Exploring the health disparity of long-term $PM_{2.5}$ exposure with advanced $PM_{2.5}$ mapping and hospital admission records

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Introduction

The negative association between ambient fine particulate matter less than 2.5μm diameter (PM2.5) and adverse health outcomes is well-known (Pope, 2000). Compared with short-term PM2.5 exposure, long-term PM2.5 exposure generates more severe and extensive impacts on human health (Di et al. 2017). One core task to understand the health risks imposed by different levels of PM2.5 exposure is to characterize and model the disease relative risk (RR) responses to various PM2.5 levels and the associated discrepancy across different diseases, regions, and demographic groups (Apte et al., 2015; Burnett et al., 2014).

With a focus on the state of Arizona, the goal of this current study is to investigate the quantitative relationships between long-term PM2.5 exposure and morbidity of all-cause and five cause-specific diseases from 2005 to 2014 (i.e., asthma, chronic respiratory, skin cancer, heart, and cerebrovascular diseases) by combining high-resolution PM2.5 concentration mapping and with large-scale longitudinal health statistics obtained through the Healthcare Cost Utilization Project (HCUP) hospital admission records. To fulfil this goal, we first estimate long-term PM2.5 exposure of each patient who visited Arizona's hospitals from 2005 to 2014 based on the patient's residential zip code. We then estimate RR of PM2.5-morbidity for all-cause and cause-specific diseases for the total population at different PM2.5 levels and develop RR functions relating PM2.5 to adverse health impacts. We examine the disparities of RRs of PM2.5-morbidity among different gender and racial/ethnic subpopulations.

Method

Health and demographic data

The State Inpatient Databases (SID) compiled by HCUP provide hospital admission records in the U.S. The HCUP database has been successfully used in many investigations on health effects of PM2.5 thanks to the long time-span and large geographic coverage. Each record indicates a patient's hospital admission, including a patient's principle and secondary diagnosis results, month and year of hospitalization, as

well as demographic group. The acquired Arizona SID dataset includes 10-year hospital admission records from 2005 and 2014. Each hospital admission record includes an inpatient's cause-specific admission and a corresponding International Classification of Disease, 9th Revision, Clinical Modification (ICD-9-CM) code. Patient's residential address at the zip code-level is available to identify each individual's activity area and to assess the associated PM2.5 exposure. Based on the ICD-9-CM code, we select admission records for asthma, chronic respiratory infection, skin cancer, heart disease, and cerebrovascular disease, all of which are related to heart, respiratory, and/or skin systems.

Demographic data are retrieved from the United States Census Bureau (USCB) that contain racial and ethnic classifications (African American, non-Hispanic White, Hispanic) and sex (male and female) for each zip code area in Arizona. For the time of study (2005-2014), such census data are only available for the years of 2000 and 2010-2014, yet not for 2001-2009. The subpopulations for missing years are estimated based on an assumption that subpopulation rates of growth/decline at each zip code area are constant from 2000 to 2010.

Exposure assessment

The gridded annual average PM2.5 concentration dataset at the 1 km spatial resolution for 2004 to 2014 is from a recently proposed machine learning based hybrid method (Liu et al., 2018). We adopt this dataset to assess human health risks due to long-term PM2.5 exposure because it has a finer spatial resolution and shows higher agreement with ground-based stations (Liu et al., 2018) compared with other commonly used methods such as van Donkelaar et al., (2016). With this new fine spatial-resolution PM2.5 dataset, we calculate each zip code's annual average PM2.5 concentrations for 2004-2014. We then assess each patient's ambient PM2.5 exposure

RRs of PM2.5-morbidtiy and PM2.5-morbidity RR functions

Based on the exposure estimation and the hospital admission, we can calculate RRs of PM2.5-attributable morbidity for all-cause and five cause-specific diseases (i.e., asthma, chronic respiratory infection, skin cancer, heart, and cerebrovascular diseases). Relative risk functions are widely used to quantify the relationship between PM2.5 exposure and its health outcomes (Pope et al., 2009; Pope et al., 2002; Burnett et al., 2014). Burnett et al. (2014) developed an integrative exposure-response (IER) PM2.5-RR function by utilizing the linear, logarithm, and power models together (Equation 1), which is widely adopted for disease burden assessment (Apte et al., 2015). The IER model is applied in the current study to establish the PM2.5-morbidity RR functions for all-cause and the cause-specific diseases.

$$RR_c^d = \begin{cases} 1 + \alpha[1 - exp(-\beta(c - c_0)^\delta)] & c > c_0 \\ 1 & c \le c_0 \end{cases} \quad (1)$$

in which RR_c^d represents the simulated RR based on the IER function at PM2.5 concentration level c, for disease d, and α, β, and δ are the three parameters shaping the curves of RRs changing with PM2.5 concentrations, and c_0 represents the threshold PM2.5 concentration level.

Results

RRs and RR functions for total population

We calculate RRs of PM2.5-morbidity and develop RR functions under the relatively low PM2.5 concentrations (<12.5 µg/m3). Following the IER approach of Burnett et al. (2014), the PM2.5-attributable morbidity RR functions for all-cause, skin cancer, asthma, chronic respiratory, and heart disease morbidity for the total population are developed (Figure 1). The increase of PM2.5 concentration from an RR increase trend is found for all-cause as well as the cause-specific diseases, excluding that of cerebrovascular disease. The RR of PM2.5-attributable cerebrovascular morbidity begins to rise above 1.0 at the PM2.5 concentration of 3.6 µg/m3, followed by continuous increases until reaching a maximum at 8.0 µg/m3, and a subsequent RR decline at PM2.5 concentrations >8.0 µg/m3. Since the IER approach assumes that the RR of PM2.5-attributatble morbidity increases with the increment of PM2.5 concentration, the PM2.5-RR function for cerebrovascular diseases does not follow this trend in the current study. We therefore fit the RR functions of cerebrovascular disease using a simple polynomial function. Excluding cerebrovascular diseases, the increasing rates (i.e., gradient or first order derivative) of the RR are gradually reduced with the increase in the PM2.5 concentration. Yet, with incrementally increasing the PM2.5 concentration, the increasing rates of the RR of skin cancer and asthma are clearly larger than those of all-cause, heart disease, and chronic respiratory disease (see Figure 1).

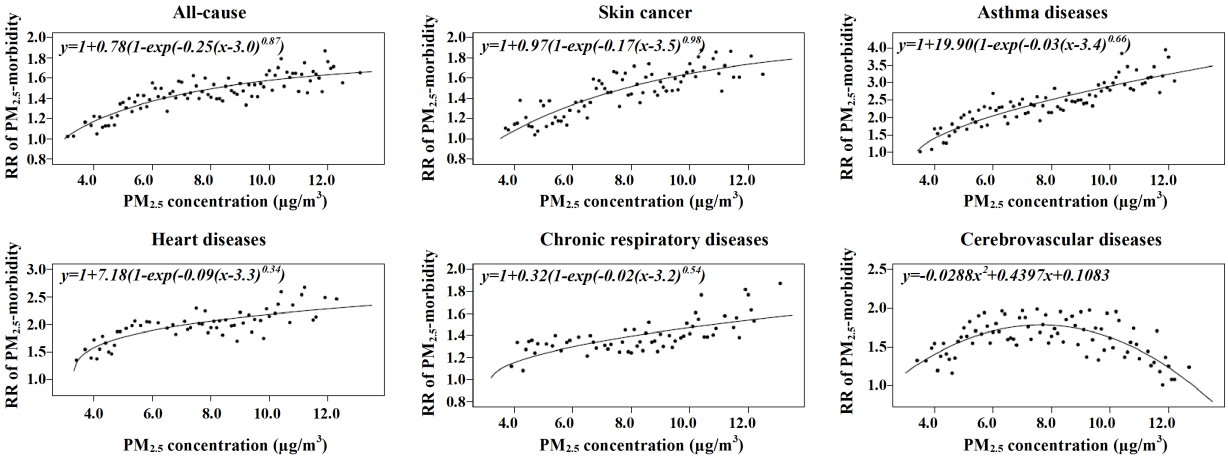

Figure 1: The RR functions of PM2.5-morbidity for all-cause and the five cause-specific diseases. Except cerebrovascular diseases (polynomial curve), the RR functions for all-cause and the other four cause-specific diseases are developed based the IER approach

RRs across subpopulations

Figure 2 displays the differences of RRs of the PM2.5-attributable morbidity between males and females at the 95% confidence interval (CI) for all-cause and the five cause-specific diseases. Women are more vulnerable than men to long-term PM2.5 exposure, whereby female's RRs for all-cause disease are significantly larger than those of male at all PM2.5 levels from 4.4 µg/m3 to 12.5 µg/m3. However, women do not always show higher risks due to exposure to PM2.5 compared to men. Females have significantly

lower PM2.5-attributable RRs for asthma than males at PM2.5-concentrations ranging from 7.3 µg/m3 to 12.0 µg/m3 and clear disparities of RRs for skin cancer cannot be found between the male and female subpopulations (Figure 2b).

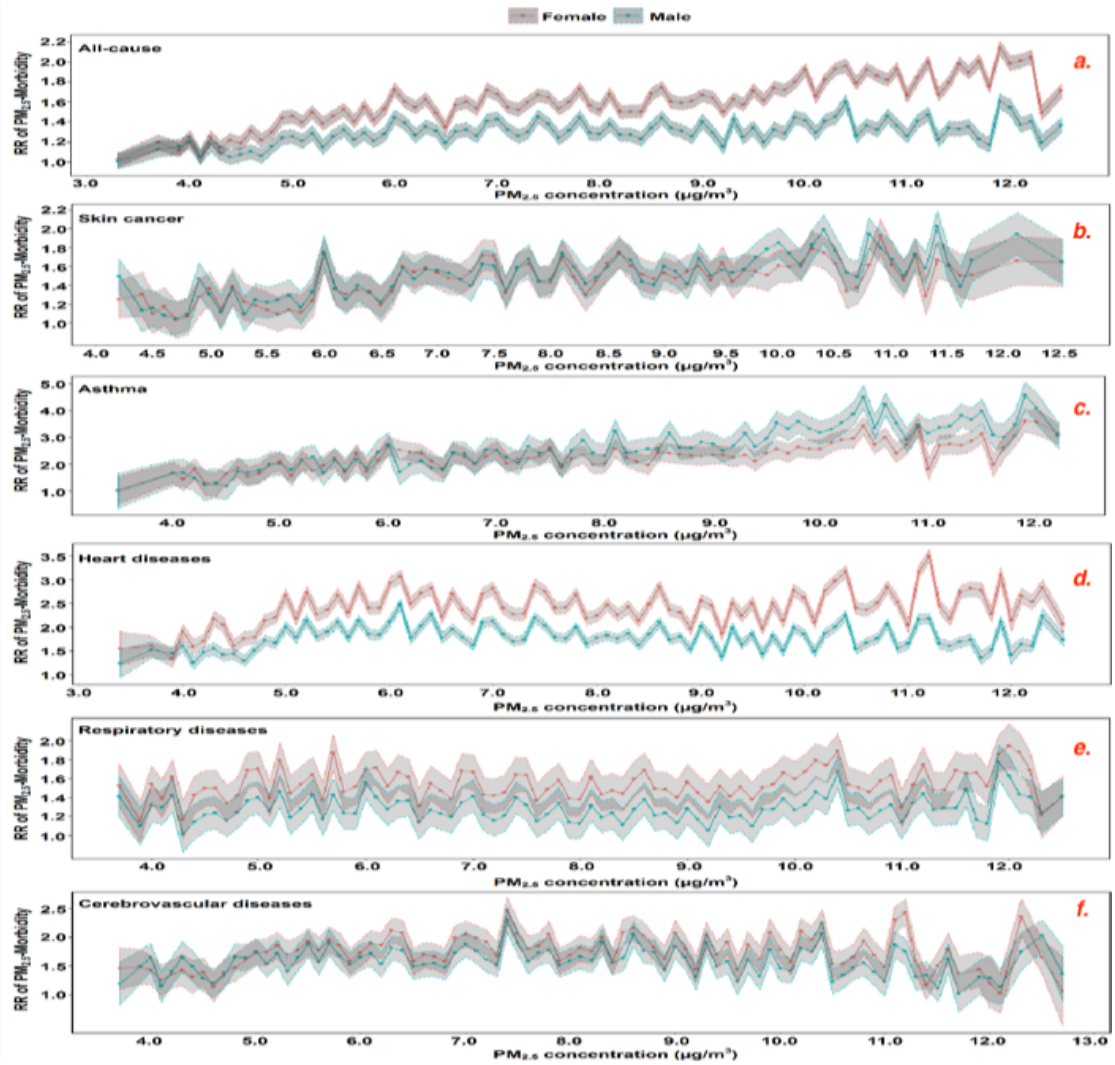

Figure 2. Disparities of PM2.5-morbidity RRs between female and male for all-cause (a) the five cause-specific diseases (b for skin c, c for asthma, d for heart diseases, e for chronic respiratory diseases, and f for cerebrovascular diseases

Figure 3a shows that the African American subpopulation has a larger RR for all-cause disease at PM2.5 concentrations > 5.7 µg/m3 and < 10.5 µg/m3. With PM2.5 concentrations > 10.5 µg/m3, the all-cause RRs for African Americans are nearly equal to Hispanics, yet remain significantly larger than those of non-Hispanic White. We do not calculate the African American RRs of PM2.5-attributable morbidity for skin cancer, asthma, and cerebrovascular diseases because the patients' hospital admission counts for these three cause-specific diseases are too low to make meaningful references across the majority of PM2.5 concentrations. However, we find that African Americans have significantly higher RRs of PM2.5-attributable morbidity for heart and chronic respiratory diseases at nearly all PM2.5 levels between 3.5 µg/m3 and 12.5 µg/m3 (Figure 3d and 3e). Hispanics show larger risks of suffering from skin cancer and heart diseases than non-Hispanic White due to long-term PM2.5 exposure at almost all PM2.5

levels from 4.2 µg/m3 to 11.5 µg/m3 and 3.4 µg/m3 to 11.9 µg/m3, respectively (Figure 3b). Additionally, Hispanics have significantly larger RRs of PM2.5 exposure for all-cause diseases than non-Hispanic White at PM2.5 concentrations larger than 6.2 µg/m3 and smaller than 12.5 µg/m3 (Figure 3a). Moreover, with an increase in PM2.5 concentrations, the difference in the RRs of all-cause diseases between Hispanics and non-Hispanic Whites tends to increase (Figure 3a). However, non-Hispanic Whites are not always the least affected by PM2.5 exposure. Non-Hispanic Whites are more susceptible to cerebrovascular diseases due to long-term PM2.5 exposure with the PM2.5 concentration between 5.8 µg/m3 and 12.4 µg/m3 (see Figure 3f).

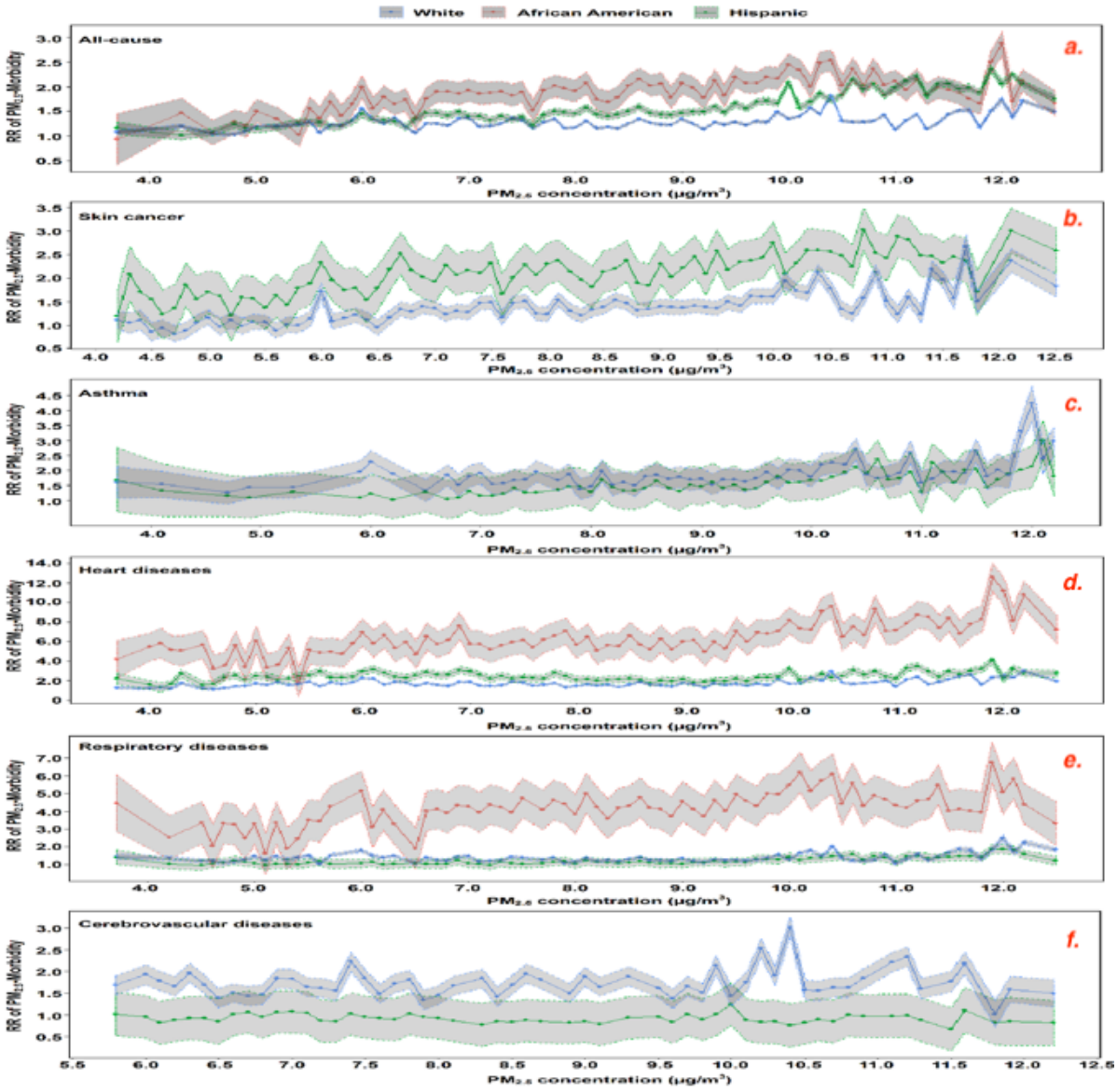

Figure 3:Disparities of PM2.5-morbidity RRs among different racial and ethnic subpopulation for all-cause (a) the five cause-specific diseases (b for skin cancer, c for asthma, d for heart diseases, e for chronic respiratory disease and f for cerebrovascular diseases.

Discussion and Conclusion

To assess the impacts of long-term PM2.5 exposure on human health, we quantify and evaluate the associations between PM2.5 exposure and morbidity in Arizona using datasets with more finely resolved spatial resolutions. We applied the newly developed 1 km × 1 km resolution PM2.5 images and a large hospital admission database (i.e., SID) from HCUP. Using these datasets, we developed the RR functions of PM2.5-morbidity based on Burnett et al. (2014)'s IER model. We found long-term exposure to PM2.5 concentrations increases the morbidity risks except for cerebrovascular ailment. Additionally, long-term PM2.5 exposure displays disparate effects on human health across different subpopulations. Generally, females are more vulnerable than males to heightened long-term PM2.5 exposure. Further, a higher long-term PM2.5 exposure has a larger negative effect on African Americans than non-Hispanic Whites and Hispanics. Compared with Hispanics, non-Hispanic White have lower risks suffering from all-cause, skin cancer, and heart disease but larger risks of cerebrovascular ailments due to long-term exposure to relatively low PM2.5 concentrations.

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