



## Fine particulate matter components and mortality in Greater Houston: Did the risk reduce from 2000 to 2011?



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### HIGHLIGHTS

- PM<sub>2.5</sub> concentrations were associated with increased mortality risk.
- A few major PM<sub>2.5</sub> components were associated with increased mortality risk.
- Associations were generally strongest in winter in Greater Houston.
- Effect estimates of PM<sub>2.5</sub> mass had reduced from 2000–2005 to 2006–2011.

### ARTICLE INFO

#### Article history:

Received 20 April 2015

Received in revised form 16 July 2015

Accepted 10 August 2015

Available online xxx

Editor: D. Barcelo

#### Keywords:

Fine particulate matter

Components

Mortality

Poisson regression

Species

Time series

### ABSTRACT

Fine particulate matter (less than 2.5 μm in aerodynamic diameter; PM<sub>2.5</sub>) pollution poses a major environmental threat in Greater Houston due to rapid economic growth and the numerous PM<sub>2.5</sub> sources including ports, vehicles, and the largest petrochemical industry in the United States (U.S.). Our objectives were to estimate the short-term associations between the PM<sub>2.5</sub> components and mortality during 2000–2011, and evaluate whether these associations have changed over time. A total of 333,317 deaths were included in our assessment, with an average of 76 deaths per day. We selected 17 PM<sub>2.5</sub> components from the U.S. Environmental Protection Agency's Chemical Speciation Network, and then applied Poisson regression models to assess the associations between the PM<sub>2.5</sub> components and mortality. Additionally, we repeated our analysis for two consecutive periods: 2000–2005 and 2006–2011. Interquartile range increases in ammonium (0.881 μg/m<sup>3</sup>), nitrate (0.487 μg/m<sup>3</sup>), sulfate (2.245 μg/m<sup>3</sup>), and vanadium (0.004 μg/m<sup>3</sup>) were associated with an increased risk in mortality of 0.69% (95% confidence interval (CI): 0.26, 1.12%), 0.38% (95% CI: 0.11, 0.66%), 0.61% (95% CI: 0.15, 1.06%), and 0.58% (95% CI: 0.12, 1.04%), respectively. Seasonal analysis suggested that the associations were strongest during the winter months. The association between PM<sub>2.5</sub> mass and mortality decreased during 2000–2011, however, the PM<sub>2.5</sub> components showed no notable changes in mortality risk over time. Our study indicates that the short-term associations between PM<sub>2.5</sub> and mortality differ across the PM<sub>2.5</sub> components and suggests that future air pollution control measures should not only focus on mass but also pollutant sources.

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### 1. Introduction

Ambient air pollution is a major global concern contributing to 3.7 million deaths worldwide in 2012 (World Health Organization [WHO], 2014). The Global Burden of Disease Study also reported that ambient particulate matter (PM) pollution accounted for 3.1 million excess deaths worldwide and 3.1% of global disability adjusted life-years (Lim et al., 2013). Greater Houston, the fifth largest metropolitan area in the United States (U.S.) with more than 6 million people in the population (U.S. Census Bureau, 2013), has many emission sources of

PM, including the largest petrochemical complex in the U.S. along the Houston Ship Channel as well as a large number of ports and vehicles (Sexton et al., 2006). Although the overall levels of PM<sub>2.5</sub> (PM with an aerodynamic diameter of ≤2.5 μm) in Greater Houston have declined since monitoring began in 1999, the reported annual PM<sub>2.5</sub> averages are still near the limit as defined by the U.S. National Ambient Air Quality Standards (NAAQS), which was set to 12 μg/m<sup>3</sup> in 2012 (Texas Commission on Environmental Quality [TCEQ], 2013). This is also indicated by the annual “design value” used by U.S. Environmental Protection Agency (EPA) that is compared to the NAAQS to determine the attainment status of an area, e.g., the PM<sub>2.5</sub> annual design values were 14.1 μg/m<sup>3</sup> for 2002 and 12.1 μg/m<sup>3</sup> for 2012 in Greater Houston (EPA, 2014).

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Although many previous studies have reported short-term associations between mass concentration of PM<sub>2.5</sub> and mortality, as recently reviewed by Atkinson et al. (2014), a relatively smaller number of studies have evaluated the short-term associations between the PM<sub>2.5</sub> components and mortality. PM<sub>2.5</sub> is a complex mixture of many chemical components that may differ in their effects on human health (Bell et al., 2007). There is growing evidence that PM<sub>2.5</sub> components may play a role in the associations between PM<sub>2.5</sub> and mortality (Chen and Lippmann, 2009; Kelly and Fussell, 2012; Reiss et al., 2007; Rohr and Wyzga, 2012; Schlesinger, 2007; Stanek et al., 2011). Although the findings are inconclusive, previous studies have more frequently reported on positive associations between certain PM<sub>2.5</sub> components (e.g., nickel, vanadium, elemental carbon, and organic carbon) and increased mortality risk compared to other PM<sub>2.5</sub> components. Further, the effect estimates often vary seasonally and geographically. For example, Ito et al. (2011) found a strong seasonal pattern between PM<sub>2.5</sub> components and the association with cardiovascular disease (CVD) mortality, with the strongest associations in winter; similarly, Ostro et al. (2007) reported stronger associations between mortality and PM<sub>2.5</sub> components (e.g., sulfate, elemental carbon, and organic carbon) in the cool season.

There are three previous short-term multi-city studies, all of which included data from Houston (Dai et al., 2014; Franklin et al., 2008; Krall et al., 2013), however these studies used data prior to 2006 and therefore the changes in PM<sub>2.5</sub> concentrations after 2006 were not reflected in their findings and conclusions. Greater Houston has been one of the fastest growing metropolitan areas in the U.S. in recent years. In fact, the population of Greater Houston grew by 14% between 2005 (5,321,501) and 2011 (6,086,538) (U.S. Census Bureau, 2014). In addition to the implementation of a number of air quality-related national and regional standards and regulations since 2000, including the National Tier 2 Vehicle and Gasoline Sulfur Program (phased in between 2004 and 2007) and the Heavy-Duty Highway Diesel Program (phased in between 2007 and 2010), the Texas State Implementation Plan (SIP) has initiated a series of air quality control measures in Greater Houston (see Supplemental material Table S1 for more details). It is not clear if these regulations and programs have influenced mortality risk attributed to PM<sub>2.5</sub> mass and the PM<sub>2.5</sub> components.

In this study, we applied generalized additive models to assess the short-term association between PM<sub>2.5</sub> mass and the PM<sub>2.5</sub> components and mortality in Greater Houston during 2000–2011. We first evaluated these associations over the entire study period and across the four seasons. We then repeated our analyses separately for two time periods (2000–2005; and 2006–2011) to assess whether the associations between PM<sub>2.5</sub> mass and the PM<sub>2.5</sub> components and mortality have changed over time.

## 2. Methods

Greater Houston (see Supplemental material Fig. S1) is a geographic area composed of nine counties, including Austin, Brazoria, Chambers, Fort Bend, Galveston, Harris, Liberty, Montgomery and Waller counties (U.S. Census Bureau, 2013). There are many sources of PM<sub>2.5</sub> in Greater Houston. First, the long-range transport of PM<sub>2.5</sub> resulting from events such as wildfires, controlled burns, continental haze, and African dust may affect PM<sub>2.5</sub> levels in Greater Houston (Bates et al., 2008; Chiou et al., 2010; TCEQ, 2013). Second, PM<sub>2.5</sub> can also be generated from regional dust storms originating in areas of West Texas and the New Mexico area as well as agricultural burning in Mexico and Central America (TCEQ, 2013). Regional sources of PM<sub>2.5</sub> also include marine vessel emissions in the Gulf of Mexico and secondary particles from upwind regions (Parrish et al., 2009). Lastly, Greater Houston has many local sources of air pollution that also contribute to the increased levels of PM<sub>2.5</sub>, including two of the four largest refineries in the U.S., the petrochemical complex along the Houston Ship Channel, the Port of Houston, numerous industrial facilities across the entire

Greater Houston, and emissions from on-road motor vehicular traffic (Bahreini et al., 2009; Chiou et al., 2010; Nowak et al., 2010; Sexton et al., 2006).

### 2.1. Air pollution and meteorological data

We retrieved PM<sub>2.5</sub> mass and speciation data from the U.S. EPA Air Quality System (AQS) ([www.epa.gov/ttn/airs/airsaqs/](http://www.epa.gov/ttn/airs/airsaqs/)). Our analysis used 24-hour (h) integrated measurements for PM<sub>2.5</sub> mass were downloaded for all 16 PM<sub>2.5</sub> monitoring sites in Greater Houston during 2000–2011 using the federal reference method (FRM) (see Supplemental material Fig. S1). Three of the 16 monitoring sites continuously monitored PM<sub>2.5</sub> mass in the study period while the others were operated over limited years over the study period. Daily average concentrations for PM<sub>2.5</sub> mass across multiple monitors in Greater Houston were calculated for each day in the study period. We extracted speciation data collected from Houston Deer Park between 2000 and 2011, the only chemical speciation network (CSN) site in Greater Houston (see Supplemental material Fig. S1). CSN typically collects PM<sub>2.5</sub> samples on a one-in-three or one-in-six day schedule. Speciation samples were not collected continuously, therefore our data were restricted to including only those observations when both daily deaths and the PM<sub>2.5</sub> component concentrations at a specific time lag were available. Based on the results from previous epidemiological studies (Dai et al., 2014; Franklin et al., 2008; Krall et al., 2013; Zanobetti et al., 2009; Ostro et al., 2007) and a local source apportionment study (Sullivan et al., 2013), we selected 17 PM<sub>2.5</sub> components to include in our assessment, including trace elements (Aluminum (Al), Bromine (Br), Chromium (Cr), Copper (Cu), Iron (Fe), Manganese (Mn), Nickel (Ni), Potassium (K), Silicon (Si), Vanadium (V), and Zinc (Zn)), ions (Ammonium (NH<sub>4</sub><sup>+</sup>), Nitrate (NO<sub>3</sub><sup>-</sup>), Sodium ion (Na<sup>+</sup>), and Sulfate (SO<sub>4</sub><sup>2-</sup>)), and carbonaceous species (elemental carbon (EC) and organic carbon (OC)). The U.S. EPA implemented a new method to analyze EC and OC at Houston Deer Park in 2009 and a validated correction method is not yet available, therefore, we included only EC and OC data that were collected between 2000 and 2009 in our assessment.

Hourly weather data collected at the George Bush Intercontinental Houston Airport (IAH) were obtained from the National Climate Data Center (NCDC, 2014). Average temperature and dew point temperature were then calculated for each day based on the hourly data.

### 2.2. Mortality data

Daily all-cause mortality data were obtained from the Texas Department of State Health Services during 2000–2011. *The International Classification of Diseases, Tenth Revision (ICD-10)* (WHO, 2007), was used to classify causes of death for the study period. In this study, we only included non-accidental mortality data (ICD-10, A00 – R99). Deaths due to external causes (ICD-10, S00 – U99) were excluded.

### 2.3. Statistical methods

We applied generalized additive models to examine the associations between daily counts of all-cause mortality and daily concentrations of PM<sub>2.5</sub> mass and the PM<sub>2.5</sub> components. The parameters specifying the distribution of death counts were assigned by quasi-Poisson distributions to account for overdispersion. The analysis was first conducted on PM<sub>2.5</sub> mass and each PM<sub>2.5</sub> component separately during 2000–2011. This was followed by a stratified analysis examining the seasonal effect. Each season was defined as: spring (March, April, and May), summer (June, July, and August), fall (September, October, and November), and winter (December, January, and February). Finally, we split the entire study period into two consecutive periods: 2000–2005 and 2006–2011 and conducted the analysis on each period

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