

NIH Public Access

Author Manuscript

Health Place. Author manuscript; available in PMC 2010 March 1.

Published in final edited form as:

Health Place. 2009 March ; 15(1): 25–36. doi:10.1016/j.healthplace.2008.02.002.

Outdoor air pollution, family and neighborhood environment, and asthma in LA FANS children

Michelle Wilhelm1,2, **Lei Qian**3, and **Beate Ritz**1,2

1*Department of Epidemiology, School of Public Health, UCLA* 2*Center for Occupational and Environmental Health, School of Public Health, UCLA* 3*Department of Biostatistics, School of Public Health, UCLA*

Abstract

We examined associations between outdoor air pollution and childhood asthma, using measures of SES, neighborhood quality and social support from the Los Angeles Family and Neighborhood Survey (LA FANS). We linked residential census tracts for 3,114 children to government air monitoring stations and estimated average pollutant concentrations for the year before interview. CO and $NO₂$ levels increased and $O₃$ levels decreased as neighborhood quality decreased, yet correlations were low. Pollutant levels were not correlated with neighborhood support. Even after adjustment for social environment characteristics, LA FANS children living in high O_3 , PM_{10} , and CO areas appeared to have worse asthma morbidity.

Keywords

air pollution; asthma; children; socioeconomic status; neighborhood

Background

Asthma is a complex, multifactorial disease. 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 the independent effects or the interplay between social and physical aspects of the community (Gold and Wright, 2005, Mielck et al., 1996). 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 (O'Neill et al., 2003, Houston et al., 2004, Kohlhuber et al., 2006). It is generally well-established that short-term increases in outdoor air pollution can worsen respiratory symptoms in asthmatic children (Gilmour et al., 2006, Trasande and Thurston, 2005, Thurston and Bates, 2003, Brunekreef and Holgate, 2002). 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 most consistently linked with exacerbation of asthma symptoms. While long-term exposures to O_3 , PM_{10} and nitrogen

Author Information and address for reprints: Michelle Wilhelm, Ph.D. (Correspondent), Department of Epidemiology, School of Public Health, UCLA, P.O. Box 951772, 650 Charles E. Young Drive, Los Angeles, CA 90095-1772, USA, Phone: (310) 206-4704, Fax: (310) 206-6039, Email: mwilhelm@ucla.edu.

Publisher's Disclaimer: This is a PDF file of an unedited manuscript that has been accepted for publication. As a service to our customers we are providing this early version of the manuscript. The manuscript will undergo copyediting, typesetting, and review of the resulting proof before it is published in its final citable form. Please note that during the production process errors may be discovered which could affect the content, and all legal disclaimers that apply to the journal pertain.

 $dioxide (NO₂)$ have been associated with chronic respiratory impairments such as reduced lung function and growth, bronchitis and chronic cough, data on asthma incidence are less conclusive (Gilmour et al., 2006, Trasande and Thurston, 2005, Brunekreef and Holgate, 2002). Recently, air pollution research has focused on the contributions of 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 are more able to penetrate cellular targets in the lung and enter systemic circulation (Kunzli et al., 2003, Li et al., 2003, Li et al., 2002, Pandya et al., 2002). 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 (Salam et al., 2008, Brauer et al., 2007, Bayer-Oglesby et al., 2006,Gauderman et al., 2007).

Low SES neighborhood impacts on asthma morbidity may also reflect differences in 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 (Gold and Wright, 2005). Higher levels of psychosocial stress have been linked to greater morbidity in asthmatic children (Sandberg et al., 2004, Wright, 2005, Chen et al., 2006, Miller and Chen, 2006), 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 (Gold and Wright, 2005, Wright et al., 2002, Wright et al., 2004). Other potentially important factors related to SES include differential access to health care and differences in health behaviors such as diet and smoking.

Thus, it has been argued that in order to adequately evaluate the contributions of the physical environment to health outcomes such as asthma, is important to consider social aspects as potential confounders and effect measure modifiers (O'Neill et al., 2003) . The first wave of The Los Angeles Family and Neighborhood Survey (LA FANS) collected extensive data on individual, family, and neighborhood characteristics of Los Angeles residents (Sastry et al., 2003), allowing us to evaluate associations between outdoor air pollution and asthma taking into account both physical and social aspects of neighborhoods. While most previous studies examining this issue relied solely on SES measures based on administrative data sources (such as census data) to reflect exposures to adverse social conditions, the LA FANS study collected information directly from participants regarding their ratings of neighborhood safety, cohesion and social support, providing us with additional measures of neighborhood social environment to consider in our analyses of outdoor air pollution's impact on asthma.

Methods

Subjects

A multistage method was used to select LA FANS neighborhoods (defined as census tracts) and participants (Sastry et al., 2003). LA County census tracts were assigned to three SES strata based on the percentage of the tract population living below the poverty line. LA FANS subjects were then selected by stratified sampling first of census tracts, followed by census blocks, and then households. Very poor and poor tracts ($\geq 90^{th}$ percentile and between the 60–89th percentiles of the poverty distribution, respectively) and households with children were oversampled. Within each household, one adult $(\geq 18$ years) and one child $(\leq 18$ years) were randomly selected for interview. Only children aged \geq 9 years 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 child had one or more siblings with the same biological or adoptive mother and the same primary caregiver, one sibling was also randomly selected for interview. LA FANS Wave One interviews were conducted between April 2000 and January 2002, and included 3,090 households in 65 census

tracts, with 30%, 31% and 39% of the households in the very poor, poor and non-poor strata, respectively. The following analyses included 3,114 children (ages 0–17 years) with data on the health outcomes of interest and census tract of residence.

Outcome Assessment

Each child's primary caregiver (PCG) was interviewed about the child's health status, including the presence of asthma. Specifically, the PCGs were asked "Has a doctor or other health professional ever told you that [child's name] has asthma?". Those who responded 'yes' were also asked "During the past 12 months has [child's name] had an episode of asthma or an asthma attack?". The responses to these questions were used to define children as asthmatics with attacks in the previous 12 months, asthmatics without attacks in the previous 12 months, or nonasthmatics.

Exposure Assessment

Exposure to outdoor air pollution was assessed based on measurements collected by the South Coast Air Quality Management District (SCAQMD) at a network of air monitoring stations located throughout the county, using methods similar to those employed in our previous studies (Ritz and Yu, 1999, Ritz et al., 2000, Ritz et al., 2002, Wilhelm and Ritz, 2005). Specifically, the locations of existing air monitoring stations were mapped and overlaid with a map of LA FANS residential neighborhoods (census tracts) using ArcView GIS software (Version 3.3; ESRI, Redlands, CA). Using U.S. Census Block (2000) population densities, we also located the population-weighted centroid of each census tract. The final overlay was an elevation map for the air basin. The LA FANS residential census tracts (based on year 2000 boundaries) were then linked (manually) to existing monitoring stations taking into account not only distance, but geographical factors and the population distribution within tracts. For example, in cases where major geographical features (mountains) lay in between the nearest monitoring station and a tract, a linkage was not made. The next nearest station was used, or in some cases, the tract was left unlinked (see below). For larger census tracts, the population-weighted centroid helped confirm whether the majority of the population was in relatively close proximity to the nearest station even though other (less populated) portions of the tract were much farther away. This only impacted two census tracts, since most of the study region was urbanized with small tracts (average area of 3.9 km²). There were 14 CO stations, 15 NO_2 and O_3 stations, 10 $PM_{2.5}$ stations and 8 PM₁₀ stations with measurement data available for the study period. On average, population-weighted census tract centroids were 6.8 km from CO, $NO₂$, and $O₃$ monitoring stations (range of $0.23-15.4$ km), 8.2 km from PM_{2.5} stations (range of $0.23-25.7$ km), and 9.7 km from PM_{10} stations (range of 0.23–24.3 km).

For each subject, we estimated annual average concentrations of carbon monoxide (CO), $NO₂, O₃, PM₁₀$ and $PM_{2.5}$ for the one-year period prior to the interview date (to correspond to the time period for assessment of asthma symptoms) based on hourly measurements for the gaseous pollutants (CO, $NO₂$, and $O₃$); 24-hour average measurements taken daily or every 6 and 3 days were used for PM_{10} and $PM_{2.5}$, respectively. Two (out of 90) census tracts were not linked to a monitoring station because the census tracts were located too far from and/or in a different geographic region (mountain or desert) than the nearest monitoring station (resulting in exclusion of 67 children). One additional census tract was not assigned to a $PM_{2.5}$ station for the same reasons (resulting in 93 children with missing $PM_{2.5}$ data).

Individual, Family and Neighborhood Level Risk Factors

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 and one or more well visits during the previous 12 months (Table 1).

At the family level, we examined the following measures of socioeconomic status (SES): family income (as percent of the federal poverty limit [FPL]), homeowner status (yes/no), nonhousing assets, use of public assistance in previous 12 months, primary caregiver (PCG) education (years), and parents' employment status. 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 1 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 1 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 and level of closeness to neighbors. Based on the design of the LA FANS survey, these perception questions were answered by the PCG for 52% of the families, by the PCG's spouse or partner for 28% of the families and by another adult in the household for 14% of the families (6% had missing data on this variable). The neighborhood definition was not specified for these responses, i.e., responses pertained to each individual's own definition of their neighborhood. Finally, we examined the importance of PCG nativity (US- or foreign-born), family type (single or dual parent family), PCG current martial status, and PCG current smoking status.

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, and percent male unemployment. Similar to Cohen et al. (2006), a census cross-walk was used to relate 2000 data to 1990 tract boundaries (the original sampling frame for LA FANS). Other censusbased 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) (Prentice, 2006). Finally, we averaged the neighborhood cohesion scores and opinions of neighborhood safety across all LA FANS adults living in the same census tract as additional measures of neighborhood quality.

Statistical analysis

To examine associations between air pollution and individual, family and neighborhood characteristics, we calculated correlation coefficients and performed a factor analysis (using principal components analysis for initial factor extraction and varimax rotation). We used logistic regression to evaluate associations between outdoor air pollution and asthma, comparing asthmatics with and without exacerbations to nonasthmatics, and also asthmatics with exacerbations to asthmatics without exacerbations. 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 evaluated univariate associations and 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 into the models. We adjusted for the following variables in all models based on subject-matter criteria, correlations between variables, and impact on estimates (i.e., whether their inclusion changed associations estimates by at least 10% (Rothman and Greenland, 1998): child's age, gender, race/ethnicity, and insurance status, homeowner status (yes/no), PCG education, PCG marital status, PCG nativity,

and number of relatives in neighborhood. This research was approved by the UCLA Office for Protection of Research Subjects.

Results

Of the 3,114 LA FANS Wave One children included in our analyses, 345 had ever received a doctor's diagnosis of asthma (11%) and 144 of these reported suffering asthma attacks in the past 12 months (42%), according to PCG interview responses. Table 1 shows univariate associations between individual, family and neighborhood-level characteristics and asthma. At the individual level, likelihood of reporting an asthma diagnosis and attacks in the previous 12 months was higher for boys and African American children, and increased with age. The odds of reporting asthma with attacks was lower for Hispanic children, but not the odds of having received an asthma diagnosis. Those who were uninsured or only partially insured during the previous year and children of foreign-born PCGs had lower odds of asthma, especially asthma with attacks. Based on family-level characteristics, children with lower SES (measured by family income, homeowner status, non-housing assets, and PCG education level) were less likely to have asthma with attacks reported than higher SES children. This pattern was not evident or not as strong for asthma without attacks. Children in families reporting low neighborhood support had higher odds of both asthma outcomes, while those from families reporting no relatives living in the neighborhood were only more likely to report asthma with attacks. Children from single parent families and unmarried families had higher odds of both asthma outcomes, as did children of PCGs who reported smoking. Based on neighborhoodlevel characteristics, children living in less cohesive and safe neighborhoods (based on the average opinion of all LA FANS adults in a given census tract) and with more economic disadvantage (based on US Census data also averaged at the census tract level) had lower odds of asthma with attacks being reported. A similar pattern emerged for asthma without attacks, but relations were not as strong. Odds of both asthma outcomes appeared elevated for children living in census tracts where there was not one predominant race/ethnicity.

Associations between Family and Neighborhood-Level Characteristics and Air Pollution

Correlation coefficients and a factor analysis were used to evaluate relations among annual average air pollution exposure estimates and selected family and neighborhood level sociodemographic variables (see Table 2 for factor analysis results; correlation coefficients are discussed below). Extraction using principal components followed by varimax rotation suggest the 22 selected variables can be summarized by three factors. The first "neighborhood quality" factor reflects both subjective (opinions of cohesion and safety at the neighborhood and family level) and objective (neighborhood level economic disadvantage, family level income and nonhousing assets) measures of neighborhood quality. Pearson correlations suggested that CO and NO2 levels tended to increase as neighborhood quality decreased, while the opposite trend was observed for O_3 , but overall the air pollution metrics were not strongly correlated with the neighborhood quality measures (r~0.3 or lower). The second "air pollution" factor reflects the relatively strong correlations between average pollutant concentrations, especially for CO, NO₂ and O₃. While CO and NO₂ were positively correlated (Pearson correlation coefficient (r) of 0.67), both were negatively correlated with O₃ (r~−0.7). Correlations between PM_{2.5} and PM_{10} and these pollutants, however, were more moderate (PM2.5: r~0.4 for CO and NO₂ and −0.4 for O3; PM10: r=0.49 for NO2, 0.25 for CO and −0.23 for O3). The third "neighborhood support, social ties" factor reflected the correlations among family level ratings of neighborhood support and social ties/networks. Air pollutant levels were not correlated with family ratings of neighborhood support and social ties reflected in both low loadings of the air pollution variables on this factor and low Pearson correlation coefficients (r of 0.16 or lower). Two variables, family level opinion of neighborhood cohesion and number of relatives living

in the neighborhood loaded similarly on the first and third factors suggesting these variables represented aspects of both groupings.

Spatially, neighborhoods in the downtown, urban core areas of the County had the highest CO and $NO₂$ levels, while $O₃$ was elevated in the more eastern and south-eastern parts of the County. Levels of PM_{10} were most elevated in the eastern part of the County, especially in the south east, but also somewhat elevated in the downtown urban area. Levels of $PM_{2.5}$ were elevated in the urban core, but also in the south east. Although "neighborhood quality" as defined by variables such as the census tract level disadvantage score tended to be lowest in the downtown urban core, there were also other areas characterized by higher deprivation, including some areas of the San Fernando Valley and near the Harbors. The overall low correlations between the "neighborhood quality", "air pollution", and "neighborhood support" factors described above suggest that SES, social support, and air pollution follow complex patterns in LA County. These factors identified in our analyses only explained about 45% of the variance of the original variables. Thus, rather than relying on such weak proxies in our logistic regression models for asthma, we considered the relations identified between variables loading on the same factor when constructing our final most parsimonious adjusted model.

Associations between Air Pollution and Reported Asthma

When comparing children with doctor-diagnosed asthma and reports of one or more attacks in the previous year to nonasthmatics, we estimated an approximately 64% increase in odds of this outcome per 1 pphm increase in annual average O_3 in an unadjusted model (OR=1.64, 95% CI=1.15–2.33). However, adjustment for home ownership, insurance status of the child, PCG nativity, education, and marital status, and the family's report of relatives living in their neighborhood reduced the estimate to essentially a null finding (Table 3). Adding PM_{10} and $PM_{2.5}$ to the model did not change this result (CO, NO₂ and O₃ concentrations were considered too highly correlated to be included in the same models).

We also estimated a 46% increase in odds of reporting a doctor-diagnosis of asthma without attacks in the previous year per 1 pphm increase in O_3 based on a crude model (OR=1.46, 95%) CI=1.07–1.99). However, this effect estimate did not change appreciably when any of the variables noted above were added (Table 3). Adjusting for all variables simultaneously and adding PM_{10} and $PM_{2.5}$ to the model resulted in an OR essentially the same as the crude result but less precise (OR=1.45, 95% CI=0.93–2.25). Most of the association between O_3 and asthma without attacks appeared isolated to those children experiencing very high exposures (greater than the 90th percentile of 2.38 pphm). Very weak crude associations between PM_{10} and asthma without attacks in the previous 12 months increased when we added O_3 and PM_2 , to the model (OR=1.46, 95% CI=0.96–2.22) (Table 3). When focusing on children most highly exposed to PM_{10} (>45.9 µg/m³), we observed an 86% increase in the odds of asthma without attacks in the previous 12 months (OR=1.86, 95% CI=1.13–3.08).

In a sub-analysis, we compared asthmatics with attacks to asthmatics without attacks in the past year and observed associations that distinguished these two groups only for CO. We estimated a 57% increase in odds of attacks in the previous 12 months per 1 ppm increase in annual average CO among asthmatic children (OR=1.57, 95% CI=0.71–3.48) (Table 4). Adjustment for race/ethnicity and age increased this estimate to a 64% increase per 1 ppm CO. Further adjustment for home ownership, child's insurance status, PCG nativity, education level and marital status, and whether the family had relatives in the neighborhood did not change the estimate appreciably. Adjusting for these variables simultaneously, we estimated an approximately 2-fold increase in risk of attacks in the previous 12 months per 1 ppm increase in CO (OR=2.33, 95% CI=1.03–5.25). Similar to what we observed for O_3 and PM₁₀, most of the association between CO and asthma attacks appeared isolated to children with very high exposures (greater than the 90th percentile of 1.77 ppm).

Discussion

Using the LA FANS dataset in combination with US Census and government air monitoring data, we were able to examine associations between outdoor air pollution and a large number of family- and neighborhood-level characteristics in Los Angeles neighborhoods. In general, we found that CO and $NO₂$ levels increased and $O₃$ levels decreased with decreasing neighborhood quality, assessed based on objective measures such as economic disadvantage and subjective measures such as average ratings of neighborhood safety and cohesion by LA FANS participants. However, correlations between these factors and pollution levels were fairly low (r~0.3 or less). Air pollution levels were not correlated with family-level ratings of neighborhood support and level of social ties and networks.

Based on our models, LA FANS children living in high O_3 areas had higher odds of doctordiagnosed asthma but not higher odds of reporting attacks in the previous 12 months. Similar to O_3 , associations between PM_{10} levels and asthma were limited to asthmatics without attacks in the previous 12 months, and the very highly exposed (i.e., exposures above the $90th$ percentile of 45.9 μ g/m³). When limiting comparisons to diagnosed asthmatics only, children living in high CO areas had higher odds of reporting attacks in the previous 12 months. Since CO is emitted directly by motor vehicles and does not readily react in the atmosphere to form other compounds, it is often considered a marker for the suite of pollutants released in exhaust. Measurement data indicate levels of CO are spatially correlated with other exhaust constituents, such as ultrafine particles in the LA Basin (Zhu et al., 2002a, Zhu et al., 2002b). There is a growing literature linking various traffic metrics to asthma symptoms and exacerbations (Salam et al., 2008, Brauer et al., 2007, Bayer-Oglesby et al., 2006, Gauderman et al., 2007). Thus, the observed associations with CO could reflect toxic action of unmeasured traffic exhaust compounds.

We also performed analyses stratifying on the census tract level indicators of neighborhood quality and two family-level measures of social support (number of relatives in the neighborhood and the overall neighborhood social support rating). When stratifying on median values of the census tract level variables, in general, O_3 effect estimates for asthmatics without attacks in the previous 12 months appeared greater in neighborhoods considered to be more cohesive and safe and with lower economic disadvantage, although 95% CIs for most stratumspecific estimates overlapped widely due to our limited sample size. The strongest difference was observed for the census tract level rating of neighborhood cohesion; we estimated a 2-fold increase in odds of asthma without attacks per 1 pphm increase in O_3 (OR=2.07, 95% CI=1.09– 3.93) for children living in neighborhoods considered to be more cohesive and essentially no association with O_3 for children living in less cohesive neighborhoods (OR=1.03, 95%) CI=0.52–2.02). This could be indicative of better reporting by parents and/or less exposure misclassification (due to less residential mobility) among subjects in high SES areas. We did not observe differences in effect estimates when stratifying on median values of the social support variables.

One limitation of the present analyses is the cross-sectional nature of the data; specifically, potential bias caused by temporal ambiguity between exposure and disease. We did not have lifetime residential histories for these children (we assigned monitoring stations based on current home location) and did not know the date of asthma diagnosis. The magnitude of resulting bias depends on the residential mobility patterns of the LA FANS children. Information on residential history was collected for the two years prior to the interview date. Based on these data, approximately 67% of the children lived regularly with the interviewed family and did not move during this period. Of the approximately 27% of children who lived regularly with a family that did move, only 35% moved to a different census tract (~9% of total families) and only 13% moved to a census tract with a different air monitor assignment (~4%

Wilhelm et al. Page 8

of total families) based on residential data for the year prior to the interview. Based on these data, the majority (94%) of children lived in the same home for at least a year prior to interview or if they did move, tended to stay in the same census tract or in the same "monitoring area". When we stratified on the median value of our census measure of residential stability (i.e., percent of tract population living in the same home as 5 years ago), point estimates for CO and to a lesser extent PM_{10} were greater for those living in neighborhoods with greater stability. This suggests that exposure misclassification (if assumed non-differential) may be impacting our estimates. Ozone effect estimates were largely similar when stratifying on residential stability, which may reflect the spatially more homogeneous distribution of this pollutant.

A second issue is the outcome assessment which relied on parental reports of health care provider-diagnosed asthma and attacks in the previous 12 months. The higher odds of these outcomes for wealthier, educated individuals with healthcare could reflect under-reporting among the more disadvantaged. The strong negative relation between PCG nativity and reporting asthma with attacks (and to a lesser extent also for asthma without attacks) suggests that under-reporting may be an issue especially for children of foreign-born parents. Based on data from a LA county-wide random sample of 6,004 children collected during September 1999-April 2000 using the same questions regarding asthma, Simon et al. (2003) reported the highest prevalence of asthma among black children (15.8%), followed by whites (7.3%) and Asians (6%), and the lowest among children of Latino ethnicity (3.9%); differences persisted after adjustment for income, measures of health care access, and other covariates. Asthma prevalence was inversely related to income in all racial/ethnic groups except for Spanishspeaking Latinos. The authors concluded the lower prevalence and lack of an association with income among Latino children from Spanish-speaking households could be due to either health-protective influences in the family and community in the less acculturated or a higher level of undiagnosed asthma or less ability to communicate a health care provider's asthma diagnosis in an interview format. For LA FANS children, Sastry and Pebley (2003) previously reported that Latino children were more likely to be rated by care givers as being in fair health compared to whites and Asians, despite the lower percentages of Latino children reported to have had asthma, asthma attacks in the past 12 months and chronic ear infections than all other children. Further analysis based on mother's place of birth showed that Latino children from US-born or non-Mexican-born mothers were more likely to be rated as being in excellent health compared to children of Mexican-born mothers, while the latter had the highest percentage of children in fair health. However, Latino children from US-born or non-Mexican born mothers had an asthma and chronic ear infection prevalence similar to or slightly higher than whites, while children of Mexican-born mothers again had a lower prevalence. Assuming that ear infections and asthma make an important contribution to overall child health, this contradictory observation suggests some reporting bias may be present among recent Mexican immigrants.

Relying on ambient air monitoring stations to assess air pollution exposure likely resulted in exposure misclassification, especially for pollutants that are known to have concentrations that vary over short distances such as CO and related exhaust toxins (e.g., ultrafine particles) (Sioutas et al., 2005, Zhou and Levy, 2007). We have discussed this issue extensively in previous reports (Ritz and Yu, 1999, Ritz et al., 2000, Wilhelm and Ritz, 2003, Wilhelm and Ritz, 2005). We were also missing data on some potentially important confounders such as sources of indoor allergens (e.g., pets and molds). In a previous study of Southern Californian children, adjustment for presence of one or more pets in the home, mildew or cockroaches did not substantially alter reported cross-sectional relations between community-level measures of air pollution (PM_{10} and NO_2) and bronchitic symptoms in asthmatic children after adjustment for age, sex, race/ethnicity, school grade, and membership in a health insurance plan (McConnell et al., 1999). In regards to assessing environmental tobacco smoke exposure, our data was limited to knowing whether the PCG was a current smoker and we did not know which other household members also smoked. However, children may be most exposed to

second-hand smoke from PCGs and our data showed strong associations with PCG smoking status among children who had suffered asthma attacks in the past 12 months and to a lesser extent for asthmatic without attacks.

A second LA FANS survey (Wave Two) that includes families who participated in Wave One plus a sample of new entrants into these neighborhoods is currently being conducted. The asthma outcome assessment has been expanded to include date of diagnosis, symptoms in addition to diagnoses, family history of asthma, and measures of lung function using portable spirometers in children 5 years and older. In addition, questions on residential history and the home environment have been added. In conjunction with this interview, we are collecting measurements of nitrogen oxides (NO_x) at approximately 200 locations throughout the LA FANS neighborhoods as a marker of vehicle exhaust and these data will be used to generate air pollution exposure surfaces for the children in Wave Two through land use-based regression (LUR) modeling (e.g., Sahsuvaroglu et al., 2006). Thus, we will be able to address the limitations noted above in future analyses.

Despite the limitations noted, the LA FANS data provide a unique way to look at environmental exposures in conjunction with other potentially important co-factors for asthma. Since asthma is a multifactorial disease, information on family and neighborhood-level characteristics that may be indicative of psychosocial stress and other risk factors are needed if these either confound the air pollution relations or increase susceptibility to air pollutants in children. After adjusting for a variety of SES and psychosocial factors at the family and neighborhood level, we still observed increased odds of asthma for children residing in areas of high air pollution in LA. The LA FANS Wave Two data will increase our ability to examine the interplay of these factors on asthma in disadvantaged children, i.e. to assess effect measure modification due to psychosocial factors.

Conclusions

In LA FANS both outdoor air pollution and a number of family and neighborhood-level characteristics were associated with asthma in children. In general, we found that CO and $NO₂$ levels increased and $O₃$ levels decreased as neighborhood quality decreased. However, correlations between these factors and pollution levels were fairly low (r~0.3 or less). Air pollution levels were not correlated with family ratings of neighborhood support and level of social ties and networks. Based on our models, LA FANS children living in areas with high O_3 and PM₁₀ levels had greater odds of reporting doctor-diagnosed asthma without attacks in the previous 12 months. However, similar relations were not observed for doctor-diagnosed asthma with attacks in the previous 12 months. This may be due to under-reporting of diagnoses and symptoms among certain subpopulations. We also found that LA FANS children diagnosed with asthma and living in areas with high CO levels had greater odds of reporting attacks in previous 12 months. Since CO is directly emitted from motor vehicles and does not readily react in the atmosphere to form other compounds, this association may be indicative of the influence of a co-occurring pollutant or mixture of pollutants in traffic exhaust. We will be able to examine these questions further once Wave Two data collection is complete.

Acknowledgements

This work was supported by the RAND Center for Population Health and Health Disparities (NIEHS Grant 1 P50 ES012383) and by the Southern California Environmental Health Sciences Center (NIEHS Grant 5 P30 ES07048-07). We thank the LA FANS Principal Investigators – Anne Pebley (UCLA) and Narayan Sastry (RAND and University of Michigan) – for their collaboration and input. We also thank Katherine Hoggatt (University of Michigan) for statistical advice and Jo Kay Ghosh for help with editing.

References

- Bayer-Oglesby L, Schindler C, Hazenkamp-von Arx M, Braun-Fahrlander C, Keidel D, Rapp R, Kunzli N, Braenli O, Burdet L, Liu LJS, Leuenberger P, Ackermann-Liebrich U, the SAPALDIA Team. Living near main streets and respiratory symptoms in adults: the Swiss Cohort Study on Air Pollution and Lung Diseases in Adults. Am J Epidemiol 2006;164:1190–1198. [PubMed: 17032694]
- Brauer M, Hoek G, Smit H, de Jongste JC, Gerritsen J, Postma DS, Kerkhof M, Brunekreef B. Air pollution and the development of asthma, allergy and infections in a birth cohortc. European Respir J 2007;29:879–888. [PubMed: 17251230]
- Brunekreef B, Holgate ST. Air pollution and health. Lancet 2002;360:1233–1242. [PubMed: 12401268]
- Chen E, Hanson MD, Paterson LQ, Griffin MJ, Walker HA, Miller GE. Socioeconomic status and inflammatory processes in childhood asthma: The role of psychological stress. J Allergy Clin Immunol 2006;117:1014–1020. [PubMed: 16675327]
- Cohen DA, Finch BK, Bower A, Sastry N. Collective efficacy and obesity: The potential influence of social factors on health. Social Science and Medicine 2006;62:769–778. [PubMed: 16039767]
- English P, Neutra R, Scalf R, Sullivan M, Waller L, Zhu L. Examining Associations between Childhood Asthma and Traffic Flow Using a Geographic Information System. 1999;107:761–767.
- Gauderman WJ, Avol E, Lurmann F, Kuenzli N, Gilliland F, Peters J, McConnell R. Childhood asthma and exposure to traffic and nitrogen dioxide. Epidemiology 2005;16:737–743. [PubMed: 16222162]
- Gauderman WJ, Vora H, McConnell R, Berhane K, Gilliland F, Thomas D, Lurmann F, Avol E, Kunzli N, Jerrett M, Peters J. Effect of exposure to traffic on lung development from 10 to 18 years of age: a cohort study. Lancet 2007;369:571–577. [PubMed: 17307103]
- Gilmour MI, Jaakola MS, London SJ, Nel AE, Rogers CA. How exposure to environmental tobacco smoke, outdoor air pollutants, and increased pollen burdens influences the incidence of asthma. Environmental Health Perspectives 2006;114:627–633. [PubMed: 16581557]
- Gold DR, Wright R. Population disparities in asthma. Annu Rev Public Health 2005;26:89–113. [PubMed: 15760282]
- Houston D, Wu J, Ong P, Winer AM. Structural Disparities of Urban Traffic in Southern California: Implications for Vehicle-related Air Pollution Exposure in Minority and High-poverty Neighborhoods. Journal of Urban Affairs 2004;26:565–592.
- Kim JJ, Smorodinsky S, Lipsett M, Singer B, Hodgson AT, Ostro B. Traffic-related air pollution near busy roads: the East Bay Children's Respiratory Health Study. Am J Resp Crit Care Med 2004;170:520–526. [PubMed: 15184208]
- Kohlhuber M, Heinrich J, van den Hazel P, Zuurbier M, Bisrup ML, Koppe JG, Bolte G. Children's environmental health: why should social disparities be considered? Acta Paediatr Suppl 2006;95:26– 30. [PubMed: 17000566]
- Kunzli N, McConnell R, Bates DV, Bastain T, Hricko A, Lurmann F, Avol E, Gilliland F, Peters J. Breathless in Los Angeles: The Exhausting Search for Clean Air 2003;93:1494–1499.
- Li N, Sioutas C, Cho A, Schmitz D, Misra C, Sempf JM, Wang MTDO, Froines J, Nel AE. Ultrafine particulate pollutants induce oxidative stress and mitochondrial damage. 2003;111:455–460.
- Li N, Wang MTDO, Sempf JM, Nel AE. Comparison of the pro-oxidative and proinflammatory effects of organic diesel exhaust particle chemicals in bronchial epithelial cellS and macrophages. 2002;169:4531–4541.
- McConnell R, Berhane K, Gilliland F, London SJ, Vora H, Avol EWJGHGM, Lurmann F, Thomas DC, Peters JM. Air pollution and bronchitic symptoms in Southern California children with asthma 1999;107:757–760.
- McConnell R, Berhane K, Yao L, Jerrett M, Lurmann F, Gilliland F, Kunzli N, Gauderman J, Avol E, Thomas D, Peters J. Traffic, susceptibility, and childhood asthma. Environ Health Perspect 2006;114:766–772. [PubMed: 16675435]
- Mielck A, Reitmeir P, Wjst M. Severity of childhood asthma by socioeconomic status. Int J Epidemiol 1996;25:388–393. [PubMed: 9119565]
- Miller GE, Chen E. Life stress and diminished expression of genes encoding glucocorticoid receptor and B2-adrenergic receptor in children with asthma. PNAS 2006;103:5496–5501. [PubMed: 16567656]

- O'Neill MS, Jerrett M, Kawachi I, Levy JI, Cohen AJ, Gouveia N, Wilkinson P, Fletcher T, Cifuentes L, Schwartz J. Health, wealth, and air pollution: advancing theory and methods. Environmental Health Perspectives 2003;111:1861–1870. [PubMed: 14644658]
- Pandya RJ, Solomon G, Kinner A, Balmes JR. Diesel exhaust and asthma: hypotheses and molecular mechanisms of action. Environ Health Perspect 2002;110:103–112. [PubMed: 11834468]
- Prentice J. Neighborhood effects on primary care access in Los Angeles. Social Science and Medicine 2006;62:1291–1303. [PubMed: 16129534]
- Ritz B, Yu F. The effect of ambient carbon monoxide on low birth weight among children born in Southern California between 1989 and 1993. 1999;107:17–25.
- Ritz B, Yu F, Chapa G, Fruin S. Effect of air pollution on preterm birth among children born in Southern California between 1989 and 1993. 2000;11:502–511.
- Ritz B, Yu F, Fruin S, Chapa G, Shaw GM, Harris JA. Ambient air pollution and risk of birth defects in Southern California. American Journal of Epidemiology 2002;155:17–25. [PubMed: 11772780]
- Rothman, KJ.; Greenland, S. Modern Epidemiology. Philadephia: Lippincott-Raven; 1998.
- Sahsuvaroglu T, Arain A, Kanaroglou P, Finkelstein N, Newbold B, Jerrett M, Beckerman B, Brook J, Finkelstein M, Gilbert N. A land use regression model for predicting ambient concentratons of nitrogen dioxide in Hamilton, Ontario, Canada. J Air Waste Manage Assoc 2006;56:1059–1069.
- Salam MT, Islam T, Gilliland F. Recent evidence for adverse effects of residential proximity to traffic sources on asthma. Curr Opin Pulm Med 2008;14:3–8. [PubMed: 18043269]
- Sandberg A, Jarvenpaa S, Penttinen A, Paton JY, McCann DC. Asthma exacerbations in children immediately following stressful life events: a Cox's hierarchical regression. Thorax 2004;59:1046– 1051. [PubMed: 15563703]
- Sastry N, Ghosh-Dastidar B, Adams J, Pebley A. The Design of a Multilevel Survey of Children, Families, and Communities: The Los Angeles Family and Neighborhood Survey. Soc Sci Res 2006;35:1000– 1024.
- Sastry, N.; Pebley, A. Neighborhood and Family Effects on Children's Health in Los Angeles. Santa Monica, CA: RAND; 2003. Labor and Population Program, Working Paper Series 03-25
- Simon PA, Zeng Z, Wold CM, Haddock W, Fielding JE. Prevalence of childhood asthma and associated morbidity in Los Angeles County: Impacts of race/ethnicity and income. Journal of Asthma 2003;40:535–543. [PubMed: 14529103]
- Sioutas C, Delfino RJ, Singh M. Exposure assessment for atmospheric ultrafine particles (UFPs) and implications in epidemiologic research. Environ Health Perspect 2005;113:947–955. [PubMed: 16079062]
- Thurston GD, Bates DV. Air pollution as an underappreciated cause of asthma symptoms. 2003;290:1915–1917.
- Trasande L, Thurston GD. The role of air pollution in asthma and other pediatric morbidities. J Allergy Clin Immunol 2005;115:689–699. [PubMed: 15805986]
- Wilhelm M, Ritz B. Residential proximity to traffic and adverse birth outcomes in Los Angeles County, California, 1994–1996 2003;111:207–216.
- Wilhelm, M.; Ritz, B. Local variations in CO and particulate air pollution and adverse birth outcomes in Los Angeles County. Vol. 113. California, USA: 2005. p. 1212-1221.
- Wright RJ. Stress and atopic disorders. J Allergy Clin Immunol 2005;116:1301–1306. [PubMed: 16337463]
- Wright RJ, Cohen S, Carey V, Weiss ST, Gold DR. Parental stress as a predictor of wheezing in infancy: a prospective birth-cohort study. Am J Resp Crit Care Med 2002;165:358–365. [PubMed: 11818321]
- Wright RJ, Finn P, Contreras JP, Cohen S, Wright RO, Staudenmayer J, Wand M, Perkins D, Weiss ST, Gold DR. Chronic caregiver stress and IgE expression, allergen-induced proliferation, and cytokine profiles in a birth cohort predisposed to atopy. J Allergy Clin Immunol 2004;113:1051–1057. [PubMed: 15208584]
- Zhou Y, Levy JI. Factors influencing the spatial extent of mobile source air pollution impacts: a metaanalysis. BMC Public Health 2007;7:89–99. [PubMed: 17519039]
- Zhu YF, Hinds WC, Kim S, Shen S, Sioutas C. Study of ultrafine particles near a major highway with heavy-duty diesel traffic. Atmosperic Environment 2002a;36:4323–4335.

Zhu YF, Hinds WC, Kim S, Sioutas C. Concentration and size distribution of ultrafine particles near a major highway. 2002b;52:1032–1042.

 NIH-PA Author Manuscript NIH-PA Author Manuscript

Table 1
Number (Percent) of Subjects in Each Outcome Group by Individual-, Family-, and Neighborhood-Level Demographic Characteristics Number (Percent) of Subjects in Each Outcome Group by Individual-, Family-, and Neighborhood-Level Demographic Characteristics and Crude Odds Ratios (95% CI) for Asthma and Crude Odds Ratios (95% CI) for Asthma

Health Place. Author manuscript; available in PMC 2010 March 1.

 $N = \frac{1}{2}$ (32.9) $N = \frac{1}{2}$ (32.9) $N = \frac{1}{2}$ (32.9) $N = \frac{1}{2}$ (32.9) $N = \frac{1}{2}$ (32.9) 17

Wilhelm et al. Page 14

Wilhelm et al. Page 15

Wilhelm et al. Page 16

Ŕ

Í

incording to

Wilhelm et al. Page 17

 $^{(4)}$ This is the average of the neighborhood safety responses for all adult respondents in a given census tract using the following numeric responses for each response: 1=completely safe, 2=fairly safe, 3=somewhat danger $^{(4)}$ This is the average of the neighborhood safety responses for all 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.

⁽⁵⁾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, percent male ⁵⁵This 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, percent male unemployment.

 $^{(6)}$ Based on U.S. Census 2000 data. *(6)*Based on U.S. Census 2000 data.

Varimax rotation, principal component factors for annual average pollution concentrations and family- and census tract- level sociodemographic variables Varimax rotation, principal component factors for annual average pollution concentrations and family- and census tract- level sociodemographic variables

 $\ensuremath{^{(1)}}\xspace$ High values for these measures indicate low ratings. $\binom{1}{1}$ High values for these measures indicate low ratings.

⁽²⁾The mean (range) of annual average air pollution concentrations were: CO: 1.1 (0.33-2.2) ppm; NO₂ 3.5 (1.6-5.1) pphm; O3 1.9 (1.1-3.8) pphm; PM_{2.5} 20.7 (10.6-26.7) µg/m³; PM₁₀ 40.8 (26.5- $^{2/2}$ The mean (range) of annual average air pollution concentrations were: CO: 1.1 (0.33–2.2) ppm; NO₂ 3.5 (1.6–5.1.) pphm; O3 1.9 (1.1–3.8) pphm; PM2.5 20.7 (10.6–26.7) µg/m³; PM₁₀ 40.8 (26.5–

55.6) µg/m3

 $^{(3)}$ These variables are dichomotously coded such that a high value (1) means less support (see also Table 1). *(3)*These variables are dichomotously coded such that a high value (1) means less support (see also Table 1).

Table 3

Association (Odds Ratio, 95% CI) between annual average O₃ and PM ₁₀ concentrations and asthma among LA FANS participants ages 0–17 years

Table 4

Association (Odds Ratio, 95% CI) between annual average CO concentrations and asthma attacks among LA FANS participants ages 0–17 years

