were measured for height and weight in the L.A. FANS-2 study.
(2) Average of responses for the following questions (with reverse coding where necessary): (a) This is a close-knit neighborhood; (b) There are adults kids can look up to; (c) People are willing to help their neighbors; (d) Neighbors generally don't get along; (e) Adults watch out that kids are safe; (f) People in neighborhood don't share same values; (g) People in neighborhood can be trusted; (h) Parents in neighborhood know kids friends; (i) Adults in neighborhood know local kids' (j) Parents in neighborhood know each other; (k) Neighbors do something if kid hangs out; (l) Would do something if kid does graffiti; (m) Would scold kid if showing disrespect. Responses for a-j were: 1=strongly agree, 2=agree, 3=unsure, 4=disagree, 5=strongly disagree; Responses for k-m were: 1=very likely, 2=likely, 3=unsure, 4=unlikely, 5=very unlikely.
(3) Average of responses (1=often, 2=sometimes, 3=rarely, 4=never) for the following questions: (a) How often do neighbors do favors for each other; (b) How often do neighbors watch each others property; (c) How often do neighbors ask advice.
(4) This is the average of the neighborhood cohesion score for adult respondents in a given census tract.
(5) This is the average of the neighborhood safety responses for adult respondents in a given census tract using the following numeric responses for each response: 1=completely safe, 2=fairly safe, 3=somewhat dangerous, 4=extremely dangerous.
(6) This is the average of the following four variables for each census tract (based on U.S. Census 2000 data): percent poor families, percent households on public assistance, percent female headed families with children under the age of 18 years, percent male unemployment.
(7) Based on U.S. Census 2000 data.

**Associations between Air Pollution Exposure Metrics and Respiratory Health Endpoints**

Of the 1,387 L.A. FANS-2 children included in our analyses, 191 (13.8%) had received a doctor's diagnosis of asthma, while 145 (10.5%) suffered from wheeze, 162 (11.7%) used medications for asthma or wheeze, and 270 (19.5%) had sneezing or runny/blocked nose apart from colds in the past 12 months, according to PCG interview responses. Also according to PCG responses, 140 children (10.1%) had more than 3 doctor-diagnosed ear infections in one year during their lifetime. Table 13 provides a cross-classification of asthma, wheeze, and asthma medication use status. Approximately 37% (n=71) of the diagnosed asthmatics did not wheeze or use asthma medications in the previous year, 13% (n=24) did not report wheezing but did use medications, and 50% (n=95) reported wheezing whether on medication or not. Of the children reported to have suffered from wheeze in the previous 12 months (n=145), 34% (n=49) were not diagnosed with asthma. Twenty-six children who were not diagnosed with asthma and reported no wheeze, reported taking asthma medications in the previous year.

**Table 13. Distribution (Number, Percent) of Wheeze and Medication Use Outcomes Among L.A. FANS-2 Child Participants With and Without a Doctor-Diagnosis of Asthma (n=1,387)**

| | Doctor-diagnosed Asthma (n=191) | No Doctor-diagnosed Asthma (n=1196) |
|---|---|---|
| Wheeze in past 12 mos, medication use | 89 (46.6%) | 23 (1.9%) |
| Wheeze in past 12 mos, no medication use | 7 (3.7%) | 26 (2.2%) |
| No wheeze in past 12 mos, medication use | 24 (12.6%) | 26 (2.2%) |
| No wheeze in past 12 mos, no medication use | 71 (37.1%) | 1121 (93.7%) |

We report univariate associations between individual, family and neighborhood-level characteristics and the selected respiratory endpoints in Tables 14-19 (located at end of report). In univariate models, the odds of reporting an asthma diagnosis was higher for boys, overweight children and African American children, and lower for children who were uninsured in the previous month and who did not have a usual source of sick care, although the number of children without insurance and sick care was small. Based on family-level characteristics, children with lower SES (measured by family income and PCG education level) were less likely to have an asthma diagnosis reported by PCGs than higher SES children. Children of foreign born PCGs were also less likely to have an asthma diagnosis reported while children of single parents were more likely to have diagnosed asthma reported. Presence of smokers in the home, especially smoking PCGs, increased the odds of diagnosed asthma, as did reports of pests in the home in the previous 12 months. Maternal asthma was a strong predictor of doctor-diagnosed asthma in the child. Children in families where adults felt the neighborhood was dangerous or reported no relatives in the neighborhood were more likely to have diagnosed asthma, but the other characteristics of the neighborhood as rated by the individual family did not appear to be related to this outcome (Table 14). Odds of diagnosed asthma appeared to be marginally lower in lower SES neighborhoods based on census tract level ratings of neighborhood cohesion and safety.

Univariate associations between demographic characteristics and odds of wheeze in the previous 12 months (current wheeze) were similar to those for doctor-diagnosed asthma (Table 15). Main differences were that health insurance in the previous month and usual source of sick care were not as strongly related to this outcome as diagnosed asthma, and associations between family-level SES variables (income, homeowner status, and PCG education) appeared somewhat

stronger, whereby lower SES families reported less current wheeze in children. Also, reports of mold in the home in the previous 12 months and presence of furry pets increased the odds of reports of current wheeze more than of diagnosed asthma. Interestingly, less neighborhood cohesion and support networks as reported by an adult participant were associated with reporting less current wheeze in the children. Similarly, a lower census-tract rating of neighborhood cohesion and higher tract-level disadvantage were also negatively associated with current wheeze. There were only 73 children for whom PCG's reported wheeze with night waking in the previous 12 months. Univariate associations between demographic variables and this outcome were, in general, similar to those for current wheeze (Table 16), but the smaller sample size resulted in wider 95% confidence intervals for all univariate odds ratio estimates.

For the outcome medication use for asthma or wheeze in the past 12 months, univariate associations for demographic variables were also very similar to those for doctor-diagnosed asthma (Table 17), except that lack of a usual source of sick care was not negatively related to this outcome; again, these odds ratio estimates were imprecise due to small numbers. Negative associations between family- and neighborhood-level ratings of neighborhood cohesion and support and this outcome were similar to those for current wheeze.

Univariate associations for the outcomes sneezing, runny/blocked nosed apart from cold in past 12 months and lifetime occurrence of frequent ear infections (defined as more than three doctor-diagnosed ear infections in one year) are reported in Tables 18 and 19, respectively. For both outcomes, across several different indicators of family-level SES (income, homeowner status, PCG education), children from lower SES families were less likely to have these outcomes reported. This trend was also apparent for the census-level measures of neighborhood quality (i.e., census tract level ratings of neighborhood safety and cohesion and tract-level economic disadvantage). For all outcomes considered, foreign born PCGs were less likely to report that children had adverse respiratory symptoms.

Among all respiratory outcomes and exposure measures we evaluated, the strongest associations we observed were between wheeze in the past 12 months (current wheeze) and NO, $NO_2$ and $NO_x$ predicted by the more local traffic LUR model (Table 20). We estimated a 29% (95% CI=1.05-1.59) and 26% (95% CI=1.03-1.54) increase in odds of current wheeze per interquartile increase in 12-month average NO and $NO_x$, respectively, after adjusting for age, race/ethnicity, sex and family income; the association for more local traffic $NO_2$ was weaker (OR=1.19, 95% CI=0.95-1.50) but the 95% confidence interval for this estimate overlapped almost completely with those for NO and $NO_x$ measures. Adjustment for maternal asthma slightly moved estimates towards the null, while adjustment for overweight, group participation in the previous 12 months and census-tract level economic disadvantage slightly increased effect estimates; all other covariates evaluated did not result in changes in estimates >5% after adjustment for age, race/ethnicity, sex and family income. Point estimates of effect based on more local traffic NO, $NO_2$ and $NO_x$ exposures when evaluated for the current home and averaging over 2 years prior to interview were slightly lower, while averaging over 5 years prior to interview slightly higher than the 12-month estimates, but again 95% confidence intervals were widely overlapping (results not shown). Adjusting for peak 8-hour $O_3$ did not change estimates of association between more local traffic NO, $NO_2$ and $NO_x$ exposure metrics and current wheeze, while incorporating concentrations at school locations into exposure estimates tended to strengthen these associations (Table 21), although the 95% CIs for effect estimates based on home only versus home and school averages were widely overlapping. When analyses were stratified based on median level of census tract-level economic disadvantage, associations

**Table 20. Associations (Odds ratios, 95% CIs) between Annual Average Air Pollution Exposure Metrics (12 Mos prior to Interview) and Wheeze in the Past 12 Months Among L.A. FANS-2 Participants Ages 0–17 Years**

| Pollutant | Crude (per IQR increase)[1] | Model 1: Adjusting for age, race/ethnicity, sex, income | Model 1+Maternal Asthma | Model 1+ No. of Groups Participated in past 12 mos | Model 1+ Tract-level disadvantage | Model 1+Overweight | Model 1+ Maternal Asthma, No. of Groups Participated in past 12 mos, Overweight | Model 1 + Maternal Asthma, No. of Groups Participated in past 12 mos, Tract-level disadvantage, Overweight |
|---|---|---|---|---|---|---|---|---|
| NO | 0.92 (0.74, 1.15) | 1.19 (0.93, 1.52) | 1.14 (0.88, 1.46) | 1.26 (0.96, 1.65) | 1.21 (0.94, 1.56) | 1.23 (0.94, 1.62) | 1.13 (0.84, 1.51) | 1.15 (0.85, 1.55) |
| $NO_2$ | 0.91 (0.74, 1.13) | 1.14 (0.89, 1.47) | 1.09 (0.84, 1.40) | 1.13 (0.86, 1.48) | 1.15 (0.89, 1.49) | 1.17 (0.88, 1.55) | 1.02 (0.76, 1.37) | 1.02 (0.76, 1.37) |
| $NO_x$ | 0.92 (0.74, 1.14) | 1.18 (0.92, 1.51) | 1.12 (0.87, 1.44) | 1.24 (0.94, 1.64) | 1.20 (0.93, 1.55) | 1.23 (0.93, 1.63) | 1.10 (0.82, 1.48) | 1.11 (0.82, 1.51) |
| NO-LT[2] | 1.08 (0.89, 1.31) | 1.29 (1.05, 1.59) | 1.24 (1.00, 1.53) | 1.39 (1.11, 1.74) | 1.30 (1.06, 1.60) | 1.37 (1.09, 1.72) | 1.30 (1.02, 1.66) | 1.31 (1.02, 1.67) |
| $NO_2$-LT | 0.98 (0.80, 1.20) | 1.19 (0.95, 1.50) | 1.14 (0.90, 1.43) | 1.17 (0.92, 1.50) | 1.19 (0.95, 1.50) | 1.22 (0.94, 1.57) | 1.08 (0.83, 1.40) | 1.08 (0.83, 1.40) |
| $NO_x$-LT | 1.06 (0.88, 1.28) | 1.26 (1.03, 1.54) | 1.20 (0.98, 1.47) | 1.34 (1.07, 1.67) | 1.27 (1.04, 1.55) | 1.33 (1.06, 1.67) | 1.23 (0.97, 1.57) | 1.24 (0.97, 1.57) |
| $O_3$ | 1.03 (0.79, 1.33) | 0.90 (0.68, 1.20) | 0.94 (0.70, 1.25) | 0.81 (0.59, 1.11) | 0.89 (0.67, 1.19) | 0.94 (0.68, 1.29) | 0.95 (0.67, 1.36) | 0.95 (0.66, 1.36) |
| $PM_{2.5}$ | 0.95 (0.82, 1.10) | 1.04 (0.88, 1.23) | 1.02 (0.86, 1.21) | 0.99 (0.83, 1.18) | 1.04 (0.88, 1.23) | 1.01 (0.84, 1.22) | 0.91 (0.75, 1.10) | 0.91 (0.75, 1.10) |

(1) Interquartile ranges (IQRs) for each pollutant were: NO=10.7 ppb; $NO_2$=6.1 ppb; $NO_x$=16.9 ppb; NO-LT=11.8 ppb; $NO_2$-LT=6.1; $NO_x$-LT=16.9 ppb; $O_3$=29.1 ppb; $PM_{2.5}$=2.4 µg/$m^3$.
(2) "LT" stands for "more local traffic impact" LUR model estimates.

**Table 21. Associations (Odds ratios[1], 95% CIs) between Annual Average Air Pollution Exposure Metrics (12 Mo prior to Interview) and Wheeze in the Past 12 Months Among L.A. FANS-2 Participants Ages 0–17 Years: Incorporating School Locations and Stratifying on Neighborhood Disadvantage**

| Pollutant | Two Pollutant Model[2] HOME ONLY | Two Pollutant Model[2] HOME+SCHOOL | Tract-level disadvantage[3] <0.13 (median) HOME ONLY *Higher SES* | Tract-level disadvantage[3] ≥0.13 HOME ONLY *Lower SES* | Tract-level disadvantage[3] <0.13 (median) HOME+SCHOOL | Tract-level disadvantage[3] ≥0.13 HOME+SCHOOL |
|---|---|---|---|---|---|---|
| NO-LT[4] | 1.31 (1.02, 1.68) | 1.42 (1.07, 1.87) | 1.41 (0.98, 2.02) | 1.40 (0.99, 1.98) | 1.63 (1.07, 2.49) | 1.46 (1.00, 2.12) |
| $O_3$ | 1.02 (0.69, 1.49) | 1.03 (0.69, 1.54) | 0.70 (0.42, 1.16) | 1.80 (0.99, 3.24) | 0.74 (0.43, 1.28) | 1.60 (0.87, 2.96) |
| $NO_2$-LT | 1.08 (0.83, 1.41) | 1.14 (0.85, 1.53) | 1.35 (0.89, 2.04) | 1.09 (0.80, 1.55) | 1.60 (0.98, 2.59) | 1.09 (0.75, 1.58) |
| $O_3$ | 0.95 (0.66, 1.37) | 0.94 (0.64, 1.39) | 0.62 (0.37, 1.06) | 1.51 (0.85, 2.70) | 0.62 (0.35, 1.09) | 1.33 (0.73, 2.42) |
| $NO_x$-LT | 1.24 (0.97, 1.58) | 1.34 (1.02, 1.75) | 1.39 (1.00, 1.94) | 1.30 (0.91, 1.85) | 1.60 (1.09, 2.36) | 1.33 (0.91, 1.96) |
| $O_3$ | 1.00 (0.69, 1.47) | 1.02 (0.68, 1.53) | 0.69 (0.41, 1.14) | 1.76 (0.96, 3.23) | 0.72 (0.42, 1.25) | 1.60 (0.84, 2.95) |

(1) Odds ratios are per interquartile range (IQR) increase in each pollutant: NO=10.7 ppb; $NO_2$=6.1 ppb; $NO_x$=16.9 ppb; NO-LT=11.8 ppb; $NO_2$-LT=6.1; $NO_x$-LT=16.9 ppb; $O_3$=29.1 ppb; $PM_{2.5}$=2.4 µg/$m^3$.
(2) Adjusting for age, race/ethnicity, sex, income, maternal asthma, overweight, no groups participated in past 12 months and tract-level disadvantage.
(3) Stratified models are two-pollutant models, i.e., LUR variables or $PM_{2.5}$ plus $O_3$ in each model. Stratified models do not include adjustment for tract-level disadvantage.

between more local traffic NO and $NO_x$ were similar in magnitude across strata, with an approximately 40% increase in odds of current wheeze per IQR increase in more local traffic NO and $NO_x$ (10.7 and 16.9 ppb increase, respectively) after adjusting for important covariates and peak $O_3$ (Table 21). Interestingly, although no associations between peak 8-hour $O_3$ and current wheeze were observed in higher SES neighborhoods (i.e., those with a tract-level disadvantage score lower than the median value of 0.13), we estimated an approximately 80% increase in current wheeze per IQR increase in $O_3$ (29 ppb) for children living in lower SES areas, based on home only exposure averages (Table 21). Again, incorporating school locations into exposure averages tended to strengthen associations estimated by stratified analyses, but 95% CIs were widely overlapping with those for stratified analyses based on home only averages. Effect estimates stratified on median census tract-level rating of neighborhood safety followed a similar pattern as those for analyses stratified on tract-level disadvantage, but differences across strata were weaker (results not shown). There were no substantive differences in effect estimates across strata of tract-level ratings of neighborhood cohesion or percent in the same home as five years ago (results not shown). Associations between wheeze with night waking in the previous 12 months and more local traffic LUR estimates for NO, $NO_2$ and $NO_x$ were very similar to those for current wheeze with wider 95% confidence intervals due to smaller sample sizes for this outcome (results not shown); there was insufficient sample size for this outcome to perform stratified analyses.

Associations between local traffic LUR exposure metrics and medication use for asthma or wheeze in the previous 12 months followed the same pattern as associations described for current wheeze, but were weaker (Table 22): odds of medication use increased 15% per interquartile increase in local traffic NO (95% CI=0.94-1.41) and $NO_x$ (95% CI=0.94-1.39), after adjustment for age, race/ethnicity, sex and income. Adjustment for maternal asthma slightly reduced point estimates, while adjustment for group participation in the previous 12 months and census-tract level rating of neighborhood cohesion slightly increased effect estimates; all other covariates evaluated did not result in changes in estimates >5% after adjustment for age, race/ethnicity, sex and family income. Effect estimates for exposures based on the current home only, on homes lived in 2 years prior to interview, and on homes lived in 5 years prior to interview were the same as, slightly lower and slightly higher, respectively, than those for the based on residences lived in during the 12-month prior to interview, but again, 95% confidence intervals widely overlapped (results not shown). Adjusting for peak 8-hour $O_3$ did not change effect estimates for more local traffic NO, $NO_2$ or NOx exposure metrics, while incorporating estimated concentrations at school locations into exposure averages slightly strengthened associations (Table 23). Similar to current wheeze, analyses stratified based on median tract-level economic disadvantage suggested relatively strong associations between peak $O_3$ and medication use for asthma or wheeze in lower SES areas, with an approximately two-fold increase in odds of this outcome per 29 ppb increase (IQR) in $O_3$, while inverse associations between peak $O_3$ and this outcome were observed in higher SES areas (Table 23). Similar to unstratified analyses, incorporating school locations tended to strengthen effect estimates, but 95% CIs were widely overlapping with those for stratified analyses based on home only averages. Stratified analyses also suggested stronger associations between more local traffic NO and $NO_x$ and asthma medication use in higher SES areas, but again effect estimates were fairly imprecise with widely overlapping confidence intervals between the two strata. Effect estimates stratified on median census tract-level rating of neighborhood safety followed a similar pattern as those for analyses stratified on tract-level disadvantage, but differences across strata were weaker

**Table 22. Associations (Odds ratios, 95% CIs) between Annual Average Air Pollution Exposure Metrics (12 Mos prior to Interview) and Medication Use for Asthma or Wheeze in the Past 12 Months Among L.A. FANS-2 Participants Ages 0–17 Years**

| Pollutant | Crude (per IQR increase)[1] | Model 1: Adjusting for age, race/ethnicity, sex, income | Model 1+Maternal Asthma | Model 1+ No. of Groups Participated in past 12 mos | Model 1+ Tract-level rating of neighborhood cohesion | Model 1+ Maternal Asthma, No. of Groups Participated in past 12 mos | Model 1+ Maternal Asthma, No. of Groups Participated in past 12 mos, Tract-level neighborhood cohesion |
|---|---|---|---|---|---|---|---|
| NO | 0.89 (0.72, 1.10) | 1.04 (0.81, 1.32) | 1.00 (0.78, 1.28) | 1.04 (0.79, 1.37) | 1.11 (0.86, 1.43) | 0.99 (0.75, 1.30) | 1.03 (0.78, 1.37) |
| $NO_2$ | 0.87 (0.71, 1.06) | 1.01 (0.80, 1.28) | 0.96 (0.76, 1.23) | 0.96 (0.74, 1.24) | 1.06 (0.83, 1.36) | 0.91 (0.70, 1.18) | 0.96 (0.73, 1.25) |
| $NO_x$ | 0.89 (0.72, 1.09) | 1.04 (0.82, 1.33) | 1.00 (0.78, 1.28) | 1.04 (0.80, 1.37) | 1.11 (0.87, 1.43) | 0.98 (0.75, 1.29) | 1.03 (0.78, 1.35) |
| NO-LT[2] | 1.04 (0.86, 1.25) | 1.15 (0.94, 1.41) | 1.11 (0.90, 1.36) | 1.18 (0.94, 1.48) | 1.20 (0.98, 1.48) | 1.12 (0.89, 1.41) | 1.14 (0.91, 1.44) |
| $NO_2$-LT | 0.94 (0.78, 1.14) | 1.08 (0.87, 1.34) | 1.03 (0.83, 1.29) | 1.03 (0.82, 1.30) | 1.12 (0.90, 1.40) | 0.97 (0.77, 1.23) | 1.02 (0.80, 1.30) |
| $NO_x$-LT | 1.03 (0.86, 1.24) | 1.14 (0.94, 1.39) | 1.10 (0.90, 1.34) | 1.17 (0.94, 1.45) | 1.19 (0.98, 1.45) | 1.10 (0.88, 1.37) | 1.13 (0.91, 1.41) |
| $O_3$ | 1.09 (0.85, 1.39) | 0.99 (0.76, 1.30) | 1.03 (0.78, 1.36) | 0.95 (0.71, 1.28) | 0.98 (0.74, 1.29) | 0.99 (0.73, 1.35) | 0.99 (0.73, 1.34) |
| $PM_{2.5}$ | 0.90 (0.78, 1.03) | 0.98 (0.84, 1.15) | 0.96 (0.82, 1.13) | 0.93 (0.79, 1.09) | 1.00 (0.85, 1.17) | 0.91 (0.77, 1.07) | 0.92 (0.78, 1.10) |

(1) Interquartile ranges (IQRs) for each pollutant were: NO=10.7 ppb; $NO_2$=6.1 ppb; $NO_x$=16.9 ppb; NO-LT=11.8 ppb; $NO_2$-LT=6.1; $NO_x$-LT=16.9 ppb; $O_3$=29.1 ppb; $PM_{2.5}$=2.4 μg/$m^3$.
(2) "LT" stands for "more local traffic impact" LUR model estimates.

**Table 23. Associations (Odds ratios[1], 95% CIs) between Annual Average Air Pollution Exposure Metrics (12 Mo prior to Interview) and Medication Use for Asthma or Wheeze in the Past 12 Months Among L.A. FANS-2 Participants Ages 0–17 Years: Incorporating School Locations and Stratifying on Neighborhood Disadvantage**

| Pollutant | Two Pollutant Model[2] HOME ONLY | Two Pollutant Model[2] HOME+SCHOOL | Tract-level disadvantage[3] <0.13 (median) HOME ONLY | Tract-level disadvantage[3] ≥0.13 HOME ONLY | Tract-level disadvantage[3] <0.13 (median) HOME+SCHOOL | Tract-level disadvantage[3] ≥0.13 HOME+SCHOOL |
|---|---|---|---|---|---|---|
| NO-LT[4] | 1.15 (0.91, 1.45) | 1.20 (0.93, 1.56) | 1.34 (0.94, 1.92) | 1.15 (0.84, 1.57) | 1.44 (0.96, 2.15) | 1.19 (0.85, 1.68) |
| $O_3$ | 1.03 (0.75, 1.41) | 1.01 (0.71, 1.42) | 0.59 (0.37, 0.93) | 2.25 (1.37, 3.68) | 0.54 (0.33, 0.89) | 2.30 (1.36, 3.88) |
| $NO_2$-LT | 1.02 (0.80, 1.30) | 1.03 (0.79, 1.34) | 1.35 (0.92, 1.97) | 1.02 (0.75, 1.38) | 1.45 (0.94, 2.22) | 1.02 (0.74, 1.42) |
| $O_3$ | 0.99 (0.73, 1.35) | 0.95 (0.69, 1.32) | 0.53 (0.33, 0.85) | 2.07 (1.27, 3.36) | 0.47 (0.28, 0.80) | 2.06 (1.23, 3.46) |
| $NO_x$-LT | 1.13 (0.91, 1.42) | 1.17 (0.91, 1.50) | 1.39 (1.00, 1.92) | 1.12 (0.82, 1.53) | 1.48 (1.02, 2.13) | 1.16 (0.82, 1.63) |
| $O_3$ | 1.03 (0.75, 1.41) | 1.00 (0.71, 1.41) | 0.58 (0.36, 0.91) | 2.24 (1.34, 3.74) | 0.53 (0.32, 0.88) | 2.30 (1.33, 3.97) |

(1) Odds ratios are per interquartile range (IQR) increase in each pollutant: NO=10.7 ppb; $NO_2$=6.1 ppb; $NO_x$=16.9 ppb; NO-LT=11.8 ppb; $NO_2$-LT=6.1; $NO_x$-LT=16.9 ppb; $O_3$=29.1 ppb; $PM_{2.5}$=2.4 μg/$m^3$.
(2) Adjusting for age, race/ethnicity, sex, income, maternal asthma, no groups participated in past 12 months and census tract-level neighborhood cohesion.
(3) Stratified models are two-pollutant models, i.e., LUR variables or $PM_{2.5}$ plus $O_3$ in each model. Stratified models do not include adjustment for census tract-level neighborhood cohesion.
(4) "LT" stands for "more local traffic impact" LUR model estimates.

(results not shown). There were no substantive differences in effect estimates across strata of tract-level ratings of neighborhood cohesion or percent in the same home as five years ago (results not shown).

We also observed marginally statistically significant associations between more local traffic LUR exposure metrics and odds of doctor-diagnosed asthma. We estimated an approximately 15% increase in odds of diagnosed-asthma per interquartile increase in more local traffic NO, $NO_2$ and $NO_x$, after adjusting for age, race/ethnicity, sex, and income (Table 24). Additional adjustment for factors that changed point estimates at least 5% did not result in substantially different findings (maternal asthma, any versus no relatives in the neighborhood, and census tract-level measures of neighborhood safety). Effect estimates for exposures based on the current home only or homes 2 years prior to interview were slightly lower, and estimates based on 5 years prior to interview exposures measures slightly higher, than those for the 12-month prior to interview measure, but again, 95% confidence intervals widely overlapped (results not shown). Results incorporating concentrations at school locations and stratifying on tract-level disadvantage followed the same patterns as for the outcome medication use for asthma or wheeze in the past 12 months, but point estimates were, in general, weaker (Table 25). One exception was stronger associations between doctor-diagnosed asthma and more local traffic NO, $NO_2$ and $NO_x$ in higher SES areas (Table 25) compared to associations between these exposure metrics and asthma medication use in higher SES areas.

In general, results from analysis stratified on census tract-level ratings of neighborhood quality (i.e., cohesion, safety, disadvantage and percent in same home as five years ago), should be interpreted with caution due to the small sample size available to us. Originally, the L.A. FANS-2 study planned to enroll approximately 4,000 children (all of the L.A. FANS-1 children plus a sample of new entrants into each neighborhood). Despite extending data collection by approximately 1.5 years, the total number of L.A. FANS-2 child participants was approximately 35% of the originally planned enrollment number. This issue is addressed further in the Discussion section.

We did not observe associations between any of the pollutant exposure measures we evaluated and the outcomes sneezing or runny/blocked nose apart from cold in the previous 12 months and more than three doctor-diagnosed ear infections in one year.

**Table 24. Associations (Odds ratios, 95% CIs) between Annual Average Air Pollution Exposure Metrics (12 Mos prior to Interview) and Doctor-Diagnosed Asthma Among L.A. FANS-2 Participants Ages 0–17 Years**

| Pollutant | Crude (per IQR increase)[1] | Model 1: Adjusting for age, race/ethnicity, sex, income | Model 1+Maternal Asthma | Model 1+ No. of Relatives in Neighborhood | Model 1+ Tract-level neighborhood safety | Model 1+ Maternal Asthma, No. of Relatives in Neighborhood | Model 1+ Maternal Asthma, No. of Relatives in Neighborhood, Tract-level neighborhood safety |
|---|---|---|---|---|---|---|---|
| NO | 0.92 (0.75, 1.12) | 1.05 (0.84, 1.32) | 1.01 (0.80. 1.27) | 1.13 (0.88, 1.44) | 1.10 (0.87, 1.38) | 1.06 (0.83, 1.36) | 1.07 (0.83, 1.39) |
| $NO_2$ | 0.95 (0.79, 1.16) | 1.11 (0.88, 1.38) | 1.05 (0.84, 1.33) | 1.16 (0.91, 1.47) | 1.13 (0.90, 1.42) | 1.09 (0.85, 1.40) | 1.10 (0.85, 1.41) |
| $NO_x$ | 0.93 (0.77, 1.13) | 1.08 (0.86, 1.35) | 1.03 (0.82, 1.29) | 1.16 (0.91, 1.48) | 1.12 (0.89, 1.41) | 1.09 (0.85, 1.40) | 1.10 (0.85, 1.41) |
| NO-LT[2] | 1.01 (0.85, 1.21) | 1.12 (0.93, 1.36) | 1.08 (0.89, 1.31) | 1.19 (0.97, 1.46) | 1.16 (0.96, 1.41) | 1.13 (0.91, 1.39) | 1.14 (0.92, 1.41) |
| $NO_2$-LT | 1.02 (0.85, 1.22) | 1.16 (0.95, 1.42) | 1.11 (0.90, 1.37) | 1.20 (0.97, 1.50) | 1.18 (0.96, 1.44) | 1.14 (0.91, 1.42) | 1.14 (0.91, 1.43) |
| $NO_x$-LT | 1.03 (0.87, 1.22) | 1.14 (0.95, 1.37) | 1.09 (0.90, 1.31) | 1.21 (0.99, 1.47) | 1.17 (0.98, 1.42) | 1.14 (0.93, 1.39) | 1.15 (0.95, 1.41) |
| $O_3$ | 0.99 (0.79, 1.25) | 0.90 (0.70, 1.16) | 0.94 (0.73, 1.22) | 0.82 (0.62, 1.09) | 0.86 (0.66, 1.12) | 0.86 (0.64, 1.15) | 0.86 (0.65, 1.15) |
| $PM_{2.5}$ | 0.98 (0.85, 1.12) | 1.06 (0.90, 1.23) | 1.04 (0.89, 1.22) | 1.08 (0.91, 1.28) | 1.08 (0.92, 1.27) | 1.07 (0.90, 1.27) | 1.05 (0.89, 1.25) |

(1) Interquartile ranges (IQRs) for each pollutant were: NO=10.7 ppb; $NO_2$=6.1 ppb; $NO_x$=16.9 ppb; NO-LT=11.8 ppb; $NO_2$-LT=6.1; $NO_x$-LT=16.9 ppb; $O_3$=29.1 ppb; $PM_{2.5}$=2.4 μg/$m^3$.
(2) "LT" stands for "more local traffic impact" LUR model estimates.

**Table 25. Associations (Odds ratios[1], 95% CIs) between Annual Average Air Pollution Exposure Metrics (12 Mo prior to Interview) and Doctor-Diagnosed Asthma Among L.A. FANS-2 Participants Ages 0–17 Years: Incorporating School Locations and Stratifying on Neighborhood Disadvantage**

| Pollutant | Two Pollutant Model[2] HOME ONLY | Two Pollutant Model[2] HOME+SCHOOL | Tract-level disadvantage[3] <0.13 (median) HOME ONLY *Higher SES* | Tract-level disadvantage[3] ≥0.13 HOME ONLY *Lower SES* | Tract-level disadvantage[3] <0.13 (median) HOME+SCHOOL | Tract-level disadvantage[3] ≥0.13 HOME+SCHOOL |
|---|---|---|---|---|---|---|
| NO-LT[4] | 1.12 (0.90, 1.40) | 1.13 (0.89, 1.45) | 1.39 (0.98, 1.96) | 1.05 (0.78, 1.41) | 1.46 (1.00, 2.15) | 1.07 (0.77, 1.49) |
| $O_3$ | 0.89 (0.66, 1.19) | 0.87 (0.64, 1.20) | 0.58 (0.38, 0.88) | 1.46 (0.92, 2.31) | 0.55 (0.35, 0.86) | 1.47 (0.90, 2.41) |
| $NO_2$-LT | 1.14 (0.91, 1.44) | 1.12 (0.88, 1.44) | 1.45 (1.01, 2.10) | 1.16 (0.86, 1.55) | 1.54 (1.02, 2.30) | 1.14 (0.83, 1.56) |
| $O_3$ | 0.86 (0.64, 1.16) | 0.85 (0.62, 1.16) | 0.51 (0.32, 0.80) | 1.55 (0.98, 2.45) | 0.48, (0.30, 0.77) | 1.53 (0.94, 2.50) |
| $NO_x$-LT | 1.13 (0.92, 1.40) | 1.13 (0.90, 1.43) | 1.44 (1.04, 1.97) | 1.05 (0.78, 1.42) | 1.50 (1.05, 2.14) | 1.07 (0.77, 1.49) |
| $O_3$ | 0.89 (0.66, 1.20) | 0.88 (0.64, 1.20) | 0.57 (0.37, 0.87) | 1.47 (0.91, 2.38) | 0.54 (0.34, 0.85) | 1.49 (0.89, 2.49) |

(1) Odds ratios are per interquartile range (IQR) increase in each pollutant: NO=10.7 ppb; $NO_2$=6.1 ppb; $NO_x$=16.9 ppb; NO-LT=11.8 ppb; $NO_2$-LT=6.1; $NO_x$-LT=16.9 ppb; $O_3$=29.1 ppb; $PM_{2.5}$=2.4 μg/$m^3$.
(2) Adjusting for age, race/ethnicity, sex, income, maternal asthma, no. relatives in neighborhood and census tract-level neighborhood safety.
(3) Stratified models are two-pollutant models, i.e., LUR variables or $PM_{2.5}$ plus $O_3$ in each model. Stratified models do not include adjustment for census tract-level neighborhood safety.
(4) "LT" stands for "more local traffic impact" LUR model estimates.

**Associations between Air Pollution Exposure Metrics and Lung Function**

Distributions of lung function parameters are presented in Table 26. Overall, mean lung volumes and flows, as expected, were greater for boys than for girls, and were also greater for the 395 children with three acceptable and reproducible curves, as defined by 1994 American Thoracic Society guidelines[127] compared to the 890 children with one or more acceptable curves. Table 27 provides a breakdown of the number of acceptable and reproducible curves by age group. Children 5-<10 years of age were less likely to have three acceptable and reproducible curves than children 10 years and older.

**Table 26. Mean (SD) Lung Function in L.A. FANS-2 Participants Ages 5-17 Years**

| Lung Function | Boys (1 or More Acceptable Curves, n=486) | Boys (3 Acceptable and Reproducible Curves, n=221) | Girls (1 or More Acceptable Curves, n=404) | Girls (3 Acceptable and Reproducible Curves, n=174) |
|---|---|---|---|---|
| $FEV_1$ (mL) | 2629 (1036) | 2730 (1018) | 2369 (744) | 2500 (616) |
| FVC (mL) | 3193 (1231) | 3267 (1198) | 2800 (861) | 2958 (733) |
| PEF (mL/s) | 5758 (2291) | 6039 (2130) | 5347 (1947) | 5712 (1552) |
| $FEF_{75}$ (mL/s) | 1455 (818) | 1491 (793) | 1409 (707) | 1419 (633) |
| $FEF_{25-75}$ (mL/s) | 2807 (1332) | 2940 (1263) | 2695 (1069) | 2828 (932) |

**Table 27. Summary of Acceptable and Reproducible Spirometry Curves by Age Group (N, percent)**

| Age group | 1 acceptable curve | 2 acceptable curves, not reproducible | 2 acceptable and reproducible curves | 3 acceptable curves, not reproducible | 3 acceptable and reproducible curves |
|---|---|---|---|---|---|
| 5-<10 years[1] | 46 (21) | 24 (11) | 62 (28) | 12 (5) | 78 (35) |
| 10-<15 years | 41 (10) | 40 (10) | 94 (22) | 50 (12) | 190 (46) |
| ≥15 years | 30 (12) | 22 (9) | 46 (18) | 28 (11) | 127 (50) |
| All ages[2] | 117 (13) | 86 (10) | 202 (23) | 90 (10) | 395 (44) |

(1) Percents are based on all children of given age group with one or more acceptable curves (i.e., 222 for 5-<10 years, 415 for 10-<15 years and 253 for ≥15 years).

(2) Percents are based on all children with one or more acceptable curves (n=890).

For boys with one or more acceptable maneuvers, we estimated decrements across all lung function measures with increasing exposure to LUR estimates of NO, $NO_2$ and $NO_x$ based on current home exposure averages; decrements were also observed with increasing exposure to annual average $PM_{2.5}$ as estimated by a kriged surface for the L.A. Basin (Table 28). We estimated an approximately 50-70 mL decrease in the two volume measures $FEV_1$ and FVC per interquartile increase in LUR NO, $NO_2$ and $NO_x$ (10.9, 5.9 and 17.1 ppb, respectively), after adjusting for age, race/ethnicity, height and height$^2$ and being overweight. Estimated decrements per IQR increase in $PM_{2.5}$ (2.4 μg/m$^3$) were smaller at approximately 30 mL. For the flow measures, we estimated an approximately 70 mL/s reduction in $FEF_{75}$ and 100 mL/s reduction in $FEF_{25-75}$ per IQR increase in NO, $NO_2$ and $NO_x$ (Table 28). Decrements in PEF were similar in magnitude to those for $FEF_{75}$, but all estimates were non-significant with wide 95% confidence intervals (results not shown). Additional adjustment for important covariates slightly increased effect estimates, while adjustment for peak 8-hour $O_3$ did not change estimates (Table 28). Incorporating concentrations at school locations in exposure estimates slightly strengthened

**Table 28. Associations (Betas[1], 95% CIs) between Annual Average Air Pollution Exposure Metrics (Current Home) and Lung Function Among L.A. FANS-2 Participants, Boys Ages 5–17 Years**

| Lung Function | Pollutant | 1 OR MORE ACCEPTABLE CURVES (n=486) | | | | 3 ACCEPTABLE AND REPRODUCIBLE CURVES (n=221) | | | |
|---|---|---|---|---|---|---|---|---|---|
| | | Adjusting for age, race/ethnicity, height, height$^2$, overweight | Final Model[2] HOME ONLY | Two-Pollutant Model[3] HOME ONLY | Two-Pollutant Model[3] HOME +SCHOOL | Adjusting for age, race/ethnicity, height, height$^2$, overweight | Final Model[2] HOME ONLY | Two-Pollutant Model[3] HOME ONLY | Two-Pollutant Model[3] HOME +SCHOOL |
| **$FEV_1$ (mL)** | | | | | | | | | |
| | NO | -63 (-119, -8) | -75 (-144, -6) | -69 (-141, 2) | -97(-176, -18) | -70 (-148, 7) | -45 (-147, 56) | -48 (-146, 50) | -44 (-149, 60) |
| | $_2$ | -60 (-113, -7) | -75 (-137 ,-14) | -75 (-138, -12) | -72 (-138, -6) | -60 (-129, 8) | -78 (-157, 0.5) | -73 (-151, 5) | -67 (-149, 16) |
| | $_x$ | -73 (-128, -17) | -91 (-159, -24) | -90 (-162, -18) | -100 (-177, -22) | -67 (-142, 8) | -54 (-150, 43) | -63 (-157, 30) | -64 (-164, 36) |
| | $_{2.5}$ | -33 (-71, 5) | -52 (-98, -7) | -50 (-99, -2) | -46 (-95, 3) | -47 (-96, 3) | -62 (-121, -4) | -50 (-111, 11) | -55 (-114, 5) |
| **FVC (mL)** | NO | | | | | | | | |
| | NO | -52 (-119, 15) | -60 (-141, 22) | -56 (-141, 28) | -86 (-179, 7) | -46 (-134, 42) | -43 (-164, 78) | -50 (-170, 70) | -80 (-205, 45) |
| | PM | -46 (-111, 18) | -67 (-139, 4) | -66 (-139, 7) | -70 (-147, 6) | -56 (-136, 25) | -80 (-175, 16) | -75 (-171, 22) | -76 (-176, 24) |
| | $NO_2$ | -56 (-123, 12) | -76 (-155, 4) | -75 (-159, 9) | -95 (-184, -5) | -62 (-150, 25) | -50 (-165, 65) | -59 (-175, 56) | -78 (-199, 43) |
| | $NO_x$ | -28 (-73, 17) | -45 (-97, 6) | -47 (-102, 8) | -44 (-99, 11) | -49 (-107, 9) | -56 (-126, 14) | -48 (-121, 26) | -58 (-129, 14) |
| | $PM_{2.5}$ | | | | | | | | |
| | | | | | | | | | |
| **$FEF_{75}$ (mL/s)** | | | | | | | | | |
| | NO | -62 (-126, 2) | -39 (-119, 40) | -39 (-122 43) | -58 (-150, 35) | -39 (-133, 54) | -30 (-154, 93) | -11 (-135, 113) | 20 (-113, 153) |
| | $NO_2$ | -67 (-128, -5) | -63 (-133, 7) | -64 (-135, 8) | -74 (-150, 1) | -23 (-107, 62) | -75 (-176, 26) | -63 (-164, 38) | -54 (-159, 51) |
| | $NO_x$ | -67 (-131, -2) | -60 (-137, 18) | -62 (–144, 19) | -77 (-167, 12) | -37 (-128, 54) | -52 (-171, 67) | -38 (-158, 83) | -20 (-148, 108) |
| | $PM_{2.5}$ | -47 (-91, -2) | -56 (-108, -4) | -62 (-117, -7) | -62 (-119, -5) | -34 (-95, 26) | -103 (-174, -33) | -68 (-143, 8) | -78 (-154, -3) |
| | | | | | | | | | |
| **$FEF_{25-75}$ (mL/s)** | | | | | | | | | |
| | NO | -105 (-206, -4) | -65 (-187, 57) | -55 (-181, 72) | -85 (-225, 54) | -129 (-282, 25) | -45 (-250, 159) | -61 (-257, 135) | -27 (-231, 178) |
| | $NO_2$ | -93 (–188, 2) | -104 (-212, 3) | -99 (-208, 10) | -115 (-229, -0.4) | -91 (-227, 44) | -137 (-297, 23) | -114 (-272, 45) | -114 (-275, 48) |
| | $_x$ | -108 (-208, -9) | -101 (-220, 18) | -93 (-218, 31) | -116 (-251, 18) | -151 (-301, -2) | -99 (-289, 91) | -101 (-289, 88) | -83 (-278, 112) |
| | $PM_{2.5}$ | -53 (-123, 16) | -90 (-170, -10) | -89 (-174, -4) | -92 (-177, -7) | -67 (-164, 30) | -139 (-251, -26) | -112 (-228, 4) | -122 (-237, -6) |

(1) Interquartile ranges (IQRs) for each pollutant were: NO=10.9 ppb; $NO_2$=5.9 ppb; $NO_x$=17.1 ppb; $PM_{2.5}$=2.4 μg/$m^3$.

(2) Adjusting for age, race/ethnicity, height, height$^2$, overweight, income, no usual source of sick care, maternal asthma, furry pets in home, PCG smoking status, PCG education level, no. of neighbors talked to for 10 min in past 30 days, and census tract-level disadvantage.

(3) LUR variables or $PM_{2.5}$ plus $O_3$ in each model.

estimated effect sizes. Similar or slightly lower decrements in lung function as presented in Table 28 were estimated for the other exposure periods evaluated (1-year, 2-years and 5-years prior to interview) and associations for the more local traffic LUR exposure estimates were 25-65% lower than those for the final, optimized model LUR exposure metrics and not statistically significant (results not shown).

When restricting analyses to boys with three acceptable and reproducible curves, similar magnitude decrements in $FEV_1$ and FVC were observed per IQR increases in LUR estimates of NO, $NO_2$ and $NO_x$ and kriged estimates of $PM_{2.5}$, but effect estimates were less precise and not statistically significant (Table 28). For the flow measures $FEF_{75}$ and $FEF_{25-75}$, associations with $NO_2$ and especially $PM_{2.5}$ were similar in magnitude, while associations with NO and $NO_x$ weakened with 95% CIs spanning the null value (Table 28).

Analyses stratified on census-tract level indicators of neighborhood quality for boys with one or more acceptable curves suggested greater lung function decrements with increasing air pollution for children living in more cohesive and higher SES neighborhoods for some endpoints and pollutants, but none of these interactions were statistically significant (i.e., 95% CIs for point estimates across strata widely overlapped). Associations also appeared greater for boys who had a doctor-diagnosis of asthma versus those without an asthma diagnosis, but again, 95% CIs across strata overlapped. There was insufficient sample size for meaningful stratified analyses when restricting to boys with three acceptable and reproducible curves.

For girls with one or more acceptable spirometry curves, we estimated 40-80 mL decrements in $FEV_1$ per IQR increase in NO, $NO_2$, and $NO_x$ and 30 mL decrement per IQR increase in $PM_{2.5}$ averaged over the five years prior to interview, after adjusting for important covariates, peak 8-hour $O_3$ and taking concentrations at school locations into account in exposure averages (Table 29). However, unlike for boys, we did not observe associations between any of our exposure metrics and FVC in girls. Associations between the traffic markers NO, $NO_2$ and $NO_x$ and the flow measures PEF and $FEF_{25-75}$ were much stronger in girls than in boys. We estimated 300-350 mL/s decrements in PEF and 200-300 ml/s decrements in $FEF_{25-75}$ per IQR increase in NO, $NO_2$ and $NO_x$ in final models adjusted for $O_3$ and taking school concentrations into account (Table 29). Reductions in $FEF_{75}$ with increases in NO, $NO_2$ and $NO_x$ were similar to those estimated for boys (~60-90 ml/s), but less precisely estimated. Associations for the more local traffic LUR metrics were very similar those those for the final, optimized model exposure metrics. Estimated negative associations between LUR measures for NO, $NO_2$ and $NO_x$ and lung function decreased when considering shorter time periods prior to interview (2-years and 1-year prior to interview), and no associations were observed for current home exposure estimates (results not shown).

No associations between the LUR traffic metrics and lung function were observed when we restricted analyses to girls with three acceptable and reproducible curves. When restricting to this subset of girls, we observed decrements in lung function only for the parameter PEF (mL/s) with increasing exposure to annual average peak $O_3$, based on kriged estimates extracted for the current home (Table 30). Specifically, we estimated an approximately 400 mL/s decrement in PEF per interquartile increase in $O_3$ (29.9 ppb) in final models adjusting for LUR $NO_2$ and taking into account concentrations at school locations ($O_3$ effect estimates when adjusting for NO, $NO_x$ and $PM_{2.5}$ were similar). Although we also estimated PEF decrements with increasing $O_3$ for girls with one or more acceptable curves, point estimates were lower and 95% confidence intervals spanned zero (-123 mL/s, 95% CI=-323, 63). Point estimates for the other time periods evaluated (1-, 2-, and 5-year $O_3$ estimates) were similar to those for exposures based on the

**Table 29. Associations (Betas[1], 95% CIs) between Annual Average Air Pollution Exposure Metrics (5-Years Prior to Interview) and Lung Function Among L.A. FANS-2 Participants, Girls Ages 5–17 Years**

| Lung Function | Pollutant | 1 OR MORE ACCEPTABLE CURVES (n=404) | | | |
|---|---|---|---|---|---|
| | | Adjusting for age, race/ethnicity, height, height$^2$, weight, weight$^2$, overweight | Final Model[2] HOME ONLY | Two-Pollutant Model[3] HOME ONLY | Two-Pollutant Model[3] HOME+SCHOOL |
| **$FEV_1$** | | | | | |
| | NO | -69 (-125, -14) | -74 (-143, -5) | -59 (-128, 9) | -43 (-131, 45) |
| | $_2$ | -52 (-106, 3) | -81 (-142, -19) | -66 (-127, -4) | -65 (-137, 7) |
| | $_x$ | -63 (-118, -7) | -79 (-145, -14) | -66 (-133, 0.7) | -78 (-157, 1) |
| | $_{2.5}$ | -33 (-73, 7) | -40 (-86, 7) | -42 (-90, 5) | -30 (-83, 24) |
| **FVC** NO | | | | | |
| NO | NO | -38 (-100, 23) | -2 (-75, 72) | 10 (-65, 85) | 17 (-79, 113) |
| PM | $NO_2$ | -21 (-80, 38) | -5 (-73, 63) | 19 (-49, 87) | 8 (-70, 86) |
| | $NO_x$ | -38 (-98, 22) | -4 (-74, 67) | 9 9-64, 81) | -23 (-111, 65) |
| | $PM_{2.5}$ | -24 (-67, 18) | -20 (-68, 27) | 0.3 (-52, 52) | -5 (-62, 52) |
| | | | | | |
| **PEF** | NO | -129 (-290, 32) | -123 (-326, 79) | -178 (-383, 28) | -322 (-572, -72) |
| | $NO_2$ | -183 (-333, -32) | -204 (-387, -21) | -260 (-439, -81) | -332 (-532, -132) |
| | $NO_x$ | -158 (-314, -1) | -155 (-347, 38) | -229 (-428, -31) | -355 (-585, -125) |
| | $PM_{2.5}$ | 29 (-85, 142) | -49 (-179, 80) | -69 (-215, 77) | -64 (-218, 91) |
| | | | | | |
| **$FEF_{75}$** | NO | -52 (-133, 30) | -94 (-186, -1) | -79 (-170, 12) | -89 (-200, 22) |
| | $NO_2$ | -18 (-95, 58) | -48 (-132, 36) | -46 (-126, 33) | -62 (-155, 30) |
| | $NO_x$ | -37 (-116, 42) | -80 (-168, 7) | -70 (-157, 18) | -82 (-185, 22) |
| | $PM_{2.5}$ | -2 (-62, 57) | -13 (-74, 48) | -9 (-73, 54) | -36 (-109, 36) |
| | | | | | |
| **$FEF_{25-75}$** | NO | -157 (-279, -36) | -187 (-324, -50) | -202 (-340, -63) | -279 (-448, -110) |
| | $NO_2$ | -103 (-220, 13) | -171 (-297, -44) | -150 (-278, -22) | -207 (-356, -59) |
| | $NO_x$ | -151 (-269, -33) | -173 (-302, -45) | -220 (-354, -85) | -287 (-447, -127) |
| | $PM_{2.5}$ | -45 (-131, 42) | -43 (-139, 53) | -35 (-140, 71) | -86 (-202, 29) |

(1) Interquartile ranges (IQRs) for each pollutant were: NO=10.9 ppb; $NO_2$=5.8 ppb; $NO_x$=16.7 ppb; $PM_{2.5}$=2.4 µg/$m^3$.
(2) Adjusting for age, race/ethnicity, height, height$^2$, weight, weight$^2$, overweight, income, no usual source of sick care, maternal asthma, furry pets in home, PCG smoking status, foreign born status of PCG, group participation in past 12 months, no. of adults recogonize in neighborhood, and census tract-level disadvantage.
(3) LUR variables or $PM_{2.5}$ plus $O_3$ in each model.

**Table 30. Associations (Betas[1], 95% CIs) between Annual Average Peak 8-Hour Ozone (Current Home) and Lung Function Among L.A. FANS-2 Participants, Girls Ages 5–17 Years**

| Lung Function | Adjusting for age, race/ethnicity, height, height$^2$, weight, weight$^2$, overweight | Final Model[2] HOME ONLY | Two-Pollutant Model[3] HOME ONLY | Two-Pollutant Model[3] HOME+SCHOOL |
|---|---|---|---|---|
| **Girls with 1 or More Acceptable Curves (n=404)** | | | | |
| **PEF** | 41 (-120, 202) | -85 (-288, 118) | -135 (-333, 63) | -123 (-323, 76) |
| **Girls with 3 Acceptable and Reproducible Curves (n=174)** | | | | |
| **PEF** | -183 (-381, 15) | -398 (-641, -155) | -435 (-681, -188) | -420 (-668, -171) |

(1) Betas are per IQR increase of 29.9 ppb $O_3$.
(2) Adjusting for age, race/ethnicity, height, height$^2$, weight, weight$^2$, overweight, income, no usual source of sick care, maternal asthma, furry pets in home, PCG smoking status, foreign born status of PCG, group participation in past 12 months, no. of adults recogonize in neighborhood, and census tract-level disadvantage.
(3) Two-pollutant model presented is $O_3$ plus $NO_2$; $O_3$ effect estimates adjusting for NO, $NO_x$ and $PM_{2.5}$ were very similar.

current home.

Similar to results for boys, analyses stratified on census-tract level indicators of neighborhood quality for girls with one or more acceptable curves suggested greater lung function decrements with increasing air pollution in more cohesive and higher SES neighborhoods for some endpoints and pollutants, but none of these interactions were statistically significant (i.e., 95% CIs for point estimates across strata widely overlapped). There were only 18 asthmatic girls with one or more acceptable spirometry curves, so analyses stratified on asthma status were not possible. There was also insufficient sample size for meaningful stratified analyses when restricting to girls with three acceptable and reproducible curves.

In final logistic and linear models reported above, adding a second-level with a random intercept for family to account for non-independence of siblings (i.e., clustering at the family level) changed effect estimates minimally.

## V. DISCUSSION

### LUR Model Development

We modeled NO, $NO_2$ and $NO_x$ concentrations for the LA metropolitan area using "A Distance Decay REgression Selection Strategy" (ADDRESS). Our final three prediction models explained 81%, 86% and 85% of NO, $NO_2$ and $NO_x$ variances, respectively. Thus, these models have a higher prediction power ($R^2$) than a large majority of previously published LUR surfaces.[133,134] To our knowledge, this is the first application of an intensive air pollution monitoring campaign (with 201 samplers) to model traffic-related air pollution in a large, distributed urban area like Los Angeles. Our LUR models and semivariograms suggest that the distance of influence for highways and major roads in this region is greater than 10 km. The spatial extent of traffic impacts on NO, $NO_2$ and $NO_x$ were much greater than previously reported in the literature. For example, Zhou and Levy[7] reported spatial extents for traffic impacts on the order of 100-300 and 200-500 m for NO and $NO_2$, respectively, based on a compilation of published LUR models. Because of LA's geography, infrastructure, road network and population characteristics, land use regression models based on a limited number of buffer

distances (up to 5000 m) were found inadequate to optimize models to predict the spatial distribution of traffic related air pollution, as represented by NO, $NO_2$ and $NO_x$. Most previous LUR models used circular area buffers of less than 1500 m for examining roadway and traffic variables, and land use and population density within a maximum distance of 3000 m.[29,132] Typically, for a medium-sized city (2-5 million population), the influence of traffic diminishes with increasing distance from roads, and local sources of pollution dominate over background effects.[29,133] Highways (including primary and secondary) have a total length of more than 3000 km, and highway and major road densities are 435 and 1669 m per $km^2$, respectively, in the L.A. Basin, much higher than corresponding road categories for a typical medium-sized city.

Differences in NO and $NO_2$ spatial surfaces across cities may also be due to differences in spatial distributions of ozone. NO reacts with ambient ozone to form $NO_2$, and the combination of this reaction and dilution in the surrounding air mass near roadways typically results in a rapid decrease in concentration with downwind distance.[7] For $NO_2$, on the other hand, the dominant formation process ($NO + O_3 \rightarrow NO_2$) slows down its dilution and concentrations decrease at a more gradual rate. Though levels of $O_3$ are relatively high in LA, concentrations are lower in the morning during the high traffic commuting period and also lower in the winter. By contrast, $NO_x$ concentrations are high during those time periods. For a mega-city such as LA, the intertwined high density highway and major road network probably make $O_3$ concentrations insufficient to remove high NO emissions from traffic (especially during the winter and morning rush hours) near sources and thus the formation of $NO_2$ appears to continue to even greater distances. The semivariograms (Figure 7) and spatial distance decay of pollutant concentrations (Figures 9 and 10) demonstrate the slow decrease of NO and $NO_2$ concentrations from highways and major roadways in L.A. compared to values summarized by Zhou and Levy.[7]

However, we also generated a second set of "more local traffic" LUR models which excluded traffic volumes on highways and major roads beyond 5 km. The prediction power for NO, $NO_2$ and NOx in these models that ignored traffic within a buffer distance greater than 5000 m were 0.64, 0.78 and 0.68, respectively, i.e. comparable to previous LUR models published in the literature but much lower than the full models developed here. If from the point of prediction power, the previous LUR models successfully predicted local sources of air pollution, they paid little attention to contributions from background traffic sources (e.g., at buffer distances >5 km), that we found important in a mega-city like L.A. Earlier regional studies in England similarly reported even larger influence areas for $NO_2$ — suggesting that regional patterns are an important contributor to $NO_2$ levels in this locale.[135] Based on the trend curves in Figures 5 and 6, 50% reductions in near source concentrations of NO and $NO_2$ are not reached until 3000 and 5000 m from highways, respectively, while 10% reductions occur at distances greater than 10000 m. Annual average concentrations based on the 15 SCAQMD monitoring sites were 24.7 and 24.3 ppb for NO and $NO_2$, respectively. If we consider these as "background" levels, then such levels are still 45.8% and 73.2% as high as near source concentrations (53.9 and 33.2 ppb for NO and $NO_2$, respectively). Thus, background concentrations were high even though both NO and $NO_2$ are generally considered reactive pollutants. As background concentrations increase, spatial extents increase correspondingly.[7] In addition, high emission rates also increase the spatial extent of traffic impacts for absolute comparisons.[7] Because of the limited sample size of previous studies (mostly <100 monitoring sites) and smaller urban areas studied versus a mega-city such as LA, the greater distance of influence of background traffic effects has not been fully identified previously. Here, we found that levels of $NO_x$ pollution at a residence in a mega-city may be influenced by local traffic sources, and in addition may depend on strongly on urban-

scale background traffic sources.

It should be noted, however, that although incorporation of traffic impacts from distances as far away as 11 km improved LUR model prediction ability for NO, $NO_2$ and $NO_x$ specifically, it is currently unknown how well these models predict other pollutants released directly in motor vehicle exhaust that are also of biologic interest, such as polycyclic aromatic hydrocarbons (PAHs) sorbed to particles from diesel engines and ultrafine particles (less than 0.1 microns in aerodynamic diameter). Ultrafine particles are of concern from a health standpoint because they are more able to penetrate cellular targets in the lung and enter systemic circulation than larger size particles.[1,22,24,136] Measurement studies at urban sites in LA indicate a large portion of UF consist of organic carbon, followed by elemental carbon, as primary products from vehicle emissions and that UF contain the largest fraction of PAHs by mass.[51,52,137] Organic components of PM, which comprise a large proportion of freshly emitted exhaust, have been shown to induce a broad polyclonal expression of cytokines and chemokines in respiratory epithelium.[52,138] This may be due to the action of PAHs, metals and related compounds that lead to the production of cytotoxic reactive oxygen species (ROS). In L.A., concentrations of ultrafine particles have been shown to decrease exponentially with distance from freeways, reaching background concentrations within approximately 150 meters (500 feet).[8-13] Similarly, measurement data for $PM_{10}$ and $PM_{2.5}$ absorbance, black smoke, particle-bound PAHs, and elemental carbon (EC) – all markers of exhaust particle emissions – indicate strong spatial gradients in concentrations with peaks near roadway sources.[7,12,13,139-148] Thus, it may be that the "more local traffic" LUR models we developed here provide better surrogate estimates of exposure for fresh vehicle exhaust including UF and associated toxics; hwoever, additional measurement data would be needed to examine this hypothesis further.

A novel aspect of our modeling process was use of satellite remote sensing data to help build prediction models. Satellite remote sensing of air quality has evolved dramatically over the last decade. Global observations are now available for a wide range of species including aerosols, tropospheric $O_3$, tropospheric $NO_2$, CO (carbon monoxide), HCHO (formaldehyde), and $SO_2$ (sulfur dioxide).[149] However, the resolution of these sensors is coarser than 10 km. To help model small area variation (e.g., 30 m resolution) of pollutant concentrations and compare its effects with land use variables, Landsat ETM+ data of resolution 30 m were used. The satellite data may over-estimate roadway emissions in some degree, but will not be overwhelmingly biased because the spectral information between a tar-roofed building and an often vehicle traveled roadway/parking lot are different. Places with high vegetation cover have the effect of reducing pollutant concentrations, while roadways and tar-roofed buildings do not. The slight over-estimation of pollutant concentrations at places with tar-roofed buildings might better represent the spatial pattern of pollutant concentrations at those places. Compared to open land use, the degree of greenness or soil brightness from remote sensed data should be more accurately characterizing ground land use. Overall, we demonstrated that remote sensing derived data such as vegetation greenness and soil brightness can be useful model inputs that will improve the estimation of spatial variability in NO, $NO_2$ and $NO_x$ concentrations, especially greenness which correlated highly with these concentrations ($r = 0.40$-$0.50$). The advantage of using ETM+ data for LUR is its global coverage and free access (http://landsat.gsfc.nasa.gov/). In locations where other spatial covariates are not readily available such as certain land use data, Landsat ETM+ data might provide effective surrogate measures. The model we developed here provides a relatively easy and feasible way to improve exposure analysis.

Most previous land use regression models[29,132] have included population density as a

predictor. However, in our modeling process, population density was not included for all three prediction models because of high VIFs (VIF>2). Our sensitivity analyses demonstrated that if population density was added, the model prediction power increased only by 0.50%, 1.28% and 0.71%, respectively, for NO, $NO_2$ and NOx; thus, omitting population density from our LUR did not substantially decrease the predictive power of our models.

**Respiratory Health Analyses**

We observed the most consistent and strongest positive associations for the outcome current wheeze and NO and $NO_x$ from local traffic as estimated by our LUR models. We observed stronger associations with LUR exposure metrics that estimated the influence of more local traffic compared to metrics from LUR models that also incorporated traffic influences from as far away as 11 km. This suggests that pollutants released directly in exhaust emissions with sharp concentration peaks close to sources (e.g., UF and associated toxics) may be most relevant for asthmatic symptoms such as current wheeze. Our findings are in agreement with previous European cross-sectional studies reporting associations between various residence- and school-based traffic metrics and the prevalence of wheeze in children.[36,40,43,67-71,74,75] They are also in agreement with previous studies in California, that reported higher odds of wheeze for school-aged children residing close to freeways[2] and major roads,[150] and higher odds of current asthma (i.e., asthma episode in the previous 12 months) for school-aged children with higher levels of traffic (especially freeway traffic) within 150 m of homes.[5]

Since low socioeconomic status is associated with higher traffic exposures,[34,90,91] residual confounding by low SES and associated factors (e.g., access to health care, stress, exposure to tobacco smoke) is a concern in studies examining spatial differences in respiratory health outcomes. Our findings for local traffic NO and $NO_x$ impacts on current wheeze were robust to adjustment for many different measures of socioeconomic status at the family- and neighborhood-level. We also examined differences in effect estimates across several indicators of neighborhood quality. Stratifying on median level of economic disadvantage, we observed relatively similar increases in odds of current wheeze (an approximately 40% increase per IQR increase in NO and $NO_x$) in both higher and lower SES areas.

Although we did not observe associations between $O_3$ and current wheeze in unstratified analyses, an approximately 80% increase in odds of this outcome per IQR increase in peak 8-hour $O_3$ was observed in lower SES areas, while negative associations were observed with $O_3$ in higher SES areas. This difference in effect estimates across higher versus lower income areas may reflect differences in children's behaviors and resulting air pollution exposures during peak $O_3$ episodes. For example if children in higher SES areas (especially those with a doctor's diagnosis of asthma or on medications for asthma) spend more time inside while children in lower SES areas spend more time outdoors during these warm, sunny periods. However, these results may also be an artifact of our very small sample size when stratifying in this manner.

While we observed positive associations between exposures to local traffic NO and $NO_x$ and doctor-diagnosed asthma, they were weaker than those observed for current wheeze. This might partly be due to under-diagnosis and/or under-reporting of asthma among study participants; 66% of subjects with current wheeze were also diagnosed asthmatics, however, as many as 34% of children with wheeze had not been not diagnosed as asthmatics, according to PCG responses. Differences in access to health care and physician practices in diagnosing asthma across communities may be factors affecting our results.[151] Low income children and children of foreign born PCGs were more likely to be highly exposed to traffic pollution based

on our LUR model estimates, but were less likely to have been diagnosed with asthma. Thus, even though we controlled for confounding due to income, PCG education level, PCG birth place, insurance status, and whether children had a usual source of sick care in our analyses, this approach does not help to reduce outcome misclassification, especially differential misclassification due to more highly exposed, low SES, immigrant children being less likely to receive high quality medical care that may result in the diagnosis and treatment of asthma. Thus, our results for self-reported physician diagnoses of asthma and treatment received for asthma may be biased towards the null of no association for traffic-related exposures. Even though lower income and foreign born PCGs were also less likely to report that their children currently suffered from wheeze, positive associations between LUR NO and $NO_x$ and wheeze still emerged in on our models, perhaps because the reporting of this asthma symptom was less misclassified than the medical care system-dependent outcomes. Furthermore, when we stratified on median census-tract level economic disadvantage, associations between more local traffic NO and $NO_x$ and asthma of similar magnitude as for current wheeze emerged for children residing in higher SES areas, again suggesting that under-diagnosis and/or under-reporting may be an important bias impacting our results for children in lower SES areas.

Another potential explanation for the weaker associations we observed between LUR NO, $NO_2$ and $NO_x$ exposure measures and doctor-diagnosed asthma in comparison to associations observed for current wheeze, may be additional misclassification of the relevant exposure period for this outcome. We constructed annual average exposure estimates based on the child's current home, as well as weighting for all homes the child resided in 1, 2 and 5 years prior to the interview. A more relevant exposure estimate, however, may be early life or life-time exposures that would require weighting LUR metrics for homes the child resided in from birth to the age of asthma diagnosis. We did observe slightly higher point estimates for associations between 5-year average exposures to local traffic NO and $NO_x$ and diagnosed asthma in our models, suggesting that longer-term exposure (or perhaps exposure during early life) may be more relevant for this outcome. Unfortunately, the number of children for who we had lifetime residential histories was too small for meaningful statistical analyses of associations between lifetime air pollution exposures and doctor-diagnosed asthma.

Stratifying on median level of economic disadvantage, we observed positive associations in lower SES areas but negative associations in higher SES areas between peak $O_3$ and doctor-diagnosed asthma, similar to stratified results for current wheeze. Again, this may be due to differences in children's behavior patterns during peak $O_3$ episodes. The relatively strong negative association with peak $O_3$ in higher SES areas may reflect avoidance of these air pollution exposures by children with asthma. For children in lower SES areas, we observed an approximately 50% increase in odds of asthma per IQR increase in peak $O_3$. These weaker associations in comparison to those for current wheeze in higher SES areas may be due to more under-diagnosis or under-reporting of asthma for these children, as discussed above. Under-diagnosis and under-reporting may also explain why associations between asthma and more local traffic NO and $NO_x$ only were seen for children living in higher SES areas.

Associations between medication use for asthma and wheeze and LUR exposure metrics followed a similar pattern as associations for current wheeze, but were weaker. As already mentioned above, similar to doctor-diagnosed asthma, our effect estimates for this outcome may be impacted by differential access to health care and/or differences in physician practices across communities. Again, analyses stratified on census-tract level economic disadvantage indicated associations between local traffic LUR exposure metrics and medication use for asthma and

wheeze similar in magnitude to the outcome current wheeze (approximately 40% increases per IQR increase in pollution) in higher SES areas, with much lower and less precisely estimated associations in lower SES areas, suggesting under-diagnosis and/or under-reporting is impacting results for these children. Similar to current wheeze and doctor-diagnosed asthma, we observed negative associations between peak $O_3$ and medication use for asthma or wheeze in higher SES areas but two-fold increases in this outcome with increases in peak $O_3$ in lower SES areas, again potentially reflecting differences in $O_3$ exposure patterns due to avoidance behavior among asthmatics living in higher SES areas as discussed above.

Unlike the majority of previous European studies reporting associations between traffic related air pollution and allergic sensitization as assessed by questionnaire, skin prick tests and/or levels of IgE antibodies to specific allergens,[26,32,40,67-69,82,84] we did not observe associations between any of our air pollution exposure metrics and odds of an allergy symptom as assessed by the ISAAC questionnaire definition "sneezing, or runny/blocked nose apart from colds in the previous 12 months". Reasons for this null finding could include exposure misclassification as many of the previous European reporting positive associations for allergic sensitization focused on early life exposures to traffic.[26,32,82,84] Outcome misclassification could be another explanation, as most of the previous studies also performed skin prick tests and/or measured levels of specific IgE antibodies in blood in addition to collecting questionnaire reports of allergic symptoms. Finally, reporting of these symptoms was highly related to income in L.A. FANS-2 participants, with children from low income families having the least symptoms reported, suggesting that under-and or mis-reporting may be impacting our estimates.

As part of the TRAPCA study, Brauer et al.[85] reported positive associations between LUR measures of traffic exposure ($PM_{2.5}$, soot and $NO_2$) at birth residences and odds of doctor-diagnosed ear infections in the first two years of life. We did not find similar associations between any of our air pollution exposure metrics and odds of more than three doctor-diagnosed ear infections in L.A. FANS-2 children. Since most ear infections occur prior to age two,[85] early life exposures may provide more relevant exposure metrics for this outcome than exposures averaged over the years prior to the L.A. FANS-2 interview. Also, similar to the outcome sneezing, or runny/blocked nose apart from colds in the previous year, low income children were less likely reported as having doctor-diagnosed ear infections than high income children, suggesting that systemic under-diagnosis and under-reporting of these outcomes may also be impacting these estimates.

**Lung Function Analyses**

Several studies from Europe have reported decrements in cross-sectional measures of lung function in children more highly exposed to traffic pollution.[36-39,41,44,49] Yet not all findings have been consistent.[40,42,43] Here, we observed negative associations between LUR estimates of traffic exposure and lung function in boys with one or more acceptable spirometry curves. We estimated 70-100 mL reductions in the volume measures $FEV_1$ and FVC and 60-100 mL/s reductions in the flow measures $FEF_{75}$ and $FEF_{25-75}$ with IQR increases in the traffic markers NO, $NO_2$ and $NO_x$. Increases in $PM_{2.5}$ were also associated with approximately 50 mL reductions in $FEV_1$ and FVC and 60-90 mL/s reductions in $FEF_{75}$ and $FEF_{25-75}$ (per 2.4 $\mu g/m^3$ increase). Although similar magnitude reductions in PEF were observed, effect estimates were very imprecise and not statistically significant. The stronger associations observed when incorporating school concentrations into exposure estimates likely is due to restricting analyses to older children with higher quality spirometry measurements (since exposure estimates

incorporating homes only versus homes and schools were very highly correlated, r>0.9). Associations between the more local traffic LUR estimates and lung function were much weaker than associations with the final, optimized model LUR estimates (25-65% lower in magnitude and not statistically significant).

In general, we estimated similar magnitude reductions in lung function with increasing exposure to LUR estimates of traffic pollution and $PM_{2.5}$ when restricting analyses to boys with three acceptable and reproducible curves, but effect estimates were more imprecise due to the smaller sample size available (221 boys versus 486 boys with one or more acceptable curves). Also, associations between LUR estimates of $NO_2$ and kriged estimates of $PM_{2.5}$ and $FEF_{75}$ and $FEF_{25-75}$ remained when limiting to boys with three or more curves while associations with NO and $NO_x$ were reduced or disappeared.

For girls with one or more acceptable curves, we estimated weaker effects between IQR increases in LUR traffic exposure metrics and measures of lung volume compared to boys (40-80 mL reductions in $FEV_1$ and no associations with FVC) but much greater reductions with measures of expiratory flow (300-350 mL/s reductions in PEF and 200-300 mL/s reductions in $FEF_{25-75}$). Again, the stronger associations observed for exposure metrics incorporating homes and schools likely is due to restricting analyses to older children with more accurate spirometry measures.

Similar to our findings, other cross-sectional studies have also reported stronger air pollution impacts on expiratory flow versus volume measures of lung function, and in girls versus boys. Roselund et al.[44] reported reductions of 62 mL/s and 85 mL/s in $FEF_{25-75}$ and PEF, respectively, per 10 $\mu g/m^3$ increase in LUR-modeled $NO_2$ (based on current home locations) in a study of children ages 9-10 years in Rome, and the effect of $FEF_{25-75}$ was isolated to girls. Oftedal et al.[49] reported approximately 100 mL/s decreases in PEF, $FEF_{25}$ and $FEF_{50}$ per interquartile increases in $NO_2$, $PM_{10}$ and $PM_{2.5}$ for 9-10 year old girls living in Oslo, Norway. Decrements in volume measures were not observed, and decrements in boys were half as strong as in girls and did not reach statistical significance. Exposures measures were derived from emissions and air dispersion models and included both local and background source contributions. Interestingly, both life-time and early life (i.e., first year) exposures were found to be important. Here, we observed the strongest decrements in PEF, $FEF_{75}$ and $FEF_{25-75}$ with increasing NO, $NO_2$ and $NO_x$ levels when averaging LUR metrics over all homes during five years prior to interview, with estimates gradually attenuating for shorter time periods (2-years and 1-year prior to interview). This observation may also suggest that earlier life and long-term exposure to traffic pollutants are most important for lowering lung function, at least in girls. We did not observe strong differences in effect estimates across exposure averaging periods for boys. Unfortunately, the number of children for who we had lifetime residential histories was too small for meaningful statistical analyses of associations between lifetime air pollution exposures and lung function. Furthermore, decrements in lung function with increased exposure to LUR-modeled NO, $NO_2$ and $NO_x$ were only observed in analyses including girls with one or more acceptable curves, and were not seen when restricting to the sample of girls with three acceptable and reproducible curves for whom lung function values are more accurate and precise.

In the first cross-sectional analysis of ambient air pollution and lung function in the Children's Health Study (CHS), decrements in lung function with increasing air pollution were also isolated to girls. Specifically decrements in volume measures ($FEV_1$ and FVC) were observed with increasing exposure to $PM_{10}$, $PM_{2.5}$, and $NO_2$ as measured by central monitoring sites in each community studied, and these effects appeared strongest in girls spending more time

outdoors, while observed decrements in flow measures (PEF and $FEF_{25-75}$) did not differ much with the girls' time-activity patterns (i.e., more time spent out- versus indoors). The strongest effect reported across all pollutants and endpoints was a 250 mL/s decrement in PEF per 40 ppb increase in peak $O_3$ in girls. We estimated a greater decrement in PEF per an increase in peak $O_3$ (~400 mL/s per 30 ppb $O_3$) when using a kriging model to extrapolate levels from ambient stations to the LA Basin; however, our estimates were less precise due to the smaller sample size of the L.A. FANS cohort. Adjusting for measures of neighborhood social support appeared important; i.e.our effect estimate sizes increased with adjustment. The University of California Berkeley Ozone Studies also reported negative effects of life-time exposure to $O_3$ on lung expiratory flows, but not volumes, in college freshman ages 17-21 years.[15,16] On the other hand, reductions in lung function growth between ages 10-18 years were not observed in the CHS in those more highly exposed to $O_3$.[53-55] Here, we did not observe differences in $O_3$ effect estimates for PEF for the various time period evaluated (current home, 1-year, 2-years and 5-years prior to interview estimates).

Peak expiratory flow (PEF) rate, $FEF_{75}$, and $FEF_{25-75}$ are considered markers of small airway function, converse to the volume measures that provide information on the larger, central airways.[49,56] Studies of smoking impacts on lung function and some previous studies of air pollution indicate stronger impacts on flow rather than volume measures of lung function.[49] These findings may be indicative of the biologic impact of small particles (in UF size range) in cigarette smoke and traffic exhaust which can reach the small, peripheral airways and alveoli. Thus, flow measures may be more sensitive indicators of air pollution impacts on lung health and reflect preclinical structural changes before the larger airways are affected.[56]

As mentioned previously, decrements in lung function with increased exposure to LUR-modeled NO, $NO_2$ and $NO_x$ were only observed in analyses including girls with one or more acceptable curves, and were not seen when restricting to girls with three acceptable and reproducible curves for whom values are more accurate and precise. Similarly, estimated decrements in lung function with increases in NO, $NO_2$ and $NO_x$ were more imprecisely estimated and not statistically significant when restricting to boys with three acceptable and reproducible curves. However, negative associations between exposure to peak daily $O_3$ and PEF in girls were much stronger for the subgroup with three curves. Overall, children with three acceptable and reproducible curves were more likely to come from families with higher incomes, where the PCG had more education and was U.S. born, and from families that reported higher levels of neighborhood safety and support. These children also had greater variability in exposure to $O_3$ and slightly lower mean exposure values and narrower interquartile ranges for NO, $NO_2$ and $NO_x$ levels based on LUR modeling. Thus, our estimates of associations between traffic pollutants and lung function parameters for children with three acceptable and reproducible curves may be affected, in part, by the differential loss of individuals who were more highly exposed to traffic. The subset of children with valid lung function data tended to be a higher socioeconomic status, suburban population that is more highly exposed to regionally distributed pollutants versus local traffic pollutants in the urban core of LA. Our analyses may also be impacted by quality of the spirometry measurements: we only had available one cross-sectional measure of lung function taken in homes during a study field visit by multiple interviewers rather than at schools or at clinics where differences in technician and test procedures are more tightly controllable and the test results less influenced by such variability in the field. Suggestions of greater air pollution impacts on lung function in higher versus lower SES areas based on analyses stratified on median census tract-level economic disadvantage,

might reflect in part better quality spirometry measurements in those areas, but again, sample sizes for these analyses were very small, and 95% CIs for point estimates across strata were widely overlapping. Furthermore, there was insufficient sample size for stratified analyses restricing to children with three acceptable and reproducible curves. Originally, the L.A. FANS-2 study planned to enroll approximately 4,000 children (all of the L.A. FANS-1 children plus a sample of new entrants into each neighborhood). Despite extending data collection by approximately one and a half years, the total number of L.A. FANS-2 child participants was approximately 35% of the originally planned enrollment number. This was most likely due to the longitudinal nature of the study and the unanticipated additional efforts required to trace and re-enroll L.A. FANS-1 study participants after approximately 5 years without any study contact in a highly mobile community of mostly low SES and immigrant parents. Also, the extensive collection of physical measurements newly included in the L.A. FANS-2 protocol added logistical complexities to implement the survey and further increased participant burden. Finally, as illustrated by findings from the CHS, consideration of time-activity patterns (i.e., time spent outdoors) may be important in estimating air pollution impacts on lung function, especially for the large airway measures of $FEV_1$ and FVC, and unfortunately detailed time-activity information was not collected as part of L.A. FANS-2.

Although several studies have silmiliarly reported greater air pollution impacts on girls compared boys, reasons for differential suspectibility are currently unknown.[49] One possible explanation is differences in growth during adolescence, with girls achieving their full height and maximum lung size considerably earlier than boys. However, whether this difference in the growth process affects susceptibility to air pollution is not known.[152] As girls enter the reproductive period of their life, and airways become subject to cyclical fluctuation of their sex hormones, they exhibit premenstrual changes including increases in airway responsiveness to methacholine, in chemosensitivity, in ventilatory demands, especially on exercise and decreases in spirometric lung function.[153] Whether these hormonal differences affect defense mechanisms and/or response to air pollutants is not known. Differences in the response to cigarette smoking also have been observed, with larger effects seen in young women.[154] Differences in airway susceptibility to air pollution in girls compared to boys is an area that requires further investigation.

**Study Strengths and Limitations**

One strength of this study was the use of a large neighborhood-level campaign of air monitoring and geostatistical modeling to estimate spatial variability in exposures to traffic-related air pollution on a small spatial scale. The use of LUR modeling likely resulted in traffic air pollution exposure estimates with less misclassification than estimates based on pollutant measurements at existing government air monitors, [28-30] a method which has been used in most previous epidemiologic studies of respiratory health impacts. The latter approach assumes concentrations of air pollutants are relatively homogenous over large geographic areas, which is particularly problematic for traffic exhaust pollutants known to have a more heterogeneous spatial distribution with peaks near roadway sources.[8-13] Nonetheless, our estimates are still biased to some extent by exposure misclassification since although the LUR model provides estimates of air pollution levels outside of children's homes and schools, it does not account for variability in personal exposures due to differences in time-activity patterns (time spent outdoors, indoors and in vehicles) and levels of air pollutants in indoor and in-vehicle microenvironments. It has been shown that exposures to traffic exhaust particles in the UF size range are much higher

while driving in vehicles.[155-157] Unfortunately, we did not have detailed time-activity information for children to take into account such exposures. As discussed previously, the differences in $O_3$ effects estimates for wheeze and asthma in higher compared to lower SES neighborhoods could in part reflect important differences in time-activity patterns and resulting exposures across neighborhoods, but may also be reflective of differential access to asthma care and diagnosis and treatment as well. Additionally, the LUR model was built relying on measurements of NO, $NO_2$ and $NO_x$ as markers of the suite of pollutants present in exhaust emissions. Additional measurements would be required to determine how well such pollutants reflect spatial patterns of and personal exposure to other pollutants of biologic interest for respiratory health such as ultrafine particles and PAHs.

Another source of error in our analysis is outcome misclassification. We relied on parental reports of wheeze and asthma, as discussed above. Analyses stratified on census tract-level economic disadvantage indicated stronger associations between wheeze and asthma and traffic air pollution in higher SES areas. This may be due in part to better reporting of these outcomes (i.e. less outcome misclassification) in such areas. Odd ratios for current wheeze, which was asked of all parents whether or not the child had been diagnosed with asthma, were similar in lower and higher SES areas, further demonstrating how access to health care and differential reporting might impact population-based studies of traffic impacts on asthma symptoms.

The L.A. FANS-2 survey included collection of detailed information on perceptions of neighborhood quality, including safety, cohesion and support, allowing us to take such factors into consideration in our analyses. We found adjustment for such factors was important in our analyses of traffic air pollution impacts on wheeze and asthma, as including such factors in statistical models increased estimated associations. This was because odds of reporting the outcomes of interest were greater in more cohesive, supportive, safe and higher SES neighorboods, while on the other hand, traffic air pollution was lower in such areas. Unfortunately, the length and complexity of the L.A. FANS-2 survey in combination with the collection of physiological measures, may have reduced participation and increased logistical complexity resulting in the lower than expected number of subjects who completed the study, as discussed previously. Thus, we had very limited statistical power to examine differences in effect measures after stratifying on neighborhood quality factors, especially for analyses of lung function, where sample sizes were small after stratifying on gender and restricting to those children with 3 acceptable curves (for example, there were 221 boys and 174 girls with three acceptable and reproducible curves). This highlights the importance of balancing data collection against study participation and logistical practicalities in future population-based studies.

## VI. SUMMARY AND CONCLUSIONS

There is a growing literature linking exposure to traffic exhaust pollutants to adverse respiratory health in children. A limited number of studies have used advanced GIS modeling techniques to estimate exposure to traffic pollutants on a fine spatial scale, versus relying on ambient monitoring data or cruder traffic metrics, such as levels of traffic surrounding homes and schools. Here we developed a land use regression (LUR) model for the Los Angeles Basin of Southern California. Novel aspects of this LUR modeling effort compared to previously published work include: (1) use of a large number of sampling sites (~200) for simultaneous passive measurement of NO, $NO_2$ and $NO_x$ over a large and complex geographic region, (2) use of "A Distance Decay REgression Selection Strategy" (ADDRESS) to explore importance of

geographic features within many different size buffers and development of spatial models highly predictive of measured concentrations, and (3) use of remote sensing data to provide additional information on geographic distribution of traffic sources and improve LUR model predictions. Our final LUR models explained 81%, 86% and 85% of the variation in NO, $NO_2$ and $NO_x$ concentrations, respectively, higher prediction powers than a large majority of previously published LUR surfaces.[132-134] LUR model results indicated traffic on highways and major roads as far away as 11 km from measurement sites still had important impacts on measured $NO_x$ concentrations, a much greater spatial extent than previously reported in the literature.[7] Although incorporating the influence of traffic at farther distances improved prediction ability for NO, $NO_2$ and $NO_x$, we generated a separate set of "more local traffic" LUR models that excluded traffic at distances greater than 5000 m (resulting $R^2$ values were 0.64, 0.78 and 0.68, respectively). It is currently unknown how well LUR models built on $NO_x$ measurements predict other pollutants released directly in motor vehicle exhaust that are also of biologic interest. In L.A., concentrations of UF particles have been shown to decrease exponentially with distance from freeways, reaching background concentrations within approximately 150 meters.[8-13] Thus, it may be that the "more local traffic" LUR models we developed here provide better surrogate estimates of exposure for fresh vehicle exhaust and UF and associated toxics; additional measurement data are needed to examine this hypothesis further. We also generated kriged exposure surfaces for peak daily $O_3$ and $PM_{2.5}$ to examine the importance of exposure to more regionally distributed background pollutants on respiratory health in children.

Using the LUR and kriged model surfaces to estimate exposures, we evaluated associations between air pollution and respiratory health in the 1,387 children who participated in L.A. FANS-2. Based on our models, children more highly exposed to traffic pollution as estimated by interquartile increases in "more local traffic" LUR model estimates for NO, $NO_2$ and $NO_x$ were approximately 30-40% more likely to report wheeze in the past 12 months. These estimates were robust to adjustment for many different family- and neighborhood-level SES factors. The stronger associations observed between exposure metrics from the more local traffic versus those from the final, optimized LUR models suggest pollutants released directly in exhaust emissions and with sharp concentration peaks close to sources (e.g., UF and associated toxics) may be the most relevant for current wheeze symptoms. We observed weaker and more marginal 15% increases in odds of medication use for asthma and wheeze in the past year and doctor-diagnosed asthma per interquartile increase in local traffic NO, $NO_2$ and $NO_x$. However, when we stratified analyses on median census tract-level economic disadvantage, odds of both asthma outcomes increased by 40% per IQR increase in traffic pollution, similar to results for current wheeze, but in higher SES areas only. While we included health insurance status, a usual source of sick care and other family SES characteristics into our models to adjust for confounding by these covariates, misclassification of outcome may still bias our estimates due to differential access to health care and differences in diagnostic and treatment practices for asthma across communities. The stronger associations with traffic pollution observed in higher SES areas may thus in part reflect differential access to health care and resulting differences in diagnosis and reporting in higher compared to lower SES communities. Stratified analyses indicated 80-100% increases in current wheeze and medication use for asthma and wheeze per IQR increases in peak 8-hour $O_3$, but only in lower SES areas, while null or inverse associations between peak $O_3$ and these outcomes were observed for children living in higher SES areas. This may reflect differences in children's behaviors (e.g., time spent outdoors) and resulting exposures during pollution epispodes or may be an artifact of our very small sample size when

stratifying in this manner. The weaker (50%) increases in odds of doctor-diagnosed asthma with increases in peak $O_3$ estimated for children living in lower SES areas may reflect bias due to exposure misclassification since lifetime exposure averages from birth to date of asthma diagnosis may be more relevant for doctor-diagnosed asthma, as opposed to the averages over shorter time periods prior to interview we employed. Unfortunately the number of asthmatic children for whom we had life-time residential histories was too small to perform meaningful statistical analyses of these exposure periods.

Similar to previous cross-sectional studies in Europe and the U.S., we observed reductions in lung function with increasing exposure to traffic pollution, but our results differed substantially between girls and boys and for children with lower versus higher quality spirometry curves. In boys, we estimated 70-100 mL reductions in lung volume and 60-100 mL/s decrements in expiratory flow per interquartile increase in final, optimized model LUR estimates of NO, $NO_2$ and $NO_x$ based on the current home (associations for the other averaging periods we evaluated were similar). Slightly lower associations were observed for $PM_{2.5}$ exposures (40-50 mL reductions in volume and 60-90 mL/s reductions in flow per IQR increase). However, when restricting analyses to boys with three acceptable and reproducible curves, negative associations were more imprecisely estimated and in general did not reach statistical significance, except for those between IQR increases in $PM_{2.5}$ and $FEF_{75}$ and $FEF_{25-75}$. In girls, we estimated 40-80 mL reductions in $FEV_1$ with increasing exposure to LUR-estimates of NO, $NO_2$ and $NO_x$ averaged over the 5 years prior to interview, but no associations with FVC. However, stronger associations between traffic pollution and measures of expiratory flow were observed in girls than in boys (300-350 mL/s reductions in PEF mL/s and 200-300 reductions in $FEF_{25-75}$ per IQR increase in final, optimized model LUR estimates of NO, $NO_2$ and $NO_x$). However, these effects were observed only when considering all girls with one or more acceptable spirometry curve and not seen in the group with three acceptable and reproducible curves. This may be due to the smaller sample size available and/or the characteristics of the select group with three curves available (i.e., higher SES with higher exposure to $O_3$ and lower exposure to traffic pollutants). Similar to previous U.S. cross-sectional studies,[14-16] we also observed reductions in PEF in girls more highly exposed to peak daily $O_3$ (~100 mL/s decrements and ~400 mL/s decrements per 30 ppb increase in $O_3$ for girls with one or more acceptable curves and three acceptable and reproducible curves, respectively) and again these estimated effects were robust to adjustment for family- and neighborhood-level measures of SES. Similar to previous literature, this suggests important differences in the biological impact of air pollution on lung health in boys versus girls. Differences in susceptibility to air pollution may be due to differences in growth patterns or due to hormonal differences, but additional research is needed to uncover possible underlying biological mechanisms.

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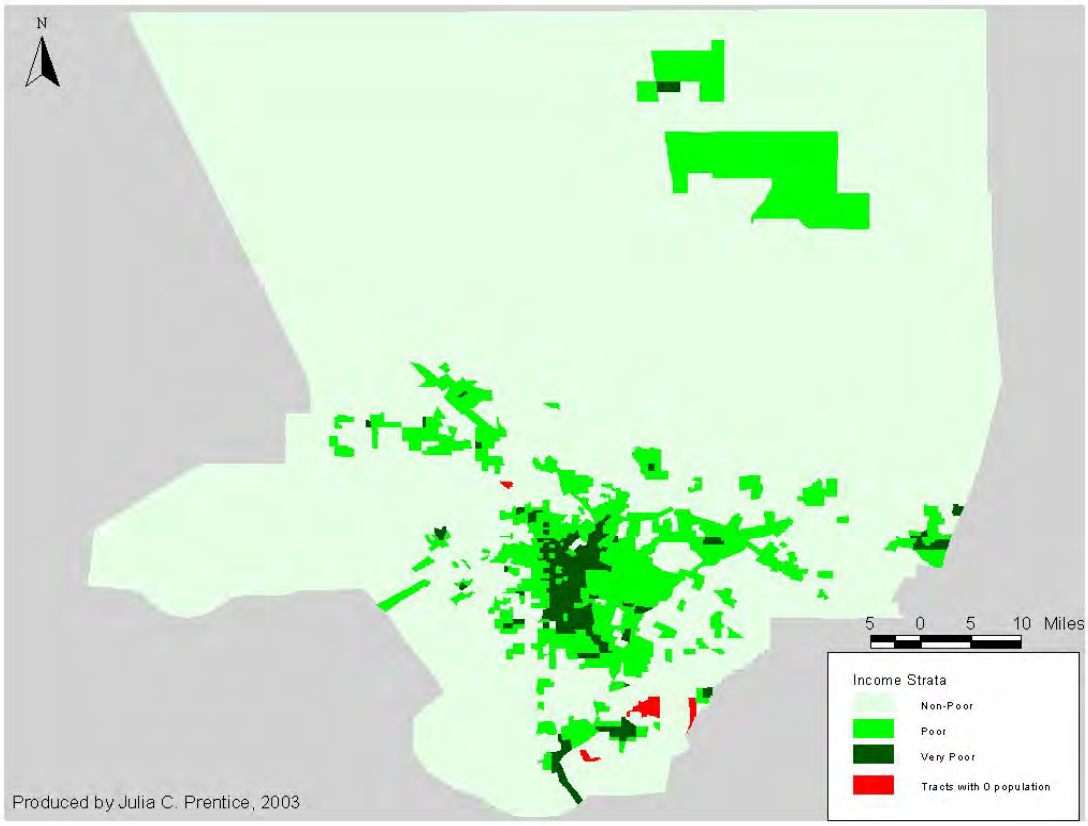


**Figure 1. Ranking of L.A. County Census Tracts by L.A. FANS Poverty definition**

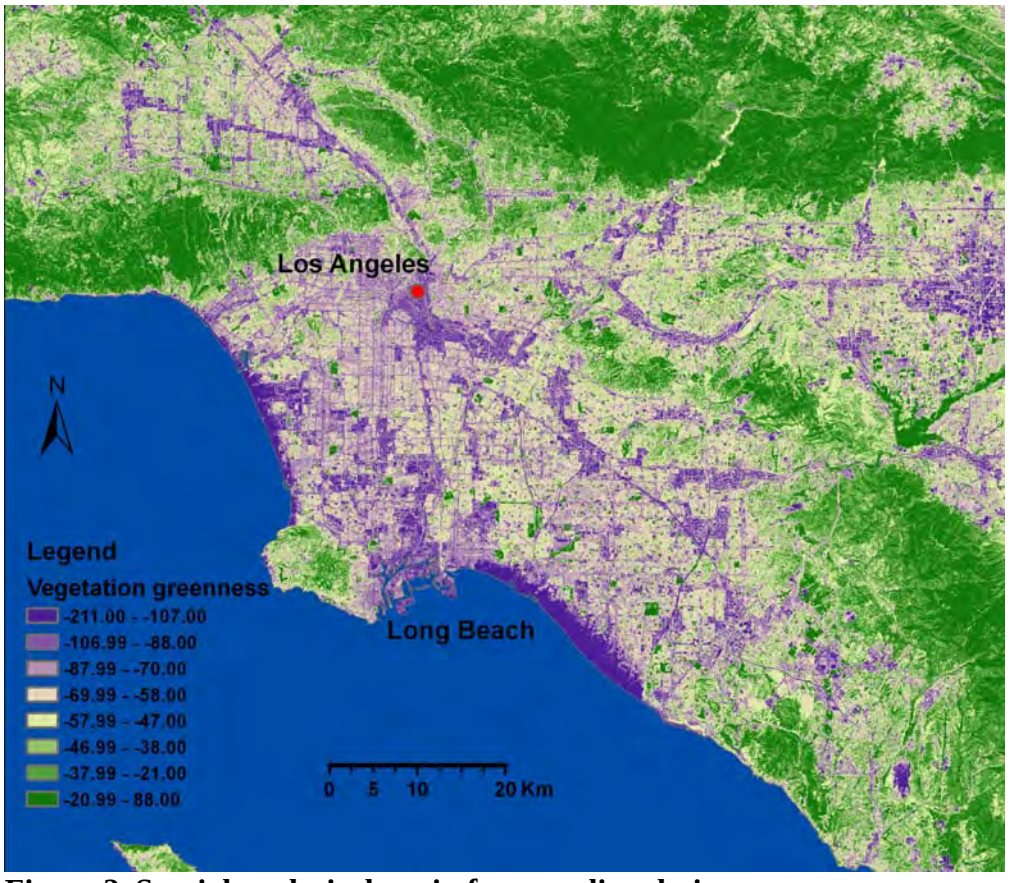


**Figure 2. Spatial analysis domain for sampling design**

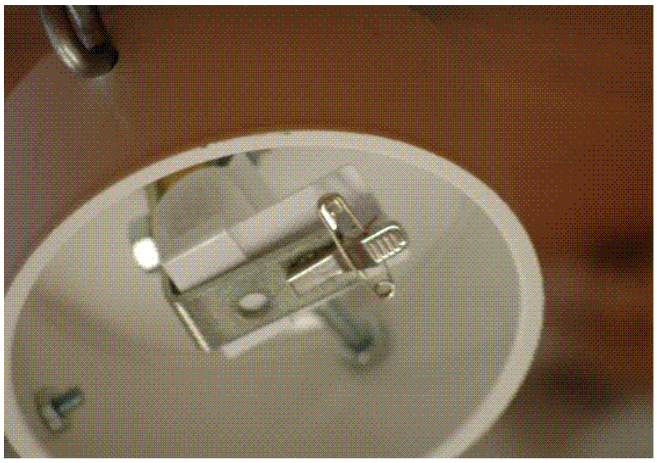

**Figure 3. Sampler inside rain shelter**

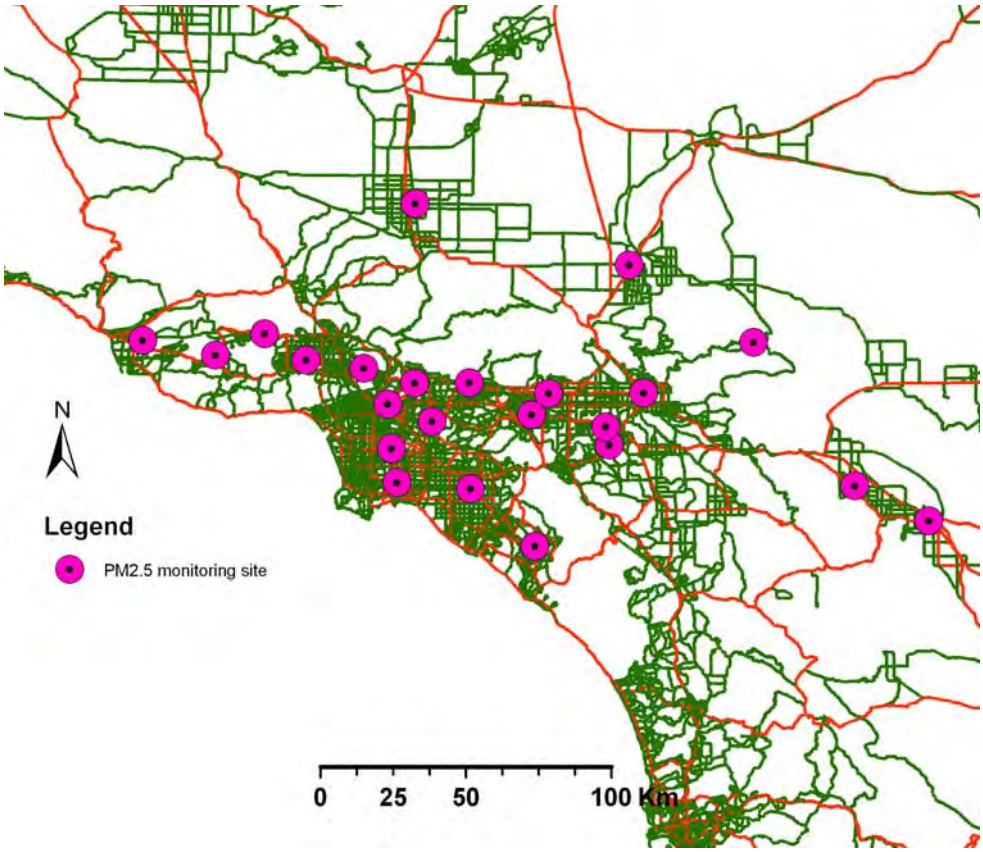


**Figure 4. Los Angeles Basin $PM_{2.5}$ monitoring sites**

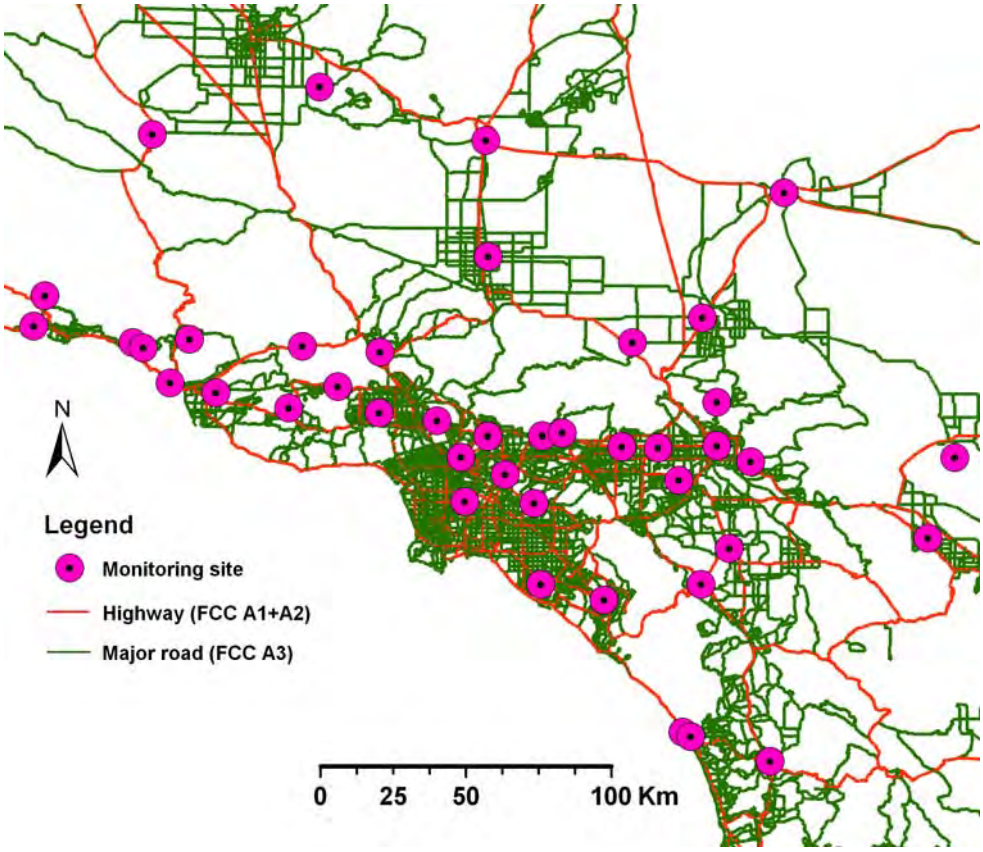


**Figure 5. The Los Angeles Basin $O_3$ monitoring sites**

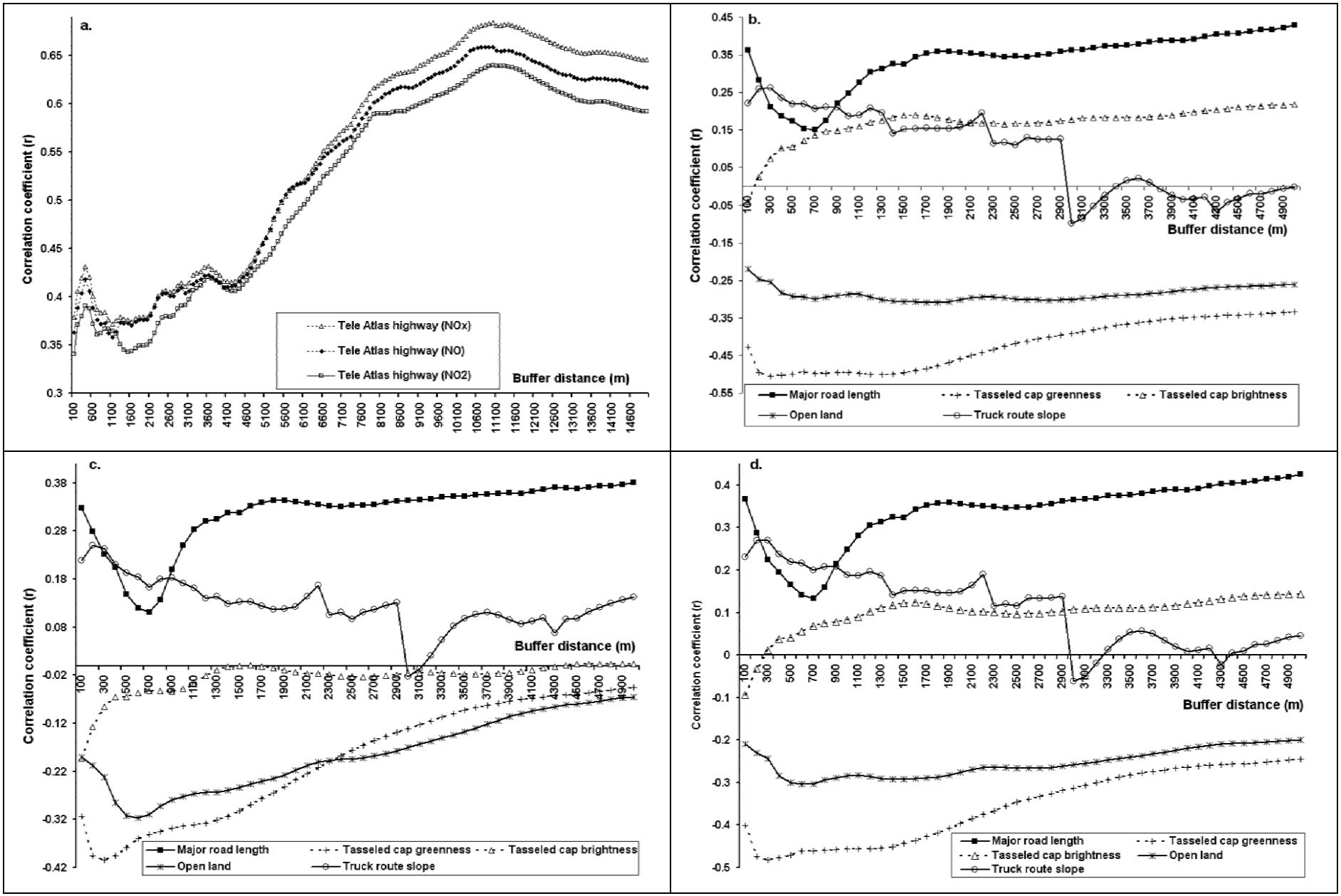


**Figure 6a-6d. Distance decay curves of correlations between selected spatial covariates and measured air pollution concentrations (6a for traffic volumes - total vehicle miles traveled, 6b for NO, 6c for $NO_2$ and 6d for $NO_x$**

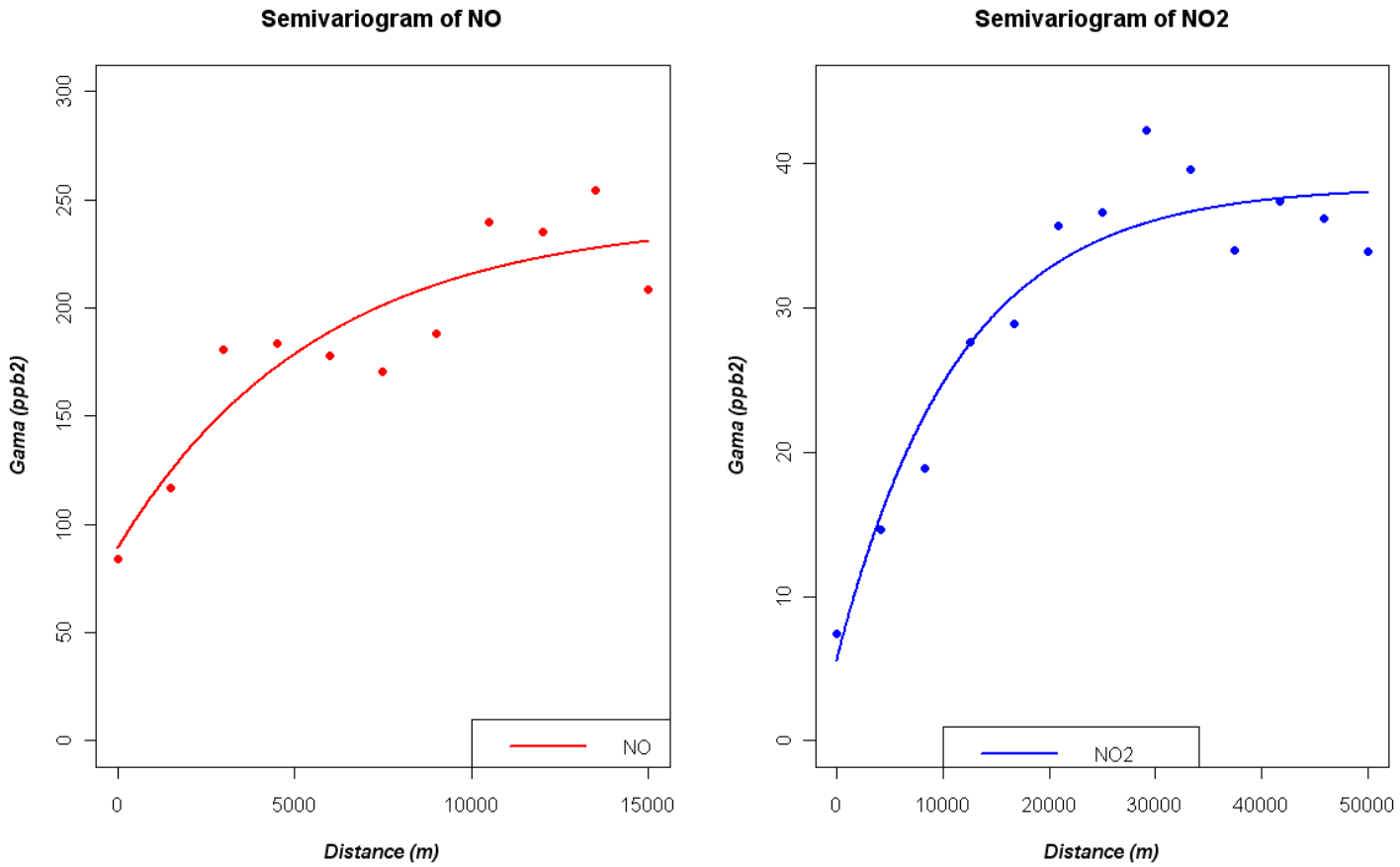


**Figure 7. Semivariograms of NO and $NO_2$ based on measurements from the 201 monitoring sites**

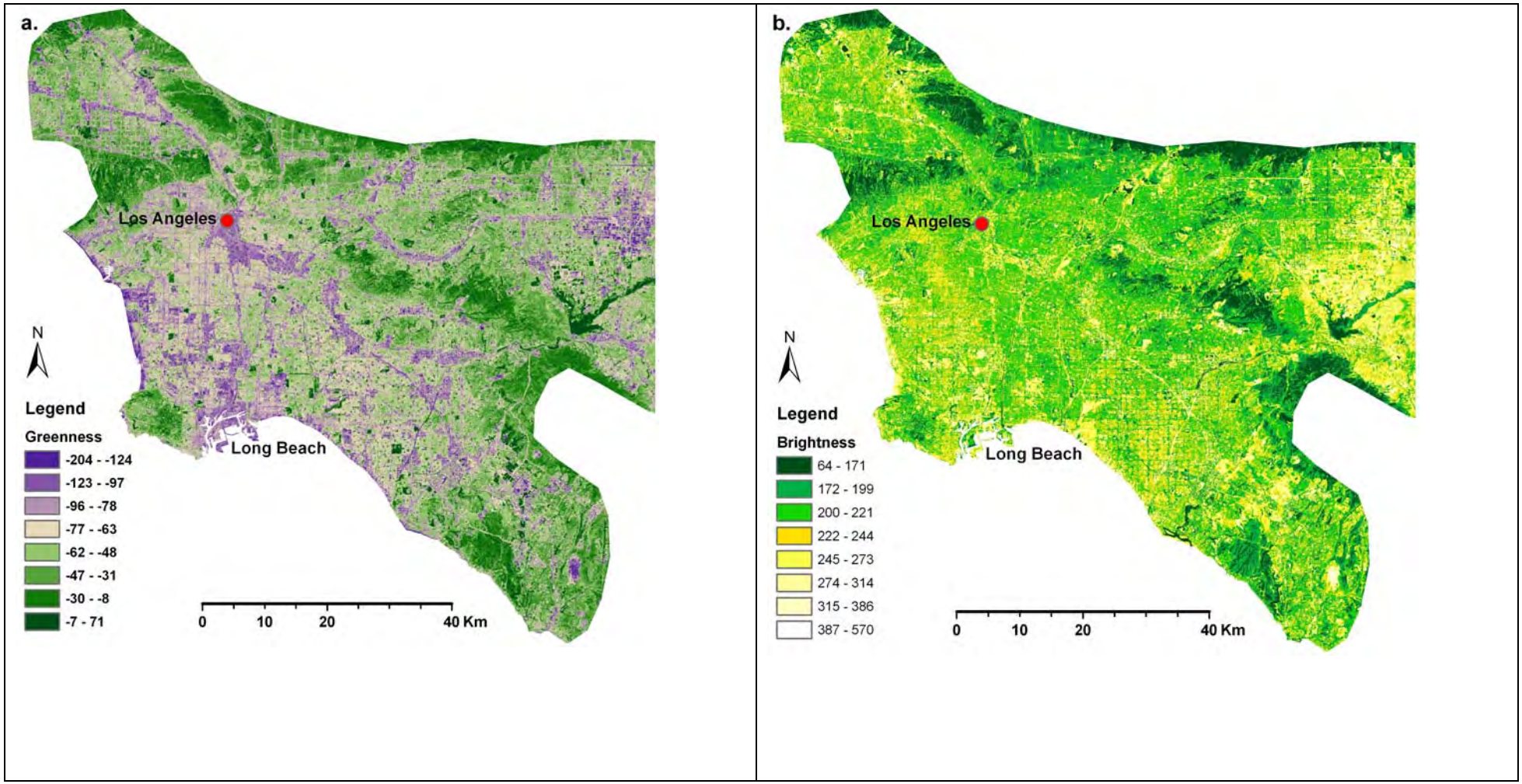


**Figure 8. Tasseled-cap greenness (8a) and soil brightness (8b)**

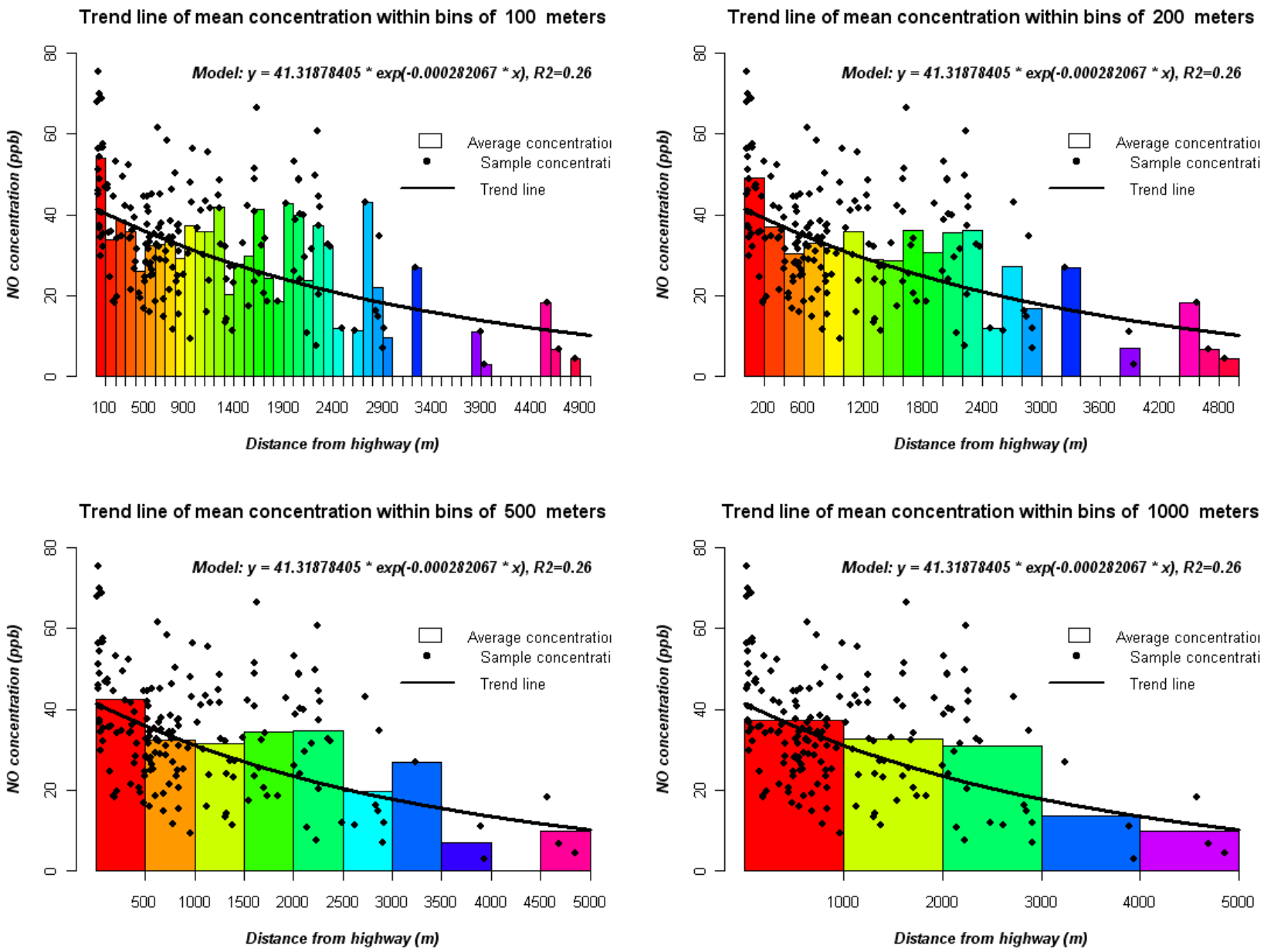


**Figure 9. Distance decay of NO concentrations further away from highways (A1 and A2) based on 201 monitoring sites in the LA metropolitan area**

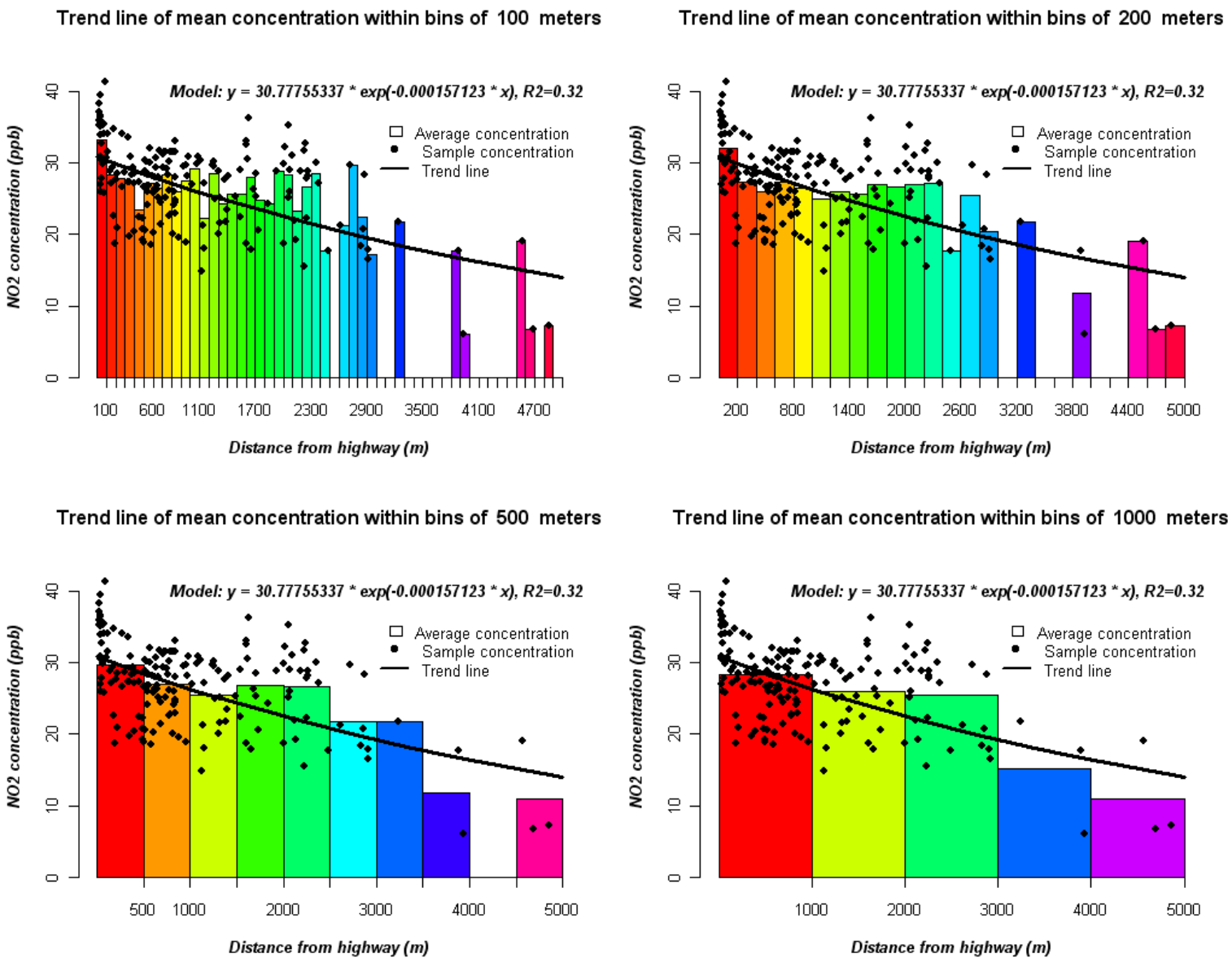


**Figure 10. Distance decay of $NO_2$ concentrations further away from highways (A1 and A2) based on 201 monitoring sites in the LA metropolitan area**

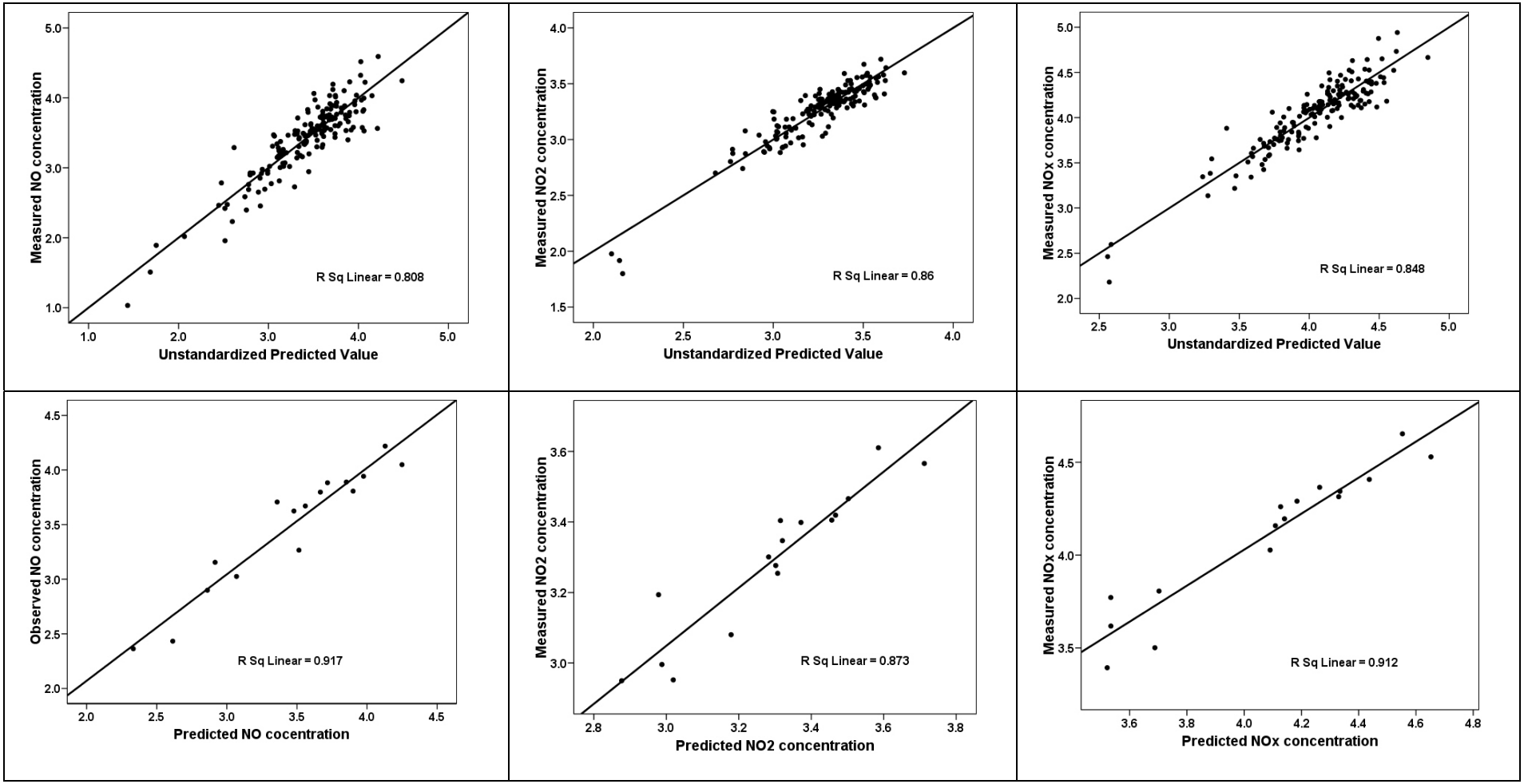


**Figure 11. Model predictions of natural log-transformed NO, $NO_2$ and $NO_x$ (11a,11b and 11c) and corresponding cross-validation results (11d, 11e and 11f)**

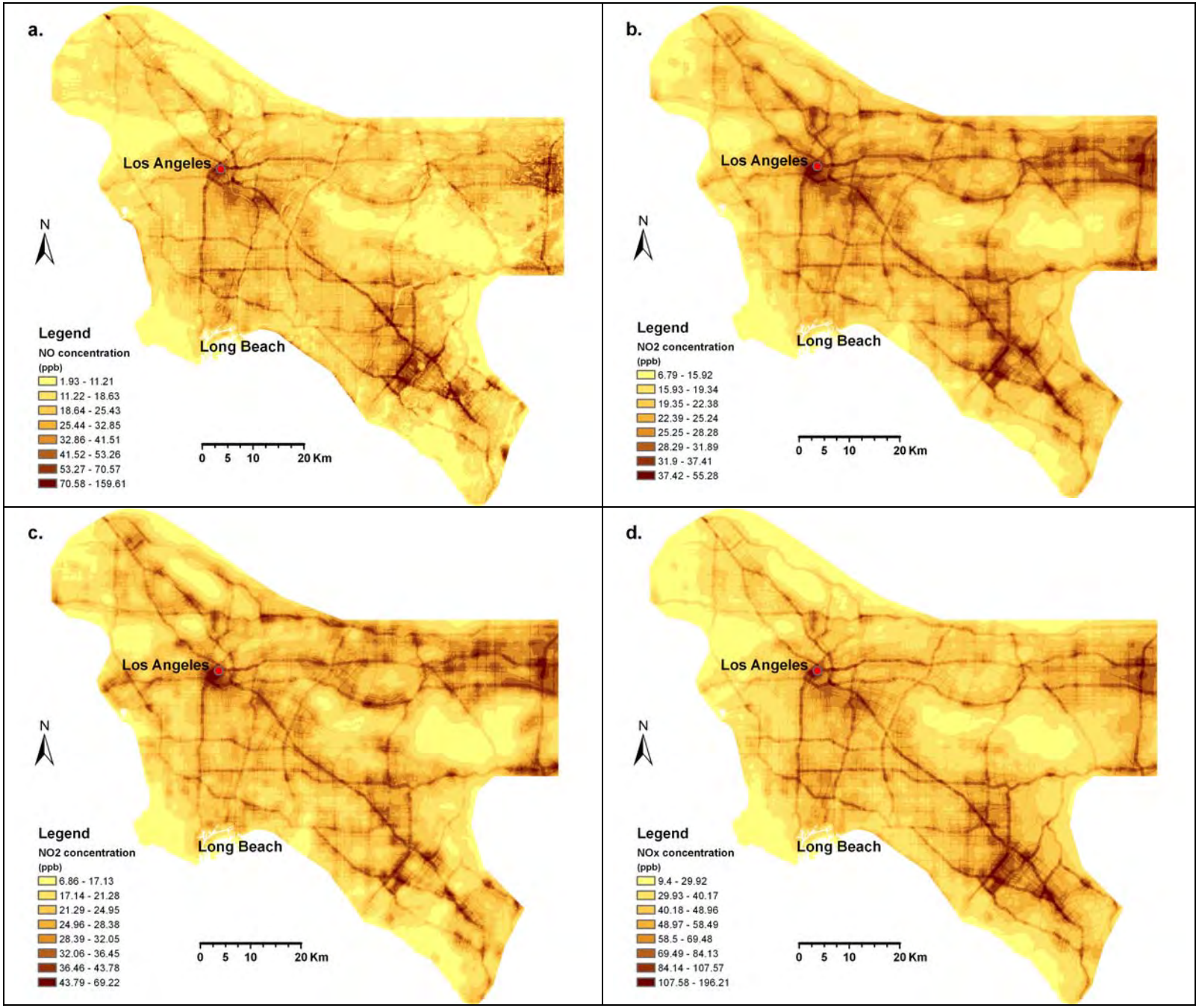


**Figure 12. Model prediction surfaces of NO (12a), $NO_2$ (12b, 12c) and $NO_x$ (12d) through an ADDRESS selection process (12b with and 12c without buffer distance within 11 km)**

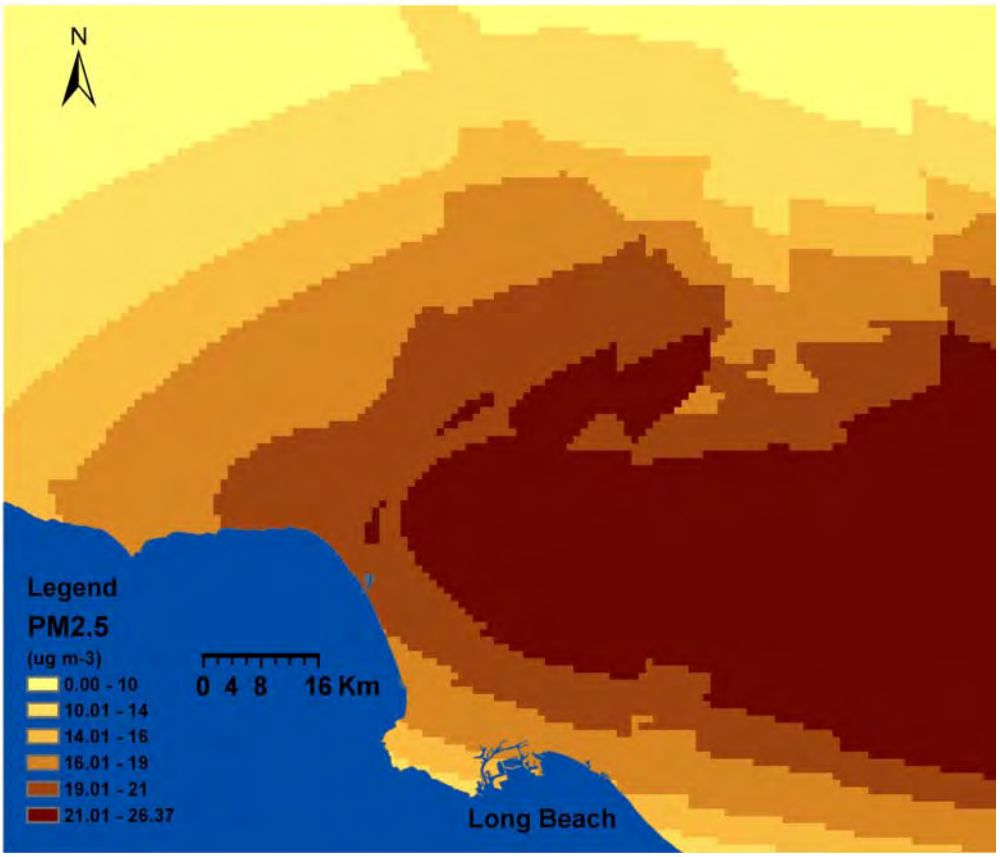


**Figure 13. $PM_{2.5}$ surface through kriging for the LA Basin**

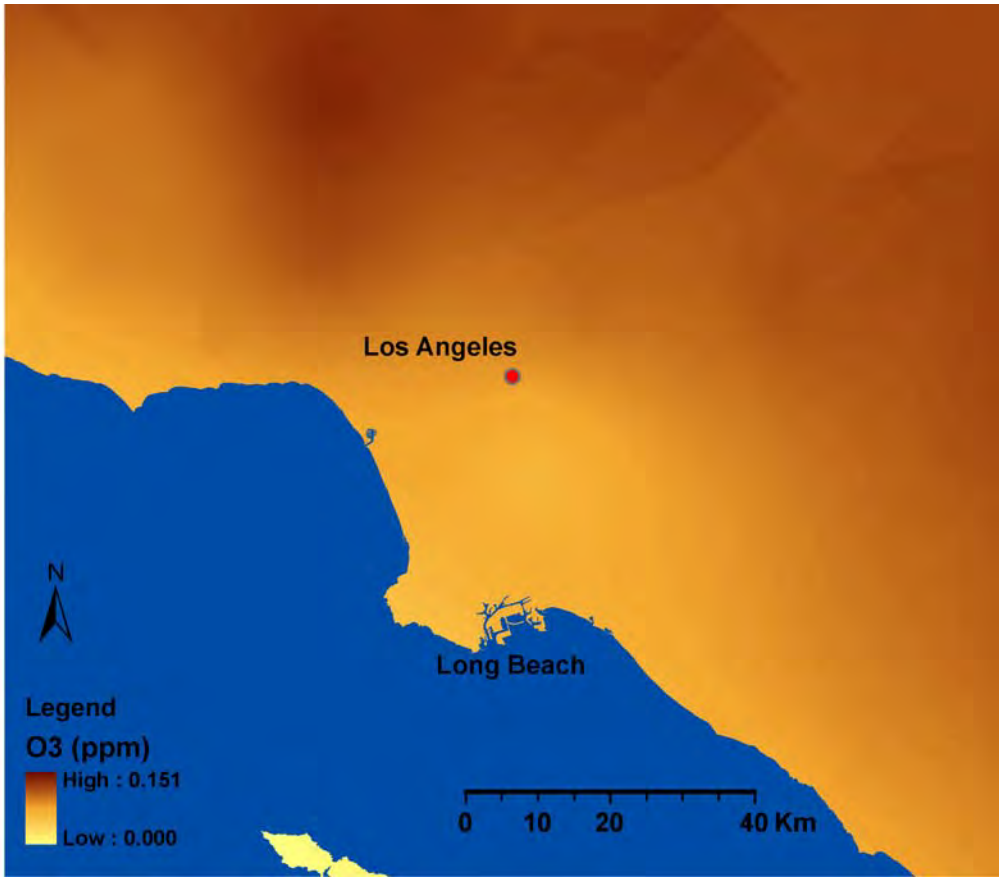


**Figure 14. $O_3$ ordinary kriging surface for the LA Basin**

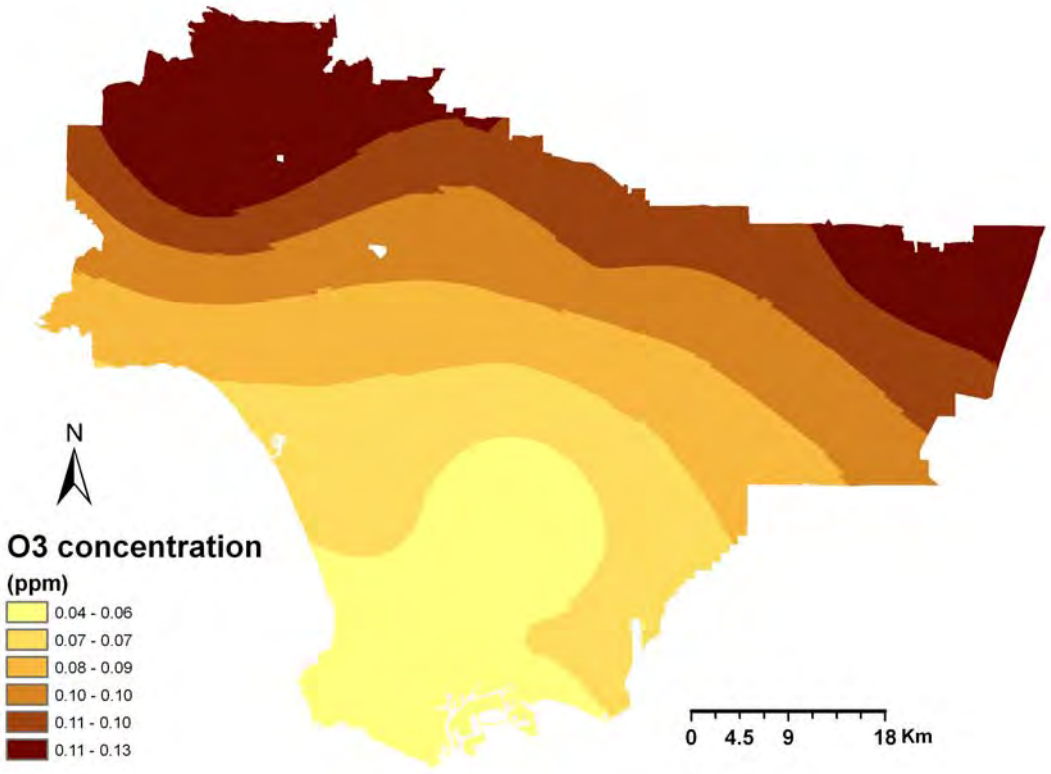


**Figure 15. $O_3$ ordinary kriging surface for the LA Basin (close up of urban core area)**

**Table 1. South Coast Air Quality Management District (SCAQMD) air monitoring stations where Ogawa samplers were co-located during each monitoring session.**

| LOCATION | ADDRESS | CARB ID | AIRS SITE ID |
|---|---|---|---|
| Azusa | 803 N. Loren Ave., Azusa, CA 91702 | 2484 | 60370002 |
| Burbank | 228 W. Palm Ave., Burbank, CA 91502 | 2492 | 60371002 |
| Downtown Los Angeles | 1630 N Main St, Los Angeles, CA 90012 | 2899 | 60371103 |
| Glendora | 840 E Laurel Ave, Glendora, CA 91741 | 2849 | 60370016 |
| Hawthorne | 7201 W Westchester Parkway, Los Angeles CA 90045 | 3683 | 60375005 |
| La Habra | 621 W Lambert Rd, La Habra, CA 90631 | 2249 | 60595001 |
| Lynwood | 11220 Long Beach Blvd. Lynwood, CA 90262 | 2583 | 60371301 |
| North Long Beach | 3648 N. Long Beach Blvd. Long Beach 90807 | 2429 | 60374002 |
| Pasadena | 752 S. Wilson Ave. Pasadera, CA 91106 | 2160 | 60372005 |
| Pico Rivera | 4144 San Gabriel River Parkway, Pico Rivera, CA 90660 | 3693 | 60371602 |
| Pomona | 924 N. Garey Ave. Pomona, CA 91767 | 2898 | 60371701 |
| Reseda | 18330 Gault St. Reseda, CA 91335 | 2420 | 60371201 |
| Santa Clarita | 22224 Placerita Canyon Road, Santa Clarita, CA 91321 | 3502 | 60376012 |
| South Long Beach | 1305 East Pacific Coast Highway, Long Beach, CA 90806 | 3679 | 60374004 |
| West Los Angeles | 11301 Wilshire Blvd., Los Angeles, CA 90073 | 2494 | 60370113 |

**Table 2. Traffic statistics for measured road segments in LA and the proportion of roads covered with measurements.**

| Road category* | Traffic volume measurements (AADT) | | | | | | TeleAtlas Dynamap Data Coverage | |
|---|---|---|---|---|---|---|---|---|
| | # roads | Minimum | Maximum | Mean | Median | Std Dev | # roads | % measured |
| A1 | 1,658 | 3,100 | 190,000 | 92,969 | 104,000 | 39,860 | 24,508 | 6.77 |
| A2 | 799 | 1,885 | 76,000 | 21,458 | 18,750 | 10,757 | 9,004 | 8.87 |
| A3 | 8,045 | 21 | 114,622 | 15,848 | 13,500 | 11,120 | 124,391 | 6.47 |
| A4 | 7,954 | 1 | 129,500 | 4,611 | 2,383 | 6,093 | 500,302 | 1.59 |
| A5 | 3 | 564 | 2,100 | 1,093 | 614 | 873 | 9,105 | 0.03 |
| A6 | 40 | 556 | 148,500 | 40,407 | 17,000 | 45,863 | 37,866 | 0.11 |
| A7 | 5 | 488 | 26,950 | 9,501 | 6,780 | 11,008 | 34,871 | 0.01 |
| Total: | 18,504 | | | | | | 740,047 | 2.50 |

*A1: Primary highway with limited access; A2: primary road without limited access; A3: secondary and connecting road; A4: local, neighborhood and rural road; A5: vehicular trail; A6: road access ramp; A7: road as other thoroughfare.

**Table 3. L.A. FANS Wave-2 Child Respiratory Health Questions (asked of PCGs)**

| |
|---|
| Has a doctor or other health professional ever told you that (child's name) has asthma (Yes, No)? |
| How old was (child's name) when the doctor first told you that (he/she) had asthma? (Age in months or years)<br>Asked of those reporting doctor-diagnosed asthma |
| During the past 12 months (or since (child's name) was born if less than 1 year old), has your child ever had wheezing or whistling in the chest (Yes, No)? |
| Let me ask you something else about the past 12 months (or the time since (child's name was born if less than 1 year old). In an average week how often has (he/she) woken up because (he/she) was wheezing (Never woken with wheezing; Less than one night per week; One or more nights per week)?<br>Asked of those reporting wheezing in past 12 months |
| During the past 12 months, has wheezing ever been severe enough to limit (child's name)'s speech to only one or two words at a time between breaths (Yes, No)?<br>Asked of those reporting wheezing in past 12 months and child is at least one year of age |
| During the past 12 months (or since (child's name) was born if less than 1 year old), has (child's name) used any medicines, pills, puffers or other medication for wheezing or asthma (Yes, No)? |
| During the past 12 months (or since (child's name) was born if less than 1 year old), has (he/she) had a problem with sneezing, or a runny or blocked nose when he/she DID NOT have a cold or the flu (Yes, No)? |
| Has your doctor or health professional ever said that (child's name) had more than 3 ear infections in a year (Yes, No)? |

**Table 4. Basic spirometry measures and definitions**[127]

| Spirometry measurement | Abbreviation | Description |
|---|---|---|
| Forced Vital Capacity | FVC | This is the total amount of air forcibly blown out after full inspiration, measured in liters. |
| Forced Expiratory Volume in 1 Second | $FEV_1$ | This is the amount of air forcibly blown out in one second, measured in liters. Along with FVC it is considered one of the primary indicators of lung function. |
| Peak Expiratory Flow | PEF | This is the speed of the air moving out of the lungs at the beginning of the expiration, measured in liters per second. |
| Forced Expiratory Time | FET | This measures the length of the expiration in seconds. |
| Forced Expiratory Flow at 25% of FVC | $FEF_{25}$ | This is the flow of air measured at the time when 25% of the entire FVC has been expelled. |
| Forced Expiratory Flow at 75% of FVC | $FEF_{75}$ | This is the flow of air measured at the time when 75% of the entire FVC has been expelled. |
| Forced Expiratory Flow between 25% and 75% of FVC[2] | $FEF_{25-75}$ | This is average flow of air measured during the interval between the time when 25% and 75% of the entire FVC has been expelled. |

(1) $FEF_{25-75}$ is also called Maximum Midexpiratory Flow (MMEF)

**Table 5. Mean, median and range of measured $NO_2$ and $NO_x$ at all neighborhood locations (including duplicates) and AQMD stations, adjusted for blanks. NO concentrations were calculated as the difference between $NO_x$ and $NO_2$ measures.**

| | Mean (ppb) | Median (ppb) | Range (ppb) |
|---|---|---|---|
| Sept 2006, $NO_2$ | 29.2 | 29.6 | 5.4 – 42.7 |
| Sept 2006, $NO_x$ | 58.2 | 57.9 | 8.1 – 122.8 |
| Sept 2006, NO | 29.0 | 27.4 | 2.6 – 80.1 |
| Feb 2007, $NO_2$ | 24.5 | 25.6 | 5.3 – 39.8 |
| Feb 2007, $NO_x$ | 64.1 | 64.3 | 9.6 – 157.0 |
| Feb 2007, NO | 39.6 | 38.6 | 3.0 – 117.2 |

**Table 6. Model prediction results using ADDRESS model, ADDRESS model with clustering considered, and GEE model for NO, $NO_2$ and $NO_x$.**

| Pollutant | Variable | ADDRESS model* | | | | | ADDRESS model with clustering** | | | | GEE model*** | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | Coef. | Std. Err. | t | P>\|t\| | VIF* | Coef. | Std. Err. | t | P>\|t\| | Coef. | Std. Err. | t | P>\|t\| |
| 6a. NO | Intercept | -2.6407240 | 0.654861 | -4.03 | 0.000 | | -2.6407240 | 0.792661 | -3.33 | 0.001 | -2.6407240 | 0.634952 | -4.16 | 0.000 |
| | TeleAtlas traffic highway and major roads (11000 m) | 0.00000003 | 0.000000 | 11.93 | 0.000 | 1.35 | 0.00000003 | 0.000000 | 10.58 | 0.000 | 0.00000003 | 0.000000 | 12.30 | 0.000 |
| | TeleAtlas traffic all roads (400 m) | 0.00000210 | 0.000000 | 6.75 | 0.000 | 1.27 | 0.00000210 | 0.000000 | 5.91 | 0.000 | 0.00000210 | 0.000000 | 6.96 | 0.000 |
| | Distance to truck routes (m) | -0.00003880 | 0.000013 | -3.06 | 0.003 | 1.49 | -0.00003880 | 0.000014 | -2.87 | 0.005 | -0.00003880 | 0.000012 | -3.15 | 0.002 |
| | Major road (100 m) | 0.00053750 | 0.000143 | 3.77 | 0.000 | 1.13 | 0.00053750 | 0.000178 | 3.01 | 0.004 | 0.00053750 | 0.000138 | 3.89 | 0.000 |
| | Industrial (2700 m) | 0.00036130 | 0.000084 | 4.29 | 0.000 | 1.20 | 0.00036130 | 0.000089 | 4.08 | 0.000 | 0.00036130 | 0.000082 | 4.42 | 0.000 |
| | Commercial (1200 m) | 0.00277730 | 0.000533 | 5.21 | 0.000 | 1.19 | 0.00277730 | 0.000514 | 5.40 | 0.000 | 0.00277730 | 0.000517 | 5.37 | 0.000 |
| | Soil brightness (700 m) | 0.01005310 | 0.001727 | 5.82 | 0.000 | 1.32 | 0.01005310 | 0.001935 | 5.20 | 0.000 | 0.01005310 | 0.001674 | 6.00 | 0.000 |
| | X coordinate | 0.00000660 | 0.000001 | 5.76 | 0.000 | 1.09 | 0.00000660 | 0.000001 | 4.80 | 0.000 | 0.00000660 | 0.000001 | 5.94 | 0.000 |
| | Open (100 m) | -0.1542625 | 0.049998 | -3.09 | 0.002 | 1.20 | -0.1542625 | 0.047074 | -3.28 | 0.002 | -0.1542625 | 0.048478 | -3.18 | 0.001 |
| | $R^2$ (p) \| $R^2$ (p)**** | 0.81 (< 0.0001) \| 0.92 (< 0.0001) | | | | | 0.81 (< 0.0001) | | | | | | | |
| 6b. $NO_2$ | Intercept | -11.282530 | 2.443303 | -4.62 | 0.000 | | -11.282530 | 3.725334 | -3.03 | 0.003 | -11.2825300 | 2.369021 | -4.76 | 0.000 |
| | TeleAtlas traffic highway and major roads (11000 m) | 0.00000001 | 0.000000 | 9.72 | 0.000 | 1.44 | 0.00000001 | 0.000000 | 7.23 | 0.000 | 0.00000001 | 0.000000 | 10.03 | 0.000 |
| | TeleAtlas traffic all roads (400 m) | 0.00000072 | 0.000000 | 5.26 | 0.000 | 1.28 | 0.00000072 | 0.000000 | 4.04 | 0.000 | 0.00000072 | 0.000000 | 5.43 | 0.000 |
| | Distance to truck routes (m) | -0.0000439 | 0.000006 | -7.90 | 0.000 | 1.49 | -0.00004390 | 0.000014 | -3.04 | 0.003 | -0.00004390 | 0.000005 | -8.15 | 0.000 |
| | Major road (100) | 0.00018990 | 0.000063 | 3.01 | 0.003 | 1.15 | 0.00018990 | 0.000070 | 2.72 | 0.008 | 0.00018990 | 0.000061 | 3.11 | 0.002 |
| | Local road (1400) | 0.00000234 | 0.000001 | 2.82 | 0.005 | 1.56 | 0.00000234 | 0.000001 | 2.73 | 0.008 | 0.00000234 | 0.000001 | 2.91 | 0.004 |
| | Industrial (1700 m) | 0.00059240 | 0.000096 | 6.16 | 0.000 | 1.36 | 0.00059240 | 0.000145 | 4.10 | 0.000 | 0.00059240 | 0.000093 | 6.35 | 0.000 |
| | Commercial (1000 m) | 0.00261960 | 0.000308 | 8.50 | 0.000 | 1.16 | 0.00261960 | 0.000376 | 6.96 | 0.000 | 0.00261960 | 0.000299 | 8.77 | 0.000 |
| | X coordinate (m) | 0.00000515 | 0.000001 | 10.11 | 0.000 | 1.12 | 0.00000515 | 0.000001 | 7.38 | 0.000 | 0.00000515 | 0.000000 | 10.43 | 0.000 |
| | Y coordinate (m) | 0.00000316 | 0.000001 | 4.98 | 0.000 | 1.20 | 0.00000316 | 0.000001 | 3.26 | 0.002 | 0.00000316 | 0.000001 | 5.14 | 0.000 |
| | $R^2$ (p) \| $R^2$ (p) | 0.86 (< 0.0001) \| 0.87 (< 0.0001) | | | | | 0.86 (< 0.0001) | | | | | | | |
| 6c. $NO_x$ | Intercept | -0.0590325 | 0.429163 | -0.14 | 0.891 | | -0.0590325 | 0.543035 | -0.11 | 0.914 | -0.05903250 | 0.417438 | -0.14 | 0.888 |
| | TeleAtlas traffic highway and major roads (11000 m) | 0.00000002 | 0.000000 | 13.13 | 0.000 | 1.30 | 0.00000002 | 0.000000 | 10.83 | 0.000 | 0.00000002 | 0.000000 | 13.5 | 0.000 |
| | TeleAtlas traffic all roads (400 m) | 0.00000144 | 0.000000 | 7.11 | 0.000 | 1.27 | 0.00000144 | 0.000000 | 5.20 | 0.000 | 0.00000144 | 0.000000 | 7.31 | 0.000 |
| | TeleAtlas traffic major road (100 m) | 0.00002710 | 0.000007 | 3.92 | 0.000 | 1.14 | 0.00002710 | 0.000008 | 3.22 | 0.002 | 0.00002710 | 0.000007 | 4.03 | 0.000 |
| | Distance to truck routes (m) | -0.0000450 | 0.000008 | -5.47 | 0.000 | 1.47 | -0.0000450 | 0.000011 | -3.94 | 0.000 | -0.0000450 | 0.000008 | -5.63 | 0.000 |
| | Industrial (2700 m) | 0.00029010 | 0.000054 | 5.38 | 0.000 | 1.16 | 0.00029010 | 0.000070 | 4.15 | 0.000 | 0.00029010 | 0.000053 | 5.53 | 0.000 |
| | Commercial (1000 m) | 0.00328070 | 0.000450 | 7.29 | 0.000 | 1.12 | 0.00328070 | 0.000488 | 6.72 | 0.000 | 0.00328070 | 0.000438 | 7.49 | 0.000 |
| | Soil brightness (1700 m) | 0.00442720 | 0.001104 | 4.01 | 0.000 | 1.19 | 0.00442720 | 0.001500 | 2.95 | 0.004 | 0.00442720 | 0.001074 | 4.12 | 0.000 |
| | X coordinate (m) | 0.00000572 | 0.000001 | 7.62 | 0.000 | 1.10 | 0.00000572 | 0.000001 | 6.67 | 0.000 | 0.00000572 | 0.000001 | 7.83 | 0.000 |
| | $R^2$ (p) \| $R^2$ (p) | 0.85 (< 0.0001) \| 0.92 (< 0.0001) | | | | | 0.85 (< 0.0001) | | | | | | | |

* ADDRESS model: An optimized distance decay model selection strategy for our land use regression models. VIF = variance inflation factor. ** For clustering analysis, observations were grouped using census tract. We assumed that measurements from multiple sites within a census tract might be correlated but, across census tracts, they were uncorrelated.*** GEE model: Generalized estimation equation model to analyze correlated data within census tracts.**** R2 (p) | R2 (p): the left side part is for model prediction power and right side for cross-validation result.

**Table 7. Description of Available Residential Air Pollution Estimates by Exposure Period for n=1,387 Children included in L.A. FANS-2**

| Air Pollution Averaging period | No. (%) of subjects with NO, $NO_2$, $NO_x$ and $O_3$ values | No. (%) of subjects with NO, $NO_2$, $NO_x$, $O_3$ and $PM_{2.5}$ values | No. of subjects with LUR or $O_3$ values missing for one or more homes[1] | No. of additional subjects missing $PM_{2.5}$ values for one or more homes[2] |
|---|---|---|---|---|
| Current home | 1378 (99%) | 1364 (98%) | 9 | 14 |
| 12 months prior to interview | 1311 (95%) | 1301 (94%) | 76 | 10 |
| 24 months prior to interview | 1288 (93%) | 1278 (92%) | 99 | 10 |
| 5 years prior to interview | 1223 (88%) | 1213 (88%) | 164 | 10 |

(1) Subjects are missing data because: (a) one or more homes not geocoded and/or (b) homes dates (i.e., dates in each home) do not span this period or are missing data and/or (c) one or more homes fell outside the modeling domain.
(2) These homes were successfully geocoded, but fell outside the modeling domain for the $PM_{2.5}$ exposure surface which covered a slightly different area than the LUR and $O_3$ modeling domains.

**Table 8. Description of Available School Air Pollution Estimates by Exposure Period for n=1,387 Children included in L.A. FANS-2**

| Air Pollution Averaging period | No. (%) of subjects with NO, $NO_2$, $NO_x$ and $O_3$ values[1] | No. (%) of subjects with NO, $NO_2$, $NO_x$, $O_3$ and $PM_{2.5}$ values[1] | No. of subjects with LUR or $O_3$ values missing for one or more schools[1] | No. of additional subjects missing $PM_{2.5}$ values for one or more schools[2] |
|---|---|---|---|---|
| Current school | 1253 (99%) | 1237 (98%) | 13 | 16 |
| 12 months prior to interview | 1220 (96%) | 1206 (95%) | 46 | 14 |
| 24 months prior to interview | 1180 (93%) | 1170 (92%) | 86 | 10 |
| 5 years prior to interview | 912 (72%) | 903 (71%) | 354 | 9 |

(1) Percentages are based on a total of 1,266 children reported as having started school. Subjects are missing data because: (a) one or more schools not geocoded and/or (b) school dates (i.e., dates in each school) do not span this period or are missing data and/or (c) one or more schools fell outside the modeling domain.
(2) These schools were successfully geocoded, but fell outside the modeling domain for the $PM_{2.5}$ exposure surface which covered a slightly different area than the LUR and $O_3$ modeling domains.

**Table 9. Pollutant Disributions and Pearson Correlation Coefficients for LUR, $O_3$ and $PM_{2.5}$ Annual Averages (12 months prior to interview) for n=1,387 Children with Questionnaire Data**

| Pollutant | Median (range) | IQR | Pearson Correlation Coefficients | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | | | NO | $NO_2$ | $NO_x$ | NO-LT[2] | $NO_2$-LT | $NO_x$-LT | $O_3$ |
| NO (ppb)[1] | 23.5 (2.5-69.0) | 10.7 | 1.0 | | | | | | |
| $NO_2$ (ppb)[1] | 23.7 (6.2-36.9) | 6.1 | 0.86 | 1.0 | | | | | |
| $NO_x$ (ppb)[1] | 47.9 (11.3-97.5) | 16.9 | 0.97 | 0.93 | 1.0 | | | | |
| NO-LT (ppb)[2] | 28.7 (2.8-73.5) | 11.8 | 0.86 | 0.82 | 0.88 | 1.0 | | | |
| $NO_2$-LT (ppb) | 25.6 (6.0-43.0) | 6.1 | 0.75 | 0.95 | 0.83 | 0.85 | 1.0 | | |
| $NO_x$-LT (ppb) | 55.3 (6.3-126.2) | 16.9 | 0.83 | 0.87 | 0.89 | 0.98 | 0.89 | 1.0 | |
| $O_3$ (ppb)[3] | 71.1 (46.2-129.8) | 29.1 | -0.35 | -0.18 | -0.36 | -0.27 | -0.08 | -0.27 | 1.0 |
| $PM_{2.5}$ ($\mu m/m^3$) | 21.5 (8.5-23.7) | 2.4 | 0.51 | 0.68 | 0.59 | 0.37 | 0.56 | 0.44 | -0.36 |

(1) Estimates are from the final, optimized LUR model.
(2) "LT" stands for "more local traffic impact"; these estimates are from the LUR model excluding traffic within 11 km buffers.
(3) Kriged $O_3$ estimates based on 8-hour maximum concentrations.

**Table 10. Pollutant averages (ranges) and Pearson Correlation Coefficients for LUR, $O_3$ and $PM_{2.5}$ Annual Averages (12 months prior to interview) - For n=890 Children with 1 or More Acceptable Spirometry Curves**

| Pollutant | Median (range) | IQR | Pearson Correlation Coefficients | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | | | NO | $NO_2$ | $NO_x$ | NO-LT[2] | $NO_2$-LT | $NO_x$-LT | $O_3$ |
| NO (ppb)[1] | 23.5 (2.9-60.4) | 10.5 | 1.0 | | | | | | |
| $NO_2$ (ppb)[1] | 23.7 (6.2-36.9) | 5.7 | 0.86 | 1.0 | | | | | |
| $NO_x$ (ppb)[1] | 48.0 (11.9-94.8) | 16.5 | 0.97 | 0.93 | 1.0 | | | | |
| NO-LT (ppb)[2] | 28.5 (2.8-73.5) | 11.4 | 0.85 | 0.82 | 0.88 | 1.0 | | | |
| $NO_2$-LT (ppb) | 25.5 (6.0-43.0) | 5.7 | 0.73 | 0.94 | 0.82 | 0.84 | 1.0 | | |
| $NO_x$-LT (ppb) | 55.4 (6.3-126.2) | 16.2 | 0.82 | 0.87 | 0.87 | 0.98 | 0.89 | 1.0 | |
| $O_3$ (ppb)[3] | 70.1 (46.2-129.8) | 29.4 | -0.40 | -0.23 | -0.41 | -0.32 | -0.12 | -0.31 | 1.0 |
| $PM_{2.5}$ ($\mu m/m^3$) | 21.5 (8.5-23.6) | 2.3 | 0.51 | 0.69 | 0.60 | 0.38 | 0.57 | 0.46 | -0.40 |

(1) Estimates are from the final, optimized LUR model.
(2) "LT" stands for "more local traffic impact"; these estimates are from the LUR model excluding traffic within 11 km buffers.
(3) Kriged $O_3$ estimates based on 8-hour maximum concentrations.

**Table 11. Pollutant averages (ranges) and Pearson Correlation Coefficients for LUR, $O_3$ and $PM_{2.5}$ Annual Averages (12 months prior to interview) - For n=395 Children with 3 Acceptable and Reproducible Spirometry Curves**

| Pollutant | Median (range) | IQR | Pearson Correlation Coefficients | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | | | NO | $NO_2$ | $NO_x$ | NO-LT[2] | $NO_2$-LT | $NO_x$-LT | $O_3$ |
| NO (ppb)[1] | 23.1 (3.4-56.1) | 10.6 | 1.0 | | | | | | |
| $NO_2$ (ppb)[1] | 23.6 (6.3-36.9) | 5.0 | 0.84 | 1.0 | | | | | |
| $NO_x$ (ppb)[1] | 47.4 (11.9-94.8) | 15.7 | 0.97 | 0.92 | 1.0 | | | | |
| NO-LT (ppb)[2] | 27.7 (2.8-73.5) | 10.8 | 0.84 | 0.80 | 0.87 | 1.0 | | | |
| $NO_2$-LT (ppb) | 25.5 (6.1-43.0) | 5.0 | 0.70 | 0.93 | 0.80 | 0.83 | 1.0 | | |
| $NO_x$-LT (ppb) | 54.2 (6.4-126.2) | 15.9 | 0.80 | 0.85 | 0.87 | 0.98 | 0.88 | 1.0 | |
| $O_3$ (ppb)[3] | 71.1 (46.2-129.8) | 36.5 | -0.43 | -0.21 | -0.42 | -0.31 | -0.06 | -0.28 | 1.0 |
| $PM_{2.5}$ ($\mu m/m^3$) | 21.5 (8.5-23.6) | 2.4 | 0.47 | 0.65 | 0.57 | 0.30 | 0.48 | 0.37 | -0.35 |

(1) Estimates are from the final, optimized LUR model.
(2) "LT" stands for "more local traffic impact"; these estimates are from the LUR model excluding traffic within 11 km buffers.
(3) Kriged $O_3$ estimates based on 8-hour maximum concentrations.

**Table 12. Demographic Characteristics (Number, Percent) of L.A. FANS-2 Child Participants**

| Parameter | All Subjects with Questionnaire Data (n=1,387) | Subjects with One or More Acceptable Spirometry Curve (n=890) | Subjects with Three Acceptable and Reproducible Spirometry Curves (n=395) |
|---|---|---|---|
| *Individual Level* | | | |
| Gender | | | |
| Female | 716 (51.6) | 404 (45.4) | 174 (44.1) |
| Male | 671 (48.4) | 486 (54.6) | 221 (55.9) |
| Age (years) | | | |
| <5 | 100 (7.2) | -- | -- |
| 5-<10 | 379 (27.3) | 222 (25.0) | 78 (19.8) |
| 10-<15 | 551 (39.7) | 415 (46.6) | 190 (48.1) |
| ≥15 | 357 (25.7) | 253 (28.4) | 127 (32.1) |
| Race/ethnicity | | | |
| Non-Hispanic White | 262 (18.9) | 165 (18.6) | 73 (18.5) |
| Hispanic | 921 (66.4) | 594 (66.7) | 246 (62.3) |
| African American | 105 (7.6) | 65 (7.3) | 36 (9.1) |
| Asian/Other | 99 (7.1) | 66 (7.4) | 40 (10.1) |
| Health insurance during past month | | | |
| Yes | 1187 (85.8) | 758 (85.3) | 335 (84.8) |
| No | 196 (14.2) | 131 (14.7) | 60 (15.2) |
| Missing | 4 | 1 | |
| Usual source of sick care | | | |
| Yes | 1298 (93.7) | 833 (93.7) | 370 (93.9) |
| No | 88 (6.4) | 56 (6.3) | 24 (6.1) |
| Missing | 1 | 1 | 1 |
| Overweight[1] | | | |
| Yes | 531 (44.7) | 411 (46.6) | 179 (45.8) |
| No | 658 (55.3) | 472 (53.4) | 212 (54.2) |
| Missing | 198 | 7 | 4 |
| Doctor-diagnosed asthma (ever) | | | |
| Yes | 191 (13.8) | 131 (14.7) | 67 (17.0) |
| No | 1196 (86.2) | 759 (85.3) | 328 (83.0) |
| Age of asthma diagnosis (years) – only for diagnosed asthmatics | | | |
| ≤5 | 125 (67.6) | 84 (66.1) | 38 (58.5) |
| 6-<10 | 33 (17.8) | 25 (19.7) | 17 (26.2) |
| 10-<15 | 22 (11.9) | 15 (11.8) | 9 (13.8) |
| ≥15 | 5 (2.7) | 3 (2.4) | 1 (1.5) |
| Missing | 6 | 4 | 2 |
| Wheeze in past 12 months | | | |
| Yes | 145 (10.5) | 101 (11.4) | 54 (13.7) |
| No | 1242 (89.6) | 789 (88.6) | 341 (86.3) |
| Wheeze with any night waking in past 12 months | | | |
| Yes | 73 (5.3) | 52 (5.8) | 29 (7.3) |
| No | 1314 (94.7) | 838 (94.2) | 366 (92.7) |
| Medication use for asthma or wheeze in past 12 months | | | |
| Yes | 162 (11.7) | 106 (11.9) | 57 (14.4) |
| No | 1225 (88.3) | 784 (88.1) | 338 (85.6) |
| Sneezing or runny/block nose apart from cold in past 12 months | | | |
| Yes | 270 (19.5) | 179 (20.1) | 86 (21.8) |
| No | 1116 (80.5) | 710 (79.9) | 308 (78.2) |
| Missing | 1 | 1 | 1 |
| More than 3 doctor-diagnosed ear infections in 1 year | | | |
| Yes | 140 (10.1) | 94 (10.6) | 50 (12.7) |
| No | 1246 (89.9) | 795 (89.4) | 344 (87.3) |
| Missing | 1 | | 1 |

| Parameter | All Subjects with Questionnaire Data (n=1,387) | Subjects with One or More Acceptable Spirometry Curve (n=890) | Subjects with Three Acceptable and Reproducible Spirometry Curves (n=395) |
|---|---|---|---|
| ***Family Level*** | | | |
| Family Income (dollars) | | | |
| <20,000 | 310 (24.5) | 208 (24.9) | 91 (25.0) |
| 20,000-<35,000 | 321 (25.4) | 200 (24.0) | 70 (19.2) |
| 35,000-<65,000 | 318 (25.2) | 209 (25.0) | 95 (26.1) |
| ≥65,000 | 315 (24.9) | 218 (26.1) | 108 (29.7) |
| Missing | 123 | 55 | 31 |
| Homeowner | | | |
| Yes | 548 (41.6) | 359 (41.9) | 178 (46.8) |
| No | 768 (58.4) | 497 (58.1) | 202 (53.2) |
| Missing | 71 | 34 | 15 |
| PCG's education (years) | | | |
| <12 | 563 (40.8) | 362 (40.9) | 148 (37.6) |
| 12 | 242 (17.5) | 154 (17.4) | 67 (17.0) |
| >12 | 576 (41.7) | 369 (41.7) | 179 (45.4) |
| Missing | 6 | 5 | 1 |
| Foreign born status of PCG | | | |
| US born | 529 (38.1) | 336 (37.8) | 166 (42.0) |
| Foreign born | 858 (61.9) | 554 (62.3) | 229 (58.0) |
| PCG current marital status | | | |
| Married or living with partner | 1071 (77.2) | 689 (77.4) | 294 (74.4) |
| Unmarried/not living with partner | 316 (22.8) | 201 (22.6) | 101 (25.6) |
| Current household smokers | | | |
| Yes | 297 (21.4) | 190 (21.4) | 84 (21.3) |
| No | 1088 (78.6) | 698 (78.6) | 311 (78.7) |
| Missing | 2 | 2 | |
| PCG current smoking status | | | |
| Yes | 126 (9.1) | 85 (9.6) | 52 (13.2) |
| No | 1260 (90.9) | 804 (90.4) | 342 (86.8) |
| Missing | 1 | 1 | 1 |
| Current smoker in home or PCG smoker | | | |
| Yes | 325 (23.5) | 207 (23.3) | 97 (24.6) |
| No | 1059 (76.5) | 680 (76.7) | 297 (75.4) |
| Missing | 3 | 3 | 1 |
| Either parent has asthma | | | |
| Yes | 144 (10.7) | 98 (11.3) | 49 (12.7) |
| No | 1204 (89.3) | 770 (88.7) | 336 (87.3) |
| Missing | 39 | 22 | 10 |
| Mother has asthma | | | |
| Yes | 96 (7.0) | 62 (7.0) | 34 (8.7) |
| No | 1277 (93.0) | 823 (93.0) | 357 (91.3) |
| Missing | 14 | 5 | 4 |
| Gas appliance in home (stove, range or oven) | | | |
| Yes | 1287 (92.8) | 821 (92.2) | 370 (93.7) |
| No | 100 (7.2) | 69 (7.8) | 25 (6.3) |
| Gas appliance with pilot light (stove, range or oven) | | | |
| Yes | 579 (42.5) | 384 (43.7) | 172 (44.3) |
| No | 782 (57.5) | 494 (56.3) | 216 (55.7) |
| Missing | 26 | 12 | 7 |
| Pests in home in past 12 months | | | |
| Yes | 1047 (75.5) | 677 (76.1) | 301 (76.2) |
| No | 340 (24.5) | 213 (23.9) | 94 (23.8) |
| Cockroaches in home in past 12 months | | | |
| Yes | 404 (29.1) | 253 (28.4) | 100 (25.3) |
| No | 983 (70.9) | 637 (71.6) | 295 (74.7) |
| Mold in home in past 12 months | | | |
| Yes | 296 (21.4) | 185 (20.9) | 79 (20.1) |
| No | 1087 (78.6) | 702 (79.1) | 314 (79.9) |
| Missing | 4 | 3 | 2 |

| Parameter | All Subjects with Questionnaire Data (n=1,387) | Subjects with One or More Acceptable Spirometry Curve (n=890) | Subjects with Three Acceptable and Reproducible Spirometry Curves (n=395) |
|---|---|---|---|
| Furry pets in home | | | |
| Yes | 467 (34.7) | 311 (36.0) | 144 (37.4) |
| No | 879 (65.3) | 554 (64.0) | 241 (62.6) |
| Missing | 41 | 25 | 10 |
| Neighborhood satisfaction | | | |
| Very satisfied | 283 (24.3) | 179 (23.5) | 90 (26.6) |
| Satisfied or Neutral (if volunteered) | 705 (60.5) | 475 (62.2) | 201 (59.5) |
| Dissatisfied or Very dissatisfied | 178 (15.3) | 109 (14.3) | 47 (13.9) |
| Missing | 221 | 127 | 57 |
| How safe to walk alone after dark in this neighborhood | | | |
| Completely safe | 181 (15.6) | 121 (15.9) | 54 (16.0) |
| Fairly safe or Somewhat dangerous | 921 (79.5) | 600 (79.1) | 267 (79.0) |
| Extremely dangerous | 57 (4.9) | 38 (5.0) | 17 (5.0) |
| Missing | 228 | 131 | 57 |
| No. adults you recognize in neighborhood | | | |
| Many adults or most or all adults | 595 (51.0) | 396 (51.9) | 179 (53.0) |
| A few adults or no adults | 571 (49.0) | 367 (48.1) | 159 (47.0) |
| Missing | 221 | 127 | 57 |
| Neighborhood cohesion score[2] | | | |
| <2.52 (median) (higher) | 631 (54.7) | 421 (55.6) | 196 (58.2) |
| ≥2.52 | 523 (45.3) | 336 (44.4) | 141 (41.8) |
| Missing | 233 | 133 | 58 |
| No. relatives living in neighborhood | | | |
| Any | 468 (40.3) | 295 (38.8) | 125 (37.1) |
| None | 694 (59.7) | 465 (61.1) | 212 (62.9) |
| Missing | 233 | 130 | 58 |
| No. friends living in neighborhood | | | |
| Any | 804 (67.0) | 532 (69.7) | 232 (68.6) |
| None | 362 (31.1) | 231 (30.3) | 106 (31.4) |
| Missing | 221 | 127 | 57 |
| No. of neighbors talked to for 10 min in past 30 days | | | |
| Any | 1021 (87.6) | 671 (88.1) | 294 (87.2) |
| None | 144 (12.4) | 91 (11.9) | 43 (12.8) |
| Missing | 222 | 128 | 58 |
| No. groups participated in past 12 months | | | |
| Any | 420 (36.1) | 279 (36.6) | 123 (36.4) |
| None | 745 (63.9) | 483 (63.4) | 215 (63.6) |
| Missing | 222 | 128 | 57 |
| Neighborhood support score[3] | | | |
| 1-<2 (higher) | 326 (28.3) | 222 (29.4) | 109 (32.6) |
| 2-<4 | 737 (63.9) | 475 (63.0) | 197 (59.0) |
| ≥4 | 90 (7.8) | 57 (7.6) | 28 (8.4) |
| Missing | 234 | 136 | 61 |
| *Neighborhood Level* | | | |
| Census tract level rating of neighborhood cohesion[4] | | | |
| <2.53 (median) (higher) | 675 (51.1) | 446 (51.8) | 202 (52.7) |
| ≥2.53 | 646 (48.9) | 415 (48.2) | 181 (47.3) |
| Missing | 66 | 29 | 12 |
| Census tract level rating of neighborhood safety[5] | | | |
| <2.13 (median) (higher) | 658 (49.8) | 433 (50.2) | 194 (50.7) |
| ≥2.13 | 663 (50.2) | 429 (49.8) | 189 (49.4) |
| Missing | 66 | 28 | 12 |
| Tract-level disadvantage[6] | | | |
| <0.13 (median) (lower) | 685 (49.7) | 433 (48.8) | 213 (54.1) |
| ≥0.13 | 694 (50.3) | 454 (51.2) | 181 (45.9) |
| Missing | 8 | 3 | 1 |

| Parameter | All Subjects with Questionnaire Data (n=1,387) | Subjects with One or More Acceptable Spirometry Curve (n=890) | Subjects with Three Acceptable and Reproducible Spirometry Curves (n=395) |
|---|---|---|---|
| Percent of tract in same home 5 years ago[7] | | | |
| <0.52 (median) | 684 (49.6) | 451 (50.8) | 189 (48.0) |
| ≥0.52 | 695 (50.4) | 436 (49.2) | 205 (52.0) |
| Missing | 8 | 3 | 1 |
| Census tract predominately Latino or White[7] | | | |
| Yes | 1072 (77.7) | 688 (77.6) | 294 (74.6) |
| No | 307 (22.3) | 199 (22.4) | 100 (25.4) |
| Missing | 8 | 3 | 1 |

(1) Overweight is based on 2000 U.S. Centers for Disease Control BMI-for-age charts; separate charts are used for boys and girls. Children with BMI for age values at 85-<95$^{th}$ percentile are considered at risk for overweight; children with BMI for age values ≥95$^{th}$ percentile are considered overweight. Here the at risk of overweight and overweight groups were combined. Only children age 2 years and older that could stand on their own were measured for height and weight in the L.A. FANS-2 study.
(2) Average of responses for the following questions (with reverse coding where necessary): (a) This is a close-knit neighborhood; (b) There are adults kids can look up to; (c) People are willing to help their neighbors; (d) Neighbors generally don't get along; (e) Adults watch out that kids are safe; (f) People in neighborhood don't share same values; (g) People in neighborhood can be trusted; (h) Parents in neighborhood know kids friends; (i) Adults in neighborhood know local kids' (j) Parents in neighborhood know each other; (k) Neighbors do something if kid hangs out; (l) Would do something if kid does graffiti; (m) Would scold kid if showing disrespect. Responses for a-j were: 1=strongly agree, 2=agree, 3=unsure, 4=disagree, 5=strongly disagree; Responses for k-m were: 1=very likely, 2=likely, 3=unsure, 4=unlikely, 5=very unlikely.
(3) Average of responses (1=often, 2=sometimes, 3=rarely, 4=never) for the following questions: (a) How often do neighbors do favors for each other; (b) How often do neighbors watch each others property; (c) How often do neighbors ask advice.
(4) This is the average of the neighborhood cohesion score for adult respondents in a given census tract.
(5) This is the average of the neighborhood safety responses for adult respondents in a given census tract using the following numeric responses for each response: 1=completely safe, 2=fairly safe, 3=somewhat dangerous, 4=extremely dangerous.
(6) This is the average of the following four variables for each census tract (based on U.S. Census 2000 data): percent poor families, percent households on public assistance, percent female headed families with children under the age of 18 years, percent male unemployment.
(7) Based on U.S. Census 2000 data.

## Table 13. Distribution (Number, Percent) of Wheeze and Medication Use Outcomes Among L.A. FANS-2 Child Participants With and Without a Doctor-Diagnosis of Asthma (n=1,387)

| | Doctor-diagnosed Asthma (n=191) | No Doctor-diagnosed Asthma (n=1196) |
|---|---|---|
| Wheeze in past 12 mos, medication use | 89 (46.6%) | 23 (1.9%) |
| Wheeze in past 12 mos, no medication use | 7 (3.7%) | 26 (2.2%) |
| No wheeze in past 12 mos, medication use | 24 (12.6%) | 26 (2.2%) |
| No wheeze in past 12 mos, no medication use | 71 (37.1%) | 1121 (93.7%) |

**Table 14. Number and (Percent) of Subjects in Each Outcome Group by Individual-, Family-, and Neighborhood-Level Demographic Characteristics and Crude Odds Ratios (95% Confidence Intervals) for Doctor-Diagnosed Asthma (Ever)**

| Parameter | Doctor-Diagnosed Asthma (Ever) (n=191) | No Doctor-Diagnosed Asthma (n=1196) | Crude OR (95% CI) |
|---|---|---|---|
| ***Individual Level*** | | | |
| Gender | | | |
| Female | 75 (39.3) | 596 (49.8) | 1.00 |
| Male | 116 (60.7) | 600 (50.2) | 1.54 (1.13, 2.10) |
| Age (years) | | | |
| ≤5 | 7 (3.7) | 93 (7.8) | 0.53 (0.23, 1.22) |
| 6-<10 | 47 (24.6) | 332 (27.8) | 1.00 |
| 10-<15 | 84 (44.0) | 467 (39.1) | 1.27 (0.87, 1.87) |
| ≥15 | 53 (27.8) | 304 (25.4) | 1.23 (0.81, 1.88) |
| Race/ethnicity | | | |
| Non-Hispanic White | 40 (20.9) | 222 (18.6) | 1.00 |
| Hispanic | 107 (56.0) | 814 (68.1) | 0.86 (0.58, 1.26) |
| African American | 26 (13.6) | 79 (6.6) | 2.10 (1.24, 3.56) |
| Asian/Other | 18 (9.4) | 81 (6.8) | 1.37 (0.76, 2.48) |
| Health insurance during past month | | | |
| Yes | 177 (92.7) | 1010 (84.7) | 1.00 |
| No | 14 (7.3) | 182 (15.7) | 0.44 (0.25, 0.77) |
| Missing | | 4 | |
| Usual source of sick care | | | |
| Yes | 187 (97.9) | 1111 (93.0) | 1.00 |
| No | 4 (2.1) | 84 (7.0) | 0.28 (0.10, 0.78) |
| Missing | | 1 | |
| Overweight[1] | | | |
| Yes | 83 (51.6) | 449 (43.6) | 1.38 (0.99, 1.92) |
| No | 78 (48.4) | 580 (56.4) | 1.00 |
| Missing | 30 | 167 | |
| ***Family Level*** | | | |
| Family Income (dollars) | | | |
| <20,000 | 39 (22.4) | 271 (24.9) | 0.73 (0.47, 1.14) |
| 20,000-<35,000 | 43 (24.7) | 278 (25.5) | 0.78 (0.51, 1.21) |
| 35,000-<65,000 | 40 (23.0) | 278 (25.5) | 0.73 (0.47, 1.14) |
| ≥65,000 | 52 (29.9) | 263 (24.1) | 1.00 |
| Missing | 17 | 106 | |
| Homeowner | | | |
| Yes | 78 (43.8) | 470 (41.3) | 1.00 |
| No | 100 (56.2) | 668 (58.7) | 0.90 (0.66, 1.24) |
| Missing | 13 | 58 | |
| PCG's education (years) | | | |
| <12 | 63 (33.2) | 500 (42.0) | 0.62 (0.44, 0.86) |
| 12 | 29 (15.3) | 213 (17.9) | 0.66 (0.43, 1.04) |
| >12 | 98 (51.6) | 478 (40.1) | 1.00 |
| Missing | 1 | 5 | |
| Foreign born status of PCG | | | |
| US born | 104 (54.5) | 425 (35.5) | 1.00 |
| Foreign born | 87 (45.6) | 771 (64.5) | 0.46 (0.34, 0.63) |
| PCG current marital status | | | |
| Married or living with partner | 132 (69.1) | 939 (78.5) | 1.00 |
| Unmarried/not living with partner | 59 (30.9) | 257 (21.5) | 1.63 (1.17, 2.29) |
| Current household smokers | | | |
| Yes | 49 (25.7) | 248 (20.8) | 1.32 (0.92, 1.88) |
| No | 142 (74.3) | 946 (79.2) | 1.00 |
| Missing | | 2 | |
| PCG current smoking status | | | |
| Yes | 26 (13.6) | 100 (8.4) | 1.73 (1.09, 2.74) |
| No | 165 (86.4) | 1095 (91.6) | 1.00 |
| Missing | | 1 | |

| Parameter | Doctor-Diagnosed Asthma (Ever) (n=191) | No Doctor-Diagnosed Asthma (n=1196) | Crude OR (95% CI) |
|---|---|---|---|
| Current smoker in home or PCG smoker | | | |
| Yes | 56 (29.3) | 269 (22.6) | 1.43 (1.02, 2.00) |
| No | 135 (70.7) | 924 (77.4) | 1.00 |
| Missing | | 3 | |
| Either parent has asthma | | | |
| Yes | 55 (29.7) | 89 (7.7) | 5.11 (3.48, 7.48) |
| No | 130 (70.3) | 1074 (92.3) | 1.00 |
| Missing | 6 | 33 | |
| Mother has asthma | | | |
| Yes | 39 (20.7) | 57 (4.8) | 5.18 (3.33, 8.06) |
| No | 149 (79.3) | 1128 (95.2) | 1.00 |
| Missing | 3 | 11 | |
| Gas appliance in home (stove, range or oven) | | | |
| Yes | 171 (89.5) | 1116 (93.3) | 0.61 (0.37, 1.03) |
| No | 20 (10.5) | 80 (6.7) | 1.00 |
| Gas appliance with pilot light (stove, range or oven) | | | |
| Yes | 67 (36.4) | 512 (43.5) | 0.74 (0.54, 1.03) |
| No | 117 (63.6) | 665 (56.5) | 1.00 |
| Missing | 7 | 19 | |
| Pests in home in past 12 months | | | |
| Yes | 156 (81.7) | 891 (74.5) | 1.53 (1.03, 2.25) |
| No | 35 (18.3) | 305 (25.5) | 1.00 |
| Cockroaches in home in past 12 months | | | |
| Yes | 55 (28.8) | 349 (29.2) | 0.98 (0.70, 1.38) |
| No | 136 (71.2) | 847 (70.8) | 1.00 |
| Mold in home in past 12 months | | | |
| Yes | 41 (21.6) | 255 (21.4) | 1.01 (0.70, 1.47) |
| No | 149 (78.4) | 938 (78.6) | 1.00 |
| | 1 | 3 | |
| Furry pets in home | | | |
| Yes | 71 (38.2) | 396 (34.1) | 1.19 (0.87, 1.64) |
| No | 115 (61.8) | 764 (65.9) | 1.00 |
| Missing | 5 | 36 | |
| Neighborhood satisfaction | | | |
| Very satisfied | 37 (23.3) | 246 (24.4) | 1.00 |
| Satisfied or Neutral (if volunteered) | 96 (60.4) | 609 (60.5) | 1.05 (0.70, 1.58) |
| Dissatisfied or Very dissatisfied | 26 (16.4) | 152 (15.1) | 1.14 (0.66, 1.95) |
| Missing | 32 | 189 | |
| How safe to walk alone after dark in this neighborhood | | | |
| Completely safe | 14 (8.9) | 167 (16.7) | 1.00 |
| Fairly safe or Somewhat dangerous | 135 (85.4) | 786 (78.5) | 2.05 (1.15, 3.64) |
| Extremely dangerous | 9 (5.7) | 48 (4.8) | 2.24 (0.91, 5.48) |
| Missing | 33 | 195 | |
| No. adults you recognize in neighborhood | | | |
| Many adults or most or all adults | 83 (52.2) | 512 (50.8) | 1.00 |
| A few adults or no adults | 76 (47.8) | 495 (49.2) | 0.95 (0.68, 1.32) |
| Missing | 32 | 189 | |
| Neighborhood cohesion score[2] | | | |
| <2.52 (median) (higher) | 82 (51.6) | 549 (55.2) | 1.00 |
| ≥2.52 | 77 (48.4) | 446 (44.8) | 1.16 (0.83, 1.62) |
| Missing | 32 | 201 | |
| No. relatives living in neighborhood | | | |
| Any | 54 (34.0) | 414 (41.3) | 1.00 |
| None | 105 (66.0) | 589 (58.7) | 1.37 (0.96, 1.94) |
| Missing | 32 | 193 | |
| No. friends living in neighborhood | | | |
| Any | 105 (66.0) | 699 (69.4) | 1.00 |
| None | 54 (34.0) | 308 (30.6) | 1.17 (0.82, 1.66) |
| Missing | 32 | 189 | |

| Parameter | Doctor-Diagnosed Asthma (Ever) (n=191) | No Doctor-Diagnosed Asthma (n=1196) | Crude OR (95% CI) |
|---|---|---|---|
| No. of neighbors talked to for 10 min in past 30 days | | | |
| Any | 140 (88.1) | 881 (87.6) | 1.00 |
| None | 19 (11.9) | 125 (12.4) | 0.96 (0.57, 1.60) |
| Missing | 32 | 190 | |
| No. groups participated in past 12 months | | | |
| Any | 60 (37.7) | 360 (35.8) | 1.00 |
| None | 99 (62.3) | 646 (64.2) | 0.92 (0.65, 1.30) |
| Missing | 32 | 190 | |
| Neighborhood support score[3] | | | |
| 1-<2 (higher) | 42 (27.4) | 284 (28.4) | 1.00 |
| 2-<4 | 102 (66.7) | 635 (63.5) | 1.09 (0.74, 1.60) |
| ≥4 | 9 (5.9) | 81 (8.1) | 0.75 (0.35, 1.61) |
| Missing | 38 | 196 | |
| ***Neighborhood Level*** | | | |
| Census tract level rating of neighborhood cohesion[4] | | | |
| <2.53 (median) (higher) | 98 (53.9) | 577 (50.7) | 1.00 |
| ≥2.53 | 84 (46.1) | 562 (49.3) | 0.88 (0.64, 1.20) |
| Missing | 9 | 57 | |
| Census tract level rating of neighborhood safety[5] | | | |
| <2.13 (median) (higher) | 97 (53.6) | 561 (49.2) | 1.00 |
| ≥2.13 | 84 (46.4) | 579 (50.8) | 0.84 (0.61, 1.15) |
| Missing | 10 | 56 | |
| Tract-level disadvantage[6] | | | |
| <0.13 (median) (lower) | 95 (50.0) | 590 (49.6) | 1.00 |
| ≥0.13 | 95 (50.0) | 599 (50.4) | 0.99 (0.73, 1.34) |
| Missing | 1 | 7 | |
| Percent of tract in same home 5 years ago[7] | | | |
| <0.52 (median) | 97 (51.0) | 598 (50.3) | 0.97 (0.71, 1.32) |
| ≥0.52 | 93 (49.0) | 591 (49.7) | 1.00 |
| Missing | 1 | 7 | |
| Census tract predominately Latino or White[7] | | | |
| Yes | 141 (74.2) | 931 (78.3) | 0.80 (0.56, 1.14) |
| No | 49 (25.8) | 228 (21.7) | 1.00 |
| Missing | 1 | 7 | |

(1) Based on 2000 U.S. Centers for Disease Control BMI-for-age charts; separate charts are used for boys and girls. Children with BMI for age values at 85-<95$^{th}$ percentile are considered at risk for overweight; children with BMI for age values ≥95$^{th}$ percentile are considered overweight. Only children age 2 years and older that could stand on their own were measured for height and weight in the L.A. FANS-2 study.

(2) Average of responses for the following questions (with reverse coding where necessary): (a) This is a close-knit neighborhood; (b) There are adults kids can look up to; (c) People are willing to help their neighbors; (d) Neighbors generally don't get along; (e) Adults watch out that kids are safe; (f) People in neighborhood don't share same values; (g) People in neighborhood can be trusted; (h) Parents in neighborhood know kids friends; (i) Adults in neighborhood know local kids' (j) Parents in neighborhood know each other; (k) Neighbors do something if kid hangs out; (l) Would do something if kid does graffiti; (m) Would scold kid if showing disrespect. Responses for a-j were: 1=strongly agree, 2=agree, 3=unsure, 4=disagree, 5=strongly disagree; Responses for k-m were: 1=very likely, 2=likely, 3=unsure, 4=unlikely, 5=very unlikely.

(3) Average of responses (1=often, 2=sometimes, 3=rarely, 4=never) for the following questions: (a) How often do neighbors do favors for each other; (b) How often do neighbors watch each others property; (c) How often do neighbors ask advice.

(4) This is the average of the neighborhood cohesion score for adult respondents in a given census tract.

(5) This is the average of the neighborhood safety responses for adult respondents in a given census tract using the following numeric responses for each response: 1=completely safe, 2=fairly safe, 3=somewhat dangerous, 4=extremely dangerous.

(6) This is the average of the following four variables for each census tract (based on U.S. Census 2000 data): percent poor families, percent households on public assistance, percent female headed families with children under the age of 18 years, percent male unemployment.

(7) Based on U.S. Census 2000 data.

**Table 15. Number and (Percent) of Subjects in Each Outcome Group by Individual-, Family-, and Neighborhood-Level Demographic Characteristics and Crude Odds Ratios (95% Confidence Intervals) for Wheeze in Past 12 Months**

| Parameter | Wheeze in past 12 months (n=145) | No Wheeze in past 12 months (n=1242) | Crude OR (95% CI) |
|---|---|---|---|
| ***Individual Level*** | | | |
| Gender | | | |
| Female | 51 (35.2) | 620 (49.9) | 1.00 |
| Male | 94 (64.8) | 622 (50.1) | 1.84 (1.28, 2.63) |
| Age (years) | | | |
| ≤5 | 9 (6.2) | 91 (7.3) | 0.84 (0.39, 1.79) |
| 6-<10 | 40 (27.6) | 339 (27.3) | 1.00 |
| 10-<15 | 60 (41.4) | 491 (39.5) | 1.04 (0.68, 1.58) |
| ≥15 | 36 (24.8) | 321 (25.9) | 0.95 (0.59, 1.53) |
| Race/ethnicity | | | |
| Non-Hispanic White | 32 (22.0) | 230 (18.5) | 1.00 |
| Hispanic | 80 (55.2) | 841 (67.7) | 0.80 (0.52, 1.24) |
| African American | 20 (13.8) | 85 (6.8) | 1.86 (1.03, 3.35) |
| Asian/Other | 13 (9.0) | 86 (6.9) | 1.21 (0.62, 2.38) |
| Health insurance during past month | | | |
| Yes | 128 (88.3) | 1059 (85.5) | 1.00 |
| No | 17 (11.7) | 179 (14.5) | 0.79 (0.46, 1.34) |
| Missing | | 4 | |
| Usual source of sick care | | | |
| Yes | 137 (94.5) | 1161 (93.6) | 1.00 |
| No | 8 (5.5) | 80 (6.4) | 0.85 (0.40, 1.79) |
| Missing | | 1 | |
| Overweight[1] | | | |
| Yes | 67 (55.8) | 465 (43.5) | 1.65 (1.13, 2.41) |
| No | 53 (44.2) | 605 (56.5) | 1.00 |
| Missing | 25 | 172 | |
| ***Family Level*** | | | |
| Family Income (dollars) | | | |
| <20,000 | 28 (21.5) | 282 (24.9) | 0.66 (0.40, 1.10) |
| 20,000-<35,000 | 30 (23.1) | 291 (25.7) | 0.69 (0.42, 1.13) |
| 35,000-<65,000 | 31 (23.9) | 287 (25.3) | 0.72 (0.44, 1.18) |
| ≥65,000 | 41 (31.5) | 274 (24.1) | 1.00 |
| Missing | 15 | 108 | |
| Homeowner | | | |
| Yes | 64 (46.0) | 484 (41.1) | 1.00 |
| No | 75 (54.0) | 693 (58.9) | 0.82 (0.58, 1.17) |
| Missing | 6 | 65 | |
| PCG's education (years) | | | |
| <12 | 42 (29.2) | 521 (42.1) | 0.52 (0.35, 0.78) |
| 12 | 25 (17.3) | 217 (17.6) | 0.75 (0.46, 1.21) |
| >12 | 77 (53.5) | 499 (40.3) | 1.00 |
| Missing | 1 | 5 | |
| Foreign born status of PCG | | | |
| US born | 80 (55.2) | 449 (36.2) | 1.00 |
| Foreign born | 65 (44.8) | 793 (63.8) | 0.46 (0.33, 0.65) |
| PCG current marital status | | | |
| Married or living with partner | 97 (66.9) | 974 (78.4) | 1.00 |
| Unmarried/not living with partner | 48 (33.1) | 268 (21.6) | 1.80 (1.24, 2.61) |
| Current household smokers | | | |
| Yes | 33 (22.8) | 264 (21.3) | 1.09 (0.72, 1.64) |
| No | 112 (77.2) | 976 (78.7) | 1.00 |
| Missing | | 2 | |
| PCG current smoking status | | | |
| Yes | 20 (13.8) | 106 (8.5) | 1.71 (1.03, 2.86) |
| No | 125 (86.2) | 1135 (91.5) | 1.00 |
| Missing | | 1 | |

| Parameter | Wheeze in past 12 months (n=145) | No Wheeze in past 12 months (n=1242) | Crude OR (95% CI) |
|---|---|---|---|
| Current smoker in home or PCG smoker | | | |
| Yes | 39 (26.9) | 286 (23.1) | 1.23 (0.83, 1.81) |
| No | 106 (73.1) | 953 (76.9) | 1.00 |
| Missing | | 3 | |
| Either parent has asthma | | | |
| Yes | 34 (24.5) | 110 (9.1) | 3.24 (2.10, 4.99) |
| No | 105 (75.5) | 1099 (90.9) | 1.00 |
| Missing | 6 | 33 | |
| Mother has asthma | | | |
| Yes | 28 (19.4) | 68 (5.5) | 4.12 (2.55, 6.66) |
| No | 116 (80.6) | 1161 (94.5) | 1.00 |
| Missing | 1 | 13 | |
| Gas appliance in home (stove, range or oven) | | | |
| Yes | 132 (91.0) | 1155 (93.0) | 0.77 (0.42, 1.41) |
| No | 13 (9.0) | 87 (7.0) | 1.00 |
| Gas appliance with pilot light (stove, range or oven) | | | |
| Yes | 57 (41.0) | 522 (42.7) | 0.93 (0.65, 1.33) |
| No | 82 (59.0) | 700 (57.3) | 1.00 |
| Missing | 6 | 20 | |
| Pests in home in past 12 months | | | |
| Yes | 117 (80.7) | 930 (74.9) | 1.40 (0.91, 2.16) |
| No | 28 (19.3) | 312 (25.1) | 1.00 |
| Cockroaches in home in past 12 months | | | |
| Yes | 45 (31.0) | 359 (28.9) | 1.11 (0.76, 1.61) |
| No | 100 (69.0) | 883 (71.1) | 1.00 |
| Mold in home in past 12 months | | | |
| Yes | 36 (25.0) | 260 (21.0) | 1.26 (0.84, 1.88) |
| No | 108 (75.0) | 979 (79.0) | 1.00 |
| Missing | 1 | 3 | |
| Furry pets in home | | | |
| Yes | 63 (44.7) | 404 (33.5) | 1.60 (1.13, 2.28) |
| No | 78 (55.3) | 801 (66.5) | 1.00 |
| Missing | 4 | 37 | |
| Neighborhood satisfaction | | | |
| Very satisfied | 35 (28.2) | 248 (23.8) | 1.00 |
| Satisfied or Neutral (if volunteered) | 68 (54.8) | 637 (61.1) | 0.76 (0.49, 1.17) |
| Dissatisfied or Very dissatisfied | 21 (17.0) | 157 (15.1) | 0.95 (0.53, 1.69) |
| Missing | 56 | 165 | |
| How safe to walk alone after dark in this neighborhood | | | |
| Completely safe | 12 (9.8) | 169 (16.3) | 1.00 |
| Fairly safe or Somewhat dangerous | 105 (85.4) | 816 (78.8) | 1.81 (0.98, 3.37) |
| Extremely dangerous | 6 (4.8) | 51 (4.9) | 1.66 (0.59, 4.64) |
| Missing | 22 | 206 | |
| No. adults you recognize in neighborhood | | | |
| Many adults or most or all adults | 73 (58.9) | 522 (50.1) | 1.00 |
| A few adults or no adults | 51 (41.1) | 520 (49.9) | 0.70 (0.48, 1.02) |
| Missing | 21 | 200 | |
| Neighborhood cohesion score | | | |
| <2.52 (median) (higher) | 75 (60.5) | 556 (54.0) | 1.00 |
| ≥2.52 | 49 (39.5) | 474 (46.0) | 0.77 (0.52, 1.12) |
| Missing=233 | 21 | 212 | |
| No. relatives living in neighborhood | | | |
| Any | 45 (36.3) | 423 (40.8) | 1.00 |
| None | 79 (63.7) | 615 (59.2) | 1.21 (0.82, 1.78) |
| Missing | 21 | 204 | |
| No. friends living in neighborhood | | | |
| Any | 88 (71.0) | 716 (68.7) | 1.00 |
| None | 36 (29.0) | 326 (31.3) | 0.90 (0.60, 1.35) |
| Missing | 21 | 200 | |

| Parameter | Wheeze in past 12 months (n=145) | No Wheeze in past 12 months (n=1242) | Crude OR (95% CI) |
|---|---|---|---|
| No. of neighbors talked to for 10 min in past 30 days | | | |
| Any | 106 (85.5) | 915 (87.9) | 1.00 |
| None | 18 (14.5) | 126 (12.1) | 1.23 (0.72, 2.10) |
| Missing | 21 | 201 | |
| No. groups participated in past 12 months | | | |
| Any | 56 (45.2) | 364 (35.0) | 1.00 |
| None | 68 (54.8) | 677 (65.0) | 0.65 (0.45, 0.95) |
| Missing | 21 | 201 | |
| Neighborhood support score | | | |
| 1-<2 (higher) | 43 (36.1) | 283 (27.4) | 1.00 |
| 2-<4 | 70 (58.8) | 667 (64.5) | 0.69 (0.46, 1.04) |
| ≥4 | 6 (5.1) | 84 (8.1) | 0.47 (0.19, 1.14) |
| Missing | 26 | 208 | |
| ***Neighborhood Level*** | | | |
| Census tract level rating of neighborhood cohesion | | | |
| <2.53 (median) (higher) | 79 (56.8) | 596 (50.4) | 1.00 |
| ≥2.53 | 60 (43.2) | 586 (49.6) | 0.77 (0.54, 1.10) |
| Missing | 6 | 60 | |
| Census tract level rating of neighborhood safety | | | |
| <2.13 (median) (higher) | 70 (50.7) | 588 (49.7) | 1.00 |
| ≥2.13 | 68 (49.3) | 595 (50.3) | 0.96 (0.68, 1.37) |
| Missing | 7 | 59 | |
| Tract-level disadvantage | | | |
| <0.13 (median) (lower) | 80 (55.6) | 605 (49.0) | 1.00 |
| ≥0.13 | 64 (44.4) | 630 (51.0) | 0.77 (0.54, 1.09) |
| Missing | 1 | 7 | |
| Percent of tract in same home 5 years ago | | | |
| <0.52 (median) | 78 (54.2) | 617 (50.0) | 0.85 (0.60, 1.19) |
| ≥0.52 | 66 (45.8) | 618 (50.0) | 1.00 |
| Missing | 1 | 7 | |
| Census tract predominately Latino or White | | | |
| Yes | 111 (77.1) | 961 (77.8) | 0.96 (0.64, 1.45) |
| No | 33 (22.9) | 274 (22.2) | 1.00 |
| Missing | 1 | 7 | |

**Table 16. Number and (Percent) of Subjects in Each Outcome Group by Individual-, Family-, and Neighborhood-Level Demographic Characteristics and Crude Odds Ratios (95% Confidence Intervals) for Wheeze in Past 12 Months with Any Night Waking**

| Parameter | Wheeze in past 12 months with any night waking (n=73) | No Wheeze in past 12 months or wheeze without night waking (n=1314) | Crude OR (95% CI) |
|---|---|---|---|
| ***Family Level*** | | | |
| Gender | | | |
| Female | 24 (32.9) | 647 (49.2) | 1.00 |
| Male | 49 (67.1) | 667 (50.8) | 1.98 (1.20, 3.27) |
| Age (years) | | | |
| ≤5 | 5 (6.8) | 117 (8.9) | 0.71 (0.26, 1.93) |
| 6-<10 | 20 (27.4) | 331 (25.2) | 1.00 |
| 10-<15 | 31 (42.5) | 519 (39.5) | 0.99 (0.55, 1.76) |
| ≥15 | 17 (23.3) | 347 (26.4) | 0.81 (0.42, 1.58) |
| Race/ethnicity | | | |
| Non-Hispanic White | 14 (19.2) | 248 (18.9) | 1.00 |
| Hispanic | 42 (57.5) | 879 (66.9) | 0.98 (0.53, 1.81) |
| African American | 12 (16.4) | 93 (7.1) | 2.46 (1.15, 5.29) |
| Asian/Other | 5 (6.9) | 94 (7.1) | 1.01 (0.37, 2.77) |
| Health insurance during past month | | | |
| Yes | 65 (89.0) | 1122 (85.7) | 1.00 |
| No | 8 (11.0) | 188 (14.3) | 0.74 (0.35, 1.56) |
| Missing | | 4 | |
| Usual source of sick care | | | |
| Yes | 69 (94.5) | 1229 (93.6) | 1.00 |
| No | 4 (5.5) | 84 (6.4) | 0.85 (0.30, 2.38) |
| Missing | | 1 | |
| Overweight[1] | | | |
| Yes | 37 (59.7) | 495 (43.4) | 1.89 (1.12, 3.19) |
| No | 25 (40.3) | 633 (56.1) | 1.00 |
| Missing | 11 | 186 | |
| ***Family Level*** | | | |
| Family Income (dollars) | | | |
| <20,000 | 14 (21.9) | 296 (24.7) | 0.83 (0.40, 1.71) |
| 20,000-<35,000 | 17 (26.6) | 304 (25.3) | 0.98 (0.49, 1.96) |
| 35,000-<65,000 | 16 (25.0) | 302 (25.2) | 0.93 (0.46, 1.87) |
| ≥65,000 | 17 (26.5) | 298 (24.8) | 1.00 |
| Missing | 64 | 59 | |
| Homeowner | | | |
| Yes | 30 (43.5) | 518 (41.5) | 1.00 |
| No | 39 (56.5) | 729 (58.5) | 0.92 (0.57, 1.51) |
| Missing | 4 | 67 | |
| PCG's education (years) | | | |
| <12 | 21 (29.2) | 542 (41.4) | 0.58 (0.34, 1.01) |
| 12 | 15 (20.8) | 227 (17.3) | 0.99 (0.53, 1.85) |
| >12 | 36 (50.0) | 540 (41.3) | 1.00 |
| Missing | 1 | 5 | |
| Foreign born status of PCG | | | |
| US born | 39 (53.4) | 490 (37.3) | 1.00 |
| Foreign born | 34 (46.6) | 824 (62.7) | 0.52 (0.32, 0.83) |
| PCG current marital status | | | |
| Married or living with partner | 46 (63.0) | 1025 (78.0) | 1.00 |
| Unmarried/not living with partner | 27 (37.0) | 289 (22.0) | 2.08 (1.27, 3.41) |
| Current household smokers | | | |
| Yes | 19 (26.0) | 278 (21.2) | 1.31 (0.76, 2.24) |
| No | 54 (74.0) | 1034 (78.8) | 1.00 |
| Missing | | 2 | |
| PCG current smoking status | | | |
| Yes | 13 (17.8) | 113 (8.6) | 2.30 (1.23, 4.32) |
| No | 60 (82.2) | 1200 (91.4) | 1.00 |
| Missing | | 1 | |

| Parameter | Wheeze in past 12 months with any night waking (n=73) | No Wheeze in past 12 months or wheeze without night waking (n=1314) | Crude OR (95% CI) |
|---|---|---|---|
| Current smoker in home or PCG smoker | | | |
| Yes | 22 (30.1) | 303 (23.1) | 1.44 (0.86, 2.41) |
| No | 51 (69.9) | 1008 (76.9) | 1.00 |
| Missing | | 3 | |
| Either parent has asthma | | | |
| Yes | 19 (27.5) | 125 (9.8) | 3.51 (2.01, 6.14) |
| No | 50 (72.5) | 1154 (90.2) | 1.00 |
| Missing | 4 | 35 | |
| Mother has asthma | | | |
| Yes | 14 (19.4) | 82 (6.3) | 3.59 (1.92, 6.70) |
| No | 58 (80.6) | 1219 (93.7) | 1.00 |
| Missing | 1 | 13 | |
| Gas appliance in home (stove, range or oven) | | | |
| Yes | 68 (93.2) | 1219 (92.8) | 1.06 (0.42, 2.69) |
| No | 5 (6.8) | 95 (7.2) | 1.00 |
| Gas appliance with pilot light (stove, range or oven) | | | |
| Yes | 30 (42.3) | 549 (42.6) | 0.99 (0.61, 1.60) |
| No | 41 (57.7) | 741 (57.4) | 1.00 |
| Missing | 2 | 24 | |
| Pests in home in past 12 months | | | |
| Yes | 60 (82.2) | 987 (75.1) | 1.53 (0.83, 2.82) |
| No | 13 (17.8) | 327 (24.9) | 1.00 |
| Cockroaches in home in past 12 months | | | |
| Yes | 19 (26.0) | 385 (29.3) | 0.85 (0.50, 1.45) |
| No | 54 (74.0) | 929 (70.7) | 1.00 |
| Mold in home in past 12 months | | | |
| Yes | 18 (24.7) | 278 (21.2) | 1.22 (0.70, 2.10) |
| No | 55 (75.3) | 1032 (78.8) | 1.00 |
| Missing | | 4 | |
| Furry pets in home | | | |
| Yes | 30 (42.3) | 437 (34.3) | 1.40 (0.86, 2.28) |
| No | 41 (57.7) | 838 (65.7) | 1.00 |
| Missing | 2 | 39 | |
| Neighborhood satisfaction | | | |
| Very satisfied | 13 (20.0) | 270 (24.5) | 1.00 |
| Satisfied or Neutral (if volunteered) | 41 (63.1) | 664 (60.3) | 1.28 (0.68, 2.43) |
| Dissatisfied or Very dissatisfied | 11 (16.9) | 167 (15.2) | 1.37 (0.60, 3.12) |
| Missing | 8 | 213 | |
| How safe to walk alone after dark in this neighborhood | | | |
| Completely safe | 4 (6.3) | 177 (16.2) | 1.00 |
| Fairly safe or Somewhat dangerous | 58 (90.6) | 863 (78.8) | 2.97 (1.07, 8.30) |
| Extremely dangerous | 2 (3.1) | 55 (5.0) | 1.61 (0.29, 9.02) |
| Missing | 9 | 219 | |
| No. adults you recognize in neighborhood | | | |
| Many adults or most or all adults | 38 (58.5) | 557 (50.6) | 1.00 |
| A few adults or no adults | 27 (41.5) | 544 (49.4) | 0.73 (0.44, 1.21) |
| Missing | 8 | 213 | |
| Neighborhood cohesion score | | | |
| <2.52 (median) (higher) | 38 (58.5) | 593 (54.5) | 1.00 |
| ≥2.52 | 27 (41.5) | 496 (45.5) | 0.85 (0.51, 1.41) |
| Missing | 8 | 225 | |
| No. relatives living in neighborhood | | | |
| Any | 25 (38.5) | 443 (40.4) | 1.00 |
| None | 40 (61.5) | 654 (59.6) | 1.08 (0.65, 1.81) |
| Missing | 8 | 217 | |
| No. friends living in neighborhood | | | |
| Any | 51 (78.5) | 753 (68.4) | 1.00 |
| None | 14 (21.5) | 348 (31.6) | 0.59 (0.32, 1.09) |
| Missing | 8 | 213 | |

| Parameter | Wheeze in past 12 months with any night waking (n=73) | No Wheeze in past 12 months or wheeze without night waking (n=1314) | Crude OR (95% CI) |
|---|---|---|---|
| No. of neighbors talked to for 10 min in past 30 days | | | |
| Any | 58 (89.2) | 963 (87.5) | 1.00 |
| None | 7 (10.8) | 137 (12.5) | 0.85 (0.38, 1.90) |
| Missing | 8 | 214 | |
| No. groups participated in past 12 months | | | |
| Any | 27 (41.5) | 393 (35.7) | 1.00 |
| None | 38 (58.5) | 707 (64.3) | 0.78 (0.47, 1.30) |
| Missing | 8 | 214 | |
| Neighborhood support score | | | |
| 1-<2 (higher) | 21 (33.3) | 305 (28.0) | 1.00 |
| 2-<4 | 40 (63.5) | 697 (63.9) | 0.83 (0.48, 1.44) |
| ≥4 | 2 (3.2) | 88 (8.1) | 0.33 (0.08, 1.44) |
| Missing | 10 | 224 | |
| ***Neighborhood Level*** | | | |
| Census tract level rating of neighborhood cohesion | | | |
| <2.53 (median) (higher) | 35 (48.6) | 640 (51.2) | 1.00 |
| ≥2.53 | 37 (51.4) | 609 (48.8) | 1.11 (0.69, 1.79) |
| Missing | 1 | 65 | |
| Census tract level rating of neighborhood safety | | | |
| <2.13 (median) (higher) | 33 (46.5) | 625 (50.0) | 1.00 |
| ≥2.13 | 38 (53.5) | 625 (50.0) | 1.15 (0.71, 1.86) |
| Missing | 1 | 7 | |
| Tract-level disadvantage | | | |
| <0.13 (median) (lower) | 38 (52.8) | 647 (49.5) | 1.00 |
| ≥0.13 | 35 (47.2) | 660 (50.5) | 0.88 (0.55, 1.41) |
| Missing | 1 | 7 | |
| Percent of tract in same home 5 years ago | | | |
| <0.52 (median) | 33 (45.8) | 662 (50.7) | 1.21 (0.75, 1.95) |
| ≥0.52 | 39 (54.2) | 645 (49.4) | 1.00 |
| Missing | 1 | 7 | |
| Census tract predominately Latino or White | | | |
| Yes | 56 (77.8) | 1016 (77.7) | 1.00 (0.57, 1.77) |
| No | 16 (22.2) | 291 (22.3) | 1.00 |
| Missing | 1 | 7 | |

**Table 17. Number and (Percent) of Subjects in Each Outcome Group by Individual-, Family-, and Neighborhood-Level Demographic Characteristics and Crude Odds Ratios (95% Confidence Intervals) for Medication Use for Asthma or Wheeze in Past 12 Months**

| Parameter | Medication use for asthma or wheeze in past 12 months (n=162) | No Medication use for asthma or wheeze in past 12 months (n=1225) | Crude OR (95% CI) |
|---|---|---|---|
| ***Individual Level*** | | | |
| Gender | | | |
| Female | 57 (35.2) | 614 (50.1) | 1.00 |
| Male | 105 (64.8) | 611 (49.9) | 1.85 (1.32, 2.60) |
| Age (years) | | | |
| ≤5 | 10 (6.2) | 90 (7.4) | 0.77 (0.37, 1.57) |
| 6-<10 | 48 (29.6) | 331 (27.0) | 1.00 |
| 10-<15 | 65 (40.1) | 486 (39.7) | 0.92 (0.62, 1.37) |
| ≥15 | 39 (24.1) | 318 (26.0) | 0.85 (0.54, 1.33) |
| Race/ethnicity | | | |
| Non-Hispanic White | 36 (22.2) | 226 (18.5) | 1.00 |
| Hispanic | 86 (53.1) | 835 (68.2) | 0.65 (0.43, 0.97) |
| African American | 26 (16.1) | 79 (6.4) | 2.07 (1.21, 3.54) |
| Asian/Other | 14 (8.6) | 85 (6.9) | 1.04 (0.55, 1.98) |
| Health insurance during past month | | | |
| Yes | 148 (91.4) | 1039 (85.1) | 1.00 |
| No | 14 (8.6) | 182 (14.9) | 0.54 (0.31, 0.96) |
| Missing | | 4 | |
| Usual source of sick care | | | |
| Yes | 154 (95.1) | 1144 (93.5) | 1.00 |
| No | 8 (4.9) | 80 (6.5) | 0.74 (0.35, 1.57) |
| Missing | | 1 | |
| Overweight[1] | | | |
| Yes | 70 (54.3) | 462 (43.5) | 1.54 (1.07, 2.22) |
| No | 59 (45.7) | 599 (56.5) | 1.00 |
| Missing | 33 | 164 | |
| ***Family Level*** | | | |
| Family Income (dollars) | | | |
| <20,000 | 36 (24.7) | 274 (24.5) | 0.88 (0.54, 1.42) |
| 20,000-<35,000 | 35 (24.0) | 286 (25.6) | 0.82 (0.51, 1.32) |
| 35,000-<65,000 | 34 (23.3) | 284 (25.4) | 0.80 (0.49, 1.30) |
| ≥65,000 | 41 (28.1) | 274 (24.5) | 1.00 |
| Missing | 16 | 107 | |
| Homeowner | | | |
| Yes | 65 (43.0) | 483 (41.5) | 1.00 |
| No | 86 (57.0) | 682 (58.5) | 0.94 (0.67, 1.32) |
| Missing | 11 | 60 | |
| PCG's education (years) | | | |
| <12 | 50 (31.1) | 513 (42.1) | 0.56 (0.39, 0.82) |
| 12 | 26 (16.1) | 216 (17.7) | 0.70 (0.44, 1.11) |
| >12 | 85 (52.8) | 491 (40.3) | 1.00 |
| Missing | 1 | 5 | |
| Foreign born status of PCG | | | |
| US born | 83 (51.2) | 446 (36.4) | 1.00 |
| Foreign born | 79 (48.8) | 779 (63.6) | 0.55 (0.39, 0.76) |
| PCG current marital status | | | |
| Married or living with partner | 109 (67.3) | 962 (78.5) | 1.00 |
| Unmarried/not living with partner | 53 (32.7) | 263 (21.5) | 1.78 (1.25, 2.54) |
| Current household smokers | | | |
| Yes | 34 (21.0) | 263 (21.5) | 0.97 (0.65, 1.45) |
| No | 128 (79.0) | 960 (78.5) | 1.00 |
| Missing | | 2 | |
| PCG current smoking status | | | |
| Yes | 20 (12.4) | 106 (8.7) | 1.49 (0.89, 2.47) |
| No | 142 (87.6) | 1118 (91.3) | 1.00 |
| Missing | | 1 | |

| Parameter | Medication use for asthma or wheeze in past 12 months (n=162) | No Medication use for asthma or wheeze in past 12 months (n=1225) | Crude OR (95% CI) |
|---|---|---|---|
| Current smoker in home or PCG smoker | | | |
| Yes | 40 (24.7) | 285 (23.3) | 1.08 (0.74, 1.58) |
| No | 122 (75.3) | 937 (76.7) | 1.00 |
| Missing | | 3 | |
| Either parent has asthma | | | |
| Yes | 43 (27.6) | 101 (8.5) | 4.11 (2.74, 6.17) |
| No | 113 (72.4) | 1091 (91.5) | 1.00 |
| Missing | 6 | 33 | |
| Mother has asthma | | | |
| Yes | 32 (20.0) | 64 (5.3) | 4.49 (2.83, 7.13) |
| No | 128 (80.0) | 1149 (94.7) | 1.00 |
| Missing | 2 | 12 | |
| Gas appliance in home (stove, range or oven) | | | |
| Yes | 148 (91.4) | 1139 (93.0) | 0.80 (0.44, 1.44) |
| No | 14 (8.6) | 86 (7.0) | 1.00 |
| Gas appliance with pilot light (stove, range or oven) | | | |
| Yes | 63 (40.4) | 516 (42.8) | 0.91 (0.64, 1.27) |
| No | 93 (59.6) | 689 (57.2) | 1.00 |
| Missing | 6 | 20 | |
| Pests in home in past 12 months | | | |
| Yes | 128 (79.0) | 919 (75.0) | 1.25 (0.84, 1.87) |
| No | 34 (21.0) | 306 (25.0) | 1.00 |
| Cockroaches in home in past 12 months | | | |
| Yes | 44 (27.2) | 360 (29.4) | 0.90 (0.62, 1.29) |
| No | 118 (72.8) | 865 (70.6) | 1.00 |
| Mold in home in past 12 months | | | |
| Yes | 35 (21.7) | 261 (21.4) | 1.02 (0.69, 1.52) |
| No | 126 (78.3) | 961 (78.6) | 1.00 |
| Missing | 1 | 3 | |
| Furry pets in home | | | |
| Yes | 60 (37.7) | 407 (34.3) | 1.16 (0.83, 1.64) |
| No | 99 (62.3) | 780 (65.7) | 1.00 |
| Missing | 29 | 12 | |
| Neighborhood satisfaction | | | |
| Very satisfied | 35 (26.3) | 248 (24.0) | 1.00 |
| Satisfied or Neutral (if volunteered) | 77 (57.9) | 628 (60.8) | 0.87 (0.57, 1.33) |
| Dissatisfied or Very dissatisfied | 21 (15.8) | 157 (15.2) | 0.95 (0.53, 1.69) |
| Missing | 29 | 192 | |
| How safe to walk alone after dark in this neighborhood | | | |
| Completely safe | 15 (11.5) | 166 (16.1) | 1.00 |
| Fairly safe or Somewhat dangerous | 108 (82.4) | 813 (79.1) | 1.47 (0.84, 2.59) |
| Extremely dangerous | 8 (6.1) | 49 (4.8) | 1.81 (0.72, 4.51) |
| Missing | 31 | 197 | |
| No. adults you recognize in neighborhood | | | |
| Many adults or most or all adults | 68 (51.1) | 527 (51.0) | 1.00 |
| A few adults or no adults | 65 (48.9) | 506 (49.0) | 1.00 (0.69, 1.43) |
| Missing | 29 | 192 | |
| Neighborhood cohesion score | | | |
| <2.52 (median) (higher) | 76 (57.1) | 555 (54.4) | 1.00 |
| ≥2.52 | 57 (42.9) | 466 (45.6) | 0.89 (0.62, 1.29) |
| Missing | 29 | 204 | |
| No. relatives living in neighborhood | | | |
| Any | 55 (41.4) | 413 (40.1) | 1.00 |
| None | 78 (58.6) | 616 (59.9) | 0.95 (0.66, 1.37) |
| Missing | 29 | 196 | |
| No. friends living in neighborhood | | | |
| Any | 91 (68.4) | 713 (69.0) | 1.00 |
| None | 42 (31.6) | 320 (31.0) | 1.03 (0.70, 1.52) |
| Missing=221 | 29 | 192 | |

| Parameter | Medication use for asthma or wheeze in past 12 months (n=162) | No Medication use for asthma or wheeze in past 12 months (n=1225) | Crude OR (95% CI) |
|---|---|---|---|
| No. of neighbors talked to for 10 min in past 30 days | | | |
| Any | 117 (88.0) | 904 (87.6) | 1.00 |
| None | 16 (12.0) | 128 (12.4) | 0.97 (0.56, 1.68) |
| Missing | 29 | 193 | |
| No. groups participated in past 12 months | | | |
| Any | 58 (43.6) | 362 (35.1) | 1.00 |
| None | 75 (56.4) | 670 (64.9) | 0.70 (0.49, 1.01) |
| Missing | 29 | 193 | |
| Neighborhood support score | | | |
| 1-<2 (higher) | 44 (34.1) | 282 (27.5) | 1.00 |
| 2-<4 | 78 (60.5) | 659 (64.4) | 0.76 (0.51, 1.13) |
| ≥4 | 7 (5.4) | 83 (8.1) | 0.54 (0.24, 1.25) |
| Missing | 33 | 201 | |
| ***Neighborhood Level*** | | | |
| Census tract level rating of neighborhood cohesion | | | |
| <2.53 (median) (higher) | 85 (56.3) | 590 (50.4) | 1.00 |
| ≥2.53 | 66 (43.7) | 580 (49.6) | 0.79 (0.56, 1.11) |
| Missing | 11 | 55 | |
| Census tract level rating of neighborhood safety | | | |
| <2.13 (median) (higher) | 75 (50.3) | 583 (49.7) | 1.00 |
| ≥2.13 | 74 (49.7) | 589 (50.3) | 0.98 (0.69, 1.37) |
| Missing | 13 | 53 | |
| Tract-level disadvantage | | | |
| <0.13 (median) (lower) | 81 (50.3) | 604 (49.6) | 1.00 |
| ≥0.13 | 80 (49.7) | 614 (50.4) | 0.97 (0.70, 1.35) |
| Missing | 1 | 7 | |
| Percent of tract in same home 5 years ago | | | |
| <0.52 (median) | 83 (51.6) | 612 (50.3) | 0.95 (0.68, 1.32) |
| ≥0.52 | 78 (48.5) | 606 (49.8) | 1.00 |
| Missing | 1 | 7 | |
| Census tract predominately Latino or White | | | |
| Yes | 122 (75.8) | 950 (78.0) | 0.88 (0.60, 1.30) |
| No | 39 (24.2) | 268 (22.0) | 1.00 |
| Missing | 1 | 7 | |

**Table 18. Number and (Percent) of Subjects in Each Outcome Group by Individual-, Family-, and Neighborhood-Level Demographic Characteristics and Crude Odds Ratios (95% Confidence Intervals) for Sneezing, Runny or Blocked Nose Without Cold in Past 12 Months**

| Parameter | Sneezing, runny or blocked nose without cold in past 12 months (n=270) | Sneezing, runny or blocked nose without cold in past 12 months (n=1116) | Crude OR (95% CI) |
|---|---|---|---|
| ***Individual Level*** | | | |
| Gender | | | |
| Female | 121 (44.8) | 549 (49.2) | 1.00 |
| Male | 149 (55.2) | 567 (50.8) | 1.19 (0.91, 1.56) |
| Age (years) | | | |
| ≤5 | 20 (7.4) | 102 (9.1) | 0.72 (0.42, 1.24) |
| 6-<10 | 75 (27.8) | 275 (24.7) | 1.00 |
| 10-<15 | 110 (40.7) | 440 (39.4) | 0.92 (0.66, 1.28) |
| ≥15 | 65 (24.1) | 299 (26.8) | 0.80 (0.55, 1.15) |
| Race/ethnicity | | | |
| Non-Hispanic White | 71 (26.3) | 191 (17.1) | 1.00 |
| Hispanic | 135 (50.0) | 785 (70.3) | 0.48 (0.35, 0.66) |
| African American | 28 (10.4) | 77 (6.9) | 1.12 (0.69, 1.83) |
| Asian/Other | 36 (13.3) | 63 (5.7) | 1.68 (1.04, 2.72) |
| Health insurance during past month | | | |
| Yes | 238 (88.5) | 948 (85.2) | 1.00 |
| No | 31 (11.5) | 165 (14.8) | 0.75 (0.50, 1.13) |
| Missing | 1 | 3 | |
| Usual source of sick care | | | |
| Yes | 251 (93.0) | 1046 (93.8) | 1.00 |
| No | 19 (7.0) | 69 (6.2) | 1.15 (0.68, 1.94) |
| Missing | | 1 | |
| Overweight[1] | | | |
| Yes | 99 (42.5) | 432 (45.2) | 0.90 (0.67, 1.20) |
| No | 134 (57.5) | 524 (54.8) | 1.00 |
| Missing | 37 | 160 | |
| ***Family Level*** | | | |
| Family Income (dollars) | | | |
| <20,000 | 37 (15.48) | 272 (26.56) | 0.35 (0.23, 0.54) |
| 20,000-<35,000 | 48 (20.08) | 273 (26.66) | 0.45 (0.31, 0.67) |
| 35,000-<65,000 | 66 (27.62) | 252 (24.61) | 0.68 (0.47, 0.97) |
| ≥65,000 | 88 (36.82) | 227 (22.17) | 1.00 |
| Missing | 31 | 92 | |
| Homeowner | | | |
| Yes | 133 (51.55) | 415 (39.26) | 1.00 |
| No | 125 (48.45) | 642 (60.74) | 0.61 (0.46, 0.80) |
| Missing | 12 | 59 | |
| PCG's education (years) | | | |
| <12 | 70 (26.02) | 492 (44.28) | 0.38 (0.28, 0.51) |
| 12 | 41 (15.24) | 201 (18.09) | 0.54 (0.37, 0.79) |
| >12 | 158 (58.74) | 418 (37.62) | 1.00 |
| Missing | 1 | 5 | |
| Foreign born status of PCG | | | |
| US born | 139 (51.48) | 390 (34.95) | 1.00 |
| Foreign born | 131 (48.52) | 726 (65.05) | 0.51 (0.39, 0.66) |
| PCG current marital status | | | |
| Married or living with partner | 204 (75.56) | 866 (77.60) | 1.00 |
| Unmarried/not living with partner | 66 (24.44) | 250 (22.40) | 1.12 (0.82, 1.53) |
| Current household smokers | | | |
| Yes | 62 (22.96) | 235 (21.10) | 1.12 (0.81, 1.53) |
| No | 208 (77.04) | 879 (78.90) | 1.00 |
| Missing | | 2 | |
| PCG current smoking status | | | |
| Yes | 26 (9.63) | 100 (8.97) | 1.08 (0.69, 1.70) |
| No | 244 (90.37) | 1015 (91.03) | 1.00 |
| Missing | | 1 | |

| Parameter | Sneezing, runny or blocked nose without cold in past 12 months (n=270) | Sneezing, runny or blocked nose without cold in past 12 months (n=1116) | Crude OR (95% CI) |
|---|---|---|---|
| Current smoker in home or PCG smoker | | | |
| Yes | 67 (24.81) | 258 (23.18) | 1.09 (0.80, 1.49) |
| No | 203 (75.19) | 855 (76.82) | 1.00 |
| Missing | | 3 | |
| Either parent has asthma | | | |
| Yes | 50 (18.94) | 94 (8.68) | 2.46 (1.69, 3.57) |
| No | 214 (81.06) | 989 (91.32) | 1.00 |
| Missing | 6 | 33 | |
| Mother has asthma | | | |
| Yes | 36 (13.43) | 60 (5.43) | 2.70 (1.74, 4.18) |
| No | 232 (86.57) | 1044 (94.57) | 1.00 |
| Missing | 2 | 12 | |
| Gas appliance in home (stove, range or oven) | | | |
| Yes | 253 (93.70) | 1033 (92.56) | 1.20 (0.70, 2.05) |
| No | 17 (6.30) | 83 (7.44) | 1.00 |
| Gas appliance with pilot light (stove, range or oven) | | | |
| Yes | 106 (39.85) | 472 (43.14) | 0.87 (0.66, 1.15) |
| No | 160 (60.15) | 622 (56.86) | 1.00 |
| Missing | | | |
| Pests in home in past 12 months | | | |
| Yes | 220 (81.48) | 827 (74.10) | 1.54 (1.10, 2.15) |
| No | 50 (18.52) | 289 (25.90) | 1.00 |
| Cockroaches in home in past 12 months | | | |
| Yes | 75 (27.78) | 329 (29.48) | 0.92 (0.68, 1.24) |
| No | 195 (72.22) | 787 (70.52) | 1.00 |
| Mold in home in past 12 months | | | |
| Yes | 72 (26.77) | 224 (20.13) | 1.45 (1.07, 1.97) |
| No | 197 (73.23) | 889 (79.87) | 1.00 |
| Missing | 1 | 3 | |
| Furry pets in home | | | |
| Yes | 105 (40.38) | 362 (33.36) | 1.35 (1.03, 1.79) |
| No | 155 (59.62) | 723 (66.64) | 1.00 |
| Missing | 10 | 31 | |
| Neighborhood satisfaction | | | |
| Very satisfied | 74 (31.09) | 209 (22.55) | 1.00 |
| Satisfied or Neutral (if volunteered) | 132 (55.46) | 572 (61.70) | 0.65 (0.47, 0.90) |
| Dissatisfied or Very dissatisfied | 32 (13.45) | 146 (15.75) | 0.62 (0.39, 0.99) |
| Missing | 32 | 189 | |
| How safe to walk alone after dark in this neighborhood | | | |
| Completely safe | 47 (19.83) | 134 (14.55) | 1.00 |
| Fairly safe or Somewhat dangerous | 184 (77.64) | 736 (79.91) | 0.71 (0.49, 1.03) |
| Extremely dangerous | 6 (2.53) | 51 (5.54) | 0.34 (0.14, 0.83) |
| Missing | | | |
| No. adults you recognize in neighborhood | | | |
| Many adults or most or all adults | 129 (54.20) | 466 (50.27) | 1.00 |
| A few adults or no adults | 109 (45.80) | 461 (49.73) | 0.85 (0.64, 1.14) |
| Missing | 32 | 189 | |
| Neighborhood cohesion score | | | |
| <2.52 (median) (higher) | 137 (57.81) | 493 (53.82) | 1.00 |
| ≥2.52 | 100 (42.19) | 423 (46.18) | 0.85 (0.64, 1.14) |
| Missing | 33 | 200 | |
| No. relatives living in neighborhood | | | |
| Any | 92 (38.82) | 376 (40.69) | 1.00 |
| None | 145 (61.18) | 548 (59.31) | 1.08 (0.81, 1.45) |
| Missing | 33 | 192 | |
| No. friends living in neighborhood | | | |
| Any | 167 (70.17) | 636 (68.61) | 1.00 |
| None | 71 (29.83) | 291 (31.39) | 0.93 (0.68, 1.27) |
| Missing | 32 | 189 | |

| Parameter | Sneezing, runny or blocked nose without cold in past 12 months (n=270) | Sneezing, runny or blocked nose without cold in past 12 months (n=1116) | Crude OR (95% CI) |
|---|---|---|---|
| No. of neighbors talked to for 10 min in past 30 days | | | |
| Any | 209 (87.82) | 811 (87.58) | 1.00 |
| None | 29 (12.18) | 115 (12.42) | 0.98 (0.63, 1.51) |
| Missing | 32 | 190 | |
| No. groups participated in past 12 months | | | |
| Any | 99 (41.60) | 321 (34.67) | 1.00 |
| None | 139 (58.40) | 605 (65.33) | 0.75 (0.56, 1.00) |
| Missing | 32 | 190 | |
| Neighborhood support score | | | |
| 1-<2 (higher) | 68 (29.06) | 258 (28.10) | 1.0 |
| 2-<4 | 150 (64.10) | 586 (63.83) | 0.97 (0.70, 1.34) |
| ≥4 | 16 (6.84) | 74 (8.06) | 0.82 (0.45, 1.50) |
| Missing | 36 | 198 | |
| ***Neighborhood Level*** | | | |
| Census tract level rating of neighborhood cohesion | | | |
| <2.53 (median) (higher) | 164 (62.4) | 510 (48.3) | 1.00 |
| ≥2.53 | 99 (37.6) | 547 (51.8) | 0.56 (0.43, 0.74) |
| Missing | 7 | 59 | |
| Census tract level rating of neighborhood safety | | | |
| <2.13 (median) (higher) | 152 (58.0) | 506 (47.8) | 1.00 |
| ≥2.13 | 110 (42.0) | 552 (52.2) | 0.66 (0.51, 0.87) |
| Missing | 8 | 58 | |
| Tract-level disadvantage | | | |
| <0.13 (median) (lower) | 164 (61.4) | 521 (46.9) | 1.00 |
| ≥0.13 | 103 (38.6) | 590 (53.1) | 0.56 (0.42, 0.73) |
| Missing | 3 | 5 | |
| Percent of tract in same home 5 years ago | | | |
| <0.52 (median) | 135 (50.6) | 560 (50.4) | 0.99 (0.76, 1.30) |
| ≥0.52 | 132 (49.4) | 551 (49.6) | 1.00 |
| Missing | 3 | 5 | |
| Census tract predominately Latino or White | | | |
| Yes | 201 (75.3) | 870 (78.3) | 0.84 (0.62, 1.15) |
| No | 66 (24.7) | 241 (21.7) | 1.00 |
| Missing | 3 | 5 | |

**Table 19. Number and (Percent) of Subjects in Each Outcome Group by Individual-, Family-, and Neighborhood-Level Demographic Characteristics and Crude Odds Ratios (95% Confidence Intervals) for More than Three Doctor-Diagnosed Ear Infections in One Year**

| Parameter | More than 3 doctor-diagnosed ear infections in 1 yr (n=140) | More than 3 doctor-diagnosed ear infections in 1 yr (n=1246) | Crude OR (95% CI) |
|---|---|---|---|
| ***Individual Level*** | | | |
| Gender | | | |
| Female | 66 (47.14) | 604 (48.48) | 1.00 |
| Male | 74 (52.86) | 642 (51.52) | 1.06 (0.74, 1.50) |
| Age (years) | | | |
| ≤5 | 11 (7.86) | 111 (8.91) | 0.75 (0.37, 1.51) |
| 6-<10 | 41 (29.29) | 310 (24.88) | 1.00 |
| 10-<15 | 57 (40.71) | 492 (39.49) | 0.88 (0.57, 1.34) |
| ≥15 | 31 (22.14) | 333 (26.73) | 0.70 (0.43, 1.15) |
| Race/ethnicity | | | |
| Non-Hispanic White | 48 (34.29) | 214 (17.17) | 1.00 |
| Hispanic | 69 (49.29) | 852 (68.38) | 0.40 (0.27, 0.60) |
| African American | 7 (5.00) | 97 (7.78) | 0.35 (0.16, 0.81) |
| Asian/Other | 16 (11.43) | 83 (6.66) | 1.00 (0.54, 1.85) |
| Health insurance during past month | | | |
| Yes | 128 (91.43) | 1058 (85.19) | 1.00 |
| No | 12 (8.57) | 184 (14.81) | 0.54 (0.29, 0.99) |
| Missing | | 4 | |
| Usual source of sick care | | | |
| Yes | 130 (92.86) | 1167 (93.73) | 1.00 |
| No | 10 (7.14) | 78 (6.27) | 1.15 (0.58, 2.28) |
| Missing | | 1 | |
| Overweight[1] | | | |
| Yes | 60 (50.4) | 472 (44.1) | 1.29 (0.88, 1.88) |
| No | 59 (49.6) | 598 (55.9) | 1.00 |
| Missing | 21 | 176 | |
| *Family Level* | | | |
| Family Income (dollars) | | | |
| <20,000 | 20 (15.87) | 290 (25.48) | 0.41 (0.24, 0.72) |
| 20,000-<35,000 | 28 (22.22) | 293 (25.75) | 0.57 (0.35, 0.95) |
| 35,000-<65,000 | 33 (26.19) | 285 (25.04) | 0.70 (0.43, 1.12) |
| ≥65,000 | 45 (35.71) | 270 (23.73) | 1.00 |
| Missing | 14 | 108 | |
| Homeowner | | | |
| Yes | 67 (50.00) | 481 (40.73) | 1.00 |
| No | 67 (50.00) | 700 (59.27) | 0.69 (0.48, 0.98) |
| Missing | 6 | 65 | |
| PCG's education (years) | | | |
| <12 | 42 (30.22) | 521 (41.98) | 0.49 (0.33, 0.72) |
| 12 | 15 (10.79) | 227 (18.29) | 0.40 (0.22, 0.70) |
| >12 | 82 (58.99) | 493 (39.73) | 1.00 |
| Missing | 1 | 5 | |
| Foreign born status of PCG | | | |
| US born | 84 (60.00) | 444 (35.63) | 1.00 |
| Foreign born | 56 (40.00) | 802 (64.37) | 0.37 (0.26, 0.53) |
| PCG current marital status | | | |
| Married or living with partner | 103 (73.57) | 967 (77.61) | 1.00 |
| Unmarried/not living with partner | 37 (26.43) | 279 (22.39) | 1.25 (0.84, 1.86) |
| Current household smokers | | | |
| Yes | 28 (20.00) | 268 (21.54) | 0.91 (0.59, 1.41) |
| No | 112 (80.00) | 976 (78.46) | 1.00 |
| Missing | | 2 | |
| PCG current smoking status | | | |
| Yes | 13 (9.29) | 113 (9.08) | 1.03 (0.56, 1.87) |
| No | 127 (90.71) | 1132 (90.92) | 1.00 |
| Missing | | 1 | |

| Parameter | More than 3 doctor-diagnosed ear infections in 1 yr (n=140) | More than 3 doctor-diagnosed ear infections in 1 yr (n=1246) | Crude OR (95% CI) |
|---|---|---|---|
| Current smoker in home or PCG smoker | | | |
| Yes | 32 (22.86) | 292 (23.49) | 0.97 (0.64, 1.46) |
| No | 108 (77.14) | 951 (76.51) | 1.00 |
| Missing | | 3 | |
| Either parent has asthma | | | |
| Yes | 25 (18.38) | 118 (9.74) | 2.09 (1.30, 3.35) |
| No | 111 (81.62) | 1093 (90.26) | 1.00 |
| Missing | 4 | 35 | |
| Mother has asthma | | | |
| Yes | 17 (12.41) | 78 (6.32) | 2.10 (1.20, 3.67) |
| No | 120 (87.59) | 1157 (93.68) | 1.00 |
| Missing | 3 | 11 | |
| Gas appliance in home (stove, range or oven) | | | |
| Yes | 120 (85.71) | 1166 (93.58) | 0.41 (0.24, 0.70) |
| No | 20 (14.29) | 80 (6.42) | 1.00 |
| Gas appliance with pilot light (stove, range or oven) | | | |
| Yes | 49 (36.03) | 530 (43.30) | 0.74 (0.51, 1.07) |
| No | 87 (63.97) | 694 (56.70) | 1.00 |
| Missing | 4 | 22 | |
| Pests in home in past 12 months | | | |
| Yes | 109 (77.86) | 938 (75.28) | 1.16 (0.76, 1.76) |
| No | 31 (22.14) | 308 (24.72) | 1.00 |
| Cockroaches in home in past 12 months | | | |
| Yes | 27 (19.29) | 377 (30.26) | 0.55 (0.36, 0.85) |
| No | 113 (80.71) | 869 (69.74) | 1.00 |
| Mold in home in past 12 months | | | |
| Yes | 39 (27.86) | 257 (20.69) | 1.48 (1.00, 2.20) |
| No | 101 (72.14) | 985 (79.31) | 1.00 |
| Missing | | 4 | |
| Furry pets in home | | | |
| Yes | 65 (47.79) | 402 (33.25) | 1.84 (1.29, 2.63) |
| No | 71 (52.21) | 807 (66.75) | 1.00 |
| Missing | 4 | 37 | |
| Neighborhood satisfaction | | | |
| Very satisfied | 29 (24.58) | 253 (24.16) | 1.00 |
| Satisfied or Neutral (if volunteered) | 73 (61.86) | 632 (60.36) | 1.01 (0.64, 1.59) |
| Dissatisfied or Very dissatisfied | 16 (13.56) | 162 (15.47) | 0.86 (0.45, 1.64) |
| Missing | 22 | 199 | |
| How safe to walk alone after dark in this neighborhood | | | |
| Completely safe | 19 (16.10) | 162 (15.58) | 1.00 |
| Fairly safe or Somewhat dangerous | 95 (80.51) | 825 (79.33) | 0.98 (0.58, 1.65) |
| Extremely dangerous | 4 (3.39) | 53 (5.10) | 0.64 (0.21, 1.98) |
| Missing | 22 | 206 | |
| No. adults you recognize in neighborhood | | | |
| Many adults or most or all adults | 61 (51.7) | 513 (49.0) | 1.00 |
| A few adults or no adults | 57 (48.3) | 534 (51.0) | 0.97 (0.67, 1.42) |
| Missing | 22 | 199 | |
| Neighborhood cohesion score1 | | | |
| <2.52 (median) (higher) | 65 (55.56) | 565 (54.54) | 1.00 |
| ≥2.52 | 52 (44.44) | 471 (45.46) | 0.96 (0.65, 1.41) |
| Missing | 23 | 210 | |
| No. relatives living in neighborhood | | | |
| Any | 40 (33.90) | 428 (41.04) | 1.00 |
| None | 78 (66.10) | 615 (58.96) | 1.36 (0.91, 2.03) |
| Missing | 22 | 203 | |
| No. friends living in neighborhood | | | |
| Any | 89 (75.42) | 715 (68.29) | 1.00 |
| None | 29 (24.58) | 332 (31.71) | 0.70 (0.45, 1.09) |
| Missing | 22 | 199 | |

| Parameter | More than 3 doctor-diagnosed ear infections in 1 yr (n=140) | More than 3 doctor-diagnosed ear infections in 1 yr (n=1246) | Crude OR (95% CI) |
|---|---|---|---|
| No. of neighbors talked to for 10 min in past 30 days | | | |
| Any | 100 (84.75) | 920 (87.95) | 1.00 |
| None | 18 (15.25) | 126 (12.05) | 1.32 (0.77, 2.25) |
| Missing | 22 | 200 | |
| No. groups participated in past 12 months | | | |
| Any | 50 (42.37) | 369 (35.28) | 1.00 |
| None | 68 (57.63) | 677 (64.72) | 0.74 (0.50, 1.09) |
| Missing | 22 | 200 | |
| Neighborhood support score | | | |
| 1-<2 (higher) | 36 (31.30) | 290 (27.97) | 1.00 |
| 2-<4 | 70 (60.87) | 666 (64.22) | 0.85 (0.55, 1.30) |
| ≥4 | 9 (7.83) | 81 (7.81) | 0.90 (0.41, 1.94) |
| Missing | 25 | 209 | |
| ***Neighborhood Level*** | | | |
| Census tract level rating of neighborhood cohesion | | | |
| <2.53 (median) (higher) | 78 (57.8) | 596 (50.3) | 1.00 |
| ≥2.53 | 57 (42.2) | 589 (49.7) | 0.74 (0.52, 1.06) |
| Missing | 5 | 62 | |
| Census tract level rating of neighborhood safety | | | |
| <2.13 (median) (higher) | 82 (60.3) | 575 (48.6) | 1.00 |
| ≥2.13 | 54 (39.7) | 609 (51.4) | 0.62 (0.43, 0.89) |
| Missing | 4 | 63 | |
| Tract-level disadvantage | | | |
| <0.13 (median) (lower) | 83 (59.3) | 601 (48.6) | 1.00 |
| ≥0.13 | 57 (40.7) | 637 (51.5) | 0.65 (0.45, 0.92) |
| Missing | | 8 | |
| Percent of tract in same home 5 years ago | | | |
| <0.52 (median) | 74 (52.9) | 620 (50.1) | 0.90 (0.63, 1.27) |
| ≥0.52 | 66 (47.1) | 618 (50.0) | 1.00 |
| Missing | | 8 | |
| Census tract predominately Latino or White | | | |
| Yes | 105 (75.0) | 967 (78.1) | 0.84 (0.56, 1.26) |
| No | 35 (25.0) | 271 (21.9) | 1.00 |
| Missing | | 8 | |

**Table 20. Associations (Odds ratios, 95% CIs) between Annual Average Air Pollution Exposure Metrics (12 Mos prior to Interview) and Wheeze in the Past 12 Months Among L.A. FANS-2 Participants Ages 0–17 Years**

| Pollutant | Crude (per IQR increase)[1] | Model 1: Adjusting for age, race/ethnicity, sex, income | Model 1+Maternal Asthma | Model 1+ No. of Groups Participated in past 12 mos | Model 1+ Tract-level disadvantage | Model 1+Overweight | Model 1+ Maternal Asthma, No. of Groups Participated in past 12 mos, Overweight | Model 1 + Maternal Asthma, No. of Groups Participated in past 12 mos, Tract-level disadvantage, Overweight |
|---|---|---|---|---|---|---|---|---|
| NO | 0.92 (0.74, 1.15) | 1.19 (0.93, 1.52) | 1.14 (0.88, 1.46) | 1.26 (0.96, 1.65) | 1.21 (0.94, 1.56) | 1.23 (0.94, 1.62) | 1.13 (0.84, 1.51) | 1.15 (0.85, 1.55) |
| $NO_2$ | 0.91 (0.74, 1.13) | 1.14 (0.89, 1.47) | 1.09 (0.84, 1.40) | 1.13 (0.86, 1.48) | 1.15 (0.89, 1.49) | 1.17 (0.88, 1.55) | 1.02 (0.76, 1.37) | 1.02 (0.76, 1.37) |
| $NO_x$ | 0.92 (0.74, 1.14) | 1.18 (0.92, 1.51) | 1.12 (0.87, 1.44) | 1.24 (0.94, 1.64) | 1.20 (0.93, 1.55) | 1.23 (0.93, 1.63) | 1.10 (0.82, 1.48) | 1.11 (0.82, 1.51) |
| NO-LT[2] | 1.08 (0.89, 1.31) | 1.29 (1.05, 1.59) | 1.24 (1.00, 1.53) | 1.39 (1.11, 1.74) | 1.30 (1.06, 1.60) | 1.37 (1.09, 1.72) | 1.30 (1.02, 1.66) | 1.31 (1.02, 1.67) |
| $NO_2$-LT | 0.98 (0.80, 1.20) | 1.19 (0.95, 1.50) | 1.14 (0.90, 1.43) | 1.17 (0.92, 1.50) | 1.19 (0.95, 1.50) | 1.22 (0.94, 1.57) | 1.08 (0.83, 1.40) | 1.08 (0.83, 1.40) |
| $NO_x$-LT | 1.06 (0.88, 1.28) | 1.26 (1.03, 1.54) | 1.20 (0.98, 1.47) | 1.34 (1.07, 1.67) | 1.27 (1.04, 1.55) | 1.33 (1.06, 1.67) | 1.23 (0.97, 1.57) | 1.24 (0.97, 1.57) |
| $O_3$ | 1.03 (0.79, 1.33) | 0.90 (0.68, 1.20) | 0.94 (0.70, 1.25) | 0.81 (0.59, 1.11) | 0.89 (0.67, 1.19) | 0.94 (0.68, 1.29) | 0.95 (0.67, 1.36) | 0.95 (0.66, 1.36) |
| $PM_{2.5}$ | 0.95 (0.82, 1.10) | 1.04 (0.88, 1.23) | 1.02 (0.86, 1.21) | 0.99 (0.83, 1.18) | 1.04 (0.88, 1.23) | 1.01 (0.84, 1.22) | 0.91 (0.75, 1.10) | 0.91 (0.75, 1.10) |

(1) Interquartile ranges (IQRs) for each pollutant were: NO=10.7 ppb; $NO_2$=6.1 ppb; $NO_x$=16.9 ppb; NO-LT=11.8 ppb; $NO_2$-LT=6.1; $NO_x$-LT=16.9 ppb; $O_3$=29.1 ppb; $PM_{2.5}$=2.4 µg/$m^3$.
(2) "LT" stands for "more local traffic impact" LUR model estimates.

**Table 21. Associations (Odds ratios[1], 95% CIs) between Annual Average Air Pollution Exposure Metrics (12 Mo prior to Interview) and Wheeze in the Past 12 Months Among L.A. FANS-2 Participants Ages 0–17 Years: Incorporating School Locations and Stratifying on Neighborhood Disadvantage**

| Pollutant | Two Pollutant Model[2] HOME ONLY | Two Pollutant Model[2] HOME+SCHOOL | Tract-level disadvantage[3] <0.13 (median) HOME ONLY *Higher SES* | Tract-level disadvantage[3] ≥0.13 HOME ONLY *Lower SES* | Tract-level disadvantage[3] <0.13 (median) HOME+SCHOOL | Tract-level disadvantage[3] ≥0.13 HOME+SCHOOL |
|---|---|---|---|---|---|---|
| NO-LT[4] | 1.31 (1.02, 1.68) | 1.42 (1.07, 1.87) | 1.41 (0.98, 2.02) | 1.40 (0.99, 1.98) | 1.63 (1.07, 2.49) | 1.46 (1.00, 2.12) |
| $O_3$ | 1.02 (0.69, 1.49) | 1.03 (0.69, 1.54) | 0.70 (0.42, 1.16) | 1.80 (0.99, 3.24) | 0.74 (0.43, 1.28) | 1.60 (0.87, 2.96) |
| $NO_2$-LT | 1.08 (0.83, 1.41) | 1.14 (0.85, 1.53) | 1.35 (0.89, 2.04) | 1.09 (0.80, 1.55) | 1.60 (0.98, 2.59) | 1.09 (0.75, 1.58) |
| $O_3$ | 0.95 (0.66, 1.37) | 0.94 (0.64, 1.39) | 0.62 (0.37, 1.06) | 1.51 (0.85, 2.70) | 0.62 (0.35, 1.09) | 1.33 (0.73, 2.42) |
| $NO_x$-LT | 1.24 (0.97, 1.58) | 1.34 (1.02, 1.75) | 1.39 (1.00, 1.94) | 1.30 (0.91, 1.85) | 1.60 (1.09, 2.36) | 1.33 (0.91, 1.96) |
| $O_3$ | 1.00 (0.69, 1.47) | 1.02 (0.68, 1.53) | 0.69 (0.41, 1.14) | 1.76 (0.96, 3.23) | 0.72 (0.42, 1.25) | 1.60 (0.84, 2.95) |

(1) Odds ratios are per interquartile range (IQR) increase in each pollutant: NO=10.7 ppb; $NO_2$=6.1 ppb; $NO_x$=16.9 ppb; NO-LT=11.8 ppb; $NO_2$-LT=6.1; $NO_x$-LT=16.9 ppb; $O_3$=29.1 ppb; $PM_{2.5}$=2.4 µg/$m^3$.
(2) Adjusting for age, race/ethnicity, sex, income, maternal asthma, overweight, no groups participated in past 12 months and tract-level disadvantage.
(3) Stratified models are two-pollutant models, i.e., LUR variables or $PM_{2.5}$ plus $O_3$ in each model. Stratified models do not include adjustment for tract-level disadvantage.

**Table 22. Associations (Odds ratios, 95% CIs) between Annual Average Air Pollution Exposure Metrics (12 Mos prior to Interview) and Medication Use for Asthma or Wheeze in the Past 12 Months Among L.A. FANS-2 Participants Ages 0–17 Years**

| Pollutant | Crude (per IQR increase)[1] | Model 1: Adjusting for age, race/ethnicity, sex, income | Model 1+Maternal Asthma | Model 1+ No. of Groups Participated in past 12 mos | Model 1+ Tract-level rating of neighborhood cohesion | Model 1+ Maternal Asthma, No. of Groups Participated in past 12 mos | Model 1+ Maternal Asthma, No. of Groups Participated in past 12 mos, Tract-level neighborhood cohesion |
|---|---|---|---|---|---|---|---|
| NO | 0.89 (0.72, 1.10) | 1.04 (0.81, 1.32) | 1.00 (0.78, 1.28) | 1.04 (0.79, 1.37) | 1.11 (0.86, 1.43) | 0.99 (0.75, 1.30) | 1.03 (0.78, 1.37) |
| $NO_2$ | 0.87 (0.71, 1.06) | 1.01 (0.80, 1.28) | 0.96 (0.76, 1.23) | 0.96 (0.74, 1.24) | 1.06 (0.83, 1.36) | 0.91 (0.70, 1.18) | 0.96 (0.73, 1.25) |
| $NO_x$ | 0.89 (0.72, 1.09) | 1.04 (0.82, 1.33) | 1.00 (0.78, 1.28) | 1.04 (0.80, 1.37) | 1.11 (0.87, 1.43) | 0.98 (0.75, 1.29) | 1.03 (0.78, 1.35) |
| NO-LT[2] | 1.04 (0.86, 1.25) | 1.15 (0.94, 1.41) | 1.11 (0.90, 1.36) | 1.18 (0.94, 1.48) | 1.20 (0.98, 1.48) | 1.12 (0.89, 1.41) | 1.14 (0.91, 1.44) |
| $NO_2$-LT | 0.94 (0.78, 1.14) | 1.08 (0.87, 1.34) | 1.03 (0.83, 1.29) | 1.03 (0.82, 1.30) | 1.12 (0.90, 1.40) | 0.97 (0.77, 1.23) | 1.02 (0.80, 1.30) |
| $NO_x$-LT | 1.03 (0.86, 1.24) | 1.14 (0.94, 1.39) | 1.10 (0.90, 1.34) | 1.17 (0.94, 1.45) | 1.19 (0.98, 1.45) | 1.10 (0.88, 1.37) | 1.13 (0.91, 1.41) |
| $O_3$ | 1.09 (0.85, 1.39) | 0.99 (0.76, 1.30) | 1.03 (0.78, 1.36) | 0.95 (0.71, 1.28) | 0.98 (0.74, 1.29) | 0.99 (0.73, 1.35) | 0.99 (0.73, 1.34) |
| $PM_{2.5}$ | 0.90 (0.78, 1.03) | 0.98 (0.84, 1.15) | 0.96 (0.82, 1.13) | 0.93 (0.79, 1.09) | 1.00 (0.85, 1.17) | 0.91 (0.77, 1.07) | 0.92 (0.78, 1.10) |

(1) Interquartile ranges (IQRs) for each pollutant were: NO=10.7 ppb; $NO_2$=6.1 ppb; $NO_x$=16.9 ppb; NO-LT=11.8 ppb; $NO_2$-LT=6.1; $NO_x$-LT=16.9 ppb; $O_3$=29.1 ppb; $PM_{2.5}$=2.4 μg/$m^3$.
(2) "LT" stands for "more local traffic impact" LUR model estimates.

**Table 23. Associations (Odds ratios[1], 95% CIs) between Annual Average Air Pollution Exposure Metrics (12 Mo prior to Interview) and Medication Use for Asthma or Wheeze in the Past 12 Months Among L.A. FANS-2 Participants Ages 0–17 Years: Incorporating School Locations and Stratifying on Neighborhood Disadvantage**

| Pollutant | Two Pollutant Model[2] HOME ONLY | Two Pollutant Model[2] HOME+SCHOOL | Tract-level disadvantage[3] <0.13 (median) HOME ONLY | Tract-level disadvantage[3] ≥0.13 HOME ONLY | Tract-level disadvantage[3] <0.13 (median) HOME+SCHOOL | Tract-level disadvantage[3] ≥0.13 HOME+SCHOOL |
|---|---|---|---|---|---|---|
| NO-LT[4] | 1.15 (0.91, 1.45) | 1.20 (0.93, 1.56) | 1.34 (0.94, 1.92) | 1.15 (0.84, 1.57) | 1.44 (0.96, 2.15) | 1.19 (0.85, 1.68) |
| $O_3$ | 1.03 (0.75, 1.41) | 1.01 (0.71, 1.42) | 0.59 (0.37, 0.93) | 2.25 (1.37, 3.68) | 0.54 (0.33, 0.89) | 2.30 (1.36, 3.88) |
| $NO_2$-LT | 1.02 (0.80, 1.30) | 1.03 (0.79, 1.34) | 1.35 (0.92, 1.97) | 1.02 (0.75, 1.38) | 1.45 (0.94, 2.22) | 1.02 (0.74, 1.42) |
| $O_3$ | 0.99 (0.73, 1.35) | 0.95 (0.69, 1.32) | 0.53 (0.33, 0.85) | 2.07 (1.27, 3.36) | 0.47 (0.28, 0.80) | 2.06 (1.23, 3.46) |
| $NO_x$-LT | 1.13 (0.91, 1.42) | 1.17 (0.91, 1.50) | 1.39 (1.00, 1.92) | 1.12 (0.82, 1.53) | 1.48 (1.02, 2.13) | 1.16 (0.82, 1.63) |
| $O_3$ | 1.03 (0.75, 1.41) | 1.00 (0.71, 1.41) | 0.58 (0.36, 0.91) | 2.24 (1.34, 3.74) | 0.53 (0.32, 0.88) | 2.30 (1.33, 3.97) |

(1) Odds ratios are per interquartile range (IQR) increase in each pollutant: NO=10.7 ppb; $NO_2$=6.1 ppb; $NO_x$=16.9 ppb; NO-LT=11.8 ppb; $NO_2$-LT=6.1; $NO_x$-LT=16.9 ppb; $O_3$=29.1 ppb; $PM_{2.5}$=2.4 μg/$m^3$.
(2) Adjusting for age, race/ethnicity, sex, income, maternal asthma, no groups participated in past 12 months and census tract-level neighborhood cohesion.
(3) Stratified models are two-pollutant models, i.e., LUR variables or $PM_{2.5}$ plus $O_3$ in each model. Stratified models do not include adjustment for census tract-level neighborhood cohesion.
(4) "LT" stands for "more local traffic impact" LUR model estimates.

**Table 24. Associations (Odds ratios, 95% CIs) between Annual Average Air Pollution Exposure Metrics (12 Mos prior to Interview) and Doctor-Diagnosed Asthma Among L.A. FANS-2 Participants Ages 0–17 Years**

| Pollutant | Crude (per IQR increase)[1] | Model 1: Adjusting for age, race/ethnicity, sex, income | Model 1+Maternal Asthma | Model 1+ No. of Relatives in Neighborhood | Model 1+ Tract-level neighborhood safety | Model 1+ Maternal Asthma, No. of Relatives in Neighborhood | Model 1+ Maternal Asthma, No. of Relatives in Neighborhood, Tract-level neighborhood safety |
|---|---|---|---|---|---|---|---|
| NO | 0.92 (0.75, 1.12) | 1.05 (0.84, 1.32) | 1.01 (0.80. 1.27) | 1.13 (0.88, 1.44) | 1.10 (0.87, 1.38) | 1.06 (0.83, 1.36) | 1.07 (0.83, 1.39) |
| $NO_2$ | 0.95 (0.79, 1.16) | 1.11 (0.88, 1.38) | 1.05 (0.84, 1.33) | 1.16 (0.91, 1.47) | 1.13 (0.90, 1.42) | 1.09 (0.85, 1.40) | 1.10 (0.85, 1.41) |
| $NO_x$ | 0.93 (0.77, 1.13) | 1.08 (0.86, 1.35) | 1.03 (0.82, 1.29) | 1.16 (0.91, 1.48) | 1.12 (0.89, 1.41) | 1.09 (0.85, 1.40) | 1.10 (0.85, 1.41) |
| NO-LT[2] | 1.01 (0.85, 1.21) | 1.12 (0.93, 1.36) | 1.08 (0.89, 1.31) | 1.19 (0.97, 1.46) | 1.16 (0.96, 1.41) | 1.13 (0.91, 1.39) | 1.14 (0.92, 1.41) |
| $NO_2$-LT | 1.02 (0.85, 1.22) | 1.16 (0.95, 1.42) | 1.11 (0.90, 1.37) | 1.20 (0.97, 1.50) | 1.18 (0.96, 1.44) | 1.14 (0.91, 1.42) | 1.14 (0.91, 1.43) |
| $NO_x$-LT | 1.03 (0.87, 1.22) | 1.14 (0.95, 1.37) | 1.09 (0.90, 1.31) | 1.21 (0.99, 1.47) | 1.17 (0.98, 1.42) | 1.14 (0.93, 1.39) | 1.15 (0.95, 1.41) |
| $O_3$ | 0.99 (0.79, 1.25) | 0.90 (0.70, 1.16) | 0.94 (0.73, 1.22) | 0.82 (0.62, 1.09) | 0.86 (0.66, 1.12) | 0.86 (0.64, 1.15) | 0.86 (0.65, 1.15) |
| $PM_{2.5}$ | 0.98 (0.85, 1.12) | 1.06 (0.90, 1.23) | 1.04 (0.89, 1.22) | 1.08 (0.91, 1.28) | 1.08 (0.92, 1.27) | 1.07 (0.90, 1.27) | 1.05 (0.89, 1.25) |

(1) Interquartile ranges (IQRs) for each pollutant were: NO=10.7 ppb; $NO_2$=6.1 ppb; $NO_x$=16.9 ppb; NO-LT=11.8 ppb; $NO_2$-LT=6.1; $NO_x$-LT=16.9 ppb; $O_3$=29.1 ppb; $PM_{2.5}$=2.4 μg/$m^3$.
(2) "LT" stands for "more local traffic impact" LUR model estimates.

**Table 25. Associations (Odds ratios[1], 95% CIs) between Annual Average Air Pollution Exposure Metrics (12 Mo prior to Interview) and Doctor-Diagnosed Asthma Among L.A. FANS-2 Participants Ages 0–17 Years: Incorporating School Locations and Stratifying on Neighborhood Disadvantage**

| Pollutant | Two Pollutant Model[2] HOME ONLY | Two Pollutant Model[2] HOME+SCHOOL | Tract-level disadvantage[3] <0.13 (median) HOME ONLY *Higher SES* | Tract-level disadvantage[3] ≥0.13 HOME ONLY *Lower SES* | Tract-level disadvantage[3] <0.13 (median) HOME+SCHOOL | Tract-level disadvantage[3] ≥0.13 HOME+SCHOOL |
|---|---|---|---|---|---|---|
| NO-LT[4] | 1.12 (0.90, 1.40) | 1.13 (0.89, 1.45) | 1.39 (0.98, 1.96) | 1.05 (0.78, 1.41) | 1.46 (1.00, 2.15) | 1.07 (0.77, 1.49) |
| $O_3$ | 0.89 (0.66, 1.19) | 0.87 (0.64, 1.20) | 0.58 (0.38, 0.88) | 1.46 (0.92, 2.31) | 0.55 (0.35, 0.86) | 1.47 (0.90, 2.41) |
| $NO_2$-LT | 1.14 (0.91, 1.44) | 1.12 (0.88, 1.44) | 1.45 (1.01, 2.10) | 1.16 (0.86, 1.55) | 1.54 (1.02, 2.30) | 1.14 (0.83, 1.56) |
| $O_3$ | 0.86 (0.64, 1.16) | 0.85 (0.62, 1.16) | 0.51 (0.32, 0.80) | 1.55 (0.98, 2.45) | 0.48, (0.30, 0.77) | 1.53 (0.94, 2.50) |
| $NO_x$-LT | 1.13 (0.92, 1.40) | 1.13 (0.90, 1.43) | 1.44 (1.04, 1.97) | 1.05 (0.78, 1.42) | 1.50 (1.05, 2.14) | 1.07 (0.77, 1.49) |
| $O_3$ | 0.89 (0.66, 1.20) | 0.88 (0.64, 1.20) | 0.57 (0.37, 0.87) | 1.47 (0.91, 2.38) | 0.54 (0.34, 0.85) | 1.49 (0.89, 2.49) |

(1) Odds ratios are per interquartile range (IQR) increase in each pollutant: NO=10.7 ppb; $NO_2$=6.1 ppb; $NO_x$=16.9 ppb; NO-LT=11.8 ppb; $NO_2$-LT=6.1; $NO_x$-LT=16.9 ppb; $O_3$=29.1 ppb; $PM_{2.5}$=2.4 μg/$m^3$.
(2) Adjusting for age, race/ethnicity, sex, income, maternal asthma, no. relatives in neighborhood and census tract-level neighborhood safety.
(3) Stratified models are two-pollutant models, i.e., LUR variables or $PM_{2.5}$ plus $O_3$ in each model. Stratified models do not include adjustment for census tract-level neighborhood safety.
(4) "LT" stands for "more local traffic impact" LUR model estimates.

**Table 26. Mean (SD) Lung Function in L.A. FANS-2 Participants Ages 5-17 Years**

| Lung Function | Boys (1 or More Acceptable Curves, n=486) | Boys (3 Acceptable and Reproducible Curves, n=221) | Girls (1 or More Acceptable Curves, n=404) | Girls (3 Acceptable and Reproducible Curves, n=174) |
|---|---|---|---|---|
| $FEV_1$ (mL) | 2629 (1036) | 2730 (1018) | 2369 (744) | 2500 (616) |
| FVC (mL) | 3193 (1231) | 3267 (1198) | 2800 (861) | 2958 (733) |
| PEF (mL/s) | 5758 (2291) | 6039 (2130) | 5347 (1947) | 5712 (1552) |
| $FEF_{75}$ (mL/s) | 1455 (818) | 1491 (793) | 1409 (707) | 1419 (633) |
| $FEF_{25-75}$ (mL/s) | 2807 (1332) | 2940 (1263) | 2695 (1069) | 2828 (932) |

**Table 27. Summary of Acceptable and Reproducible Spirometry Curves by Age Group (N, percent)**

| Age group | 1 acceptable curve | 2 acceptable curves, not reproducible | 2 acceptable and reproducible curves | 3 acceptable curves, not reproducible | 3 acceptable and reproducible curves |
|---|---|---|---|---|---|
| 5-<10 years[1] | 46 (21) | 24 (11) | 62 (28) | 12 (5) | 78 (35) |
| 10-<15 years | 41 (10) | 40 (10) | 94 (22) | 50 (12) | 190 (46) |
| ≥15 years | 30 (12) | 22 (9) | 46 (18) | 28 (11) | 127 (50) |
| All ages[2] | 117 (13) | 86 (10) | 202 (23) | 90 (10) | 395 (44) |

(1) Percents are based on all children of given age group with one or more acceptable curves (i.e., 222 for 5-<10 years, 415 for 10-<15 years and 253 for ≥15 years).
(2) Percents are based on all children with one or more acceptable curves (n=890).

**Table 28. Associations (Betas[1], 95% CIs) between Annual Average Air Pollution Exposure Metrics (Current Home) and Lung Function Among L.A. FANS-2 Participants, Boys Ages 5–17 Years**

| Lung Function | Pollutant | 1 OR MORE ACCEPTABLE CURVES (n=486) | | | | 3 ACCEPTABLE AND REPRODUCIBLE CURVES (n=221) | | | |
|---|---|---|---|---|---|---|---|---|---|
| | | Adjusting for age, race/ethnicity, height, height$^2$, overweight | Final Model[2] HOME ONLY | Two-Pollutant Model[3] HOME ONLY | Two-Pollutant Model[3] HOME+SCHOOL | Adjusting for age, race/ethnicity, height, height$^2$, overweight | Final Model[2] HOME ONLY | Two-Pollutant Model[3] HOME ONLY | Two-Pollutant Model[3] HOME+SCHOOL |
| **$FEV_1$ (mL)** | | | | | | | | | |
| | NO | -63 (-119, -8) | -75 (-144, -6) | -69 (-141, 2) | -97(-176, -18) | -70 (-148, 7) | -45 (-147, 56) | -48 (-146, 50) | -44 (-149, 60) |
| | $_2$ | -60 (-113, -7) | -75 (-137 ,-14) | -75 (-138, -12) | -72 (-138, -6) | -60 (-129, 8) | -78 (-157, 0.5) | -73 (-151, 5) | -67 (-149, 16) |
| | $_x$ | -73 (-128, -17) | -91 (-159, -24) | -90 (-162, -18) | -100 (-177, -22) | -67 (-142, 8) | -54 (-150, 43) | -63 (-157, 30) | -64 (-164, 36) |
| | $_{2.5}$ | -33 (-71, 5) | -52 (-98, -7) | -50 (-99, -2) | -46 (-95, 3) | -47 (-96, 3) | -62 (-121, -4) | -50 (-111, 11) | -55 (-114, 5) |
| **FVC (mL)** NO | | | | | | | | | |
| NO PM | NO | -52 (-119, 15) | -60 (-141, 22) | -56 (-141, 28) | -86 (-179, 7) | -46 (-134, 42) | -43 (-164, 78) | -50 (-170, 70) | -80 (-205, 45) |
| | $NO_2$ | -46 (-111, 18) | -67 (-139, 4) | -66 (-139, 7) | -70 (-147, 6) | -56 (-136, 25) | -80 (-175, 16) | -75 (-171, 22) | -76 (-176, 24) |
| | $NO_x$ | -56 (-123, 12) | -76 (-155, 4) | -75 (-159, 9) | -95 (-184, -5) | -62 (-150, 25) | -50 (-165, 65) | -59 (-175, 56) | -78 (-199, 43) |
| | $PM_{2.5}$ | -28 (-73, 17) | -45 (-97, 6) | -47 (-102, 8) | -44 (-99, 11) | -49 (-107, 9) | -56 (-126, 14) | -48 (-121, 26) | -58 (-129, 14) |
| | | | | | | | | | |
| **$FEF_{75}$ (mL/s)** | | | | | | | | | |
| | NO | -62 (-126, 2) | -39 (-119, 40) | -39 (-122 43) | -58 (-150, 35) | -39 (-133, 54) | -30 (-154, 93) | -11 (-135, 113) | 20 (-113, 153) |
| | $NO_2$ | -67 (-128, -5) | -63 (-133, 7) | -64 (-135, 8) | -74 (-150, 1) | -23 (-107, 62) | -75 (-176, 26) | -63 (-164, 38) | -54 (-159, 51) |
| | $NO_x$ | -67 (-131, -2) | -60 (-137, 18) | -62 (–144, 19) | -77 (-167, 12) | -37 (-128, 54) | -52 (-171, 67) | -38 (-158, 83) | -20 (-148, 108) |
| | $PM_{2.5}$ | -47 (-91, -2) | -56 (-108, -4) | -62 (-117, -7) | -62 (-119, -5) | -34 (-95, 26) | -103 (-174, -33) | -68 (-143, 8) | -78 (-154, -3) |
| | | | | | | | | | |
| **$FEF_{25-75}$ (mL/s)** | | | | | | | | | |
| | NO | -105 (-206, -4) | -65 (-187, 57) | -55 (-181, 72) | -85 (-225, 54) | -129 (-282, 25) | -45 (-250, 159) | -61 (-257, 135) | -27 (-231, 178) |
| | $NO_2$ | -93 (–188, 2) | -104 (-212, 3) | -99 (-208, 10) | -115 (-229, -0.4) | -91 (-227, 44) | -137 (-297, 23) | -114 (-272, 45) | -114 (-275, 48) |
| | $_x$ | -108 (-208, -9) | -101 (-220, 18) | -93 (-218, 31) | -116 (-251, 18) | -151 (-301, -2) | -99 (-289, 91) | -101 (-289, 88) | -83 (-278, 112) |
| | $PM_{2.5}$ | -53 (-123, 16) | -90 (-170, -10) | -89 (-174, -4) | -92 (-177, -7) | -67 (-164, 30) | -139 (-251, -26) | -112 (-228, 4) | -122 (-237, -6) |

(1) Interquartile ranges (IQRs) for each pollutant were: NO=10.9 ppb; $NO_2$=5.9 ppb; $NO_x$=17.1 ppb; $PM_{2.5}$=2.4 μg/$m^3$.

(2) Adjusting for age, race/ethnicity, height, height$^2$, overweight, income, no usual source of sick care, maternal asthma, furry pets in home, PCG smoking status, PCG education level, no. of neighbors talked to for 10 min in past 30 days, and census tract-level disadvantage.

NO

(3) LUR variables or $PM_{2.5}$ plus $O_3$ in each model.

**Table 29. Associations (Betas[1], 95% CIs) between Annual Average Air Pollution Exposure Metrics (5-Years Prior to Interview) and Lung Function Among L.A. FANS-2 Participants, Girls Ages 5–17 Years**

| Lung Function | Pollutant | 1 OR MORE ACCEPTABLE CURVES (n=404) | | | |
|---|---|---|---|---|---|
| | | Adjusting for age, race/ethnicity, height, height$^2$, weight, weight$^2$, overweight | Final Model[2] HOME ONLY | Two-Pollutant Model[3] HOME ONLY | Two-Pollutant Model[3] HOME+SCHOOL |
| **$FEV_1$** | | | | | |
| | NO | -69 (-125, -14) | -74 (-143, -5) | -59 (-128, 9) | -43 (-131, 45) |
| | $_2$ | -52 (-106, 3) | -81 (-142, -19) | -66 (-127, -4) | -65 (-137, 7) |
| | $_x$ | -63 (-118, -7) | -79 (-145, -14) | -66 (-133, 0.7) | -78 (-157, 1) |
| | $_{2.5}$ | -33 (-73, 7) | -40 (-86, 7) | -42 (-90, 5) | -30 (-83, 24) |
| **FVC** NO | | | | | |
| NO | NO | -38 (-100, 23) | -2 (-75, 72) | 10 (-65, 85) | 17 (-79, 113) |
| PM | $NO_2$ | -21 (-80, 38) | -5 (-73, 63) | 19 (-49, 87) | 8 (-70, 86) |
| | $NO_x$ | -38 (-98, 22) | -4 (-74, 67) | 9 9-64, 81) | -23 (-111, 65) |
| | $PM_{2.5}$ | -24 (-67, 18) | -20 (-68, 27) | 0.3 (-52, 52) | -5 (-62, 52) |
| | | | | | |
| **PEF** | NO | -129 (-290, 32) | -123 (-326, 79) | -178 (-383, 28) | -322 (-572, -72) |
| | $NO_2$ | -183 (-333, -32) | -204 (-387, -21) | -260 (-439, -81) | -332 (-532, -132) |
| | $NO_x$ | -158 (-314, -1) | -155 (-347, 38) | -229 (-428, -31) | -355 (-585, -125) |
| | $PM_{2.5}$ | 29 (-85, 142) | -49 (-179, 80) | -69 (-215, 77) | -64 (-218, 91) |
| | | | | | |
| **$FEF_{75}$** | NO | -52 (-133, 30) | -94 (-186, -1) | -79 (-170, 12) | -89 (-200, 22) |
| | $NO_2$ | -18 (-95, 58) | -48 (-132, 36) | -46 (-126, 33) | -62 (-155, 30) |
| | $NO_x$ | -37 (-116, 42) | -80 (-168, 7) | -70 (-157, 18) | -82 (-185, 22) |
| | $PM_{2.5}$ | -2 (-62, 57) | -13 (-74, 48) | -9 (-73, 54) | -36 (-109, 36) |
| | | | | | |
| **$FEF_{25-75}$** | NO | -157 (-279, -36) | -187 (-324, -50) | -202 (-340, -63) | -279 (-448, -110) |
| | $NO_2$ | -103 (-220, 13) | -171 (-297, -44) | -150 (-278, -22) | -207 (-356, -59) |
| | $NO_x$ | -151 (-269, -33) | -173 (-302, -45) | -220 (-354, -85) | -287 (-447, -127) |
| | $PM_{2.5}$ | -45 (-131, 42) | -43 (-139, 53) | -35 (-140, 71) | -86 (-202, 29) |

(1) Interquartile ranges (IQRs) for each pollutant were: NO=10.9 ppb; $NO_2$=5.8 ppb; $NO_x$=16.7 ppb; $PM_{2.5}$=2.4 µg/$m^3$.
(2) Adjusting for age, race/ethnicity, height, height$^2$, weight, weight$^2$, overweight, income, no usual source of sick care, maternal asthma, furry pets in home, PCG smoking status, foreign born status of PCG, group participation in past 12 months, no. of adults recogonize in neighborhood, and census tract-level disadvantage.
(3) LUR variables or $PM_{2.5}$ plus $O_3$ in each model.

**Table 30. Associations (Betas[1], 95% CIs) between Annual Average Peak 8-Hour Ozone (Current Home) and Lung Function Among L.A. FANS-2 Participants, Girls Ages 5–17 Years**

| Lung Function | Adjusting for age, race/ethnicity, height, height$^2$, weight, weight$^2$, overweight | Final Model[2] HOME ONLY | Two-Pollutant Model[3] HOME ONLY | Two-Pollutant Model[3] HOME+SCHOOL |
|---|---|---|---|---|
| **Girls with 1 or More Acceptable Curves (n=404)** | | | | |
| **PEF** | 41 (-120, 202) | -85 (-288, 118) | -135 (-333, 63) | -123 (-323, 76) |
| **Girls with 3 Acceptable and Reproducible Curves (n=174)** | | | | |
| **PEF** | -183 (-381, 15) | -398 (-641, -155) | -435 (-681, -188) | -420 (-668, -171) |

(1) Betas are per IQR increase of 29.9 ppb $O_3$.

(2) Adjusting for age, race/ethnicity, height, height$^2$, weight, weight$^2$, overweight, income, no usual source of sick care, maternal asthma, furry pets in home, PCG smoking status, foreign born status of PCG, group participation in past 12 months, no. of adults recogonize in neighborhood, and census tract-level disadvantage.

(3) Two-pollutant model presented is $O_3$ plus $NO_2$; $O_3$ effect estimates adjusting for NO, $NO_x$ and $PM_{2.5}$ were very similar.

## VIII. LIST OF PUBLICATIONS

Su J, Jerrett M, Beckerman B, Wilhelm M, Ghosh JK, Ritz B. Predicting traffic-related air pollution in Los Angeles using a distance decay regression selection strategy. In Review: Environmental Research.

## IX. GLOSSARY

µg – microgram
AADT – Annual Average Daily Traffic
ADDRESS – A Distance Decay REgression Selection Strategy
ATS - American Thoracic Society
BC – black carbon
BLUE - best linear unbiased estimate
BMI - body mass index
CALINE -- California Line Source Air Dispersion Model
CHS – Children's Health Study
CI – confidence interval
Coef – coefficient
CT – census tract
DEM – digital elevation model
DEP – diesel exhaust particulate
EC – elemental carbon
ECAT – elemental carbon attributable to traffic sources
EHC – Event History Calendar
EPA –Environmental Protection Agency
ETM+ - Landsat Enhanced Thematic Mapper Plus
FACES - Fresno Asthmatic Children's Environment Study
$FEF_{25-75}$ – forced expiratory mean flow between 25% and 75% of FVC
$FEF_{75}$ – forced expiratory mean flow at 75% of FVC
$FEV_1$ – forced expiratory volume after 1 second
FVC – forced vital capacity
GEE – generalized estimating equations
GIS – Geographic Information System
GPS – global positioning system
HCHO – formaldehyde
HCl – hydrochloric acid
HDL – high-density lipoprotein
$HNO_3$ – nitric acid
HPA – hypothalamic-pituitary-adrenal
HPMS – Highway Performance Monitoring System
ICC - intraclass correlation coefficient
IgE – immunoglobulin E
IQR – inter-quartile range
ISAAC - International Study of Asthma and Allergies in Childhood
km – kilometer
L.A. FANS – Los Angeles Family and Neighborhood Study
L.A. FANS-1 -- Los Angeles Family and Neighborhood Study, Wave One Survey

L.A. FANS-2 -- Los Angeles Family and Neighborhood Study, Wave Two Survey
LT – low traffic model
LUR – land use regression
m – meter
mL – milliliter
mL/s – milliliter per second
MME - normalized mean error
MMEF – maximum midexpiratory flow
MPO – Metropolitan Planning Organization
NMB - normalized mean bias
NO – nitric oxide
$NO_2$ – nitrogen dioxide
$NO_x$ – nitrogen oxides
$O_3$ – ozone (gas)
OC – organic carbon
OR – odds ratio
PAH – polycyclic aromatic hydrocarbons
PCG – primary caregiver
PEF – peak expiratory flow
$PM_{10}$ – particulate matter less than 10 μm in aerodynamic diameter
$PM_{2.5}$ – particulate matter less than 2.5 μm in aerodynamic diameter
ppb – parts per billion
ROS - reactive oxygen species
RSA - Randomly Selected Adult
RSC - Randomly Selected Child
RTI – Research Triangle Institute
SCAG – Southern California Association of Governments
SCAQMD - South Coast Air Quality Management District
SD – standard deviation
SES – socioeconomic status
SIB – sibling of the Randomly Selected Child
$SO_2$ – sulfer dioxide
Std Err – standard error
TRAPCA – Traffic-Related Air Pollution and Childhood Asthma
TSP – total suspended particles
UF – particles less than 0.1 μm in aerodynamic diameter
UTM – Universal Transverse Mercator
VIF – variance inflation factor
VMT – vehicle miles traveled
WGS84 - World Geodetic System of 1984

## X. APPENDICES

Appendix A – Example Field Log Sheet – Site Description
Appendix B – Example Field Log Sheet – Installation and Collection Times, GPS Coordinates
Appendix C – Final Report for Contract 05-311 and Contract 05-312, Spirometry Training and Grading of Spirometry Test Results in conjunction with Wave Two of the Los Angeles Family and Neighborhood Survey (L.A. FANS)
Appendix D – L.A. FANS-2 Spirometry Protocol

# Traffic-Related Air Pollution and Asthma in Economically Disadvantaged and High Traffic Density Neighborhoods in Los Angeles County, California

**Final Report - APPENDICES**
**June 12, 2009**

**ARB Contract No. 04-323**

# Appendix A
# Example Field Log Sheet – Site Description

**Log Sheet and Protocol for Ogawa Sampling**

## I. SITE INFORMATION

| | |
|---|---|
| Name of Field Monitor(s): | |
| Site ID# | |
| Shelter ID#: | |

## II. LAND USE: Please check one

| | |
|---|---|
| | **Commercial** (Store, Restaurant, Mall) |
| | **Office Building** (Business, Lawyer, Doctor, Dentist) |
| | **Residential** (Houses, Apartments, Trailer Parks) |
| | **Open Space** (Parks, Undeveloped Land, Water bodies |
| | **Government** (Hospitals, Government Offices, Schools, Courthouses) |
| | **Industrial** (Factories, Power Plants, Waterhouses, Utility Plants, Land Fill) |
| | **Parking Lots** |
| | **Transportation Centers** (Bus Stations, Train Stations, Airport) |
| | **Other** |

BRIEF SITE DESCRIPTION**:**

**Take several (2-3) pictures to show the relative location and help us find the location at the next visit ("shelter view"). Please show the site information (first box above) in the first picture you take.**

## III. SAMPLING PROTOCOL

| Step | Task | Status |
|---|---|---|
| **1** | Identify a site to mount the sampler/shelter. Mounting options include a fence, post, downspout from rain gutter, tree limb, etc. | Complete? (Please circle)<br><br>YES |
| **2** | Briefly describe the location selected for mounting | |
| **3** | Is the location away from busy driveways, garages, parking lots and other obvious sources of vehicle exhaust? | YES |
| **4** | Get one shelter and secure the cover using cable ties. If necessary, use multiple cable ties to secure the shelter. | Complete? (Please circle)<br><br>YES |
| **5** | Are the cable ties pulled **TIGHTLY** so that the samplers will stay in place for two weeks? | YES |
| **6** | Is the shelter completely flat to ensure that rain/water does not enter? | YES |
| **7** | Measure and record the height from the ground to the bottom of the shelter. **Aim for approximately 9ft or 108 inches.** | _____ft. _____in. |
| **8** | Warm up GPS for 5 minutes before taking the reading to make sure we get a good satellite signal. | Complete? (Please circle)<br><br>YES |
| **9** | Is the GPS recording in DEGREES? | YES |
| **10** | Is the NORTH AMERICAN DATUM 83 (NAD 83) being used? | YES |
| **11** | Have one team member take a reading and record Lat, Long, and Accuracy | Longitude: _____<br><br>Latitude:_____<br><br>Accuracy _____ |
| **12** | Have a second team member take a reading and record Lat, Long and Accuracy | Longitude: _____<br><br>Latitude:_____<br><br>Accuracy _____ |
| **13** | Take a third reading, called a WAYPOINT | Longitude: _____<br><br>Latitude:_____<br><br>Accuracy _____ |
| **14** | Take picture of a piece of paper with the Site ID # for this site. | Complete? (Please circle)<br><br>YES |
| **15** | Take 4 –6 photos to show complete 360 degree angle view from the shelter (“land view”) | Complete? (Please circle)<br><br>YES |

**Once you return to the office, please save your photos to CD-ROM and rename the picture files as "site ID_Land1", "site ID_Land2", etc. for land view in section III and "site ID_shelter1", "site ID_shelter2"for shelter view in section II (ie, for site ID=001, the first land view picture should be named "001_Land1")**

## IV. ADDITIONAL FIELD MONITORING NOTES:

## Appendix B
## Example Field Log Sheet – Installation and Collection Times, GPS Coordinates

| | | | |
|---|---|---|---|
| | | | |
| **Name(s) of person installing** | | | |
| **Date of installation** | **9/16/2006** | **Day of week** | **Saturday** |

| Site ID | Time of installation | Height | Need pictures? | All pictures taken? | Same site? | Latitude (1) | Longitude (1) | Accuracy (1) | Latitude (2) | Longitude (2) | Accuracy (2) |
|---|---|---|---|---|---|---|---|---|---|---|---|
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| Name(s) of person removing | | | |
|---|---|---|---|
| **Date of removal** | 9/30/2006 | **Day of the week** | Saturday |

| | | Time window for removal | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| **Site ID** | **Time installed** | **Earliest allowable time** | **Latest allowable time** | **Time monitor removed** | **Latitude (1)** | **Longitude (1)** | **Acc (1)** | **Latitude (2)** | **Longitude (2)** | **Acc (2)** |
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**Appendix C**
**Final Report for Contract 05-311 and Contract 05-312, Spirometry Training and Grading of Spirometry Test Results in conjunction with Wave Two of the Los Angeles Family and Neighborhood Survey (L.A. FANS)**

# Spirometry Training and Grading of Spirometry Test Results in conjunction with Wave Two of the Los Angeles Family and Neighborhood Survey (L.A. FANS)

**Final Report**
**April 29, 2009**

**Submitted by:**
**Dr. Kathleen Mortimer and Mr. Lucas Carlton**

**Background:**

A large literature links outdoor air pollution exposure to adverse respiratory health effects in children and adults.[1-7] Ozone ($O_3$) and particulate matter less than 10 and 2.5 microns in aerodynamic diameter ($PM_{10}$ and $PM_{2.5}$) are the pollutants that have been most consistently linked with adverse respiratory health, particularly in asthmatics. Children are likely to be particularly vulnerable to air pollution impacts due to the large volume of air inhaled each day and subsequent delivery of substantial pollutant doses to the respiratory tract; they also typically spend more time than adults exercising outdoors.[8] A growing literature links outdoor air pollution exposure to worse asthma morbidity in children.[3-10] Although the link between air pollution and exacerbation of existing illness is well-established, recent evidence has also pointed to the potential importance of air pollution exposure in the development of chronic disease.[4-5,7] Existing studies have reported associations between $PM_{10}$, $O_3$ and $NO_2$ and reductions in lung function, slowed lung growth, chronic cough and bronchitis.[11-23] Recently, focus has turned to potential adverse respiratory effects caused by exposure to specific motor vehicle exhaust components such as polycyclic aromatic hydrocarbons (PAHs) sorbed to particles from diesel engines and ultrafine particles (less than 0.1 microns in aerodynamic diameter), which can penetrate deep into the lung.[24-26] A series of recent studies (mostly in Europe) linked various measures of traffic exhaust exposure (community-level $NO_2$, home outdoor $NO_2$, residential and school proximity to traffic) to asthma prevalence, atopy, and wheezing.[27-31] In a recent school-based study, Kim et al.[32] reported associations between current asthma in Californian 3$^{rd}$ to 5$^{th}$ graders and measured concentrations of traffic-related pollutants (black carbon (BC), nitrogen oxides ($NO_x$) and nitrogen oxide (NO)). Although children are often noted as a particularly susceptible population, there is wide evidence that adults (especially asthmatics) are impacted by exposure to ambient air pollution, and recent work has also pointed towards traffic-related air pollutants in particular as an area of concern for adult health impacts.[33-48]

Although existing air monitoring networks provide a reasonable surrogate measure of long-term exposure to pollutants that are relatively homogeneously distributed within communities, this may not be the case for primary traffic-related pollutants – such as diesel exhaust particulate – which show strong spatial gradients.[7] There is currently a lack of neighborhood and individual level air pollution measurements for Californians that live in high traffic density areas and who may be more susceptible to adverse health impacts from air pollution exposure due to economic disadvantage. Although efforts have been and are being made to develop reliable models to assess exposures at a finer spatial scale, additional measurements in Los Angeles County communities with varying amounts of socioeconomic disadvantage and major air pollution sources would help inform and validate these models. Thus, Dr. Beate Ritz (UCLA) received funding from the California Air Resources Board (CARB) for a project titled "Traffic-Related Air Pollution and Asthma in Economically Disadvantaged and High Traffic Density Neighborhoods in Los Angeles, California". The objectives of this project are: (1) to conduct $NO_x$ and $NO_2$ monitoring at 200 locations within LA County neighborhoods with varying levels of economic disadvantage and varying exposures to air pollution originating from vehicular sources; (2) to use these monitoring data to help inform land use-based regression (LUR) models developed to predict traffic pollutant – i.e., $NO_x$, NO and $NO_2$ – exposures; (3) to use geostatistical models to estimate regional background concentrations of $O_3$ and $PM_{2.5}$; (4) to evaluate associations between exposure to $NO_x$, NO and $NO_2$ (as estimated by the developed

LUR models) and measures of lung function and asthma prevalence, exacerbation and possibly incidence in children ages 0-17 years in conjunction with the Los Angeles Family and Neighborhood Survey (L.A. FANS) study;[49] and (5) to evaluate whether concentrations of the more regionally distributed background pollutants ($O_3$ and $PM_{2.5}$) confound or modify the effects of exposure to the more heterogeneously distributed traffic-related pollutants ($NO_x$, NO and $NO_2$) on lung function and asthma. Because the L.A. FANS study is already established, includes follow-up of a cohort, by design focuses on disadvantaged neighborhoods and children, performs at-home interviews, and collects extensive data on neighborhood characteristics, including access to health care and neighborhood perception, it provides a unique opportunity for evaluating associations between air pollution and asthma. This report summarizes the work of Lucas Carlton to train L.A. FANS interviewers in collection of lung function measurements in conjunction with the UCLA project. It also summarizes methods used to review and grade collected spirometry data to provide feedback to field interviewers on where improvements were needed in data collection. The grading will also be used to determine which spirometry maneuvers are valid for use in statistical analyses.

The Los Angeles Family and Neighborhood Survey (L.A.FANS) is a longitudinal study of families in Los Angeles County and of the neighborhoods in which they live. The study is specifically designed to answer key research and policy questions in several areas, with a focus on understanding neighborhood, family, and peer effects on children's development and well-being. The first wave of data collection (L.A.FANS-1) was a field survey of 3,090 households conducted from April 2000 to January 2002. L.A.FANS-2 is a continuation of this study and is funded by grants from NICHD, NIA and NIEHS. It is a collaboration of three institutions: RAND, UCLA, and Research Triangle Institute (RTI). The protocol for L.A.FANS-2 is to re-interview all respondents from L.A.FANS-1 and to add a new sample of residents who have moved into each neighborhood between the two waves. L.A.FANS-2 was also expanded to collect objective physiological health measures or "biomarkers" from approximately 1,600 respondents. During the planning process for L.A.FANS-2, it was determined that the proposed approach for lung function testing (which is one of the physiologic measures being collected) in children could be greatly improved by using portable spirometers instead of peak flow meters as originally proposed.

Although the use of peak flow measurements to assess lung function has been advocated by the National Asthma Education and Prevention Program, the value of such measurements is limited because PEFR is effort dependent and reflects only flows of the large airways.[50] Furthermore, existing studies indicate peak flow meter recordings are not highly reproducible and appear to be no better at predicting asthma exacerbations than monitoring asthma symptoms alone.[50-54] Specifically, PEFR measures appear to be no better at predicting asthma than standard questions regarding doctor diagnoses and symptoms asked on questionnaires. Portable spirometers offer an advantage over peak flow meters because these instruments can measure a wide range of pulmonary function parameters (forced vital capacity (FVC), forced expiratory volume after 1 second ($FEV_1$), forced expiratory mean flow between 25% and 75% of FVC ($FEF_{25-75}$), and forced expiratory mean flow at 75% of FVC ($FEF_{75}$)) which reflect conditions in both small and large airways and are more sensitive to changes in functional status in asthma.[50,55] Most studies of air pollution and asthma reported statistically significant but clinically small effects on PEFR and $FEV_1$.[12] Therefore, more recent work – such as the Fresno Asthmatic Children's Environment Study (FACES) – focused on measures such as $FEF_{25-75}$ and $FEF_{75}$ that may be more sensitive indicators of air pollution health effects. The University of Southern

California's (USC's) Children's Health Study (CHS) focuses on asthma and lung development in 4th through 10th graders living in 12 Southern Californian communities and has reported larger percentage effects of air pollution on lung growth based on $FEF_{25-75}$ and $FEF_{75}$ measures compared to PEFR and $FEV_1$.[56] Finally, use of spirometry allows for comparisons to other studies focused on childhood asthma, such as the CHS, FACES and the CDC-funded Oakland Kicks Asthma (OKA) project. Thus, the Principal Investigators for L.A. FANS (Dr. Anne Pebley (UCLA) and Dr. Narayan Sastry (RAND/University of Michigan)) applied for and received additional NIEHS funding to use portable spirometers to measure lung function instead of peak flow meters.

The EasyOne Frontline Spirometer from ndd Medical Technologies was selected for the LA FANS Wave-2 field work (http://www.ndd.ch/English/Products/EasyOne_fs.html). The key features of this instrument that make it well suited for the study are: (1) it is small, portable, and requires minimal power (approximately 400 measurements can be completed with two AA alkaline batteries), (2) has the ability to record and store approximately 700 sessions of spirometric data in memory including full flow-volume curves, (3) includes quality control software and prompts to obtain acceptable and repeatable efforts, (4) has time and date stamping of all records, (5) allows easy transfer of specific flows and volumes to a personal computer database, (6) can be re-used to test multiple subjects with minimal cleaning, (6) allows easy calibration, and (7) complies with American Thoracic Society (ATS) criteria for spirometer performance. A recent evaluation by the FACES study team indicated this spirometer accurately and reliably measures pulmonary function in children, relative to a "gold-standard" laboratory-style instrument (see Mortimer, et. al. 2003 for details).

Based on discussions and collaboration with members of the Fresno Asthmatic Children's Environment Study (FACES) and input received from CARB internal and external reviewers during the UCLA grant proposal review process, additional training was recommended for L.A. FANS field interviewers to help increase the quality of lung function data, especially data collected for children (some of which will be as young as 5 years old). This report summarizes the work of Dr. Kathleen Mortimer and Mr. Lucas Carlton to train L.A. FANS interviewers in collection of lung function measurements in conjunction with the UCLA project. In addition, it was also recommended that all collected spirometry curves be graded on an on-going basis for quality review and to provide feedback to field interviews on where improvements were needed during data collection. Dr. Mortimer and Mr. Carlton have extensive experience in using the EasyOne spirometer to assess lung function and in reviewing spirometry curves, especially in asthmatic children where these manuevers can be most difficult, based on their work on the FACES project at UC Berkeley.

**Project Objectives:**

The objectives of this subcontract were to provide training to the L.A. FANS-2 field interviewers on how to successfully administer spirometry tests using the EasyOne portable spirometer and to review and grade spirometry test results from subjects interviewed in the L.A. FANS-2 study. Although training and subsequent data collection includes both adults and children, parts of the training were specifically tailored to performing tests with children, since that is the main focus of the CARB-funded UCLA study of air pollution impacts.

**Description of training**:

The training sessions for field supervisors and field interviewers were held on October 11, 2005 (at the RAND Corporation), and on August 14-15, 2006 and April 16, 2007 (at the Marriott Hotel, Marina Del Rey). All field interviewers were required to attend at least one training session. The April 16, 2007 training included new field interviewers who were being trained for the first time, as well as existing field interviewers who were having trouble achieving good spirometry tests in their interviews. At the first training (October 11, 2005), each of the 8 field interviewers attended a 2-hour training session led by two technical representatives from ndd Technologies, the makers of the EasyOne spirometers. These sessions were focused on the instruments themselves, and the technicians went over how to turn the machine on, how to enter subject-specific characteristics, how to place the spirette properly in the instrument prior to the maneuver and how to perform the maneuver. The interviewers then practiced performing the maneuvers themselves. In the afternoon, these 8 field interviewers attended a 4-hour spirometry training led by Dr. Kathleen Mortimer and Lucas Carlton from the U.C. Berkeley FACES study. During all sessions, Dr. Michelle Wilhelm and Jo Kay Ghosh, both from UCLA, assisted Dr. Mortimer and Mr. Carlton with the training by helping the interviewers with the device set up and acting as "participants.". At the subsequent training sessions, there were two training session each day. Each session lasted approximately 4 hours, with one group of interviewers receiving training in the morning, and a different group receiving the training in the afternoon. The training sessions in October 2005 and August 2006 were led by Dr. Mortimer and Mr. Carlton, while the April 2007 training session was led only by Dr. Mortimer.

In the first 2 hours of the training, Dr. Mortimer and Mr. Carlton began the training session by reviewing the general purpose of collecting spirometry data, emphasizing how this can provide data on a person's lung function, and how this relates to asthma outcomes (see Appendix 1 for the materials provided to the interviewers in conjunction with this part of the training). They explained the different lung function measurements that can be obtained from the spirometer (FEV1, FVC, etc.) (Table 1) and also went over the volume versus time and flow versus volume curves. They then demonstrated a typical spirometry session, including preparing the spirometer for data collection, explaining the procedure to the participant, having the participant conduct several trials until he/she achieved three "successes", and coaching the participant to give the best effort during each trial. The "participant" for this part of the training was one of the study investigators who knew how to correctly perform a spirometry test. Thus, this gave the interviewers a "first look" at the correct way to collect such data. Afterward, they asked each field interviewer to set up their own spirometer for a test, including turning on the machine, entering a subject's name, date of birth, height and weight, and placing the spirette properly in the instrument prior to the maneuver (see Appendices 2-4 for materials provided in conjunction with this part of the training). Since there was some confusion among a few of the interviewers in terms of how to enter data using the keypad on the EasyOne, they reviewed each interviewer's spirometer with them to make sure each one was set up correctly, and that the interviewers understood how to enter both numbers and letters in each field.

In the second part of the training session, in addition to demonstrating a successful spirometry trial, they demonstrated common mistakes and showed the field interviewers what these mistakes looked like in practice, and on the spirometry graph displayed on the EasyOne's screen (see Appendix 5 for example curves). Specifically, they demonstrated what these maneuvers look like when mistakes occur and also showed the resulting curves and pointed out the problem areas. They also discussed the importance of obtaining acceptable and reproducible curves. Per ATS standards and the L.A. FANS study protocol, the goal is to obtain 3 acceptable

and 2 reproducible curves within a maximum of 8 tries from each subject. Common mistakes include not blowing hard enough or fast enough, sucking in air initially before blowing, blowing in multiple blasts, hesitating before blowing, and not blowing long enough. They provided strategies for improving the participants' technique. For example, one common mistake is that the participants bend over while blowing. The trainers recommended several strategies to remind the participant to stand up straight and only bend their legs while blowing into the spirometer.

**Table 1. Basic spirometry measures and definitions**[57]

| Spirometry measurement | Abbreviation | Description |
|---|---|---|
| Forced Vital Capacity | FVC | This is the total amount of air forcibly blown out after full inspiration, measured in liters. |
| Forced Expiratory Volume in 1 Second | FEV1 | This is the amount of air forcibly blown out in one second, measured in liters. Along with FVC it is considered one of the primary indicators of lung function. |
| Peak Expiratory Flow | PEF | This is the speed of the air moving out of the lungs at the beginning of the expiration, measured in liters per second. |
| Forced Expiratory Time | FET | This measures the length of the expiration in seconds. |
| Forced Expiratory Flow at 25% of FVC | $FEF_{25}$ | This is the flow of air measured at the time when 25% of the entire FVC has been expelled. |
| Forced Expiratory Flow at 75% of FVC | $FEF_{75}$ | This is the flow of air measured at the time when 75% of the entire FVC has been expelled. |
| Forced Expiratory Flow between 25% and 75% of FVC * | $FEF_{25-75}$ | This is average flow of air measured during the interval between the time when 25% and 75% of the entire FVC has been expelled. |

* $FEF_{25-75}$ is also called Maximum Midexpiratory Flow (MMEF)

They laid out the specific instructions that need to be given to the subjects and the physical steps that should be taken to obtain a successful test. They encouraged the interviewers to write these steps down and even read them while they are practicing maneuvers so that they become second-nature (see Appendix 4 for a short "cheat-sheet" they gave to the interviewers to help them memorize the instructions given to subjects). They also encouraged the interviewers to be very energetic while testing children and demonstrate the maneuver so that the children understand what is required. One suggestion was to do the maneuver alongside the children at the same time as a guide, if necessary. Another suggestion was not to have other children in the same room if a child appears embarrassed about doing the maneuver. Although the interviewers were encouraged to speak loudly, including "Blast out", they were instructed to speak more quietly to children who might be frightened or feel they were being yelled at for poor performance. The specific steps were covered numerous times by having the field interviewers practice among themselves and with the trainers' supervision.

To give the interviewers another perspective in how to coach the spirometry maneuver, the trainers asked an interviewer to help demonstrate how to conduct a session. This was used as an example of incorporating various personal elements into the explanation of the test to the participant, coaching the participant, and correcting the participant if needed. During this example, the interviewers saw first-hand the key elements of a successful spirometry session, and how best to interact with a participant. In particular, it was important for interviewers to see the complete explanation of the procedure, the exact order of setting up the spirometer and explaining the steps to the participant, and how to encourage the participant to give their best effort during the spirometry session. Interviewers were encouraged to use certain key phrases while the participant is blowing (e.g. "Blast out", "Keep blowing", "you're doing good", "you're almost there"), and also the use of analogy in describing how the participant should take a deep breath and blow into the spirette (e.g. "take a deep breath as if you were about to dive

underwater", "blow out like you're trying to force all the air out of your lungs"). Using an interviewer to demonstrate the procedure was effective in allowing interviewers to see the procedure from start to finish, give them ideas on what's effective and what's less effective, and allow them to ask questions about specific elements of the coaching and participant interaction.

The last part of the training involved practicing maneuvers on volunteers. During the first session (October 2005), children volunteers ages 5-15 years were the practice participants. At the subsequent sessions, interviewers practiced the maneuver on one another. The trainers observed the interviewers during these practice sessions and made suggestions about how to better coach the participants.

Almost all field interviewers were able to obtain successful maneuvers by the end of the practice sessions. The field supervisors noted any interviewers who were not able to obtain successful maneuvers during the practice session, so that follow-up could be done. All field interviewers had a rolling briefcase which contained all of their required interviewing materials including the spirometer and to supplement the training, they were asked to take the spirometers home to practice on their own children and/or children in their neighborhood or friends' children. Each interviewer was also given a test by their field supervisor at the end of their total two-week training (which included many other elements besides lung function testing).

**Description of grading of spirometry test results:**

It is important to note that the acceptability criteria coded into the EasyOne software may not be applicable to all participants or all efforts. Specifically, the EasyOne software does not detect all faulty curves that can be identified only through visual inspection of the hard copies. Examples include tests which did not start at the origin (early starts) and tests with negative flow toward the end of the test (subject took a breath before the end of the test). Conversely, the EasyOne software may also reject tests that are actually acceptable upon visual inspection. For example, the EasyOne may indicate "early end of test", meaning that the subject did not expel air long enough. Upon review of the data from this subject, however, it may be noted that the curves were actually acceptable if obtained from a young child. Children will not have the lung capacity to blow out for three seconds, which is the standard programmed into the EasyOne software. This pointed to the importance of reviewing the spirometry curves of subjects to confirm their acceptability or unacceptability.

We set up a standard protocol to share and transfer data in a secure manner across the several institutions participating in this project. After each set of field interviews was completed, the interviewers sent their EasyOne spirometers to RTI for downloading of spirometry data into the EasyWare MS Access database. The database was then uploaded onto a secure FTP (sFTP) server at UC Berkeley. Only senior project staff requiring access to these data had access to the sFTP server (each user was given a unique user name and password). To maximize time efficiency and to allow for periodic monitoring of data quality, Mr. Carlton graded each batch of data as received from RTI (approximately once a month to every two months).

In addition, an electronic form was added to the database, so that all the grading and comments could be recorded directly into the database. The grading was done by looking at the shape of each curve, and some of the diagnostic criteria provided from the EasyOne spirometers, including the key spirometry measures presented in Table 1. Once the grading was complete for a batch, the data were summarized and results provided to the RAND project manager. The RAND project manager then provided these summaries to the field interviews. Figure 1 provides an example of grading summaries provided to RAND field interviewers. The percent of

curves that were acceptable and reproducible (determined as explained below) were summarized by age group and by technician identification number to help translate findings effectively to field interviewers. Field interviewers were re-trained by head interviewers as needed based on these findings.

The grading of the spirometry curves consisted of two parts: determining acceptability for each curve, and determining reproducibility in each subject. Acceptability means that the maneuver was completed correctly, for example, that the participant inhaled deeply, and exhaled fast enough and hard enough to get a good measure of lung function. Reproducibility means that the acceptable curves from the same person are similar enough (according to defined criteria) to be useful in determining the person's lung function.[57]

To determine acceptability, all grading of spirometry curves was done based on the following criteria:[57]

(1) The Back Extrapolated Volume must be ≤5% or 150mL, whichever is greater;
(2) Time to Peak Flow must be ≤120 milliseconds;
(3) No abrupt end to test;
(4) FET must be ≥ 2 seconds;
(5) Time/Volume curve must begin at origin (to ensure proper start of test);
(6) Curve must show that subject exhaled using only one continuous blast of air; and
(7) Curve must show no leaks or negative flow throughout test (i.e. no inhalation).

For each curve that was graded as "not acceptable", Mr. Carlton recorded a brief reason why the curve was rejected, such as "need to blow longer", "blow harder and faster", "don't hesitate", "late to peak flow", "bad effort", and "negative flow". These comments were used to help determine which aspects of spirometry coaching needed to be emphasized in on-going feedback to the interviewers and in subsequent the field interviewer training sessions.

After the curves were graded for acceptability, reproducibility was assessed in the acceptable curves. At minimum, a subject needed to have two acceptable curves in order to determine whether or not the curves were reproducible. We used the American Thoracic Society (ATS) guidelines, which specify that at least one FVC reading must be within 0.20 L of the largest FVC measure, and that at least one FEV1 reading must be within 0.20 L of the largest FEV1 measure for the same person. The ATS criteria are the most commonly used criteria for assessing reproducibility of spirometry curves.[57]

Overall, we graded spirometry curves for over 3,000 participants, including both children and adults. The large majority of these subjects (over 75%) were able to achieve 2 or more acceptable spirometry curves, and over 78% of these met the ATS criteria for reproducibility. Of subjects with 2 acceptable curves, 69% had curves that were reproducible. Of subjects with 3 acceptable curves, 83% had curves that were reproducible. Figures 2 and 3 provide final summaries of acceptability and reproducibility by age for L.A. FANS-2 child and adults respondents. Children <8 years of age were the least likely to be able to achieve 2 or more acceptable curves and ability to achieve 2 or more acceptable curves increased with age, reaching 80% in adults. Adults were most likely to achieve reproducible curves (over 63% were reproducible), and results were similar among teenagers (59%) and preteens (56%). However, children <8 years of age were far less likely to achieve reproducible curves (42%), partly due to many of the children being unable to obtain a minimum of 2 acceptable curves.

**Summary and recommendations**:
Overall, the field interviewers received 6 hours of spirometry training. At the end of the training day many expressed that they felt more training and practice maneuvers, especially with children, would be helpful. In future trainings, practice maneuvers spread over a few days may be ideal. The interviewers did take the spirometers home with them to practice on their own families and friends. The field interviewers were receptive to the material and efforts to improve their technique. They expressed that it gave them more confidence with the instruments. The L.A. FANS study team was also very appreciative of the training sessions and felt these sessions were essential for obtaining high quality lung function data.

After reviewing the data from the first several months of field interviews, the trainers found that the age groups having the most trouble achieving acceptable and reproducible spirometry curves were the pre-teens and teenagers. In the subsequent training sessions, they gave specific advice on how best to handle these age groups. They asked some of the more experienced interviewers and field supervisors to give their advice, which included reassuring that nobody (e.g. none of their peers) is watching, and reminding them that the sooner they get 3 acceptable curves, the sooner they will be finished. Additionally, some interviewers seemed to have more trouble with the spirometry than others. The trainers recommended that these interviewers pair up with an interviewer who had had more success with spirometry and/or that these interviewers be retrained.

Overall, the ratings based on the EasyOne pre-programmed software were in agreement with the technician ratings 96.2% percent of the time. However, using the technician grading, we were able to detect 55 additional acceptable spirometry curves (0.6%) that were rejected by the EasyOne software, and also reject 303 curves (3.2%) where problems occurred during the spirometry test (based on both adult and child curves). In regards to providing on-going feedback to field interviewers based on grading results, one recommendation for improvement would be to provide the review results on a more timely basis. Due to the logistics of the L.A. FANS study, data from each interviewer's spirometer was downloaded relatively infrequently and thus there was a lag of two to three months between data collection for some subjects and feedback. More frequent data downloading would allow field interviews to learn about and rectify mistakes more quickly.

**Figure 1: Example of Periodic Summary of Spirometry Grading Results to Provide Feedback to L.A. FANS-2 Interviewers**

**Acceptability by Age**

| Age Group | Reviewer Accept | | | |
|---|---|---|---|---|
| | No acceptable curves | 1 acceptable curve | 2-3 acceptable curves | Total |
| <8 children | 5 | 5 | 8 | 18 |
| | 27.78 | 27.78 | 44.44 | |
| 8-<12 preteen | 0 | 1 | 23 | 24 |
| | 0 | 4.17 | 95.83 | |
| 12-<18 teens | 4 | 1 | 27 | 32 |
| | 12.5 | 3.13 | 84.38 | |
| 18+ adults | 7 | 7 | 95 | 109 |
| | 6.42 | 6.42 | 87.15 | |
| **Total** | 16 | 14 | 153 | 183 |

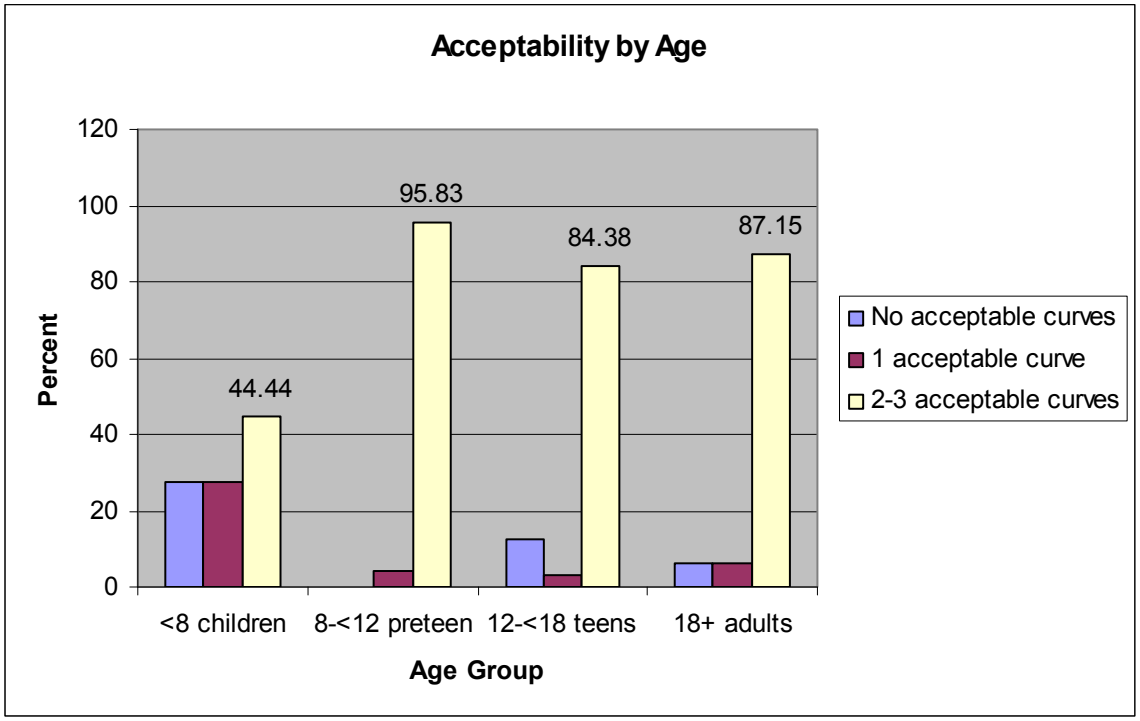


**Figure 1 (continued): Reproducibility by Age**

| Age Group | Reproducibility, using ATS criteria, based on Reviewer grading | | | |
|---|---|---|---|---|
| | NA, <2 acceptable curves | Curves NOT reproducible | Curves ARE reproducible | Total |
| <8 children | 10 | 1 | 7 | 18 |
| | 55.56 | 5.56 | 38.89 | |
| 8-<12 preteen | 1 | 6 | 17 | 24 |
| | 4.17 | 25 | 70.83 | |
| 12-<18 teens | 5 | 8 | 19 | 32 |
| | 15.63 | 25 | 59.38 | |
| 18+ adults | 14 | 13 | 82 | 109 |
| | 12.84 | 11.93 | 75.23 | |
| **Total** | 30 | 28 | 125 | 183 |

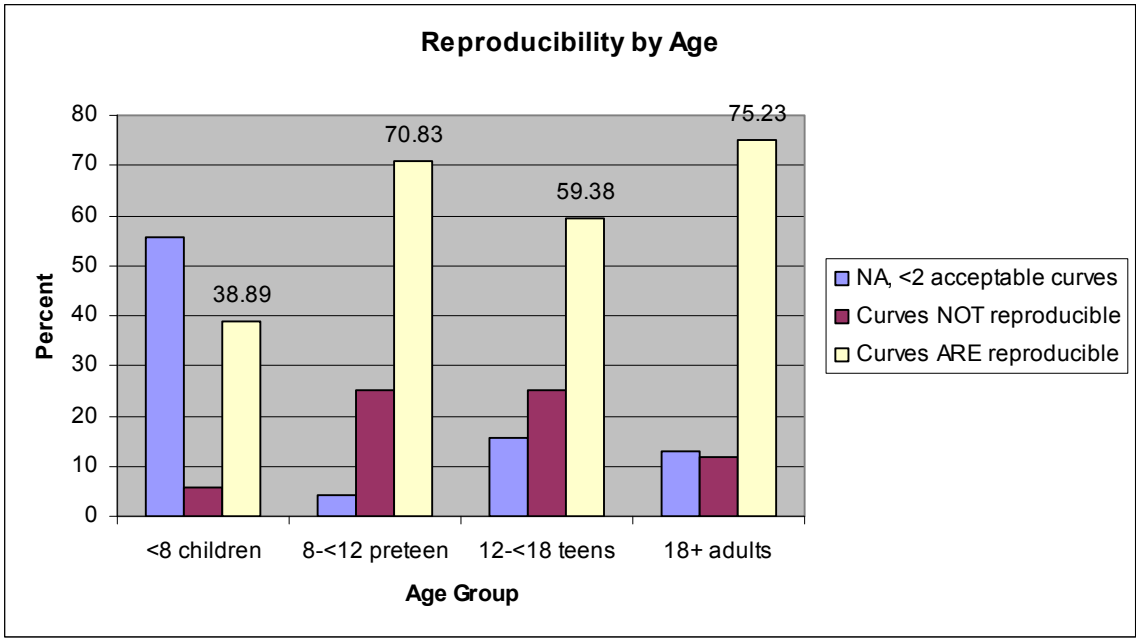


**Figure 1 (continued): Acceptability and Reproducibility by Field Interviewer**
Note: reproducibility is based on ATS criteria and reviewer grading

**Acceptability by Field Interviewer:**

| | No acceptable curves | 1 acceptable curve | 2 acceptable curves | 3 acceptable curves | TOTAL | No acceptable curves % | 1 acceptable curve % | 2-3 acceptable curves % |
|---|---|---|---|---|---|---|---|---|
| **JHAR1** | 0 | 1 | 0 | 1 | 2 | **0.0%** | **50.0%** | **50.0%** |
| **549014** | 10 | 5 | 15 | 18 | 48 | **20.8%** | **10.4%** | **68.8%** |
| **NkAS1** | 2 | 0 | 3 | 5 | 10 | **20.0%** | **0.0%** | **80.0%** |
| **TRUHL1** | 0 | 1 | 3 | 1 | 5 | **0.0%** | **20.0%** | **80.0%** |
| **664243** | 1 | 1 | 2 | 8 | 12 | **8.3%** | **8.3%** | **83.3%** |
| **LAFMLOC1ON** | 2 | 2 | 3 | 23 | 30 | **6.7%** | **6.7%** | **86.7%** |
| **LAFESPY1** | 0 | 2 | 11 | 10 | 23 | **0.0%** | **8.7%** | **91.3%** |
| **MRAV1** | 0 | 2 | 10 | 26 | 38 | **0.0%** | **5.3%** | **94.7%** |
| **IBEL1** | 0 | 0 | 0 | 9 | 9 | **0.0%** | **0.0%** | **100.0%** |
| **LAFMLOC1** | 0 | 0 | 1 | 4 | 5 | **0.0%** | **0.0%** | **100.0%** |
| | | | | | | | | |
| **Total** | 15 | 14 | 48 | 105 | 182 | **8.2%** | **7.7%** | **84.1%** |

**Reproducibility by Field Interviewer:**

| | NA, <2 acceptable curves | Curves NOT reproducible | Curves ARE reproducible | TOTAL | **NA, <2 acceptable curves %** | **Curves NOT reproducible %** | **Curves ARE reproducible %** | TOTAL w/ 2+ acceptable | **% reproducible out of acceptable** |
|---|---|---|---|---|---|---|---|---|---|
| **TRUHL1** | 1 | 2 | 2 | 5 | **20.0%** | **40.0%** | **40.0%** | 4 | 50.0% |
| **JHAR1** | 1 | 0 | 1 | 2 | **50.0%** | **0.0%** | **50.0%** | 1 | 100.0% |
| **549014** | 15 | 7 | 26 | 48 | **31.3%** | **14.6%** | **54.2%** | 33 | 78.8% |
| **664243** | 2 | 2 | 8 | 12 | **16.7%** | **16.7%** | **66.7%** | 10 | 80.0% |
| **LAFESPY1** | 2 | 5 | 16 | 23 | **8.7%** | **21.7%** | **69.6%** | 21 | 76.2% |
| **NkAS1** | 2 | 1 | 7 | 10 | **20.0%** | **10.0%** | **70.0%** | 8 | 87.5% |
| **MRAV1** | 2 | 8 | 28 | 38 | **5.3%** | **21.1%** | **73.7%** | 36 | 77.8% |
| **LAFMLOC1ON** | 4 | 3 | 23 | 30 | **13.3%** | **10.0%** | **76.7%** | 26 | 88.5% |
| **IBEL1** | 0 | 0 | 9 | 9 | **0.0%** | **0.0%** | **100.0%** | 9 | 100.0% |
| **LAFMLOC1** | 0 | 0 | 5 | 5 | **0.0%** | **0.0%** | **100.0%** | 5 | 100.0% |
| | | | | | | | | | |
| **Total** | 29 | 28 | 125 | 182 | **15.9%** | **15.4%** | **68.7%** | 153 | **81.7%** |

**Figure 2. Final Acceptability by Age for L.A.FANS-2 Child and Adult Respondents**

| Age Group | Reviewer Accept | | | |
|---|---|---|---|---|
| | No acceptable curves | 1 acceptable curve | 2-3 acceptable curves | Total |
| <8 children | 48 | 24 | 69 | 141 |
| | 34.04 | 17.02 | 48.94 | |
| 8-<12 preteen | 57 | 39 | 258 | 354 |
| | 16.1 | 11.02 | 72.88 | |
| 12-<18 teens | 75 | 55 | 442 | 572 |
| | 13.11 | 9.62 | 77.27 | |
| 18+ adults | 193 | 190 | 1528 | 1911 |
| | 10.1 | 9.94 | 79.96 | |
| **Total** | 373 | 308 | 2297 | 2978 |

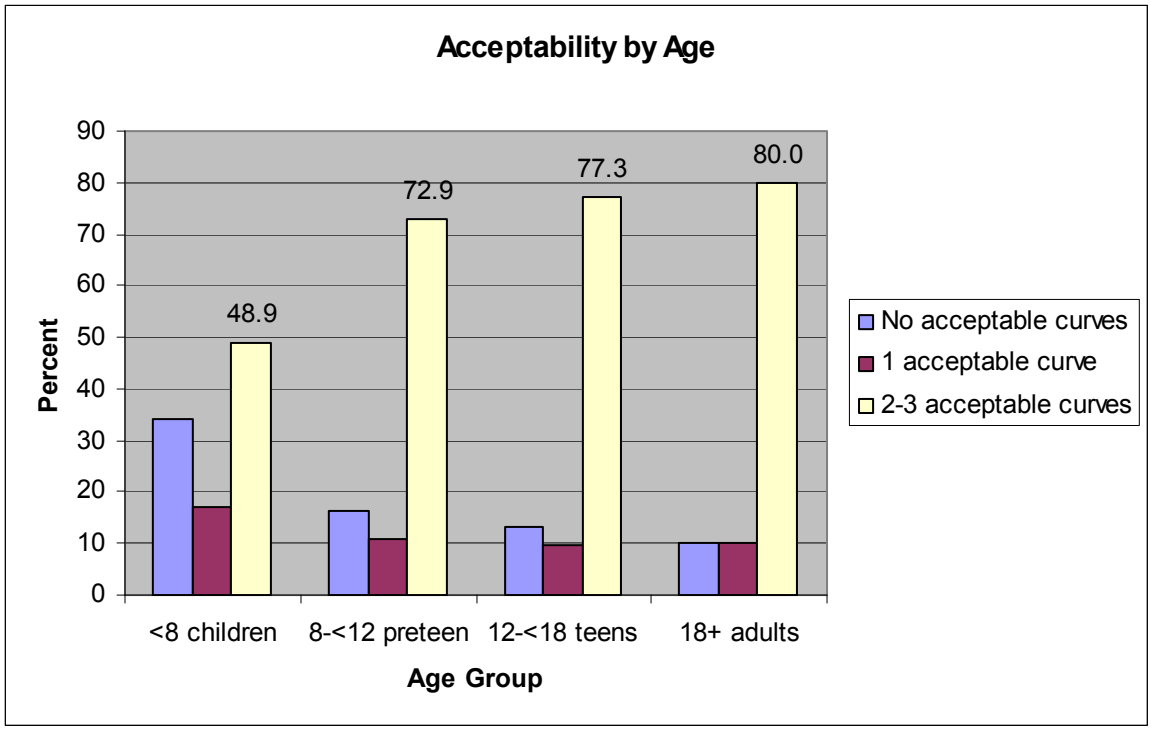
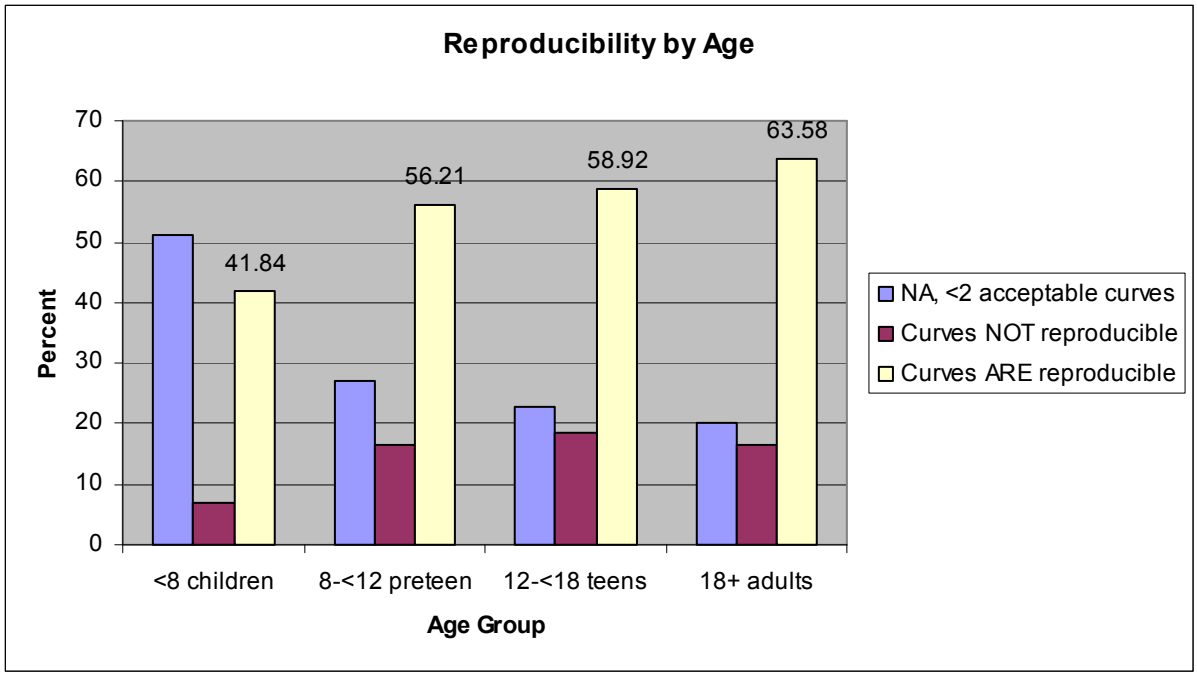


**Figure 3. Final Reproducibility by Age for L.A.FANS-2 Child and Adult Respondents**

| Age Group | Reproducibility, using ATS criteria, based on Reviewer grading | | | |
|---|---|---|---|---|
| | NA, <2 acceptable curves | Curves NOT reproducible | Curves ARE reproducible | Total |
| <8 children | 72 | 10 | 59 | 141 |
| | 51.06 | 7.09 | 41.84 | |
| 8-<12 preteen | 96 | 59 | 199 | 354 |
| | 27.12 | 16.67 | 56.21 | |
| 12-<18 teens | 130 | 105 | 337 | 572 |
| | 22.73 | 18.36 | 58.92 | |
| 18+ adults | 383 | 313 | 1215 | 1911 |
| | 20.04 | 16.38 | 63.58 | |
| **Total** | 681 | 487 | 1810 | 2978 |



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**List of Appendices:**

1. **Spirometry overview**
2. **Spirometry instructions (detailed)**
3. **EasyOne instructions**
4. **LA FANS step-by-step instructions (brief)**
5. **Sample curves for training**

# Appendix 1
# L.A. FANS Spirometry Overview

# SPIROMETRY

## I. Introduction

The principle test of lung function is known as “spirometry” or the “forced vital capacity maneuver.” The basic principal behind the test is to have a participant fill his/her lungs with as much air as s/he can and then rapidly and forcefully exhale the air until s/he feels that no more air is left in his/her lungs. The spirometer records and saves information about volume, flow rate, and time. From this information a number of measurements can be made about the mechanical function of the person’s lung.

A high level of participant cooperation is required to obtain reliable results from this test. Failure to obtain a maximum effort with each test and/or lack of attention to other details that affect the test will lead to results that are not accurate or reproducible.

## II. Measurements That Will be Made

### A. $FEV_1$ - Forced Expiratory Volume in the first second:

measures the amount of air that the participant can exhale in the first second of forced exhalation.

1. This measurement is affected adversely if the participant starts the maneuver too slowly or does not blow as hard as s/he can right from the start.

### B. FVC - Forced Vital Capacity:

measures the total amount of air that a participant can exhale after taking a full inspiration and then forcibly emptying the lung until no more air is left.

1. This measurement is affected adversely if the participant does not empty his/her lungs as completely as possible or if the participant does not blow out as forcefully as s/he can; paradoxically, the slower the participant blows, the larger the result will be (this is not what is desirable).

## C. $FEV_1$ / FVC:

$FEV_1$ expressed as a percentage of FVC.

1. This is a clinically useful index of airflow limitation for an individual, particularly if they have abnormally high or low FVC.

## D. $FEF_{25-75\%}$- Forced Expiratory Flow between 25 and 75%

the average flow of the exhale over the middle half (25% volume to 75% volume) of the FVC.

1. This is a more sensitive measure of small airway function than FEV1.

## E. PEF - Peak Expiratory Flow Rate (also called PEFR):

the highest airflow rate obtained during the test.

1. It is the top of the curve in the Flow-Volume Curve and is measured in volume/time (L/min, L/sec).
2. PEF is a direct measure of large airway function.

EasyOne™ ERS 1.5.4
(c) ndd 2000
EasyWare 1.5.0.2
SN 40614

## Patient Information

| | |
|---|---|
| Name | |
| ID | |
| Age | |
| Height | |
| Weight | |
| Gender | |
| Ethnic | |
| Smoker | |
| Asthma | |

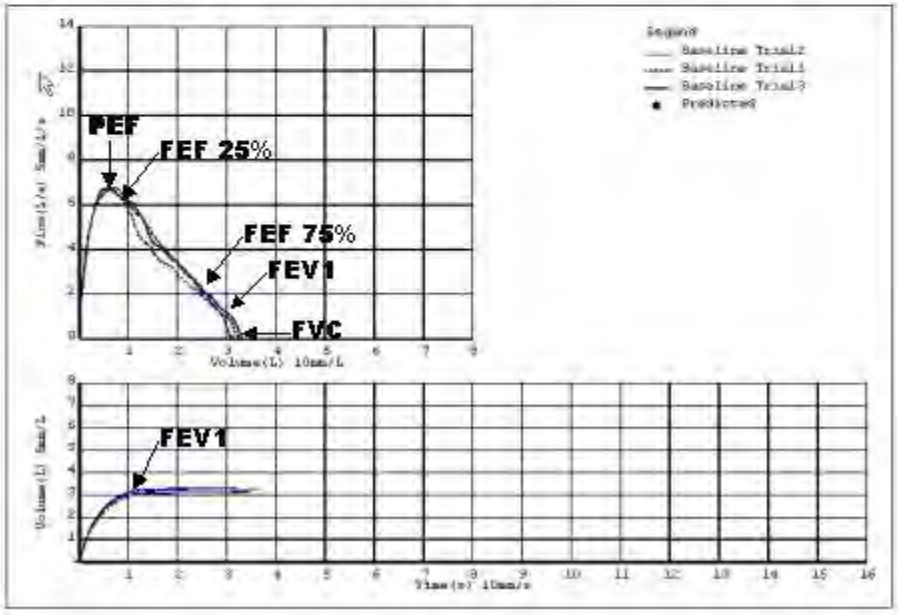
## Test Information

| | |
|---|---|
| Test Date | 06/16/05 |
| Test Time | 07:55am |
| Post Time | --:-- |
| Test Mode | DIAGNOSTIC |
| Predicted Ref | |
| Value Select | BEST VALUE |
| Tech ID | |
| Automated QC | ON |
| BTPS (IN/EX) | -.--/ 1.04 |

## Test Results

| Parameter | Baseline Best | Trial2 | Trial1 | Trial3 | Pred | %Pred |
|---|---|---|---|---|---|---|
| FVC (L) | **3.30** | 3.30 | 3.26 | 3.15 | -.-- | -.-- |
| FEV1 (L) | **3.11** | 3.11 | 2.98 | 3.00 | -.-- | -.-- |
| FEV1/FVC | **0.94** | 0.94 | 0.91 | 0.95 | -.-- | -.-- |
| PEF (L/s) | **6.76** | 6.76 | 6.67 | 6.74 | -.-- | -.-- |
| FEF25-75 (L/s) | **3.75** | 3.75 | 3.32 | 4.03 | -.-- | -.-- |
| FEF75 (L/s) | **2.30** | 2.30 | 2.02 | 2.47 | -.-- | -.-- |
| BEV (L) | **0.08** | 0.08 | 0.06 | 0.07 | -.-- | -.-- |
| PEFT (s) | **0.09** | 0.09 | 0.09 | 0.11 | -.-- | -.-- |
| EOTV (L) | **0.00** | 0.00 | 0.01 | 0.03 | -.-- | -.-- |
| FET (s) | **3.19** | 3.19 | 3.67 | 3.90 | -.-- | -.-- |

Baseline FEV1 Var = 0.12L 3.7%; FVC Var = 0.04L 1.4%; Session Quality A:
Interpretation No Interpretation possible; needed predicted value(s) can't be computed.



## III. Testing/Coaching The Participant

A. Successful spirometry maneuvers don't happen by accident. Your ability to successfully coach the participant the first time around can lead to good compliance and performance on future spirometry tests. Take the time to learn successful coaching techniques and don't worry about looking silly!

B. Start by giving a simple, but full explanation to the participant of what the forced expiratory maneuver involves, as follows:

1. **"Please sit comfortably with both feet on the floor and, whenever you are ready, take as deep a breath as you can until it feels like you cannot get any more air into your lungs. Place your mouth around the mouthpiece with your lips tightly sealed, and then breathe out as hard, as fast and as long as you can. I want you to make the air "BLAST" out of your lungs. Keep breathing out until I tell you to stop."**

    a. Observe the participant carefully during the expiration to make sure that he/she has fully understood the instructions and is performing the forced expiration adequately.

    b. Encourage the participant to keep pushing air out of the lungs throughout the entire forced expiration; for example, tell the participant to "keep going, keep blowing…"

C. Continue by having the participant perform a second attempt, judging, through examination of the curves, whether the participant is performing the expiration correctly. Additional instruction may be needed, and this can only be judged by observing the participant and the curves produced. Once the person fully understands what is expected, the curves should all provide very similar measurements of $FEV_1$ and FVC, meaning that he/she should be able to expel most of the air (FVC) in the first second of the effort. Additional instructions which may be needed include the following:

1. **“Fill your lungs fully, then stop a moment, bring the spirette up to your mouth and breathe out as fast as you can.”**

2. **“Keep the spirette away from the mouth while you are breathing in.”**

2. **“Put the spirette between your teeth and seal your mouth around the mouthpiece, not allowing any air to leak out the sides.”**

3. **“Blow out as if you are saying the word ‘haaa’.”**

4. **“Try to keep going until I tell you to stop, even though it may feel like you are out of air.”**

5. **A demonstration by the tester of what the maneuver involves may be useful, using a spirette held in the hand.**

## IV. Determining Acceptability of the Tracings[1]:

A. Determining a satisfactory end of the test:

1. An obvious plateau in the Volume Time Curve resulting in no change in volume for approximately 1 to 2 seconds (no change in volume means that volume stays within $\pm$ 40 $cm^3$).

**OR**

2. A forced exhalation of reasonable duration (between 3 and 15 seconds).

**OR**

3. The participant cannot, for legitimate reasons, continue further exhalation.

B. Determining a satisfactory start of test:

1. To achieve accurate 'time zero' (i.e. the starting point of the curve for measurement purposes) and to ensure that the $FEV_1$ comes from a maximal effort curve, the extrapolated volume should be less than 5% of the FVC or 100 $cm^3$, whichever is greater (see example of a late start with a tangent line drawn to determine time zero and extrapolated volume).

    a. Generally this means that if the curve starts with a straight vertical line, this line must be less than approx. ¼ inch.

    b. Cough during the first second of the test automatically invalidates the test (a cough of sputtering at the end of the test does not necessarily eliminate the test, if all other criteria are met).

C. Determining whether the curve is acceptable, given that the start and end of the test are acceptable:

1. The tester should observe that the participant understood the instructions and performed the maneuver with a maximum inspiration, with a good start, with a smooth continuous exhalation, with maximal effort, and <u>without</u> any of the following problems (see attached curves):

    a. coughing during the first second of the maneuver, or any other cough that, in the tester's judgement, interferes with measurement of accurate results.

    b. valsalva maneuver (glottis closure).

    c. early termination of expiration (as indicated by a sudden drop-off of the Flow Volume Curve)

    d. a leak

    e. an obstructed mouthpiece, e.g., obstruction due to the tongue being placed in front of the mouthpiece.

D. Acceptability Criteria - Overview

1. No hesitation at start of test
2. Rapid onset of flow
3. No evidence of leak
4. No cough or other evidence of stopping/starting of flow
5. Expiratory time (FET) of at least 3 seconds (possibly shorter for young children)
6. Technician assessment of participant test performance

## V. Determining Reproducibility of the Tracings:

These criteria are used to decide whether the participant has provided two reproducible tracings. Please note that the *acceptability* criteria should be applied **before** the *reproducibility* criteria are even considered.

**NO SPRIOGRAM SHOULD BE REJECTED SOLELY ON THE BASIS OF ITS POOR REPRODUCIBILITY, PROVIDED THAT 3 ACCEPTABLE TRACINGS ARE OBTAINED.**

A. Reproducibility criteria:

1. The largest FVC and the second largest FVC from the set of acceptable curves should not vary by more than 5% of the largest reading or 0.100 L, whichever is greater.

2. The largest $FEV_1$ and the second largest $FEV_1$ from the set of acceptable tracings should not vary by more than 5% of the largest reading or 0.100 L, whichever is greater.

**REFERENCE**

1. American Thoracic Society. Standardization of spirometry—1987 update. Am Rev Respir Dis 1987; 136:1285.

# Appendix 2
# L.A. Spirometry Training Instructions

# SPIROMETRY

Below is a description of the procedures for obtaining spirometry from the participant. Spirometry is the timed measurement of a person's lung volume. This is measured as the person blows out after taking a deep breath. It measures how much air is in the lungs and how effectively and quickly the lungs can be emptied. The measurements include a number of summaries, such as forced vital capacity (FVC, the volume of air that can be forcibly expelled from the lungs) and peak expiratory flow (PEF, the maximal expiratory flow rate). In L.A.FANS-2, the goal is to collect three acceptable spirometry measurements from adults and children 5 years of age and older, using a portable hand-held spirometer. The accuracy of the spirometry measurement depends on the respondent using the proper technique and exerting maximum effort. The procedure requires understanding, coordination, and cooperation between the FI and the respondent. The directions for using the device are in a separate document.

## A. General Precautions

1. Wash your hands before and after handing mouthpieces and interior surfaces of the spirometer.
2. If you have any open cuts or sores on your hands, you must wear gloves.
3. Always wash your hands between measuring different respondents.
4. Clean equipment by wiping with alcohol swabs after each use.

## B. Equipment

L.A.FANS-2 will use hand-held, portable electronic spirometers made by EasyOne. The specific model is the EasyOne Diagnostic Spirometer. The components of the system include the following:

1. The hand-held electronic spirometer.
2. 2 AA batteries.
3. A supply of single-use, disposable Spirettes (the mouthpieces).
4. Disposable nose clips.
5. Alcohol swabs to wipe equipment.
6. Disposable non-latex gloves


The device will assess whether each measurement attempt is acceptable and will provide specific guidance, such as "blow harder" or "blow longer." The read-out window on the spirometer will provide suggestions to improve performance after each attempt.

## C. Exclusions

Respondents excluded from spirometry include those who:

1. Have had any surgery on their chest or abdomen in the past three weeks.
2. Have been hospitalized for a heart problem (such as heart attack, angina or chest pain, congestive heart failure) in the past six weeks.
3. The presence of abdominal or chest pain (for any reason).
4. Oral or facial pain made worse by a mouthpiece.
5. Acute respiratory illness causing the respondent to cough, sneeze or suffer from bronchospasm.
6. Women in their 3rd trimester of pregnancy. (In general, pregnancy is not considered a medical exclusion criterion for spirometry testing. In fact, women with asthma or other respiratory conditions are often tested using spirometry throughout pregnancy in order to monitor their health. That said, for L.A. FANS-2 we will exclude women in their 3rd trimester. If any pregnant woman is anxious about the possibility that the test could be harmful to her pregnancy or fetus the field interviewer should excuse her from the test and still consider her eligible for the full health measures incentive).
7. If any respondent experiences dizziness during the procedure, testing should be stopped.

## D. Information to Collect from Respondents

Questions in the laptop instruct you to ask respondents if they:

1. Have smoked cigarettes in the past one hour?
2. Have eaten a heavy meal in the past one hour?
3. Have used any medications to help them breathe (such as bronchodilators) in the past one hour?
4. Had a cough, cold, or other acute illness in the past week?
5. Had any respiratory infection (such as the flu, pneumonia, bronchitis, or a severe cold) in the past three weeks?
6. Are currently being treated for tuberculosis?

## E. Preparing the Respondent

1. Explain that you will use your mouthpiece to demonstrate the entire procedure. Be careful to not blow into the respondent's face. Use the script in the box below to emphasize each of the following concepts:
    a. Proper placement of the mouthpiece.
    b. Proper placement of noseclip on the nose.
    c. Blasting air into the mouthpiece.
    d. Maximal inhalation. (deepest breath)

    Remember: When you demonstrate the maneuver yourself, using a mouthpiece held in your hand, demonstrate with **maximum** effort so they will use maximum effort!

2. Prepare the respondent to do an exhalation, using the following instructions as a script. Tell them that these are the steps you are going to want them to do:

   a. Stand up straight, feet flat on the floor, do not lean forward
   b. Once I hand you the spirometer, take in as MUCH air as you POSSIBLY can, until your lungs are COMPLETELY full.
   c. Quickly make a tight seal on the mouthpiece with your lips, teeth resting in the grooves. Do NOT bite down and try to keep your tongue out of the way.
   d. Then, BLAST the air out as HARD and as FAST as you POSSIBLY can and keep blowing until I tell you to stop, even though it may seem like you are out of air. Do not bend forward at your waist as you blow out. It's OK if you bend your knees and crouch down a bit.

**F. Coaching the Respondent**

Your ability to successfully coach the respondent at the start of the test will lead to the best results. Don't worry about looking silly!

1. Open a mouthpiece for the respondent and, without connecting it to the spirometer, let the respondent practice putting it in his or her mouth and getting a good seal.

2. Ask the respondent to stand up and loosen any tight clothing.

3. Just in case the participant gets dizzy, place a non-rolling chair behind the participant. Or the respondent can stand with a firm surface, such as a wall, behind him or her.

4. Insert the respondent's mouthpiece into the spirometer and begin the first effort. See "Instructions for Using the EasyOne Device" for additional details on the operation of the device.

5. Use the feedback from the spirometer to judge whether the respondent is blowing out correctly. You may need to give additional instructions to the respondent. You need to use your judgment about what to say after watching how the respondent performs the test and after reading what the spirometer says. If the respondent stops early say, "Even if your lungs feel empty, small amounts of air are still coming out, so keep pushing and blowing." Another example might be "it's okay to bend your knees but make sure you don't lean forward."

6. Continue by having the respondent perform a spirometry tests until three acceptable efforts have been obtained. If, after **8** tries, the person has not completed three acceptable sessions, discontinue the test.

7. Once the respondent fully understands how to do the test correctly, very similar measurements should be obtained when the test is repeated. This means that the respondent should be able to blow out most of the air from his or her lungs in the first second of the effort.

8. Here is a list of the different prompts that will follow each test on the display of the EasyOne device. Be prepared to know how to follow up each prompt with proper coaching:

| Prompt | Coaching response |
|---|---|
| Don't hesitate | The respondent should exhale in one breath and should not stop in-between. |
| Blast out faster | The respondent must exhale more explosively and as firmly and quickly as possible. |
| Blow out longer | The respondent has discontinued exhalation too early. The patient must exhale even more and press as much air as possible out of his/her lungs. |
| Wait until buzz before blowing out | The respondent has started to blow out before the device is ready for the test. |
| Good effort, do next | Good test. Just one to two more good tests and the test is complete. |
| Blast out harder | The test differs greatly from the previous tests. The patient can blow still more firmly and achieve a higher peak flow. |
| Deeper breath | The test differs greatly from previous tests. The patient can inhale even more deeply and exhale even more air. |
| Session complete | The test is complete. An adequate number of good tests have been conducted. |

9. Here are additional instructions you may need to include in your coaching:
   a. If the respondent starts to exhale too quickly: "Fill your lungs fully, then stop a moment, bring the mouthpiece up to your mouth and breathe out as fast as you can."
   b. "Keep the mouthpiece away from your mouth while you are breathing in."
   c. "Put the mouthpiece between your teeth and seal your mouth around the tube, not allowing any air to leak out the sides."
   d. If the respondent makes a lot of noises in his or her throat: "Blow out as if you are saying the word 'haaa'."
   e. If the respondent gives up too quickly: "Try to keep going until I tell you to stop, even though it may feel like you are out of air."
   f. For **children** with short attention spans try engaging the child in a game. For example, if they are having a hard time blowing hard enough at the start of the test, put a piece of paper on a table and challenge them to blow it off with one blast of air. If they are having trouble blowing long enough, tell them to imagine they are blowing candles out at their next birthday, and they have to keep blowing to get them all out in one breath. Although you may need to loudly encourage

them to complete the effort, be careful about yelling at the children, as this may scare them.

**G. Common Errors**

1. Not taking a deep enough breath
2. Leaking air around mouthpiece
3. Slow start to blow out
4. Poor effort in blowing out
5. Stop exhaling too soon
6. Poor posture, especially leaning forward
7. Respondent puts tongue in the mouthpiece (tell him/her not to)
8. Respondent has extra physical efforts such as coughing, vocalizing, or puffing cheeks
9. The respondent flexs his/her neck
10. The respondent pauses just before blowing out
11. The respondent makes noises in his throat while blowing
12. Too much enthusiasm during the blow on the part of the coach (you) may have a negative effect. Be aware of the affect you are having on the child or adult.

**H. Reporting Results**

We do not send spirometry results back to the respondents. One reason is because there is not a simple or easy way to summarize or interpret the spirometry results. If the participant asks for results, please explain that their results have to be put through a computer program to figure out what they mean. You can also say that you are not qualified to interpret results.

**Appendix 3**
**L.A. Spirometry Training Instructions - EasyOne**

# INSTRUCTIONS FOR USING THE EASYONE DEVICE

1. Turn the device on by pressing and holding the ON/OFF button until you hear a beep.

2. The Main menu appears (you will see “MAIN” along the left side of the screen). Select “Perform Test” by pressing the ENTER button.

3. In the next screen you will start a new test. The “NEW” option is already highlighted, so select this option by pressing ENTER.

NOTE: If at any point you need to go back to a previous screen or field, press and hold 0 Esc. You can scroll between different option within a screen be pressing the ◄ and ► buttons.
If device turns off, choose PERFORM TEST, then RECALL and LAST TEST

4. In the next screen you will enter patient data:

    a. **ID** – you will have to enter a combination of numbers and letters. For example, if you need to enter a “2” followed by an “H”, you will have to press 2 on the keypad 4 times (in order to scroll past A, B, and C) and then press 4 2 times. If you make a mistake, use the ◄ key to scroll back. Once you are finished entering the ID, press ENTER.
    b. **Name** - enter the respondent’s initials
    c. **Birth** - enter the respondent’s date of birth
    d. **Height** -Enter “150”
    e. **Weight** - accept the default value, which is 0, by pressing ENTER
    f. **Ethnicity** - accept the default value, which is Caucasian
    g. **Gender** - accept the default value, which is male
    h. **Smoker** - accept the default value, which is no

i. **Asthma** - accept the default value, which is no
j. **Tech ID** - enter your FI ID #

5. Once you have entered all of the patient data, the Test menu appears (you will see "TEST" along the left side of the screen). Select the first test titled "FVC (Expiratory)" by pressing ENTER.

6. Insert the spirette into the device: open the plastic wrap at the end with the smaller opening and insert it through the hole at the top of the device. Line up the arrows on the front of the device and then push it down until it stops. Then remove the plastic wrap from around the top of the mouthpiece (this is so you don't have to touch the mouthpiece with your hands).

7. The next screen will instruct you to block the spirette. Place your palm over the bottom of the spirette and press ENTER.

8. Keep the spirette blocked until you see "Blast Out" on the screen. Quickly hand the device to the respondent and instruct them on completing the maneuver [see "Step by Step Verbal Instructions for Spirometry"].

9. Once you hear the end-of-test beep, the maneuver is complete and you will see the results on the screen. Press ENTER to advance to the "Session Quality" screen. If the test was acceptable, you will be instructed to move on to the next effort. If the test was unacceptable, you will be instructed on how to coach the respondent and they will have to retry the effort [see "Coaching" section in main Spirometry handout].

10. Once 3 acceptable efforts have been recorded, the session will be over and you can turn the device off by pressing and holding the ON/OFF button.

11. Have respondent pull spirette from device and dispose.

**Appendix 4**
**L.A. Spirometry Training – Step by Step Verbal Instructions**

# STEP-BY-STEP VERBAL INSTRUCTIONS FOR SPIROMETRY

1. Stand up straight, feet flat on the floor, do not lean forward. Put on noseclips.

2. Once I hand you the spirometer, take in as **MUCH** air as you **POSSIBLY** can, until your lungs are **COMPLETELY** full.

3. Quickly make a tight seal on the mouthpiece with your lips, teeth resting in the grooves. Don't bite down and try to keep your tongue out of the way.

4. Then, **BLAST** the air out as **HARD** and as **FAST** as you **POSSIBLY** can. Keep blowing until I tell you to stop, even though it may seem like you are out of air. Do not bend forward at your waist as you blow out.

**Appendix 5**
**L.A. Spirometry Training – Example Curves**

EasyOne(TM) DIAGNOSTIC 2.9
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SN 54822

## Patient Information

| | |
|---|---|
| Name | LC |
| ID | 9999 |
| Age | 26 (10/10/1979) |
| Height | 150 cm |
| Weight | |
| Gender | MALE |
| Ethnic | CAUCASIAN |
| Smoker | NO |
| Asthma | NO |

## Test Information

| | |
|---|---|
| Test Date | 08/10/06 12:46am |
| Post Time | --:-- |
| Test Mode | DIAGNOSTIC |
| Interpretation | NLHEP |
| Predicted Ref | Nhanes III |
| Value Select | BEST VALUE |
| Tech ID | |
| Automated QC | ON |
| BTPS (IN/EX) | -.--/ 1.04 |

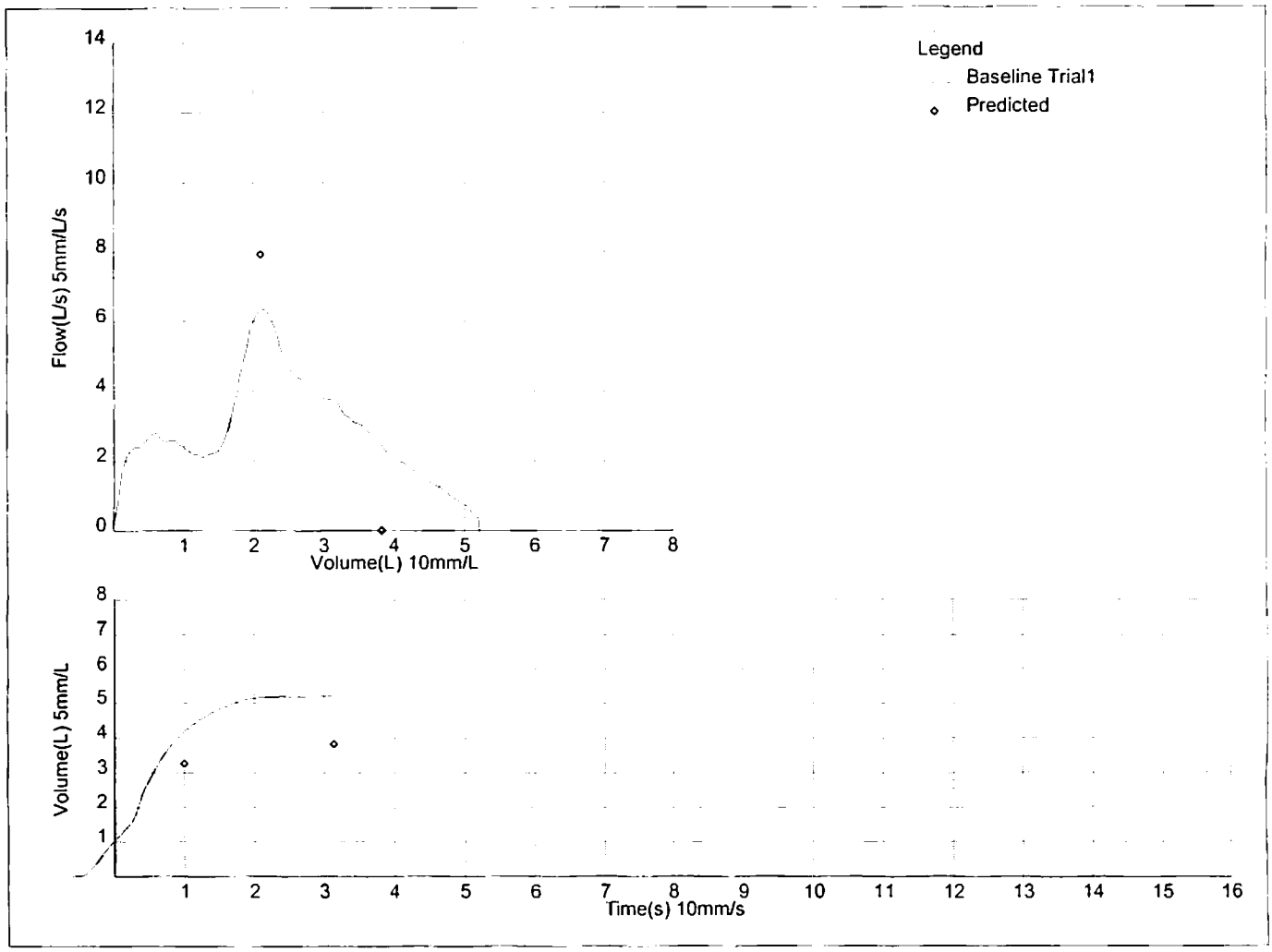
## Test Results

Your FEV1 is 129% Predicted

| Parameter | Baseline Best | Trial1# | Pred | %Pred |
|---|---|---|---|---|
| FVC(L) | 5.21 | 5.21 | 3.84 | 136 |
| FEV1(L) | 4.21 | 4.21 | 3.27 | 129 |
| FEV1/FVC | 0.81 | 0.81 | 0.83 | 98 |
| PEF(L/min) | 379 | 379 | 476 | 79 |
| FEF25-75(L/s) | 3.57 | 3.57 | 3.73 | 96 |
| FET(s) | 3.14 | 3.14 | -.-- | -- |

Baseline FEV1 Var= -.-- L; FVC Var= -.-- L; Session Quality F
Interpretation No interpretation, no acceptable maneuvers
Caution: No Acceptable Maneuvers - Interpret With Care.

DON'T HESITATE



EasyOne(TM) DIAGNOSTIC 2.9
(c)ndd 2000-2004
EasyWare 2. 7. 1. 0
SN 54822

## Patient Information

| | |
|---|---|
| Name | LC |
| ID | 99 |
| Age | 26 (10/10/1979) |
| Height | 150 cm |
| Weight | |
| Gender | MALE |
| Ethnic | CAUCASIAN |
| Smoker | NO |
| Asthma | NO |

## Test Information

| | |
|---|---|
| Test Date | 08/10/06 01:04am |
| Post Time | --:-- |
| Test Mode | DIAGNOSTIC |
| Interpretation | NLHEP |
| Predicted Ref | Nhanes III |
| Value Select | BEST VALUE |
| Tech ID | |
| Automated QC | ON |
| BTPS (IN/EX) | -.--/ 1.04 |

## Test Results

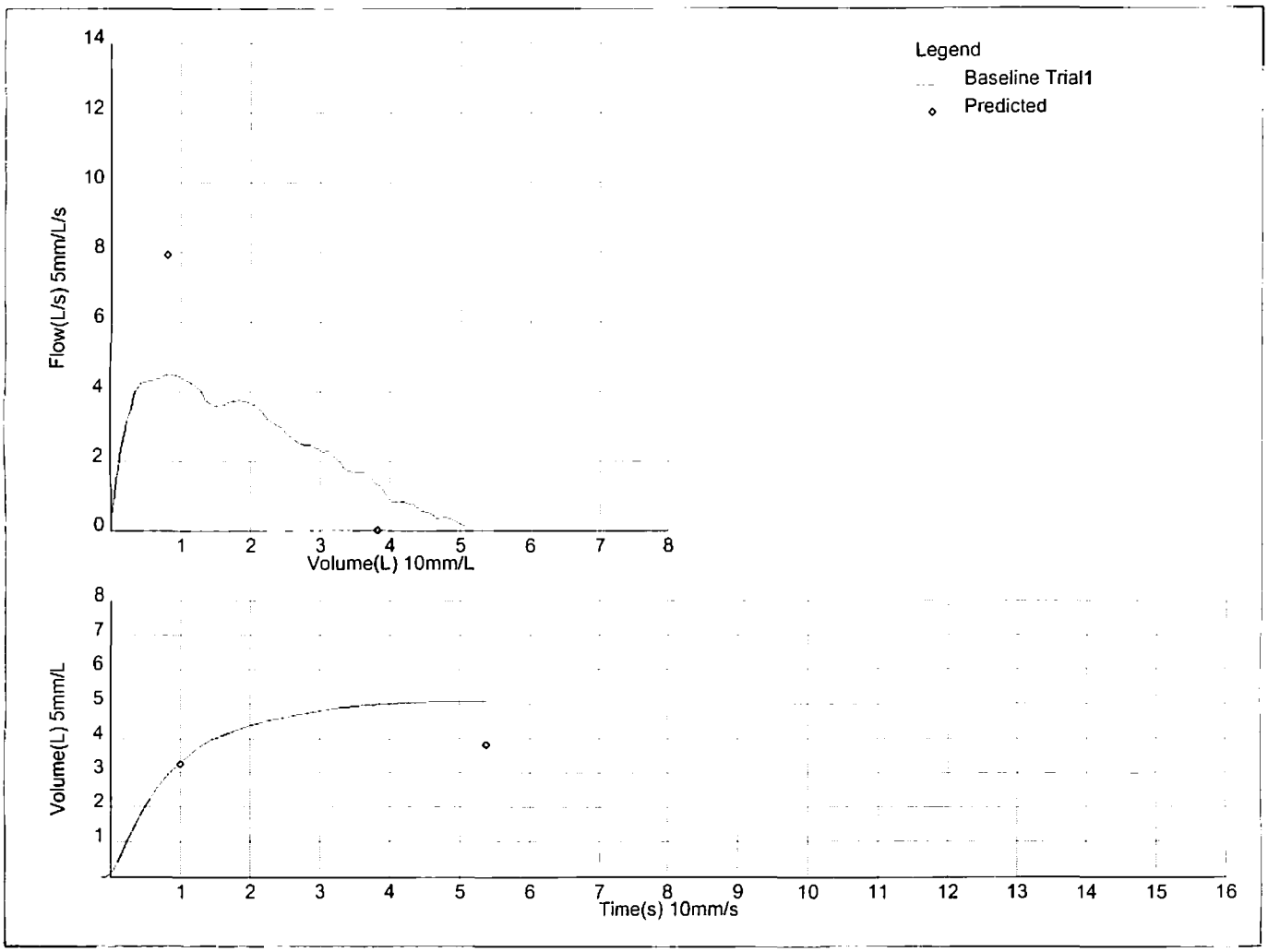
Your FEV1 is 102% Predicted

| Parameter | Baseline Best | Trial1# | Pred | %Pred |
|---|---|---|---|---|
| FVC(L) | 5.07 | 5.07 | 3.84 | 132 |
| FEV1(L) | 3.33 | 3.33 | 3.27 | 102 |
| FEV1/FVC | 0.66* | 0.66* | 0.83 | 80 |
| PEF(L/min) | 270* | 270* | 476 | 57 |
| FEF25-75(L/s) | 2.54* | 2.54* | 3.73 | 68 |
| FET(s) | 5.38 | 5.38 | -.-- | -- |

* Indicates Below LLN or Significant Post Change

Baseline FEV1 Var= -.-- L; FVC Var= -.-- L; Session Quality F
Interpretation No interpretation, no acceptable maneuvers
Caution: No Acceptable Maneuvers - Interpret With Care.

BLAST OUT FASTER



EasyOne(TM) DIAGNOSTIC 2.9
(c)ndd 2000-2004
EasyWare 2. 7. 1. 0
SN 54822

## Patient Information

| | |
|---|---|
| Name | LC |
| ID | 9999 |
| Age | 26 (10/10/1979) |
| Height | 150 cm |
| Weight | |
| Gender | MALE |
| Ethnic | CAUCASIAN |
| Smoker | NO |
| Asthma | NO |

## Test Information

| | |
|---|---|
| Test Date | 08/10/06 12:49am |
| Post Time | --:-- |
| Test Mode | DIAGNOSTIC |
| Interpretation | NLHEP |
| Predicted Ref | Nhanes III |
| Value Select | BEST VALUE |
| Tech ID | |
| Automated QC | ON |
| BTPS (IN/EX) | -.--/ 1.04 |

## Test Results

Your FEV1 is 72% Predicted

| Parameter | Baseline Best | Trial1 | Trial2# | Pred | %Pred |
|---|---|---|---|---|---|
| FVC(L) | 2.36* | 2.36* | 1.99* | 3.84 | 61 |
| FEV1(L) | 2.35* | 2.35* | 1.99* | 3.27 | 72 |
| FEV1/FVC | 1.00 | 1.00 | 1.00 | 0.83 | 121 |
| PEF(L/min) | 522 | 522 | 472 | 476 | 110 |
| FEF25-75(L/s) | 6.20 | 6.20 | 6.21 | 3.73 | 166 |
| FET(s) | 2.53 | 2.53 | 0.34 | -.-- | -- |

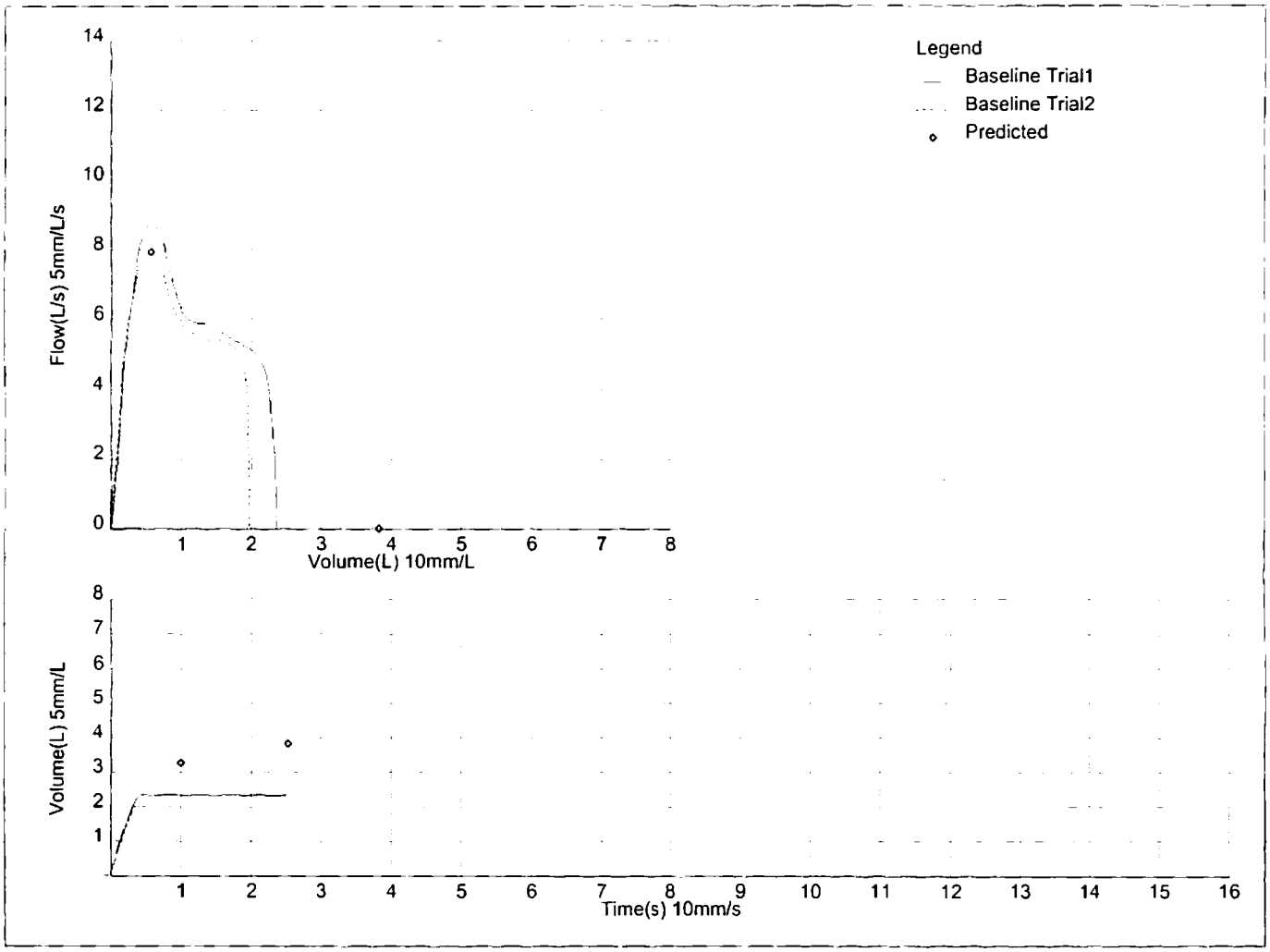
* Indicates Below LLN or Significant Post Change

Baseline FEV1 Var=0.36L 15.4%; FVC Var=0.37L 15.6%; Session Quality D

Interpretation Low vital Capacity possibly due to restriction of lung volumes

Caution: Only One Acceptable Maneuver - Interpret With Care.

BLOW OUT LONGER



EasyOne(TM) DIAGNOSTIC 2.9
(c)ndd 2000-2004
EasyWare 2. 7. 1. 0
SN 54822

## Patient Information

| | |
|---|---|
| Name | LC |
| ID | 99 |
| Age | 26 (10/10/1979) |
| Height | 150 cm |
| Weight | |
| Gender | MALE |
| Ethnic | CAUCASIAN |
| Smoker | NO |
| Asthma | NO |

## Test Information

| | |
|---|---|
| Test Date | 08/10/06 12:50am |
| Post Time | --:-- |
| Test Mode | DIAGNOSTIC |
| Interpretation | NLHEP |
| Predicted Ref | Nhanes III |
| Value Select | BEST VALUE |
| Tech ID | |
| Automated QC | ON |
| BTPS (IN/EX) | -.--/ 1.04 |

## Test Results

Your FEV1 is 120% Predicted

| Parameter | Baseline Best | Trial1 | Trial2# | Pred | %Pred |
|---|---|---|---|---|---|
| FVC(L) | 4.90 | 4.90 | 3.15* | 3.84 | 128 |
| FEV1(L) | 3.93 | 3.93 | 2.68* | 3.27 | 120 |
| FEV1/FVC | 0.80 | 0.80 | 0.85 | 0.83 | 97 |
| PEF(L/min) | 498 | 498 | 266* | 476 | 104 |
| FEF25-75(L/s) | 3.61 | 3.61 | 2.59* | 3.73 | 97 |
| FET(s) | 3.85 | 3.85 | 3.64 | -.-- | -- |

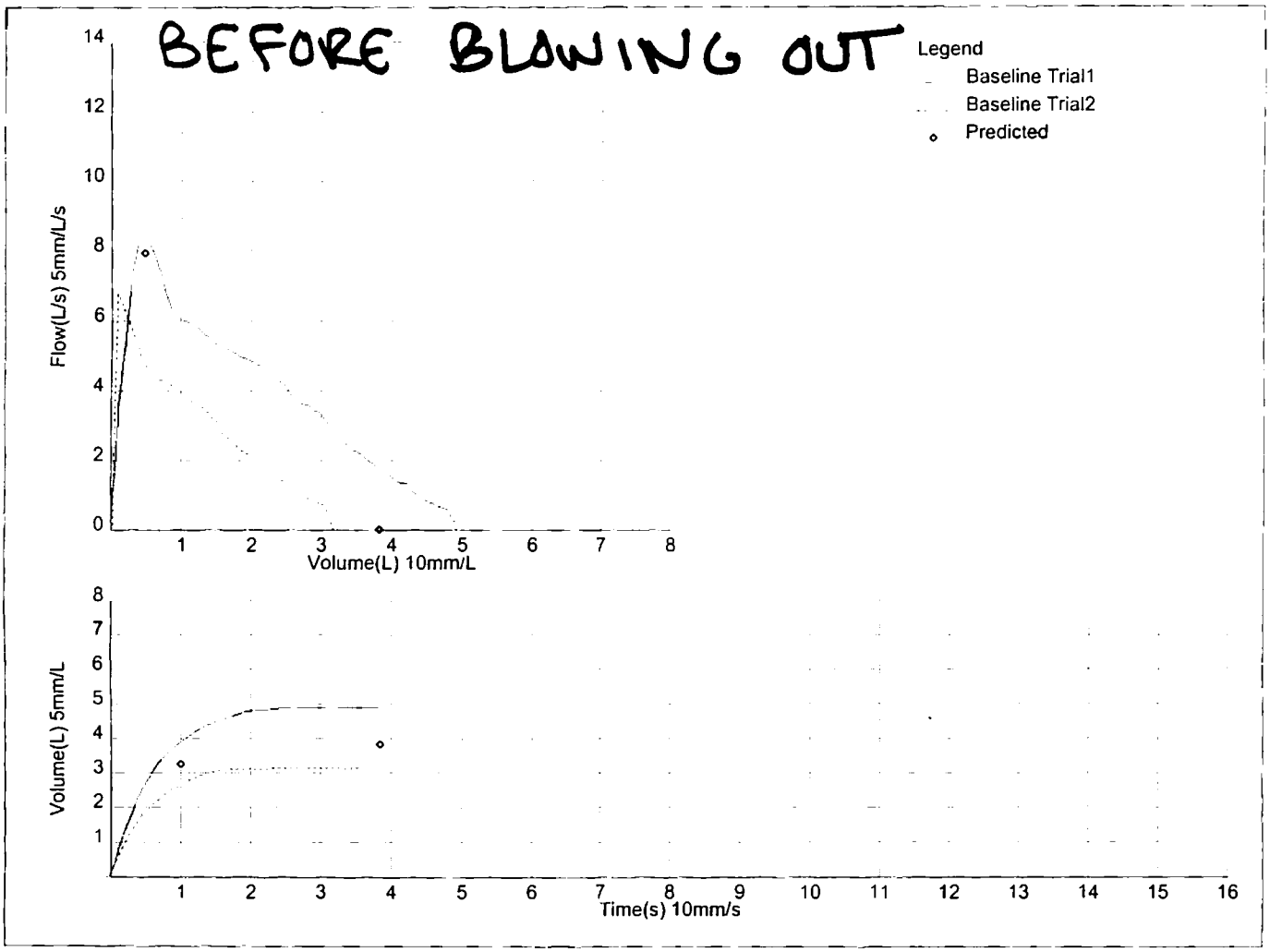
* Indicates Below LLN or Significant Post Change

Baseline FEV1 Var=1.25L 31.8%; FVC Var=1.75L 35.7%; Session Quality D

Interpretation Normal Spirometry

Caution: Only One Acceptable Maneuver - Interpret With Care.

WAIT UNTIL BUZZ BEFORE BLOWING OUT



EasyOne(TM) DIAGNOSTIC 2.9
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## Patient Information

| | |
|---|---|
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| ID | 99 |
| Age | 26 (10/10/1979) |
| Height | 150 cm |
| Weight | |
| Gender | MALE |
| Ethnic | CAUCASIAN |
| Smoker | NO |
| Asthma | NO |

## Test Information

| | |
|---|---|
| Test Date | 08/10/06 12:52am |
| Post Time | --:-- |
| Test Mode | DIAGNOSTIC |
| Interpretation | NLHEP |
| Predicted Ref | Nhanes III |
| Value Select | BEST VALUE |
| Tech ID | |
| Automated QC | ON |
| BTPS (IN/EX) | -.--/ 1.04 |

## Test Results

Your FEV1 is 128% Predicted

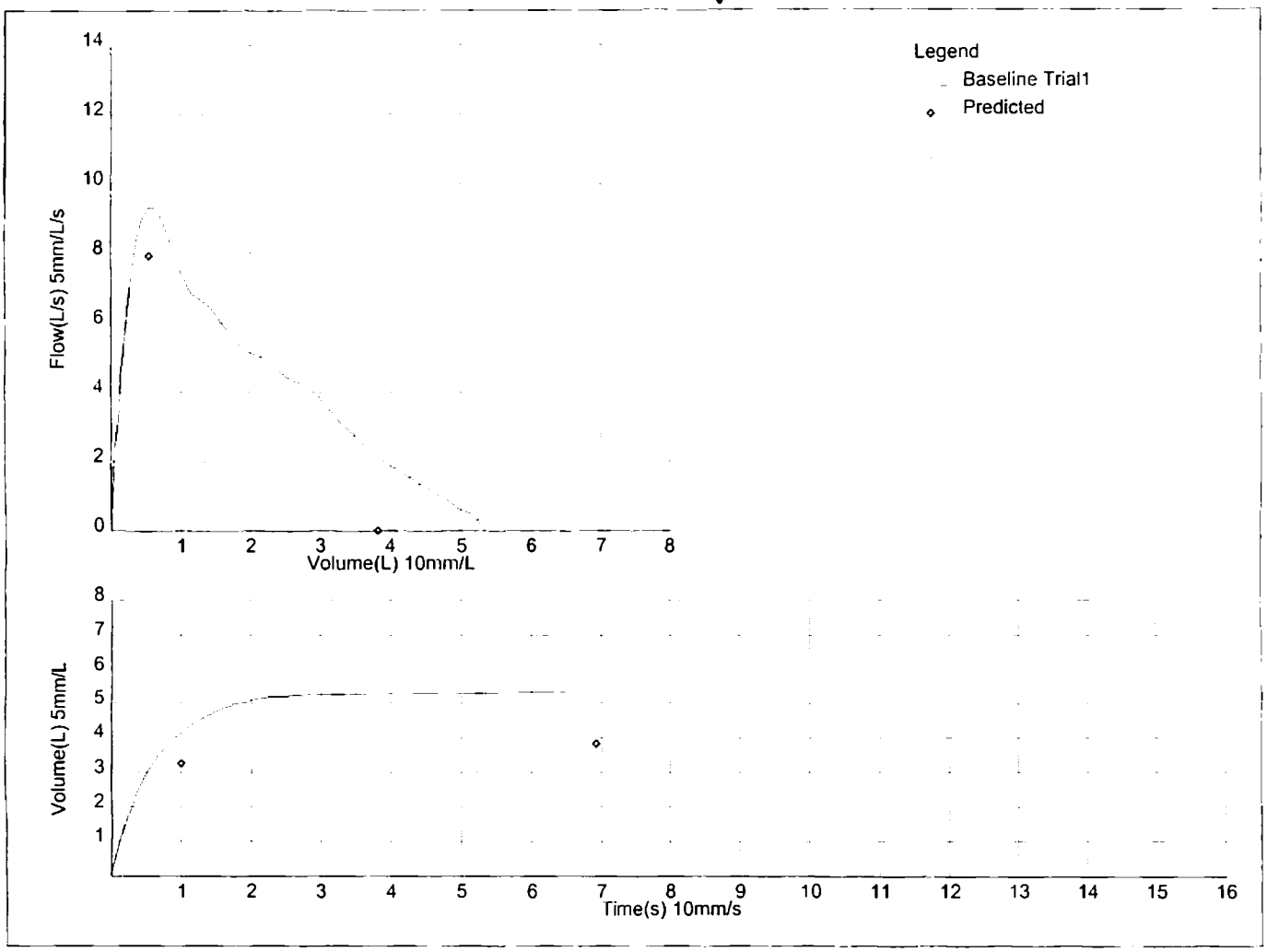
| Parameter | Baseline Best | Trial1 | Pred | %Pred |
|---|---|---|---|---|
| FVC(L) | 5.35 | 5.35 | 3.84 | 139 |
| FEV1(L) | 4.18 | 4.18 | 3.27 | 128 |
| FEV1/FVC | 0.78 | 0.78 | 0.83 | 95 |
| PEF(L/min) | 553 | 553 | 476 | 116 |
| FEF25-75(L/s) | 3.61 | 3.61 | 3.73 | 97 |
| FET(s) | 6.93 | 6.93 | -.-- | -- |

Baseline FEV1 Var= -.-- L; FVC Var= -.-- L; Session Quality D

Interpretation Normal Spirometry

Caution: Only One Acceptable Maneuver - Interpret With Care.

GOOD EFFORT, DO NEXT



EasyOne(TM) DIAGNOSTIC 2.9
(c)ndd 2000-2004
EasyWare 2. 7. 1. 0
SN 54822

## Patient Information

| | |
|---|---|
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| ID | 99 |
| Age | 26 (10/10/1979) |
| Height | 150 cm |
| Weight | |
| Gender | MALE |
| Ethnic | CAUCASIAN |
| Smoker | NO |
| Asthma | NO |

## Test Information

| | |
|---|---|
| Test Date | 08/10/06 12:54am |
| Post Time | --:-- |
| Test Mode | DIAGNOSTIC |
| Interpretation | NLHEP |
| Predicted Ref | Nhanes III |
| Value Select | BEST VALUE |
| Tech ID | |
| Automated QC | ON |
| BTPS (IN/EX) | -.--/ 1.04 |

## Test Results

**Your FEV1 is 129% Predicted**

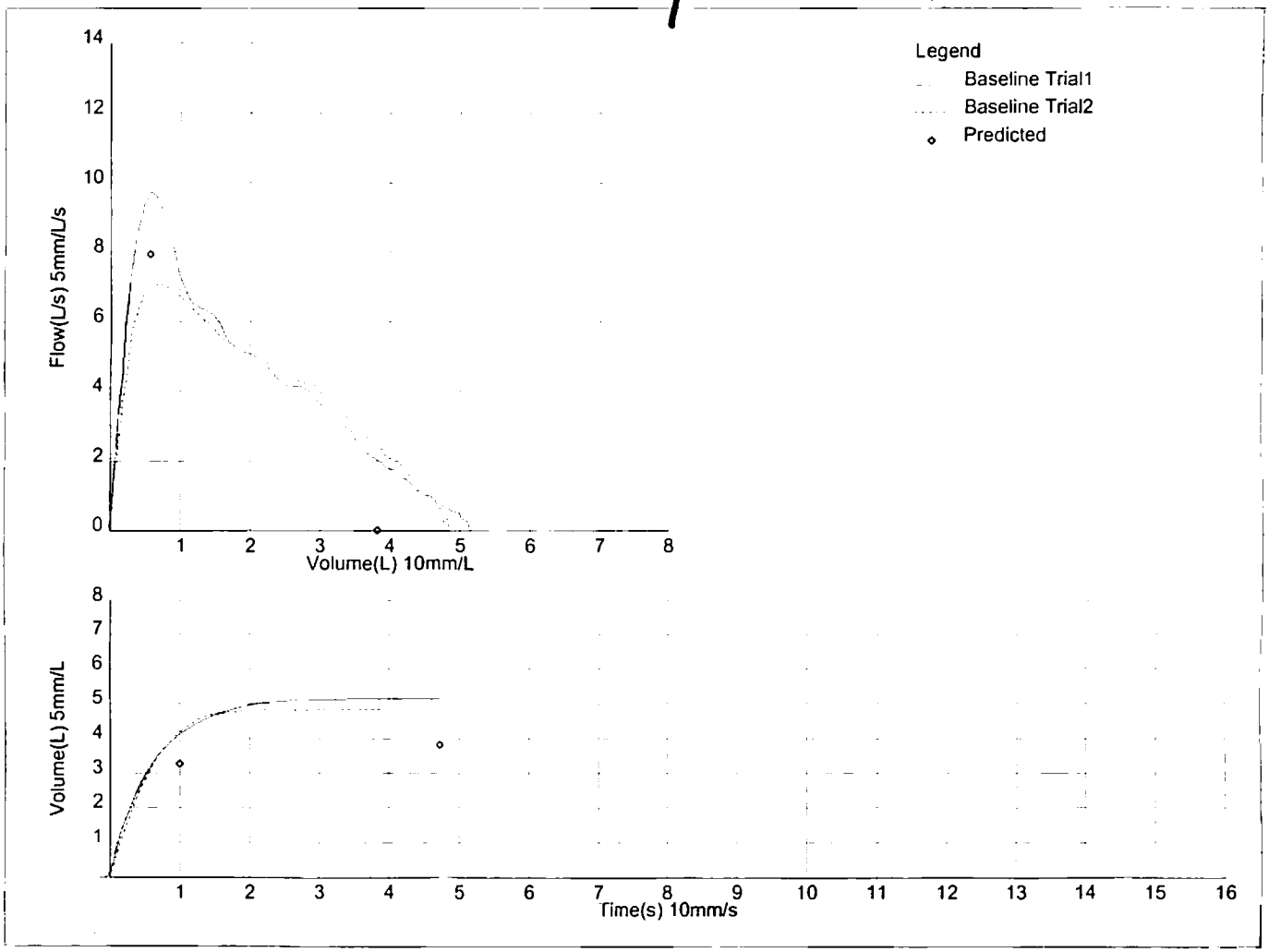
| Parameter | Baseline Best | Trial1 | Trial2 | Pred | %Pred |
|---|---|---|---|---|---|
| FVC(L) | 5.15 | 5.15 | 4.85 | 3.84 | 134 |
| FEV1(L) | 4.21 | 4.14 | 4.21 | 3.27 | 129 |
| FEV1/FVC | 0.82 | 0.80 | 0.87 | 0.83 | 99 |
| PEF(L/min) | 575 | 575 | 423 | 476 | 121 |
| FEF25-75(L/s) | 3.73 | 3.73 | 4.41 | 3.73 | 100 |
| FET(s) | 4.72 | 4.72 | 3.95 | -.-- | -- |

Baseline FEV1 Var=0.08L 1.8%; FVC Var=0.29L 5.7%; Session Quality D

Interpretation Normal Spirometry

Caution: Maneuvers Not Reproducible - Interpret With Care.

BLAST OUT HARDER / DEEPER BREATH



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(c)ndd 2000-2004
EasyWare 2. 7. 1. 0
SN 54822

## Patient Information

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| ID | 99 |
| Age | 26 (10/10/1979) |
| Height | 150 cm |
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| Gender | MALE |
| Ethnic | CAUCASIAN |
| Smoker | NO |
| Asthma | NO |

## Test Information

| | |
|---|---|
| Test Date | 08/10/06 12:54am |
| Post Time | --:-- |
| Test Mode | DIAGNOSTIC |
| Interpretation | NLHEP |
| Predicted Ref | Nhanes III |
| Value Select | BEST VALUE |
| Tech ID | |
| Automated QC | ON |
| BTPS (IN/EX) | -.--/ 1.04 |

## Test Results

**Your FEV1 is 129% Predicted**

| Parameter | Baseline Best | Trial1 | Trial2 | Pred | %Pred |
|---|---|---|---|---|---|
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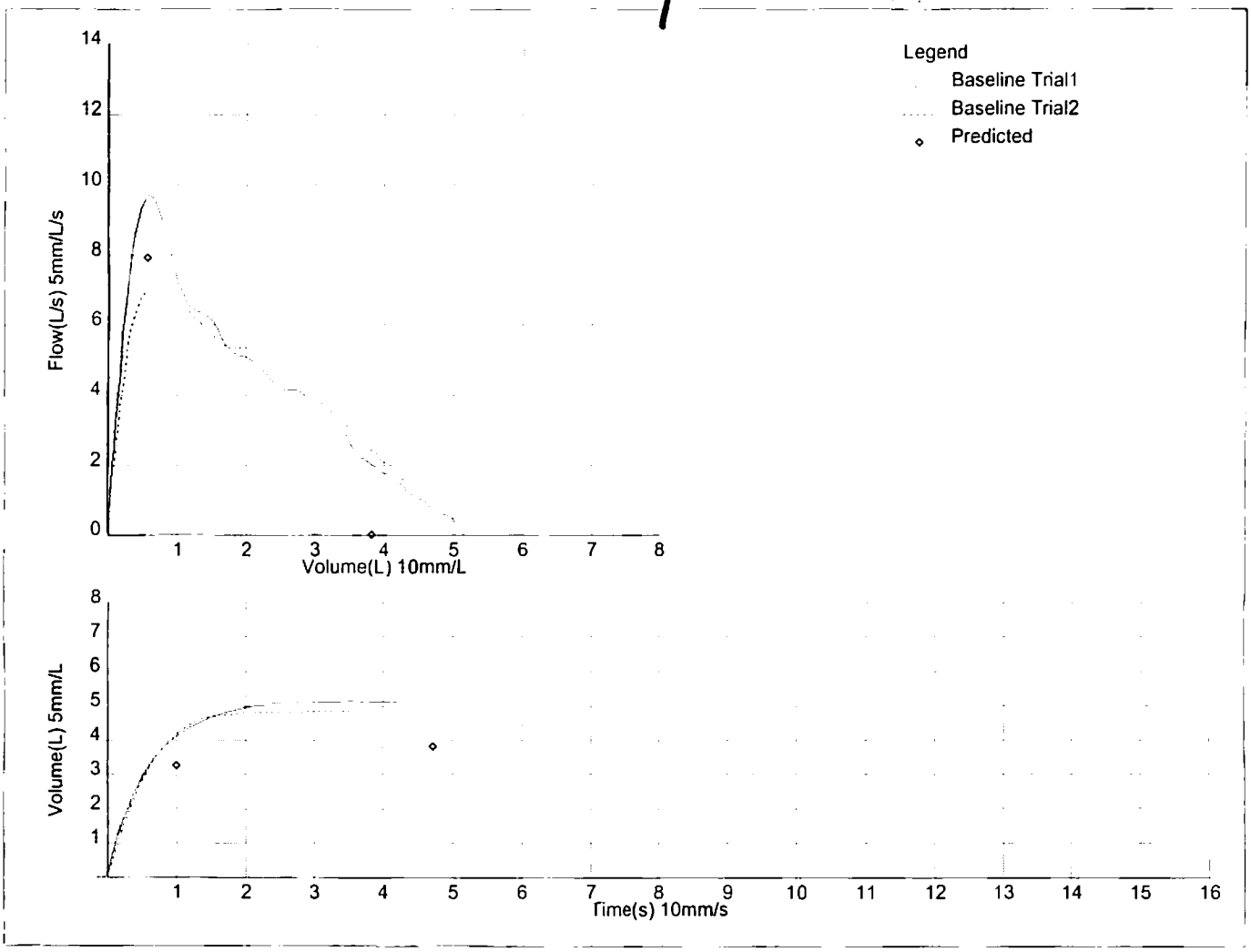
| FEV1(L) | 4.21 | 4.14 | 4.21 | 3.27 | 129 |
| FEV1/FVC | 0.82 | 0.80 | 0.87 | 0.83 | 99 |
| PEF(L/min) | 575 | 575 | 423 | 476 | 121 |
| FEF25-75(L/s) | 3.73 | 3.73 | 4.41 | 3.73 | 100 |
| FET(s) | 4.72 | 4.72 | 3.95 | -.-- | -- |

Baseline FEV1 Var=0.08L 1.8%; FVC Var=0.29L 5.7%; Session Quality D

Interpretation Normal Spirometry

Caution: Maneuvers Not Reproducible - Interpret With Care.

BLAST OUT HARDER / DEEPER BREATH



# Appendix D
# L.A. FANS-2 Spirometry Protocol

# 1. Spirometry

**Introduction**: Spirometry is the timed measurement of a person's lung volume, assessed as the person blows out after taking a deep breath. It measures how much air is in the lungs and how effectively and quickly the lungs can be emptied. The measurements include a number of indices, such as forced vital capacity (the volume of air that can be forcibly expelled from the lungs) and peak expiratory flow (the maximal expiratory flow rate).

**Protocol**: In L.A.FANS-2, you will collect three acceptable/reproducible spirometry measurements from adults and children 5 years of age and older using a portable hand-held spirometer.

**Procedure**: The accuracy of the spirometry measurement depends on the respondent using the proper technique and exerting maximum effort. The procedure requires understanding, coordination, and cooperation between the FI and the respondent.

The following procedure describes how to perform spirometry on all subjects. This information is adapted from several sources, including the American Thoracic Society "Standardization of Spirometry, 1994 Update," *American Journal of Respiratory and Critical Care Medicine* 1995 (52): 1107-1136.

## A. General Precautions

1. Wash your hands before and after handing mouthpieces and interior surfaces of the spirometer.
2. If you have any open cuts or sores on his/her hands, you must wear gloves.
3. Always wash your hands between measuring different respondents.
4. Clean equipment by wiping with alcohol swabs after each use.

## B. Equipment

L.A.FANS-2 will use hand-held, portable electronic spirometers made by EasyOne. The specific model is the EasyOne Diagnostic Spirometer. The components of the system include the following:

1. The hand-held electronic spirometer.
2. 2 AA batteries.
3. A supply of single-use, disposable Spirettes (the mouthpieces).
4. Disposable nose clips.
5. Alcohol swabs to wipe equipment.
6. Disposable non-latex gloves

The read-out window on the spirometer will provide suggestions to improve performance after each attempt. It will assess whether each measurement attempt is acceptable and will provide specific guidance, such as "blow harder" or "blow longer."

See the EasyOne instructions for further details about the equipment.

## C. Exclusions

Respondents excluded from spirometry include those who:

1. Have had any surgery on their chest or abdomen in the past three weeks.
2. Have been hospitalized for a heart problem (such as heart attack, angina or chest pain, congestive heart failure) in the past six weeks.
3. The presence of abdominal or chest pain (for any reason).
4. Oral or facial pain made worse by a mouthpiece.
5. Acute respiratory illness causing the respondent to cough, sneeze or suffer from bronchospasm.
6. Women in their 3rd trimester of pregnancy*
7. If any respondent experiences dizziness during the procedure, testing should be stopped.

*A word about women who are pregnant:

> In general, pregnancy is not considered a medical exclusion criterion for spirometry testing. In fact, women with asthma or other respiratory conditions are often tested using spirometry throughout pregnancy in order to monitor their health. That said, for LAFANS we will exclude women in their 3rd trimester and if any pregnant woman is anxious about the possibility that the test could be harmful to her pregnancy or fetus the field interviewer should simply excuse her from the test and still consider her eligible for the full health measures incentive.

## D. Information to Collect from Respondents

Questions in the laptop instruct you to ask respondents if they:

1. Have smoked cigarettes in the past one hour.
2. Have eaten a heavy meal in the past one hour.

3. Have used any medications to help them breathe (such as bronchodilators) in the past one hour.
4. Had a cough, cold, or other acute illness in the past week.
5. Had any respiratory infection (such as the flu, pneumonia, bronchitis, or a severe cold) in the past three weeks.
6. Are currently being treated for tuberculosis.

## E. Configuring the Easy One Spirometer

Each day that you use the spirometer, begin by checking its configuration. Follow these steps:

1. For the date of the tests: Choose "Configuration" from the main menu, then choose "General Settings," and check and update the field entitled "Date."
2. To be able to enter your FI ID number: Choose "Configuration" from the main menu, then choose "General Settings," and set the Tech ID field to "yes."
3. To make sure three readings are recorded and stored: Choose "Configuration" from the main menu, then choose "Test Settings," and set the Storage field to "3 Best."
4. To make sure three readings are reported: Choose "Configuration" from the main menu, then choose "Report Settings," and set the Curve field to "3 Best."
5. To set the height to centimeters: Choose "Configuration" from the main menu, then choose "General Settings," and set the Height Unit field to "m/cm."

## E. Preparing and Using the Easy One Spirometer

1. For each respondent, you must enter information into the spirometer on the "Patient Data" screen. Do the following:

    a. ID—enter the respondent's L.A. FANS-2 ID #
    b. Name—enter the respondent's initials
    c. Birth—enter the respondent's date of birth
    d. Height—enter the respondent's height in centimeters, based on the measurement you recorded earlier; if the respondent declined to have his or her height measured or you were unable to measure the respondent's height, enter "150"
    e. Weight—accept the default value, which is 0
    f. Ethnicity—accept the default value, which is Caucasian
    g. Gender—accept the default value, which is male
    h. Smoker—accept the default value, which is no
    i. Asthma—accept the default value, which is no
    j. Tech ID—enter your FI ID #

2. You can let the respondent try a maximum of eight attempts to get three satisfactory tests.
3. The respondent has performed an acceptable test when the reading in the spirometer's window says "good effort, do next."

4. The respondent has performed three acceptable tests when the reading in the spirometer's window says "session complete."
5. Results are stored on the spirometer, which will be able to hold results for about 200 respondents. You will be told how to bring or ship your spirometer to RTI so the results can be downloaded at a later time.
6. If three "acceptable" tests are not obtained, explain why in the comments field in the laptop
7. Note any deviation from the protocol or any problems encountered in the comments section in the laptop.

## F. Preparing the Respondent

1. Explain and demonstrate the entire procedure to the respondent (using your own mouthpiece) before the respondent attempts the process. Be careful to not blow into the respondent's face. Explain and demonstrate the following steps:
    a. Proper placement of the mouthpiece.
    b. Proper placement of noseclip on the nose.
    c. Maximal inhalation.
    d. Blasting air into the mouthpiece.
2. Open a mouthpiece for the respondent and, without connecting it to the spirometer, let the respondent practice putting it in his or her mouth and getting a good seal.
3. Ask the respondent to stand up and loosen any tight clothing.
4. Place a non-rolling chair behind the participant. As an alternative, the respondent can stand with a firm surface, such as a wall, behind him or her.
5. Insert the respondent's mouthpiece into the spirometer.
6. Prepare the respondent to do an exhalation, using the following instructions as a script:
    a. First put this clip on your nose.
    b. Lift up your chin to help open your airway.
    c. Now I'm going to hand you the spirometer.
    d. Take a great big deep breath of air as far as you can inhale.
    e. Without pausing, put the mouthpiece in your mouth, between your teeth, and seal your lips tightly around it.
    f. Blast out the air as hard and fast as you can!
    g. Keep on blowing out the same breath of air until I tell you to stop.
7. After each attempt give the respondent feedback based on the message from the spirometer and your own observations. If the respondent stops early say, "Even if your lungs feel empty, small amounts of air are still coming out, so keep pushing and blowing." Another example might be "it's okay to bend your knees but make sure you don't lean forward."

## G. Common Errors

1. Not taking a deep enough breath
2. Leaking air around mouthpiece
3. Slow start to blow out

4. Poor effort in blowing out
5. Stop exhaling too soon
6. Poor posture, especially leaning forward
7. Respondent puts tongue in the mouthpiece (tell him/her not to)
8. Respondent has extra physical efforts such as coughing, vocalizing, or puffing cheeks
9. The respondent is flexing his/her neck
10. The respondent pauses just before blowing out
11. The respondent makes noises in his throat while blowing
12. Too much enthusiasm during the blow on the part of the coach (you) may have a negative effect. Be aware of the affect you are having on the child or adult.

## H. Coaching the Respondent

Your ability to successfully coach the respondent at the start of the test will lead to the best results. Begin by giving a simple but full explanation to the respondent of what the spirometry test involves, as follows:

1. **"Please stand (with the wall behind you) and whenever you are ready, take as deep a breath as you can until it feels like you cannot get any more air into your lungs. Place your mouth around the mouthpiece with your lips tightly sealed, and then breathe out as hard, as fast, and as long as you can. I want you to make the air "BLAST" out of your lungs. Keep breathing out until I tell you to stop."**
    a. Observe the respondent carefully while he or she is blowing out to make sure that he or she has fully understood the instructions and is performing the test correctly.
    b. Encourage the respondent to keep pushing air out of the lungs throughout the entire test. For example, tell the participant to "keep going, keep blowing…"

2. Continue by having the respondent perform a second spirometry test. Use the feedback from the spirometer to judge whether the respondent is blowing out correctly. You may need to give additional instructions to the respondent. You need to use your judgment about what to say after watching how the respondent performs the test and after reading what the spirometer says.

    Once the respondent fully understands how to do the test correctly, very similar measurements should be obtained when the test is repeated. This means that the respondent should be able to blow out most of the air from his or her lungs in the first second of the effort.

    Here are additional instructions you may need to include in your coaching:

    - If the respondent starts to exhale too quickly: "Fill your lungs fully, then stop a moment, bring the mouthpiece up to your mouth and breathe out as fast as you can."
    - "Keep the mouthpiece away from your mouth while you are breathing in."

- "Put the mouthpiece between your teeth and seal your mouth around the tube, not allowing any air to leak out the sides."
- If the respondent makes a lot of noises in his or her throat: "Blow out as if you are saying the word 'haaa'."
- If the respondent gives up too quickly: "Try to keep going until I tell you to stop, even though it may feel like you are out of air."

3. When you demonstrate the maneuver yourself, using a mouthpiece held in your hand, demonstrate with **maximum** effort so they will use maximum effort!

4. For children with short attention spans try engaging the child in a game. For example, if they are having a hard time blowing hard enough at the start of the test, put a piece of paper on a table and challenge them to blow it off with one blast of air. If they are having trouble blowing long enough, tell them to imagine they are blowing candles out at their next birthday, and they have to keep blowing to get them all out in one breath.

## I. Reporting Results

We do not send spirometry results back to the respondents. One reason is because there is not a simple or easy way to summarize or interpret the spirometry results. Explain to the respondents that their results have to be put through a computer program to figure out what they mean. You can also say that you are not qualified to interpret results.