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Human bronchial epithelial cell injuries induced by fine particulate matter from sandstorm and non-sandstorm periods: Association with particle constituents

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ABSTRACT

Epidemiological studies have demonstrated the exacerbation of respiratory diseases following sandstorm-derived particulate matter (PM) exposure. The presence of anthropogenic and biological agents on the sandstorm PM and the escalation of PM < 2.5 μm (PM_{2.5}) pollution in China have led to serious concerns regarding the health effects of PM_{2.5} during Asian sandstorms. We investigated how changes in PM_{2.5} composition, as the weather transitioned towards a sandstorm, affected human airway epithelial cells. Six PM_{2.5} samples covering two sandstorm events and their respective background and transition periods were collected in Baotou, an industrial city near the Gobi Desert in China. PM samples from all three periods had mild cytotoxicity in human bronchial epithelial cell line BEAS-2B, which was positively correlated with the contents of polycyclic aromatic hydrocarbons and several metals. All PM samples potently increased the release of interleukin-6 (IL-6) and interleukin-8 (IL-8). Endotoxin in all samples contributed significantly to the IL-6 response, with only a minor effect on IL-8. Cr was positively correlated with both IL-6 and IL-8 release, while Si was only associated with the increase of IL-6. Our study suggests that local agricultural and industrial surroundings in addition to the sandstorm play important roles in the respiratory effects of sandstorm-derived PM.

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Introduction

An elevated level of ambient fine particulate matter (PM) with aerodynamic diameter < 2.5 μm (PM_{2.5}) is a major risk factor for increased cardiopulmonary morbidity and mortality

(Brook et al., 2008; Pope and Dockery, 2006). Epidemiological studies conducted in different continents (Chang et al., 2006; Gyan et al., 2005; Hirsch et al., 1974; Kanatani et al., 2010) have demonstrated that exposure to sandstorm particles can increase daily mortality and exacerbate cardiovascular and

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respiratory diseases. Sandstorm-derived particles are responsible for the surges of PM levels in Asia and can have both a short- and long-term impact on air quality at a local, regional, and even global scale (Duce et al., 1980; Fairlie et al., 2007; Wang et al., 2007). Sandstorm PM_{2.5} is a combination of fine sand dust from the desert surface and daily non-sandstorm PM_{2.5} accumulated prior to the sandstorm. Non-sandstorm PM_{2.5} mainly originates from local combustion and industrial emission sources or through secondary aerosol formation. These particles consist primarily of sulfate, nitrate, ammonium, organic and soil crust matter, elemental carbon, and some biological components (Wilson, 1998; Wu et al., 2014); whereas sand dust particles from Asia are mostly composed of metals (e.g., silicon, aluminum, calcium, and iron) (Honda et al., 2014). With sandstorms lasting for days, other chemical and biological agents can also attach to the sand dust particles during transport (Esmaeil et al., 2014).

Asian sandstorm particles, mainly from the Gobi Desert that covers southern Mongolia and parts of northern and northwestern China, can be transported to regions far beyond China (Duce et al., 1980; He et al., 2013). Because the combination of anthropogenic pollutants and biological agents on sandstorm PM may have an additional impact on human health, as PM_{2.5} pollution in China continues to intensify, there are serious concerns about the health effects of Asian sandstorm-derived PM among East Asian countries (He et al., 2013; Honda et al., 2014; Ichinose et al., 2009; Kanatani et al., 2010; Matsukawa et al., 2014; Mori et al., 2003). The adverse respiratory effects of non-sandstorm PM_{2.5} are mainly linked to the particles' chemical composition. Polycyclic aromatic hydrocarbons (PAHs) and metals are considered the primary components responsible for PM-induced inflammation in the lung (Charrier et al., 2014; Delfino, 2002; Li et al., 2003; Gerlofs-Nijland et al., 2009; Wu et al., 2012, 2013). Several studies have identified endotoxin as one of the biological components responsible for the inflammatory response of human airway epithelial cells and the organic contents responsible for PM-induced DNA damage; the latter has been postulated to contribute to the increase of lung cancer in China (Honda et al., 2014; Meng and Zhang, 2007; Osornio-Vargas et al., 2003; Wang et al., 2013; Zhang et al., 2009).

Currently, there is limited information on how the transition from the background weather to a sandstorm will affect PM_{2.5} composition and their cellular effects. Previous studies have focused on the adverse effects and properties of either sandstorm or non-sandstorm PM. This has resulted in a lack of information on the link between the health effects and time-resolved changes in particle composition. Moreover, there is an imperative need in understanding how the local agricultural and industrial surroundings contribute to the health effects of these particles. The objective of this study was to determine how the changes in the biological and chemical composition of PM_{2.5} affected the human airway epithelial cell response as sandstorms approached Baotou; a city surrounded by animal farming, rare earth mining, precious mineral smelting, and steel industries. The data from this study provides detailed information on the changes in the chemical and biological characteristics of ambient PM_{2.5} during a period covering three different weather conditions

(i.e., normal local weather, a sandstorm, and a transition period between the two) of two sandstorm episodes, and their effects on human bronchial epithelial cells. Correlation analyses suggest that local agricultural and industrial surrounding may affect the adverse cellular effects of these particles.

1. Materials and methods

1.1. Reagents and materials

The human bronchial epithelial cell line BEAS-2B was purchased from the American Type Culture Collection (ATCC, Manassas, VA, USA). Bronchial epithelial cell growth medium (BEGM) was obtained from Lonza (Walkersville, MD, USA). Cell culture-grade water, phosphate-buffered saline and polymyxin B (PB) were purchased from Sigma-Aldrich (St Louis, MO, USA). Trypsin-EDTA and a penicillin/streptomycin mixture were obtained from Invitrogen (San Diego, CA, USA). Limulus amoebocyte lysate (LAL) assay kit was purchased from Thermo Fisher Scientific (Rockford, IL, USA). Pierce™ Lactate Dehydrogenase Cytotoxicity Assay (LDH assay) kit was purchased from Life Technologies (Grand Island, NY, USA). Enzyme-linked immunosorbent assay (ELISA) kits for human interleukin-1β (IL-1β), interleukin-6 (IL-6), interleukin-8 (IL-8), tumor necrosis factor-α (TNF-α), and type I rat tail collagen were obtained from BD Biosciences (San Diego, CA, USA). ELISA kit for human thymic stromal lymphopoietin (TSLP) was purchased from eBioscience (San Diego, CA, USA). The PAH standards and their recovery surrogates (*p*-terphenyl D₁₄ and 2-fluorobiphenyl) were obtained from AccuStandard, Inc. (New Haven, CT, USA). Acetonitrile and *n*-hexane of Ultra Resi-Analyzed® standard were purchased from Merck KGaA (Darmstadt, Hesse-Darmstadt, Germany). Acetone and dichloromethane of Suprasolv® standard were purchased from Mallinckrodt Baker, Inc. (Phillipsburg, NJ, USA). Silica gel (100–200 mesh), neutral aluminum oxide (200–300 mesh), and granular anhydrous sodium sulfate were purchased from Beijing Chemical Reagent Co. (Beijing, China). Anhydrous sodium sulfate was baked at 600°C for 6 hr, and silica gel and neutral aluminum oxide were heated at 450°C for 8 hr to remove impurities. Silica gel and neutral aluminum oxide were further reactivated, then stored in a sealed desiccator and heated at 150°C for at least 16 hr before use. Granular anhydrous sodium sulfate was stored in a sealed glass bottle after cleaning. All glassware was cleaned with an ultrasonic cleaner (KQ-500B; Kunshan Ultrasonic Instruments Co., Jiangsu, China) for 30 min with liquid detergent, rinsed with distilled water, and finally heated at 450°C for 8 hr.

1.2. PM sampling and extraction

The details of the PM_{2.5} sampling have been described previously (Deng et al., 2007). Briefly, samples were collected using a high-volume sampler (Anderson, USA) with a quartz filter from March 6 to May 6, 2004 in Baotou. PM_{2.5} samples were collected daily for 24 hr (noon-to-noon) and six PM_{2.5} samples were used in this study. Based on local meteorological conditions and air-quality monitoring data, the selected

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