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Pro-inflammatory responses to $PM_{0.25}$ from airport and urban traffic emissions

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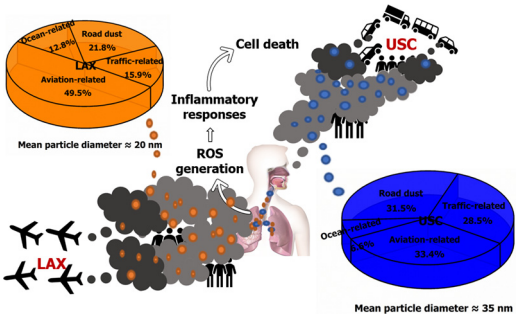
HIGHLIGHTS

- Aviation emission was the main contributor to $PM_{0.25}$ from a major airport.
- Urban area $PM_{0.25}$ was dominated by road traffic (traffic emission and road dust).
- Airport-related $PM_{0.25}$ exerts similar toxicity compared to $PM_{0.25}$ from urban traffic.

GRAPHICAL ABSTRACT



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ABSTRACT

Air traffic is rapidly growing, raising concerns about the air pollution in the surroundings of airports and its impact on public health. However, little is known about the impact of air pollution sources on air quality and health in the vicinity of airports. In this study, the sources and adverse health effects of airport-related particulate matter (PM) were investigated and compared to those of urban traffic emissions. Ambient $PM_{0.25}$ were collected at the Los Angeles International Airport (LAX) and at a central Los Angeles site (USC campus), along with $PM_{2.5}$ collected directly from turbine and diesel engines. The particle chemical composition, oxidative potential (OP) (ascorbic acid (AA), and electron spin resonance (ESR) assay) as well as their reactive oxygen species (ROS) activity, inflammatory potential (interleukin (IL) 6 and 8 and tumor necrosis factor (TNF)–α) and cytotoxicity on human bronchial epithelial (16HBE) cells were assessed. Chemical composition measurements confirmed that aircraft emissions were the major source to LAX $PM_{0.25}$, while the sources of the USC samples were more complex, including traffic emissions, suspended road and soil dust, and secondary aerosols. The traffic-related transition metals (Fe and Cu) in LAX and USC samples mainly affected OP values of particles, while multiple factors such as composition, size distribution and internalized amount of particles contributed to the promotion of ROS generation in 16HBE cells during 4 h exposure. Internalized particles in cells might also play an important role in activating inflammatory responses during cell recovery period, with LAX particles being more potent. Our results demonstrated considerable toxicity of airport-related particles, even at low exposure concentrations, suggesting that airport emission as source of $PM_{0.25}$ may also contribute to the adverse effects on public health attributable to PM. The potency of such particles is in the same range as those collected at a site in urban area impacted heavily by traffic emissions.

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1. Introduction

Due to the rapid development of the aviation industry and high demand for air transportation, the concomitant airport pollution has attracted increasing attention in recent years (Masiol and Harrison, 2014). Airport particulate matter (PM) emissions are the known source of air pollution in the proximity of an airport (Hu et al., 2009; Hudda et al., 2014). These particles, however, are not only from aircraft emissions (engine exhaust and non-exhaust emissions from aircraft), but also emissions from other sources like the ground traffic operations for transporting people and goods (Masiol and Harrison, 2014). However, current information regarding contributions of the relevant sources to airport PM emissions is inadequate, which hinders our ability to accurately assess the population risks associated with the exposure to these emissions.

Large airports are often located in the proximity of metropolises, consequently airport emissions may have considerable impact on public health in the surrounding urban areas. Barrett et al. (2010) estimated that around 8000 premature mortalities were attributable to aircraft cruise emissions globally every year (Barrett et al., 2010). Touri et al. (2013) examined the impact of airport pollution exposure on respiratory health by evaluation of studies on occupational exposure. Although the link between respiratory health effects and airport pollution exposure was shown in a few occupational studies, the correlation was weak and needs further research (Touri et al., 2013). Therefore, understanding the associated risks of exposure to airport-related PM in comparison with other major contributors to urban PM such as vehicle emissions are of great significance to public health officials and legislators.

PM is a rather complex mixture that could differ in size distribution and chemical composition depending on emission sources. Both size distribution and composition are major critical factors influencing particle toxicity (Kelly and Fussell, 2012). Particles in the nanometer-size range typically account for the majority of total particle number concentrations in airport areas (Masiol and Harrison, 2014). A recent LAX airport study shown that the mean diameter of particles from the LAX is around 20 nm that is distinctly smaller than particles emitted from the urban traffic area (USC samples: mean diameter ≈ 35 nm) (Shirmohammadi et al., 2017b). These nano-sized airport-related particles result in a higher surface area/mass ratio compared to micron-sized PM, which allows more organic and inorganic species to be adsorbed and/or absorbed on their surface. This, in turn, could increase the particle toxicity per unit inhaled mass (Li et al., 2003; Nel et al., 2006). Many relevant toxic effects of PM might be triggered through PM-induced oxidative stress due to reactive oxygen species (ROS) generation in cells (Ayres et al., 2008; Xiang et al., 2016). PM-induced ROS can oxidize lipids and damage DNA, thereby impairing natural defense mechanisms, and lead to excessive production of inflammatory mediators, which are highly related to many respiratory and lung inflammatory diseases. In particular, small particle size and PM species including elemental carbon (EC), a number of organics as well as soluble transition metals (including Ni, V, Fe, and Cu) can have significant effects on the toxicity of PM as evidenced by previous studies (Aust et al., 2002; Kelly and Fussell, 2012; Loxham et al., 2013). However, there is a general lack of information on the health hazards of aviation-released PM compared to that of other mobile transportation sources such as road traffic.

The primary goal of the study presented here was to explore the health hazards of two important PM sources in relation to their PM composition. We hypothesized that airport-related $PM_{0.25}$ can induce comparable cytotoxicity to particles collected from an urban area, impacted mostly by road traffic emissions. To test this hypothesis, we collected $PM_{0.25}$ samples downwind the Los Angeles International airport and in the downtown area of Los Angeles, CA, along with PM samples directly collected from the exhaust of a turbine and a diesel engine, then examined how these different PM samples affect the biological responses in human bronchial epithelial (16HBE) cells.

2. Materials and methods

2.1. PM sampling

2.1.1. Urban air PM samples

Ambient $PM_{0.25}$ samples were collected in two locations in Los Angeles, as discussed in more detail by Shirmohammadi et al. (2017a). One sampling site was near the residential area of Playa del Rey, at a South Coast Air Quality Management District (AQMD) LAX Hastings monitoring site, which is located adjacent to the downwind Los Angeles International Airport (LAX). The $PM_{0.25}$ collected in this site was mainly influenced by the airport emissions and to a lesser extent by shore ocean breeze, and are less affected by traffic emissions (Shirmohammadi et al., 2017a). The other site was in central Los Angeles, at the University of Southern California (USC), roughly 150 m from- and immediately downwind the major freeway (I-110). Samples collected here mainly represent urban mixed particles, mostly dominated by road traffic emissions (Minguillón et al., 2008; Sowlat et al., 2016).

Personal Cascade Impactor Samplers (PCIS) (SKC, Inc., Eighty-Four, PA, USA) were used for sampling PM at the flow rate of 9 lpm (Misra et al., 2002). The sampling duration for each sample was 7 days from late October to early December of 2016 (for a total of 5 weeks). Each site was visited once a week to collect samples and load new filters. Flow rates were checked during the visit to ensure proper performance of the PCIS samplers. PM was classified in two particle collection size ranges: accumulation mode (0.25–2.5 μm, $PM_{2.5-0.25}$) and quasi-ultrafine mode (<0.25 μm, $PM_{0.25}$). Only $PM_{0.25}$ samples have been used for the work discussed in this paper, as they were considered more representative of direct primary emissions compared to the accumulation mode range that contains a higher fraction of regional aerosols (Shirmohammadi et al., 2017a). During the sampling period, one PCIS loaded with 37-mm quartz filters (Whatman International Ltd., Maidstone, England) was used for organic chemical speciation measurements (details can be found in Shirmohammadi et al., 2017a). The other three PCISs were loaded with 37-mm Teflon filters (Pall Life Sciences, 3-μm pore, Ann Arbor, MI) for gravimetric measurements to determine mass concentration as well as for the additional chemical and biological analyses discussed in this study.

2.1.2. Turbine and diesel samples

Turbine and diesel samples were collected directly from diluted exhaust of a Fighting Falcon turbine engine (F100, Pratt & Whitney, East Hartford, Connecticut, USA) and a low-sulfur fuel diesel (EN 590) engine (Bredenoord, 35 KVA Silent, Apeldoorn, Netherlands), respectively, by means of a versatile aerosol concentration enrichment system (VACES) (Kim et al., 2001) as well as a high-volume cascade impactor (HVCI) sampler (Demokritou et al., 2002) in this study. Minor flows of the VACES were used to collect droplets containing particles into a BioSampler (SKC, Inc., Eighty-Four, PA, USA) as well as passing the concentrated aerosol through a diffusion dryer and subsequently collect PM on 47-mm Teflon filters (Pall Life Sciences, 2-μm pore, Ann Arbor, MI, USA) for the biological and chemical analyses, respectively. Albeit that the VACES was equipped with a $PM_{2.5}$ size selective inlet, the vast majority of the particles from both test engines were <0.1 μm. Sampling time for each day lasted for 8 h and a filter was collected in parallel to the BioSampler, then a new filter was loaded for the next sampling day, resulting in 1 turbine sample and 3 diesel samples were collected. Flow rates, heating and cooling temperatures, and particle number concentration as well as concentration enrichment of the VACES were checked every 2 h to ensure proper performance. The HVCI consists of different impaction stages to collect particles in different size ranges. 3 impaction stages (cut-points at 10, 2.5 and 0.1 μm, respectively) were selected in this study, and downstream of the third stage, a backup TE 38 filter (Whatman, 5-μm pore, Dassel, Niedersachsen, Germany) was placed for collecting the ultrafine particles (<0.1 μm) for chemical