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Long-term exposure to ambient air pollution (including PM_1) and metabolic syndrome: The 33 Communities Chinese Health Study (33CCHS)

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ABSTRACT

Little evidence exists about the effects of long-term exposure to ambient air pollution on metabolic syndrome (MetS). This study aimed to determine the association between long-term ambient air pollution and MetS in China. A total of 15,477 adults who participated in the 33 Communities Chinese Health Study (33CCHS) in 2009 were evaluated. MetS was defined based on the recommendation by the Joint Interim Societies. Exposure to air pollutants was assessed using data from monitoring stations and a spatial statistical model (including particles with diameters ≤ 1.0 μm (PM_1), ≤ 2.5 μm ($PM_{2.5}$), and ≤ 10 μm (PM_{10}), sulfur dioxide (SO_2), nitrogen dioxide (NO_2), and ozone (O_3)). Two-level logistic regression analyses were utilized to assess the associations between air pollutants and MetS. The prevalence of MetS was 30.37%. The adjusted odds ratio of MetS per 10 μg/m^3 increase in PM_1, $PM_{2.5}$, PM_{10}, SO_2, NO_2, and O_3 were 1.12 (95% CI = 1.00–1.24), 1.09 (95% CI = 1.00–1.18), 1.13 (95% CI = 1.08–1.19), 1.10 (95% CI = 1.02–1.18), 1.33 (95% CI = 1.12–1.57), and 1.10 (95% CI = 1.01–1.18), respectively. Stratified analyses indicated that the above associations were stronger in participants with the demographic variables of males, < 50 years of age, and higher income, as well as with the behavioral characteristics of smoking, drinking, and consuming sugar-sweetened soft drinks frequently. This study indicates that long-term exposure to ambient air pollutants may increase the risk of MetS, especially among males, the young to middle aged, those of low income, and those with unhealthy lifestyles.

1. Introduction

Ambient air pollution is a major public health concern globally. It is estimated that over 90% of the world's population currently lives in regions where air pollutant concentrations exceed the world health organization (WHO) limits (World Health Organization, 2016). Numerous epidemiological studies have linked ambient air pollution with cardiovascular diseases (Brook et al., 2010; Scheers et al., 2015), chronic obstructive pulmonary disease, asthma (Bui et al., 2013), and cancers of the lungs, cervix, and brain (Hamra et al., 2015; Raaschou-Nielsen et al., 2011). These findings indicated that exposure to ambient air pollutants may cause events at the later stages of chronic disease processes (Chen et al., 2014). However, the effects of ambient pollutants on the earlier stages of developing chronic diseases are less known (Brook et al., 2010). In particular, understanding whether air pollutants initiate or accelerate the development of modifiable risk factors for chronic diseases may have great public health implications for primary prevention.

Metabolic syndrome (MetS) is comprised of a cluster of major modifiable risk factors for non-communicable diseases (e.g.

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cardiovascular diseases and cancers) (Lorenzo et al., 2007; Li et al., 2016a, 2016b; Lee et al., 2017), including abdominal obesity, dyslipidemia, elevated blood pressure, and high glucose concentrations (Cornier et al., 2008). MetS affects approximately 20% of the global population and its prevalence is rapidly increasing worldwide (O'Neill and O'Driscoll, 2015). It has been suggested that this increase cannot be attributable to genetic factors, physical inactivity, and unhealthy lifestyles alone, but also to environmental exposures, such as ambient air pollution (Wallwork et al., 2017). Mechanistically, the inhalation of air pollutants can trigger the following: autonomic nervous system imbalance (Rajagopalan and Brook, 2012), oxidative stress, endothelium dysfunction (Brook et al., 2004), systemic inflammation (Miller, 2014), insulin resistance (Rajagopalan and Brook, 2012), and epigenetic modification (Peng et al., 2016; Wang et al., 2016). These adverse responses can independently and/or interactively be involved in the development of hypertension, obesity, dyslipidemia, and hyperglycemia (Giorgini et al., 2016)—all of which are key components in the diagnosis of MetS. Several prior experimental and epidemiological studies have explored the relationships of air pollution exposure with individual MetS components (Wallwork et al., 2017). However, few studies have focused on the complexity of metabolic alterations that constitute MetS. In the MEDLINE (Medical Literature Analysis and Retrieval System Online) database, we observed only two relevant human epidemiological studies investigating the relationships of air pollutants with MetS. They were conducted in high-income countries (Switzerland and the United States) and both reported significant associations (Eze et al., 2015a; Wallwork et al., 2017). Additionally, a very recent animal study showed that exposure to highly polluted air resulted in weight gain, as well as cardiorespiratory and metabolic dysfunction (Wei et al., 2016).

As the largest developing country, China has experienced the lifestyle and dietary changes that accompany rapid industrialization and urbanization. Simultaneously, there has also been a marked increase in MetS and its related outcomes, such as stroke, coronary heart disease, and cancers (Wang et al., 2011). Congruously, air pollution has become the most severe environmental problem in China (Guan et al., 2016). However, to our knowledge, no prior study has been conducted to evaluate the association between air pollution and MetS in the Chinese population. Therefore, considering the current MetS epidemic, the omnipresence of air pollution, and the scarcity of such an evaluation, our study aimed to assess the relationships of long-term pollution of ambient particulate matter (PM)—including particles with diameters $\leq 1.0\,\mu m$ (PM_1), $\leq 2.5\,\mu m$ ($PM_{2.5}$), and $\leq 10\,\mu m$ (PM_{10})—sulfur dioxide (SO_2), nitrogen dioxide (NO_2), and ozone (O_3) with MetS in Chinese adults. This aim was achieved by analyzing the data from the 33 Communities Chinese Health Study (33CCHS) (Dong et al., 2013; Yang et al., 2017). The comprehensive measurements of air pollutants, including $PM_{1.0}$ in particular, and the large sample size of the 33CCHS provided a unique opportunity to examine underlying exposure-response relationships.

2. Methods

2.1. Study population

The 33CCHS was a large, population-based, cross-sectional study conducted in northeastern China in 2009, with the aim of determining the association of ambient air pollution with cardiovascular diseases and main cardiometabolic risk factors. The study population has been previously described (Dong et al., 2013; Yang et al., 2017). Briefly, using a four-staged, stratified and cluster sampling method, participants were randomly selected from 33 communities (the sizes of these communities ranged from 0.25 to 0.64 km^2) of 11 districts in three Chinese cities (Shenyang, Anshan, and Jinzhou) (Fig. 1). The sample consisted of individuals in the age interval of 18–74 years, with a history of living in the community for at least five years. According to the sampling frame, 28,830 participants were selected, of whom 24,845 completed the questionnaire survey, giving an overall response rate of 86.2%. As 9368 individuals refused to provide a blood sample, finally 15,477 participants (62.3% of the 33 CCHS participants) were included in the current analysis. The study protocol was reviewed and approved by the Ethics Committee of Sun Yat-Sen University. Consistent with these measures, written informed consent was obtained from all participants after informing them of the study objectives, benefits, and confidentiality of personal information.

2.2. Air pollution estimation

Each district had one municipal air monitoring station, which was located within one kilometer of each household. Based on the protocol recommended by the State Environmental Protection Administration of China SEPA (1992), concentrations of PM_{10}, SO_2, NO_2, and O_3 were continuously measured and reported hourly using β-attentuation, ultraviolet fluorescence, chemiluminescence, and ultraviolet photometry, respectively. These measurements were then used to calculate daily mean concentrations of PM_{10}, SO_2, and NO_2, as well as an eight-hour mean of O_3, for the days in which more than 75% of hourly concentrations were available (for a detailed description, see the Supplemental material: an explanation of the air pollution data). Finally, three-year average concentrations, from 2006 to 2008, of the four pollutants were calculated and developed as surrogates for long-term air pollution exposure by linking air pollution data to residential addresses.

Daily PM_1 and $PM_{2.5}$ concentrations for the 33 communities were predicted at a $0.1^\circ \times 0.1^\circ$ spatial resolution during 2006–2008, using satellite remote sensing, meteorology, and land use information, which were detailed in our previous paper (Chen et al., 2018). In brief, two types of Moderate Resolution Imaging Spectroradiometer (MODIS) Collection six aerosol optical depth (AOD) data—Dark Target (DT) and Deep Blue (DB)—were combined. A generalized additive model was developed to link ground-monitored PM_1 and $PM_{2.5}$ data with AOD data and other spatial and temporal predictors (e.g., urban cover, forest cover, weather data, and calendar month).

2.3. Clinical measurements

Clinical measurements, including laboratory tests, have previously been described in detail (Dong et al., 2013; Song et al., 2015). All investigators and staff were required to complete a training program developed by the American Heart Association (AHA) prior to beginning data collection. Elements of the AHA training program protocol for study participants were as follows: tea, coffee, alcohol, and tobacco were not to be consumed and exercise was not to be undertaken for at least 30 min before measuring blood pressure (BP). BP was to be measured three times by carefully trained nurses using standardized mercuric-column sphygmomanometer. These measurements were to take place in a quiet and comfortable room, with the participants in the sitting position after five minutes of rest. An average of three consecutive pairs of BP measurements was to be recorded, with an interval of two minutes.

The height and weight of the participants were measured according to the standardized protocol developed by WHO (World Health Organization, 1995). Height was measured to the nearest 0.5 cm and weight was measured to the nearest 0.1 kg. Body mass index (BMI) was calculated as weight, in kilograms, divided by the square of height, in meters. Waist circumference (WC) was defined as the middle point between the lower rib and the upper margin of the iliac crest (World Health Organization, 1995). It was read to the nearest 0.5 cm by a nurse using a tape with an insertion buckle at one end.

Each study participant gave consent for trained nurses to collect a blood sample after an overnight fasting of ≥ 12 h. A Hitachi Autoanalyzer (Type 7170A; Hitachi Ltd.; Tokyo, Japan) was used to