Environmental Research 155 (2017) 77–85

Contents lists available at ScienceDirect

Environmental Research

journal homepage: www.elsevier.com/locate/envres

Allergic disease associations with regional and localized estimates of air pollution

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ARTICLE INFO

Keywords:
Air pollution
Fine particulate matter
Baysian space-time downscaler model
Allergic disease
Asthma

ABSTRACT

Background: Exposure to multiple types of air pollution may contribute to and exacerbate allergic diseases including asthma and wheezing. However, few studies have examined chronic air pollution exposure and allergic disease outcomes among an adult population. Associations between potential estimates of annual average fine particulate matter ($PM_{2.5}$), traffic related air pollution, and industrial source air emissions and three allergic disease outcomes (asthma, allergies and wheezing) were examined in a state-wide general population of adults.

Methods: The study includes a representative sample of 3381 adult Wisconsin residents who participated in the 2008–2013 Survey of the Health of Wisconsin (SHOW) program. Participant data were geographically linked to The United States Environmental Protection Agency (USEPA) Baysian space-time downscaler air pollution model for $PM_{2.5}$, the United States Census roadway, and USEPA's Toxic Release Inventory data. Self-report and lung function (FEV1) estimates were used to define prevalence of asthma, allergies and wheezing symptoms.

Results: Annual mean exposure to fine particulate matter ($PM_{2.5}$) was between 6.59 and 15.14 μg/m^3. An increase of 5 μg/m^3 in the annual mean $PM_{2.5}$ resulted in a 3.58 (2.36, 5.43) increase in the adjusted odds (95% CI) of having asthma. Exposure to vehicle traffic increased the odds of both current allergies [OR (95% CI)=1.35 (1.07, 1.35)] and current asthma [OR (95% CI)=1.51 (1.14, 2.00)]. Living within 300 m of an Interstate roadway was associated with a 3-fold increase in the odds of asthma. Those living within 800 m of an industrial site were 47% more likely to have asthma. No significant associations were seen with wheezing.

Conclusions: Within this population exposed to overall annual average levels of estimated low level chronic exposure to fine particulate matter ($PM_{2.5}$) at or near 12 μg/m^3, the USEPA standard for air quality, significant association between both modeled PM2.5 exposure and proximity to roadways with asthma and allergies but not wheezing were found. Industrial source emissions were not associated with any allergic disease outcomes.

1. Introduction

Over the past several decades there has been growing evidence air pollution is a risk factor for allergic diseases, including asthma, and there is growing concern outdoor air pollutants may contribute to the prevalence of allergic diseases (D'Amato, 2011). Allergic disease outcomes are more commonly studied among children, however, the prevalence of allergic disease among adults is also on the rise (Zhang et al., 2013). While the association between exposure to fine particulate matter and allergic disease is well supported throughout the literature (Kim and Bernstein, 2009) few of these studies have examined associations in general population-based studies of adults. Numerous epidemiological studies have demonstrated short-term exposure to elevated concentrations of ambient air pollutants, including mixtures of fine particulate matter ($PM_{2.5}$), can exacerbate pre-existing asthma and trigger wheezing (Greenbaum, 2010; Jerrett and Shankardass, 2008; McConnell et al., 2010; Nishimura and Galanter, 2013; O'Connor et al., 2008; Weinmayr and Romeo, 2010). However inconsistent results have been found between ambient and regional air pollution and allergies (Bowatte et al., 2014; Fuertes et al., 2013; Leung et al., 2012; Weir et al., 2013; Zhang et al., 2011). Furthermore, adverse effects of long-term chronic exposure to regional air pollution and allergic disease outcomes are less clear (Anderson et al., 2013; Künzli et al., 2009; Modig and Torén, 2009). Additional investigation into the association between chronic exposure to air pollutants and allergic disease is needed.

Abbreviations: USEPA, United States Environmental Protection Agency; OR, Odds Ratio; CI, Confidence Interval

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http://dx.doi.org/10.1016/j.envres.2017.01.039
Received 27 October 2016; Received in revised form 18 January 2017; Accepted 31 January 2017

Epidemiologic studies have identified exposure to traffic emissions as potentially a main driver of air pollution contributing to allergic disease health outcomes (Greenbaum, 2010). However, most studies focus on short-term exposure in children or adolescents (Batterman et al., 2014; Khreis et al., 2016; Urman et al., 2013). Less is known about the prevalence of allergic disease among adults exposed to traffic emissions over an extended period of time. Additionally, very little research has looked at allergic disease outcomes and exposure to air pollution from other stationary sources such as industries (Mirabelli and Wing, 2006; Patel et al., 2011).

The objective of this study was to assess the association of potential chronic exposure to fine particulate matter ($PM_{2.5}$), traffic related air pollution, and industrial related air pollution with the prevalence of three different allergic disease outcomes: allergies, asthma and wheezing in an adult population. Given the paucity of data on chronic exposure to air pollution and adult allergic disease prevalence, we examined associations between potential exposure to air pollution and allergic disease using not only regional ambient $PM_{2.5}$ which comes from all sources (including traffic and industries), but also estimates of traffic related and industrial related air pollution separately. Traffic related and industrial related air pollution may be greater triggers or sources of allergic disease, especially among a population exposed to regional fine particulates near or below the current United States Environmental Protection Agency (USEPA) National Ambient Air Quality Standard (NAAQS) for annual three-year mean fine-particulate matter ($PM_{2.}$) estimates of 12.0 $\mu g/m^3$ (U.S. EPA, 2012). Three different measures including annual average $PM_{2.5}$ at home address, the distance from residential address to nearest major roadway as a measure of traffic related air pollution, and the distance from residential address to nearest industrial site as a measure of industrial air pollution were used to estimate potential exposures. This study adds to the existing evidence by improving understanding of the relative associations between different potential measures of air pollution exposure and allergic disease outcomes in a randomly selected population-based sample of adults.

2. Methods

2.1. Study sample

Data came from the 2008–2013 Survey of the Health of Wisconsin (SHOW). SHOW is an ongoing health examination survey of adults ages 21–74 (n=3381). The SHOW probabilistic sampling design, procedures, and data collection are described in detail elsewhere (Nieto and Peppard, 2010). In brief, a two-stage cluster sampling strategy is used to randomly select household addresses. At the time of household based recruitment all age-eligible adults are invited to participate. After consent, information is collected through in-person interviews, self-administered questionnaires, and physical exams, including objective measures of height, weight, blood pressure and spirometry-based estimates of lung function. The SHOW protocol and informed consent documents are approved by the Health Sciences Institutional Review Board of the University of Wisconsin-Madison.

For this study subjects with missing data for any one of the three outcomes of interest were excluded from descriptive and unadjusted analyses. Subjects with a history of chronic bronchitis or emphysema (n=222) were also excluded from analysis when the outcome of interest is wheezing, resulting in final sample sizes of 3343 for current allergy, 3375 for current asthma, and 3150 for wheezing.

2.2. Exposure assessment

2.2.1. Geocoding

Each SHOW participant address at the time of consent into the SHOW program was geocoded using CENTRUS software (Pitney Bowes Inc., Stamford, CT). The geocoded addresses were used to link participants to three different types of air pollution data including ambient fine particulate matter ($PM_{2.5}$), traffic related air pollution, and industrial related air pollution using ArcGIS v10.2 software (ESRI, Redlands, CA).

2.2.2. Fine Particulate Matter ($PM_{2.5}$)

The USEPA Fused Air Quality Surface Downscaler model (FAQSD) (U.S. EPA, 2016a) was used to derive annual average $PM_{2.5}$ estimates for each SHOW participant. Data were downloaded for years 2007–2012 from the USEPA Remote Sensing Information Gateway (RSIG) data files (U.S. EPA, 2016a). FAQSD is a Bayesian space-time model that fuses together 24-h average monitoring data from the National Air Monitoring Stations and State and Local Air Monitoring Stations (NAMS/SLAMS) with 12 km gridded output from the Models-3/Community Multiscale Air Quality (CMAQ) v4.6 model (U.S. EPA, 2016b). The CMAQ model integrates USEPA's National Emissions Inventory data, daily continuous emissions monitoring data for major nitrogen oxide (NOx) point sources, and meterological data (U.S. EPA, 2016b). The final FASQD model provides 24-h $PM_{2.5}$ predictions at the 2010 US Census Tract centroid locations. Oridinary kriging was applied to the irregularly spaced FAQSD data point estimates to create a continuous raster image (pixel size=1 mi sq) for the entire state of Wisconsin. Kriging was chosen in order to reduce estimate bias by giving greater weight to values which are spatially closer. The stable variogram was selected as the best fit model based on mean standardized error (MSE) and root mean square standardized error (RMSSE).

Kriged daily and annual $PM_{2.5}$ estimates were then linked to SHOW participant data using the Spatial Analyst Tool in ArcGIS. We estimated potential chronic $PM_{2.5}$ exposures using a 1 year-lagged model. For example, 2008 participants were linked to 2007 air pollution data, and 2009 participants were linked to 2008 air pollution data and so forth. Annual average $PM_{2.5}$ was examined as a continuous variable (for every 5 $\mu g/m^3$ increase), and also by quartile (comparing those exposed to an annual average $PM_{2.5}$ in the second, third, and forth quartile to those in the lowest quartile). Additionally, we examined the number of days that exceed a 24-h mean of 30 $\mu g/m^3$ and 35 $\mu g/m^3$. The current NAAQS for 24 –hr. mean $PM_{2.5}$ is 35 $\mu g/m^3$ (U.S. EPA, 2012). Since our study population had few participants with more than 1 or 2 days 24-h mean $PM_{2.5}$ concentrations above 35 $\mu g/m^3$, we examined a threshold of 30 $\mu g/m^3$ in addition to 35 $\mu g/m^3$.

2.2.3. Traffic Related Air Pollution (TRAP)

Proximty to the nearest major roadway was used as a proxy measure for potential exposure to traffic related air pollution. Data came from the United States Census 2010, and the MAF/TIGER Feature Class Code (MTFCC) and Road Type Code (RTTYP) were used to identify roadway segments as Primary, Secondary, Primary – Interstate, and Primary – non-Interstate roadways (U.S. Census Bureau, 2010). Previous research has shown the majority of normalized pollutant concentrations diminish to background levels 100–300 m from the edge of major roadways (Karner et al., 2010; Zhou and Levy, 2007). Therefore, subjects were dichotomized into categories of potential high vs. low exposure to TRAP using a cutpoint of 300 m from each of the nearest road types. Distances were calculated using the proximity “Near” tool in ArcGIS.

2.2.4. Proximity to industrial facilities

Similar to TRAP estimates, the USEPA's Toxic Release Inventory site database (U.S. EPA, 2008) was used to examine potential industrial source air pollution exposures. Industries that are required to report fugitive or stack air emissions annually to the USEPA were downloaded for the year 2008 from the EPA website (U.S. EPA, 2008); this year of data had the most comprehensive list of industries. Only industries which had been reporting for at least 1 year prior and continued to be reporting through 2013 were included.

A dichotomous variable was created that classified participants as