



# Evaluation of individual and area-level factors as modifiers of the association between warm-season temperature and pediatric asthma morbidity in Atlanta, GA

Cassandra R. O'Lenick<sup>a,\*</sup>, Andrea Winquist<sup>a</sup>, Howard H. Chang<sup>b</sup>, Michael R. Kramer<sup>c</sup>, James A. Mulholland<sup>d</sup>, Andrew Grundstein<sup>e</sup>, Stefanie Ebel Sarnat<sup>a</sup>

<sup>a</sup> Department of Environmental Health, Rollins School of Public Health, Emory University, 1518 Clifton Road NE, Atlanta, GA 30322, USA

<sup>b</sup> Department of Biostatistics and Bioinformatics, Rollins School of Public Health, Emory University, 1518 Clifton Road NE, Atlanta, GA 30322, USA

<sup>c</sup> Department of Epidemiology, Rollins School of Public Health, Emory University, 1518 Clifton Road NE, Atlanta, GA 30322, USA

<sup>d</sup> School of Civil and Environmental Engineering, Georgia Institute of Technology, 790 Atlantic Drive NW, Atlanta, GA 30332 USA

<sup>e</sup> Department of Geography, University of Georgia, 210 Field St., Athens, GA 30602, USA

## ARTICLE INFO

### Keywords:

Climate change  
Temperature  
Childhood asthma  
Time-series  
Effect modification

## ABSTRACT

**Introduction:** Previous studies have found associations between respiratory morbidity and high temperatures; however, few studies have explored associations in potentially sensitive sub-populations.

**Methods:** We evaluated individual and area-level factors as modifiers of the association between warm-season (May–Sept.) temperature and pediatric respiratory morbidity in Atlanta. Emergency department (ED) visit data were obtained for children, 5–18 years old, with primary diagnoses of asthma or respiratory disease (diagnoses of upper respiratory infections, bronchiolitis, pneumonia, chronic obstructive pulmonary disease, asthma, or wheeze) in 20-county Atlanta during 1993–2012. Daily maximum temperature (Tmax) was acquired from the automated surface observing station at Atlanta Hartsfield International Airport. Poisson generalized linear models were used to estimate rate ratios (RR) between daily Tmax and asthma or respiratory disease ED visits, controlling for time and meteorology. Tmax effects were estimated for single-day lags of 0–6 days, for 3-, 5-, and 7-day moving averages and modeled with cubic terms to allow for non-linear relationships. Effect modification by individual factors (sex, race, insurance status) and area-level socioeconomic status (SES; ZIP code levels of poverty, education, and the neighborhood deprivation index) was examined via stratification.

**Results:** Estimated RRs for Tmax and pediatric asthma ED visits were positive and significant for lag days 1–5, with the strongest single day association observed on lag day 2 (RR = 1.06, 95% CI: 1.03, 1.09) for a change in Tmax from 27 °C to 32 °C (25th to 75th percentile). For the moving average exposure periods, associations increased as moving average periods increased. We observed stronger RRs between Tmax and asthma among males compared to females, non-white children compared to white children, children with private insurance compared to children with Medicaid, and among children living in high compared to low SES areas. Associations between Tmax and respiratory disease ED visits were weak and non-significant (p-value > 0.05).

**Conclusions:** Results suggest socio-demographic factors (race/ethnicity, insurance status, and area-level SES) may confer vulnerability to temperature-related pediatric asthma morbidity. Our findings of weaker associations among children with Medicaid compared to other health insurance types and among children living in low compared to high SES areas run counter to our belief that children from disadvantaged households or ZIP codes would be more vulnerable to the respiratory effects of temperature. The potential reasons for these unexpected results are explored in the discussion.

*List of abbreviations:* ACS, American Community Survey; CI, confidence interval; ED, emergency department; ICD-9, International Classification of Diseases, 9th Revision; NDI, Neighborhood Deprivation Index; NO<sub>2</sub>, nitrogen dioxide; RR, rate ratio; SES, socioeconomic status; ZCTA, ZIP Code Tabulation Area

\* Correspondence to: Emory University, Claudia Nance Rollins Building, 1518 Clifton Road NE, Atlanta, GA 30322, USA.

E-mail addresses: [croleni@emory.edu](mailto:croleni@emory.edu) (C.R. O'Lenick), [awinqui@emory.edu](mailto:awinqui@emory.edu) (A. Winquist), [howard.chang@emory.edu](mailto:howard.chang@emory.edu) (H.H. Chang), [mkram02@emory.edu](mailto:mkram02@emory.edu) (M.R. Kramer), [james.mulholland@ce.gatech.edu](mailto:james.mulholland@ce.gatech.edu) (J.A. Mulholland), [andrewg@uga.edu](mailto:andrewg@uga.edu) (A. Grundstein), [sebelt@emory.edu](mailto:sebelt@emory.edu) (S.E. Sarnat).

<http://dx.doi.org/10.1016/j.envres.2017.03.021>

Received 24 September 2016; Received in revised form 11 March 2017; Accepted 13 March 2017

0013-9351/ © 2017 Elsevier Inc. All rights reserved.

## 1. Introduction

Global surface temperatures have risen steadily and rapidly for the past several decades, resulting in location-specific variation in ambient temperatures and more frequent episodes of extreme heat (McMichael et al., 2011; Meehl et al., 2004). Emerging research has shown that the annual number of extreme heat events is increasing more rapidly in sprawling cities compared to compact cities (Stone et al., 2010), and climate change is expected to cause higher warm-season ambient temperatures in large metropolitan areas where temperatures are amplified by the urban heat island effect (Conlon et al., 2016; Luber et al., 2008; Stone et al., 2010; Winquist et al., 2016; Zhou et al., 2009). Although high ambient temperature is a well-documented cause of cardiorespiratory mortality, particularly among the elderly (Basu, 2002, 2009; Basu et al., 2011; Benmarhnia et al., 2015; Braga et al., 2002; Cheng et al., 2014; Laaidi et al., 2012), much less is known about the effects of high ambient temperature on respiratory morbidity, and the influence of modifying factors among sensitive subpopulations remains largely unexplored.

Among the studies that have investigated high temperature-related respiratory morbidity, there is mounting epidemiologic evidence for a lagged effect of temperature (Bunker et al., 2016; Cheng et al., 2014; Li et al., 2014b; Winquist et al., 2016; Xu et al., 2013; Ye et al., 2012), and several studies have found that the effects of temperature on respiratory morbidity remain after controlling for ambient air pollution (Anderson et al., 2013; Cheng et al., 2014; Li et al., 2014a; Lin et al., 2009; Winquist et al., 2016); these findings suggest a strong, independent effect of high temperature in addition to that potentially mediated through the effect of air pollution. There is less agreement on whether thresholds or non-linear exposure-response functions exist regarding the effects of high temperature on respiratory outcomes (Carreras et al., 2015; Green et al., 2010; Kovats et al., 2004; Li et al., 2014a, 2014b; Lin et al., 2009; Michelozzi et al., 2009; Xu et al., 2013), and multi-city studies have reported heterogeneity in the exposure-response function between study locations (Anderson et al., 2013; Michelozzi et al., 2009). Inconsistencies between studies may be due to a variety of factors including differences in the climate of the study area, different adaptive strategies employed in cities (e.g. high utilization of air conditioning, cooling centers, early warning systems), differences in population-level acclimation to climate, and the use of disparate temperature metrics to capture exposure to high ambient temperature (e.g. daily minimum temperature, daily mean temperature, daily maximum temperature, diurnal temperature ranges, heat waves, and heat stress indices) (Davis et al., 2016; Turner et al., 2012). Certain populations may also be more or less responsive to high temperature and studies that have examined age as a modifying factor have reported stronger associations among children and the elderly compared to other age groups (Bunker et al., 2016; Cheng et al., 2014; Kovats et al., 2004; Michelozzi et al., 2009; Xu et al., 2013).

Children may be inherently more susceptible than adults to temperature-related respiratory morbidity due to higher ventilation rates, developing respiratory and immunological systems, and anatomically smaller peripheral airways that predispose children to airway inflammation and obstruction (Bateson et al., 2007; Makri et al., 2008; Selgrade et al., 2008; Sharma et al., 2006). They may also spend more time outdoors during the warm-season compared to adults, thus experiencing greater exposures to high ambient temperatures. Additionally, children have an underdeveloped thermoregulatory system that results in a diminished capacity to maintain optimal internal temperatures under heat stress (Hanna et al., 2015). Impaired thermoregulation and prolonged heat exposure can result in hyperthermia and lead to increases in core body temperature, systemic inflammation, increased cardiac output, and increases in tidal volume, respiratory rate, and pulmonary ventilation (Anderson et al., 2013; Hanna et al., 2015; Leon et al., 2010; White, 2006). Finally, individual-level factors (e.g. sex, race, health care access, time-activity patterns) and area-level

socioeconomic status (SES) may further confer susceptibility and vulnerability among children to temperature related respiratory disease; however, few studies to date provide estimates of temperature-related respiratory morbidity for such susceptible and/or vulnerable groups.

To address this gap, we built on an extensive previous assessment of heat-related morbidity in Atlanta, in which we observed a positive and significant association between high warm-season temperature and asthma ED visits among children (5–18 years old), but not in other age groups (Winquist et al., 2016). Here, using a similar methodology, we focus on this previously observed association between high temperature and respiratory morbidity among children (5–18 years) to specifically examine the degree to which individual-level factors (sex, race/ethnicity, insurance status) and area-level SES modify associations, and to examine the non-linear effects of temperature across these different modifying factors.

## 2. Methods

### 2.1. Data sources

Hourly meteorological data from January 1, 1993 through December 31, 2012 were obtained from the Automated Surface Observing Station (ASOS) located at Atlanta Hartsfield International Airport. Hourly observations were used to create daily ambient meteorological metrics including daily maximum temperature and daily maximum dew-point temperature. We selected daily maximum temperature (in degrees Celsius, °C) as our primary exposure of interest based on our previous work (Winquist et al., 2016) and use of this metric by others in related health studies (Davis et al., 2016; Ebi et al., 2004; Hondula et al., 2014; Linares et al., 2008; Saha et al., 2015; Ye et al., 2001). Additionally, maximum temperature values may coincide with a time of day when children may be most active and exposed to outdoor temperatures (Barnett et al., 2010), and may represent an exposure that could cause the greatest amount of physiological stress due to temperature on a given day. For sensitivity analyses, we also acquired daily maximum and daily minimum temperature data from 13 cooperative meteorological stations located within the 20-county Atlanta study area. Cooperative stations are part of the National Weather Service (NWS) Cooperative Observer Program (COOP), and daily weather observations at COOP stations are recorded by volunteers (National Oceanic and Atmospheric Administration, 2016). Because COOP stations are maintained by volunteers, different stations may use different analytical instruments to measure ambient temperature, and some station volunteers may not be able to take daily measurements. In our sensitivity analyses, 12 of the 13 cooperative stations did not provide complete daily temperature records during the 1993–2012 study period. The names and locations of the cooperative stations used in sensitivity analyses are discussed in greater detail in Section 3.1, Descriptive Results.

Patient-level emergency department (ED) visit data from January 1, 1993 to December 31, 2012 were acquired from 41 hospitals located within the 20-county metropolitan area of Atlanta. The 20-county study area reflects the counties included in the Atlanta metropolitan statistical area definition of 1999 (United States Census Bureau, 1999), the year that our studies of ED visits and air quality in Atlanta began (Tolbert et al., 2000). All acute care hospitals in the 20-county Atlanta area were included, except for the Veterans Affairs hospital. Data from these hospitals were included if they were able to provide electronic billing records for at least part of the study period. In 1993, only 7 hospitals were contributing data. Additional hospitals were added over time as more facilities moved towards electronic records; by 2005, 40 hospitals were participating. ED visit data from 1993 to 2004 were acquired directly from individual hospitals and ED data from 2005 to 2012 were acquired from the Georgia Hospital Association (GHA). The ED visit data collected by GHA are from the same hospital billing

Download English Version:

<https://daneshyari.com/en/article/5756460>

Download Persian Version:

<https://daneshyari.com/article/5756460>

[Daneshyari.com](https://daneshyari.com)