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Annual average ambient particulate matter exposure estimates, measured home particulate matter, and hair nicotine are associated with respiratory outcomes in adults with asthma ☆

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ABSTRACT

Background: While exposure to outdoor particulate matter (PM) has been associated with poor asthma outcomes, few studies have investigated the combined effects of outdoor and indoor PM (including secondhand tobacco smoke).
Objective: To examine the associations between PM and asthma outcomes.
Methods: We analyzed data from a cohort of adults with asthma and rhinitis ($n=302$; 82% both conditions; 13% asthma only; 5% rhinitis alone) including measures of home PM, tobacco smoke exposure (hair nicotine and self-report), ambient PM from regional monitoring, distance to roadway, and season (wet or dry). The outcomes of interest were frequent respiratory symptoms and forced expiratory volume in 1 second (FEV_1) below the lower limit of normal (NHANES reference values). Multivariable regression analyses examined the associations (Odds Ratio [OR] and 95% Confidence Interval [95%CI]) between exposures and these outcomes, adjusted by sociodemographic characteristics.
Results: In adjusted analyses of each exposure, the highest tertile of home PM and season of interview were associated with increased odds for more frequent respiratory symptoms (OR=1.64 95%CI: [1.00, 2.69] and OR=1.66 95%CI: [1.09, 2.51]). The highest tertile of hair nicotine was significantly associated with FEV_1 below the lower limit of normal (OR=1.80 95%CI: [1.00, 3.25]). In a model including home PM, ambient PM, hair nicotine, and season, only two associations remained strong: hair nicotine with FEV_1 below the lower limit of normal and season of measurement (dry, April–October) with increased respiratory symptoms (OR=1.85 95%CI: [1.00, 3.41] and OR=1.54 95%CI: [1.0, 2.37]). When that model was stratified by sex, the highest tertiles of ambient PM and hair nicotine were associated with FEV_1 below the lower limit of normal among women (OR=2.23 95%CI: [1.08, 4.61] and OR=2.90 95%CI: [1.32, 6.38]), but not men. The highest tertile of hair nicotine was also associated with increased respiratory symptoms in women but not men (OR=2.38 95% CI: [1.26, 4.49]). When stratified by age, the middle quartile of ambient PM and the highest hair nicotine tertile were associated with increased respiratory symptoms (OR=2.07 95%CI: [1.01, 4.24] and OR=2.55 95% CI: [1.21, 5.36]) in those under 55 but not in the older stratum.
Conclusions: Exposure to PM from both home and ambient sources is associated with increased symptoms and lower lung function in adults with asthma, although these associations vary by type of PM, the respiratory outcome studied, sex and age.

1. Introduction

Exposure to outdoor particulate matter (PM) has been associated in multiple epidemiological studies with increased risk of various asthma outcomes, including symptoms (McConnell et al., 2003), short-term decreases in lung function (Delfino et al., 2004), and clinical exacerbations leading to health care utilization (Barnett et al., 2005). The evidence for such effects, however, is more robust for children than for adults with asthma (Patel and Miller, 2009).

Abbreviations: EPA, US Environmental Protection Agency; FEV_1, forced expiratory volume in 1 second; ICD-9, International Classification of Diseases, 9th revision; NHANES III, Third National Health and Nutrition Examination Survey; 95%CI, 95 percent confidence interval; OR, odds ratio; PM, particulate matter; $PM_{2.5}$, fine particulate matter with a mass median aerodynamic diameter <2.5 μm; PM_{10}, particulate matter with a mass median aerodynamic diameter <10 μm; $PM_{10-2.5}$, coarse particulate matter

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The health impacts of outdoor fine PM ($PM_{2.5}$), largely from combustion sources, are generally considered to be greater than those of coarse fraction PM ($PM_{10-2.5}$) (Brunekreef and Forsberg, 2005). $PM_{10-2.5}$ can also be partly anthropogenic (Gent et al., 2009), but these larger particles tend to be of crustal or biological origin. Relatively few studies have specifically focused on $PM_{10-2.5}$ and asthma outcomes (Lin et al., 2002; Mann et al., 2010; Balmes et al., 2009).

Moreover, most studies of asthma and PM (whether fine or coarse) have relied on measurements at regional air quality monitoring stations to classify individual-level exposure. These stations are usually at a considerable distance from where the individuals being studied live and investigators typically do not integrate time away from the home into the exposure estimates derived from such monitoring data, thus leading to inherent exposure misclassification. Although individual-level exposure assessment with personal monitoring may be more precise, this is not feasible to implement widely and even when possible, such monitoring may not distinguish among various sources of PM, both indoor and outdoor.

Given such challenges, it is not surprising that few studies have investigated the effects on asthma of both outdoor and indoor PM. Of note, a study of subjects with asthma or chronic obstructive pulmonary disease in four European cities reported no consistent associations between lung function and 24-h average particle number or particle mass concentrations measured at central site monitoring stations, nor did the results change when home outdoor or home indoor concentrations of PM were substituted for the central site measurements (de Hartog et al., 2010).

To address this knowledge gap, we analyzed cross-sectional data from a cohort of adults with asthma for whom we have performed home visits yielding outdoor and indoor PM measurements as well as hair nicotine measurements as a biomarker for tobacco smoke exposure, an important source of indoor PM. In addition, we have geocoded residential addresses for the cohort, allowing estimates of exposure to regional ambient PM and distance to roadway as a surrogate for traffic. These multi-factorial PM exposure data provided the opportunity to take a more integrated approach to assessing the potential associations between various sources of PM exposure and asthma outcomes in adults. We hypothesized that both outdoor and indoor PM exposures would be associated with increased asthma symptoms and decreased lung function.

2. Methods

2.1. Study cohort and subject recruitment

We used data from an established cohort of adults with asthma and rhinitis collected by both structured interviews and, in a subset of the cohort, home visits assessing lung function, measuring indoor and outdoor PM exposure, and collecting a biomarker of secondhand tobacco smoke exposure. Geocoded residential addresses allowed linkage to ambient air quality monitoring data and calculation of distances to roadways of various sizes. This asthma cohort was established through the merger of two different study groups that were recruited separately and studied independently, but following an identical study protocol. The details of the recruitment, selection, and retention for the merged asthma and rhinitis cohort have been published previously (Chen et al., 2011; Trupin et al., 2013).

The flow of subject recruitment, retention, and integration into the single cohort is illustrated in Fig. 1. In the first of the two parent study groups, the Asthma Rhinitis Cohort, recruitment of adults with asthma, rhinitis (including allergic rhinitis, chronic sinusitis, hay fever, or chronic postnasal drip), or both conditions occurred through community-based sampling of physicians or random digit dial identification of adults with report of a physician′s diagnosis of these conditions. Subjects were between age 18 and 50 at the time of enrollment, and those with concomitant diagnoses of chronic bronchitis, chronic obstructive pulmonary disease, or emphysema were ineligible. In the second study group, labeled in the figure as the Severe Asthma Cohort, the subjects were originally recruited from adult members of Northern California Kaiser Permanente. This health care maintenance organization provides health care to 25–30% of the region′s population and has demographic characteristics representative of the general population except for at the extremes of income distribution. Recruitment for that group was based on hospitalization for asthma (International Classification of Diseases 9th revision [ICD-9] code 493.xx as the primary discharge diagnosis or as a secondary code linked to an acute asthma-related respiratory condition). Potential subjects with a primary or secondary discharge diagnosis of chronic bronchitis, emphysema, or chronic obstructive pulmonary disease were excluded. Both groups were recruited within the same northern California geographical region and for the integration were limited to baseline age of 18–60 at original recruitment. The merged cohort will be referred to here as the Asthma and Rhinitis Cohort. The study protocol was approved by the University of California San Francisco Committee on Human Research. Telephone interviews took place with verbal consent by agreement to participate; home visits included a written consent document.

2.2. Subject interviews

All participants underwent a single baseline structured telephone interview administered by trained personnel using computer-assisted interviewing software. The interviews were approximately 45 min in duration and were conducted in English, although Spanish language assistance was available when needed (only two of 549 interviews). The interviews elicited data on demographics, occupation, smoking, clinical symptoms, asthma and rhinitis medication usage, and activity limitations. As shown in Fig. 1, of 711 possible participants in the study, 549 (77%) completed the baseline interview. Follow-up interview status differed significantly between the two study groups from which subjects were recruited: 85% vs. 68%. Overall, those interviewed were approximately 4 years older and less likely to be current or former smokers compared with those not interviewed.

2.3. Home visits

A home study team conducted a one-time comprehensive survey of the home environment, at which spirometry was performed and hair collected for nicotine assay. Environmental data gathered during the home visit included measures of PM inside and outside the home. The study geographic range extended throughout northern California from Fresno to the Oregon and Nevada borders, including urban, suburban, and rural dwellers. Excluding those who had moved out of this range since their original study recruitment ($n=53$), 496 subjects were eligible for home visits. A total of 302 visits were ultimately completed, 61% of those eligible (see Fig. 1). The median time elapsed between interview and home visit was 6.1 weeks.

The home interviews took place from April 2008 through September 2009. The average time elapsed between the telephone interview (at which symptoms were ascertained) and the home visit (at which lung function was measured) was 66 ± 68 days (median $=43$ days). In terms of seasonality (defined as wet [November–March] or dry [April–October] season consistent with northern California climatic conditions), 60% of the interviews and 63% of the home visits, respectively, took place during the dry season.

2.4. Exposure variables

2.4.1. Particulate matter at the home

We obtained a “snapshot” of indoor and outdoor exposures by measuring PM concentrations at the home in real time during the home visit using a nephelometer (DustTrak-8520, TSI, Inc., Shoreview, MN). Three 3-min measurements were performed at three separate home locations: just outside the front door, in the main living area, and in the kitchen. The nephelometer measurements should be considered to represent relative concentrations of PM because the instrument was not calibrated against a known concentration of a specific aerosol. The particles measured by the nephelometer are considered to approximate $PM_{2.5}$.

2.4.2. Tobacco smoke exposure

We studied hair nicotine as a biomarker of secondhand tobacco smoke exposure, which we considered a potential source of PM exposure. Overall, 288 hair samples were analyzed for nicotine (12 declined analysis or were bald and two other samples did not have sufficient hair weight collected for analysis). Concentrations of nicotine in hair were determined using liquid chromatography-tandem mass spectrometry. Deuterium-labeled nicotine (nicotine-d9) was used as an internal standard. The limit of detection was variable, depending on weight of hair obtained; 116 (38%) of study subjects had undetectable hair nicotine, which was treated as a measurement value of 0. All of these 0 value observations were included in the lowest tertile of exposure. In addition, potential tobacco smoke exposure was also assessed by interview and was categorized for this analysis as none, secondhand, or from active smoking.

2.4.3. Ambient air quality estimates

We estimated ambient PM exposure by geo-coding the subjects′ residences and linking these data to regional fixed site air pollution monitoring data. Geo-coding,