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Examining the role of location-specific associations between ambient air pollutants and adult asthma in the United States

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ABSTRACT

This study examined the association between ozone and fine particulate ($PM_{2.5}$) exposure and asthma risk by place of residence. We linked 412,832 adult respondents from the 2009 U.S. Behavioral Risk Factor Surveillance System to their residence counties. Observed and interpolated ozone and $PM_{2.5}$ concentration data from 2006 to 2009 were used as exposures. We linked self-reported current asthma status and other individual risk factors to county-level risk factors in multilevel logistic regressions. Results indicated spatially varied asthma risks and spatially varied associations between ambient air pollution and asthma risk. Residents in counties not located within a metropolitan statistical area (MSA) and in inner ring suburbs had a relatively higher asthma risk. Positive ozone–asthma associations were detected across all spatial settings, while positive $PM_{2.5}$-asthma associations were detected only in central cities of an MSA and in outer ring suburbs, indicating that residence location modified the relationship between ambient air pollution and asthma risk.

1. Introduction

Asthma is a common chronic disease that has a significant bearing on public health in the United States. In 2010, 25.7 million people in the United States had asthma, 72.76% of who were adults aged 18 years and older (Akinbami et al., 2012). Asthma is a complex disease associated with many individual- and contextual-level factors (Akinbami et al., 2011; Bener et al., 1996; Wright and Subramanian, 2007). While some studies have shown that environmental factors affect asthma causation and persistence (Asher, 2010; Bener et al., 1996; Yen et al., 2008), no nationwide study in the United States has shown an association between asthma risk and ambient air pollutants at the county level while controlling for individual risk factors (Anderson et al., 2010; Jerrett et al., 2009; Pope et al., 2009) and climate (D'amato et al., 2010; Krstić, 2011; Metintas et al., 2010; Verlato et al., 2002; Weiland et al., 2004). Most studies of environmental effects on asthma used mortality and hospital admissions at the individual level and prevalence at the population level. The separation of individual and ecological effects may lead to inconsistent findings about asthma risk from ambient air pollutants and climate (Cecchi et al., 2010; D'amato et al., 2010; Jerrett et al., 2009; Krstić, 2011; Metintas et al., 2010; Verlato et al., 2002; Zanolin et al., 2004). Because climate may influence asthma prevalence (Verlato et al., 2002; Zanolin et al., 2004), studying the relationship between asthma and ambient air pollution without controlling for climate may lead to biased results. A nationwide study on the association between long-term ozone exposure and cardiopulmonary mortality found that environmental temperature significantly modified the effect of ozone (Jerrett et al., 2009). However, the same effect on asthma has not been established. In the current study, we explored the spatial association between asthma risk and ambient air pollution and seasonal climate, while controlling for other individual risk factors.

Studies of asthma among rural United States populations are rare. Most previous studies on asthma have focused on urban areas because most Environmental Protection Agency (EPA) ambient air pollutant monitoring sites are located in metropolitan areas (Pope et al., 2009; Yip et al., 2011) and more location-specific asthma data are available for urban areas. Recognizing that findings from urban areas cannot be generalized to rural areas, Ownby (2005) reviewed rural asthma studies in the United States and elsewhere. He suggested that rural areas in the United States may have higher asthma prevalence than urban areas, even though rural residents are more likely to be immune to some asthma allergens due to less hygienic environments in rural areas (Riedler et al., 2001). However, a later study using 2005 data from the Behavioral Risk Factor Surveillance System (BRFSS) did not find significant differences in urban and rural asthma prevalence (Morrison et al., 2009). Furthermore, even within a metropolitan area, asthma prevalence may vary between its central city and suburbs due to differences in the intensity of energy consumption and pollution emissions (D'amato et al., 2010; Yip et al., 2011),

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proximity to traffic and major roads (Anderson et al., 2010), and neighborhood characteristics (Gupta et al., 2008; Yen et al., 2008). However, to our knowledge, there have been no studies that associate differential exposure and asthma risk between urban and rural areas, or among different parts of metropolitan areas in the United States. In the current study, we conducted a geographic surveillance that associated asthma risk to ozone and to fine particulate ($PM_{2.5}$) exposure in both metropolitan and non-metropolitan areas while controlling for other individual and environmental factors.

2. Methods

2.1. Data sources and study area

Individual-level data were obtained from the 2009 BRFSS, a large (415,477 records) representative sample of the U.S. adult population aged 18 years and older. In our study, the sample was restricted to the contiguous 48 U.S. states and the District of Columbia. The individual-level dependent variable was the binary response (yes/no) representing adults who "had been told by a physician or nurse that they currently had asthma." Respondents whose asthma status was unknown (2645 records) were deleted. Based on the remaining 412,832 respondents, the sample estimate of asthma prevalence was 8.5%, compared to 8.2% from the 2009 National Health Interview Survey in the United States (Akinbami et al., 2011), suggesting that the BRFSS asthma indicator had high quality. The BRFSS also has a rich set of individual and location risk factors. Based on previous literature (Akinbami et al., 2012; Jerrett et al., 2009; Morrison et al., 2009; Oraka et al., 2010; Pope et al., 2009), we selected a number of the BRFSS risk factors for independent variables (Table 1). In addition, the 2009 BRFSS included the respondent's county code (county federal information processing standards [FIPS] code), which allowed us to link individual records to their corresponding county-level information. After updating FIPS from individual states, we found that 97% of individual records could be linked to corresponding counties of residence. We linked the remaining 3% of individual records with missing county FIPS codes to their corresponding states of residence via state codes and imputed their county-level variables from the means among the rest of the counties in the state, and we labeled them as remaining county areas. Consequently, all 412,832 records were linked to 2470 residence counties and remaining county areas in the contiguous 48 U.S. states and the District of Columbia. The BRFSS also adjusts state samples to be representative within each state, and provides sample weight variables. In all analyses, the *final sample weight* was used.

In order to explore the geographic association between long-term ambient air quality and asthma risk, we obtained the maximum 8-h averages of ambient ozone concentration in parts per billion (ppb) and the maximum 24-h average ambient $PM_{2.5}$ concentration in micrograms per cubic meter ($\mu g/m^3$) from the Air Quality System of the EPA at the county level from 2006 to 2009. We calculated the 4-year average of the annual fourth-highest daily maximum ozone concentration and 4-year average of $PM_{2.5}$ concentration as the main exposure variables. We validated our calculations with the EPA calculations for 2006–2008 and updated them with the 2009 data. These calculated county-level data covered about 80% of the U.S. population, as most EPA ambient air pollutant monitoring sites are located in metropolitan areas (Yip et al., 2011). For counties without EPA monitoring sites, we opted for spatial interpolation that can be generated relatively easily and quickly. We were aware of pollutant behavior simulation models, such as the Community Multi-scale Air Quality model (Ozkaynak et al., 2009) or models that combine information from multiple satellite sensors (van Donkelaar et al., 2010). However, estimates from these models were not readily available for the spatial (county-level) and temporal (3–4 years) resolutions that would meet our exposure requirement. Another potential source for county-level ozone and $PM_{2.5}$ values is the Centers for Disease Control and Prevention (CDC). The CDC estimates use a spatial Bayesian method (McMillan et al., 2010). However, this data source had only one year of data at the time of the study, which did not meet the EPA requirements for multiple years of data in the calculations of ozone and $PM_{2.5}$ values for a particular site. For this reason, we used the most recent county-level predicted ozone and $PM_{2.5}$ values for 2007 from the CDC only for sensitivity analysis.

We used the inverse distance weight (IDW) method to estimate ozone and $PM_{2.5}$ values. Spatial interpolation methods for ozone and $PM_{2.5}$ values have been evaluated (Wong et al., 2004) and reviewed (Jerrett et al., 2004, 2005). While the IDW and Kriging methods provide highly correlated and similar estimates, the IDW method places more weight on local observations (Wong et al., 2004) and ignores pollutant behaviors, such as potential long-distance ozone transport, and global trends. Without point sources and atmospheric models for pollutant behavior in spatial interpolation methods, we felt that the IDW method produced more conservative estimates when the interpolation point was close to the monitoring site. We selected the 8 nearest counties in the interpolation, with the farthest distance being set at 300 km. The distance cut-off was determined by the empirical variogram, as there was no spatial dependency shown beyond 250–300 km, a result consistent with that of a previous study (Wong et al., 2004).

The PRISM group at Oregon State University collected county-level average maximum and minimum temperature (degrees Celisus, °C) for each month in 2000, 2005, and 2007. A previous multicenter study found that asthma prevalence was affected by seasonal changes in temperature (Verlato et al., 2002). Thus, we calculated the 3-year average maximum temperature from June to August and the 3-year average minimum temperature from December to February to measure the seasonal climate in summer and winter, respectively (Ebi et al., 2004). To provide the context, descriptive statistics are shown in Table 1.

Other county-level variables included race/ethnicity (the proportion of non-Hispanic black), education (the percentage of the population aged 25 years or older who had a least a high school diploma), and poverty (the percentage of poverty at all ages), all of which were obtained from the U.S. Census Bureau County Estimates for 2009. The 2009 unemployment rate was obtained from the U.S. Bureau of Labor Statistics. Preliminary analyses showed that county-level education and poverty variables were highly correlated (coefficient = 0.68, $p < 0.001$). We retained the education variable because it was consistently significant throughout all models. We dropped county-level race/ethnicity and unemployment variables because they were not significant in any models.

2.2. Analysis

We used multilevel logistic regression models to account for the individual-level risk factors nested within county-level risk factors. Preliminary analyses suggested significantly inverse relationships between asthma and the 2 temperature measures. Correlation analyses suggested that there was significant co-linearity between maximum temperature in summer and minimum temperature in winter (coefficient = 0.71, $p < 0.001$), and between concentration of ozone and $PM_{2.5}$ (coefficient = 0.64, $p < 0.001$). Thus, as suggested by a previous study (Jerrett et al., 2009), we implemented both a co-pollutant model (i.e., both ozone concentration and $PM_{2.5}$ concentration in a single model) and a single-pollutant models (i.e., ozone concentration and $PM_{2.5}$ concentration in separate models). Since ozone and $PM_{2.5}$ concentrations in the co-pollutant model were correlated (Corr = 0.582, $p < 0.0001$), we relied primarily on the