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Association patterns for size-fractioned indoor particulate matter and black carbon and autonomic function differ between patients with chronic obstructive pulmonary disease and their healthy spouses☆

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ABSTRACT

Background: The effect of health status on the relationship between particulate matter (PM) and black carbon (BC) and cardiac autonomic function has not been examined sufficiently directly comparing patients with healthy participants.
Objectives: To evaluate the association patterns between size-fractioned indoor PM and BC and cardiac autonomic function in chronic obstructive pulmonary disease (COPD) patients and their healthy spouses.
Methods: Twenty-four-hour heart rate variability (HRV) and heart rate (HR) was measured in eight pairs of stable COPD patients and their healthy spouses. Real-time size-fractioned indoor PM and BC levels were monitored on the same, and preceding, days. Mixed-effects models were used to estimate the changes in health indices and pollutants after controlling for potential confounding variables.
Results: Increases in size-fractioned PM and BC were associated with alterations in cardiac autonomic function in both COPD patients and their healthy spouses. However, the association patterns differed between the two groups. In COPD group, an IQR (13.65 μg/m^3) increase in $PM_{0.5}$ at 12-h moving average was associated with reductions of 14.62% (95% CI: -21.74%, -6.86%) in total power (TP) and 10.14% (95% CI: -16.11%, -3.76%) in high frequency (HF) power. In healthy volunteers, however, TP and HF declined immediately upon exposure to PM and then returned to normal levels gradually. In this group, an IQR increase in $PM_{0.5}$ at 5 min moving average was associated a 20.30% (95% CI: -25.49%, -14.73%) reduction in TP and a 31.79% (95% CI: -36.48%, -26.72%) reduction in HF.
Conclusions: Exposure to indoor PM and BC was associated with cardiac autonomic dysfunction in COPD patients and their healthy spouses. Exposure had a greater lagged effect on HRV in COPD patients than in healthy participants. These findings will aid the formulation of targeted measures to prevent the adverse effects of indoor air pollution for individuals with different health statuses.

1. Introduction

Air pollution is a burgeoning environmental problem worldwide. Megacities in China regularly suffer from air pollution levels several folds higher than those set by international air quality guidelines. Numerous epidemiological studies have found that mortality and morbidity of cardiovascular and respiratory diseases increase after long-term or short-term exposure to ambient

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particulate matter (PM) (Brook et al., 2010; Talbott et al., 2014; Weichenthal et al., 2016).

As people spend more than 80% of time indoors, a growing body of research is focusing on the health effects of indoor pollution. The Global Burden of Diseases, Injuries and Risk Factors Study reported that indoor air pollution contributed to 85.6 million global disability-adjusted life-years (DALYs) in 2015 (Forouzanfar et al., 2016). Both indoor sources (such as cooking, smoking and movement of people) and outdoor sources (such as vehicular emissions and biomass burning) contribute to the overall indoor air pollution (Custódio et al., 2014). Black carbon (BC) constitutes a significant component of indoor air pollution, the health effects of indoor BC have been far less studied compared to ambient (outdoor) PM and BC. In addition, the World Health Organization (WHO) highlights that the health effects differ across particles of different size (WHO, 2003), yet previous indoor studies tend to focus on single-size PM (e.g. $PM_{2.5}$ or PM_{10}) instead of comparing the effects of PM of different sizes.

Human epidemiological studies as well as toxicological experiments on animals have provided evidence for several plausible biological pathways through which air pollutants may increase the mortality and morbidity of cardiovascular and respiratory diseases (Burgan et al., 2010; Shrey et al., 2011; Nelin et al., 2012; Huang et al., 2013). Cardiac autonomic dysfunction is an important postulated mechanism linking exposure to PM and cardiac health effects (Mustafic et al., 2012; Shah et al., 2013). It is hypothesized that PM can activate the pulmonary autonomic nervous arc reflex through oxidative stress and inflammation, and then lead to dysfunction in the autonomic nervous system that deregulates cardiac function (Brook et al., 2010; Nelin et al., 2012). Heart rate variability (HRV) reflects the activity of autonomic nervous function via sympathetic and parasympathetic efferent impulses of the heart rhythm (Stein and Kleiger, 1999). Some evidence suggests that decreased HRV may be an early and sensitive indicator of impairment in cardiac rhythm in response to alterations in autonomic nervous function to a stressor (e.g. high level of air pollution) (Nelin et al., 2012; Weichenthal et al., 2014). The autonomic nervous system also plays an important role in the modulation of normal heart rate (HR) (Camm and Fei, 1996).

Numerous epidemiologic studies have shown the relationship between ambient PM and changes in HRV and HR (Davoodi et al., 2010; Rich et al., 2012; Bartell et al., 2013). However, most of these studies have focused on the HRV changes in healthy persons or patients with cardiovascular disease, while few studies have investigated HRV in COPD patients after exposure to PM. The biological response may be more apparent because the response to airway deposition of particles can be greater in COPD patients (Kim and Kang, 1997). Epidemiological studies have also shown that patients with COPD have a significantly higher risk of cardiovascular disease compared with the non-COPD population (W. R. Chen et al., 2015). The influence of health status on the relationship between exposure to PM and BC and cardiac autonomic function, has not been adequately investigated, and studies that directly compare COPD patients with healthy participants are especially needed.

Here we report the findings of a repeated-measure study to evaluate the associations between exposure to size-fractioned indoor PM and BC and cardiac autonomic function in COPD patients and healthy individuals to determine whether these associations differ between individuals with different health statuses. These findings will aid the formulation of differential targeted measures to prevent the adverse effects of indoor pollution for COPD patients and non-COPD population.

2. Materials and methods

2.1. Study design and subjects

A repeated-measure study was conducted from March 28th to May 21th, 2016. Eight elderly subjects with diagnosed stable COPD were recruited from Peking University Third Hospital in Beijing, and their healthy spouses were also recruited. Ambulatory 24-h ECG of all participants were measured on the same day of the week.

To reduce the influence of individual heterogeneity, we recruited participants based on the following inclusion criteria: (1) patients with stable COPD who had a baseline post-bronchodilator 1 s forced expiratory volume (FEV1) of less than 80% predicted, and a post-bronchodilator FEV1/post-bronchodilator forced vital capacity (FVC) of less than 70%; (2) patients did not have other respiratory disorders such as asthma or bronchiectasis; (3) patients have lived in Beijing for more than one year; (4) healthy spouses did not have physician-diagnosed pulmonary or endocrine conditions, or severe cardiac disease; (5) patients had not used vasoactive medication and/or other drugs that might affect heart rhythm in the preceding 6 weeks. We excluded patients with a history of severe cardiac disease (such as cardiac failure and rheumatic heart disease), a history of lung surgery or cancer. Current active or passive smokers (living with a current smoker) were also excluded. Information on participants' age, gender, weight, height, medication use was obtained from baseline questionnaires. The study protocol was approved by The Institutional Review Board of Peking University Health Science Center. Each participant gave their informed consent before the study began.

2.2. Pollution exposure measurements

Indoor exposures to size-fractioned PM ($PM_{0.5}$, $PM_{1.0}$, $PM_{2.5}$, $PM_{5.0}$ and PM_{10}) and meteorological variables were monitored continuously for 12 h prior to health measurements, using a real-time particulate counter (Model Handheld PC3016; GrayWolf Inc., USA). BC concentrations were monitored by a portable, battery-powered micro-Aethalometer (microAeth Model AE51; Magee Scientific, Berkeley, CA, USA). Real-time levels of temperature (Temp) and relative humidity (RH) were recorded by a Temp/RH meter in 1-min intervals (Model WSZY-1B; Tianjianhuayi Inc., Beijing, CHINA). All of these instruments were installed at the participants' residence, in the room with greatest occupation (except the kitchen). All exposure data were aggregated as 5-min averages in order to synchronize with the 5-min HRV and HR data.

2.3. Fractioned exhaled nitric oxide/lung function measurements

At baseline, participants, both COPD and healthy groups, were measured with fractioned exhaled nitric oxide (FeNO) and lung function [peak expiratory flow (PEF) and FEV_1] under instructions of investigators. FeNO measurements were conducted before lung function since deep inspiration affects NO concentration (Deykin et al., 1998). The NIOX VERO® machine (Aerocrine AB, Solna, Sweden) was used for FeNO measurements according to ATS/ERS Recommendations (Peltier, 2005). Participants were asked to avoid physical exercise, food and drinks 1 h before tests. Lung function measurements were conducted with electronic PEF diary meter (Model 2110; Vitalograph Ltd., Buckingham, UK) following standard ATS/ERS Task Force (Miller et al., 2005). Before the study, we adjusted all diary meters with a calibrated 3-L Precision Syringe (Vitalograph Ltd., Buckingham, UK).