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Source-to-receptor pathways of anthropogenic $PM_{2.5}$ in Detroit, Michigan: Comparison of two inhalation exposure studies

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ABSTRACT

Recent studies have attributed toxic effects of ambient fine particulate matter (aerodynamic diameter ≤ 2.5 μm; $PM_{2.5}$) to physical and/or chemical properties rather than total mass. However, identifying specific components or sources of a complex mixture of ambient $PM_{2.5}$ that are responsible for adverse health effects is still challenging. In order to improve our understanding of source-to-receptor pathways for ambient $PM_{2.5}$ (links between sources of ambient $PM_{2.5}$ and measures of biologically relevant dose), integrated inhalation toxicology studies using animal models and concentrated air particles (CAPs) were completed in southwest Detroit, a community where the pediatric asthma rate is more than twice the national average. Ambient $PM_{2.5}$ was concentrated with a Harvard fine particle concentrator housed in *AirCARE1*, a mobile air research laboratory which facilitates inhalation exposure studies in real-world settings. Detailed characterizations of ambient $PM_{2.5}$ and CAPs, identification of major emission sources of $PM_{2.5}$, and quantification of trace elements in the lung tissues of laboratory rats that were exposed to CAPs for two distinct 3-day exposure periods were completed.

This paper describes the physical/chemical properties and sources of $PM_{2.5}$, pulmonary metal concentrations and meteorology from two different 3-day exposure periods—both conducted at the southwest Detroit location in July 2003—which resulted in disparate biological effects. More specifically, during one of the exposure periods, ambient $PM_{2.5}$-derived trace metals were recovered from lung tissues of CAPs-exposed animals, and these metals were linked to local combustion point sources in southwest Detroit via receptor modeling and meteorology; whereas in the other exposure period, no such trace metals were observed. By comparing these two disparate results, this investigation was able to define possible links between $PM_{2.5}$ emitted from refineries and incinerators and biologically relevant dose, which in turn may be associated with observed health effects.

1. Introduction

Bioactivity of ambient fine particulate matter (aerodynamic diameter ≤ 2.5 μm; $PM_{2.5}$) has generally been attributed to both physical (e.g., Li et al., 2003; Oberdorster et al., 1995; Peters et al., 1997) and chemical (e.g. Prahalad et al., 1999; Valko et al., 2005) properties rather than total mass. It has also been hypothesized that combustion-derived nanoparticles act through common pathways to produce inflammation, and that oxidative stress is the lead mechanism driving adverse health effects (Donaldson et al., 2005). Other studies have shown that transition metals along with PAH and ultrafine carbon particles can exert oxidative stress on biological systems (Stohs and Bagchi, 1995). However, identifying specific sources and components from a complex mixture of ambient $PM_{2.5}$ that are associated with adverse health effects is still challenging.

In order to assess source- and component-specific health risks, a Harvard/EPA ambient particle concentrator has been widely used to concentrate fine particles from ambient air for inhalation exposure studies (Kodavanti et al., 2005; Sioutas et al., 1997). Several studies have found correlations between specific CAPs elements such as vanadium, lead and zinc, and pulmonary inflammation as indicated by neutrophils in bronchoalveolar lavage fluid (BALF) and with chemiluminescence (Clarke et al., 2000; Gurgueira et al., 2002;

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Saldiva et al., 2002). In these previous studies the biological endpoints were analyzed with respect to the elevated elemental composition of CAPs. Likewise, the present investigation studied exposure and health effects, but then went a step further, identifying pulmonary concentrations of metal composition as well as sources of the recovered metals while conducting inhalation exposure studies. In linking the adverse health effects of airborne toxicants with their sources, assessment of the pulmonary concentrations of ambient $PM_{2.5}$ is important since the health effects are likely to be associated with the dose delivered to the target tissue of concern, rather than the external exposure.

Metals are important trace constituents of ambient particles since they can function as tracers for specific emission sources, and specific metal particles from combustion emission sources may be associated with the adverse health impacts of ambient $PM_{2.5}$ (Gavett et al., 2003; Lippmann et al., 2006; Riley et al., 2003; Samet et al., 2000). Our previous inhalation exposure study using laboratory rats concluded that the CAPs-induced enhancement of allergic airway responses was not associated with the mass, and might be mediated in part by increased pulmonary deposition of potentially to toxic trace metal elements generated from refineries and incinerators in southwest Detroit (Harkema et al., 2004; Morishita et al., 2004).

In the present study we have reported that a 3-day exposure to CAPs (July 14–16, 2003) markedly enhanced the allergen-induced allergic airway disease in the CAPs-exposed animals. Specifically, airway eosinophilic inflammation and mucus hyperproduction and secretion were exacerbated when allergic animals were exposed to CAPs (Harkema et al., in press). However, we saw no CAPs-induced enhancement when we repeated this protocol on a different set of animals (July 21–23, 2003). In an effort to identify differences in air quality that might explain the disparate biological effects, the two 3-day exposure studies were closely compared in terms of source-specific ambient $PM_{2.5}$. Specifically, by comparing the physical and chemical properties, meteorology, pulmonary metal concentrations, and sources of $PM_{2.5}$ from the two exposure periods, this investigation identified sources that likely impacted the study site and caused the observed health effects.

2. Materials and methods

2.1. Site description

AirCARE1, a 53-ft mobile air research laboratory, was parked next to Maybury Elementary School in a residential neighborhood in southwest Detroit, where multiple other research studies have been conducted in order to assess the impact of ambient PM and air toxic sources on human populations and the neighborhoods (Keeler et al., 2002; Lewis et al., 2005; USEPA, 2007). Fig. 1 shows a map of Detroit area and the location of AirCARE1 during the inhalation exposure studies. The map also shows some of major $PM_{2.5}$ point sources in Wayne County (EPA 1999). These southwest Detroit communities also are subjected to heavy motor vehicle traffic due to their proximity to major interstates and the entrance to the Ambassador Bridge which is the busiest border crossing between US and Canada. Seven counties in southeast Michigan including these southwest Detroit communities have been designated as a $PM_{2.5}$ non-attainment area.

2.2. Inhalation exposure studies

Mobile air research laboratory: AirCARE1 contains whole-body inhalation chambers with a Harvard-type ambient fine particle concentrator, a biomedical lab, an inhalation exposure lab, and an atmospheric monitoring lab. The fine particle concentrator is a three-stage aerosol concentrator that utilizes virtual impactors to

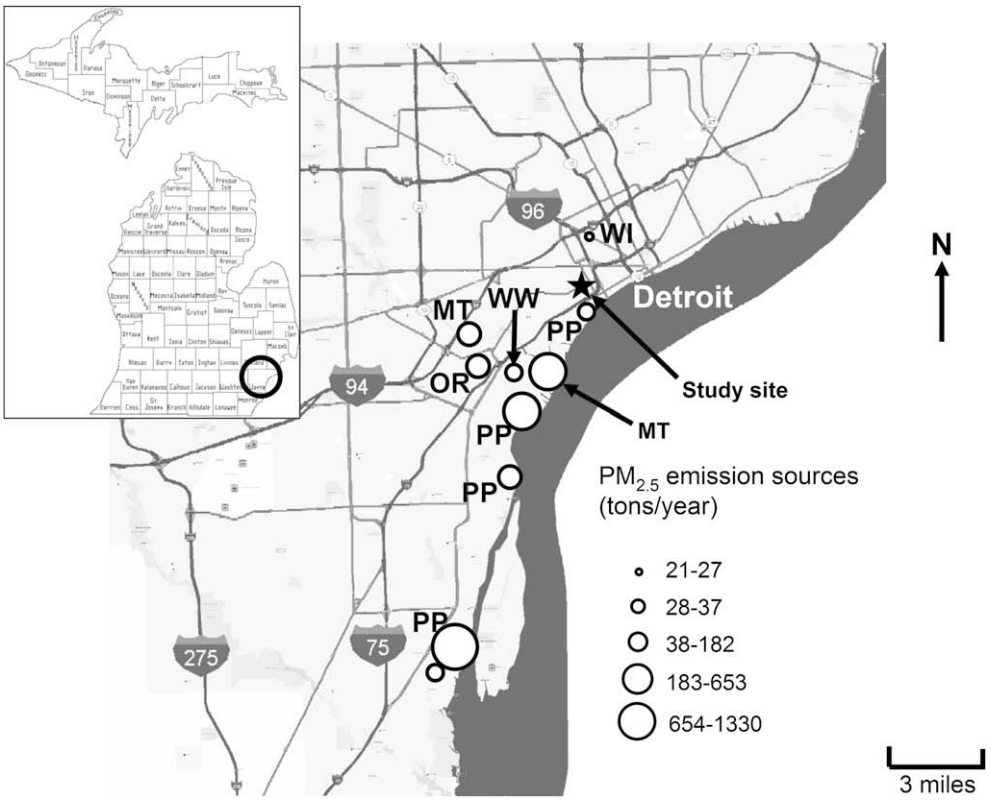

Fig. 1. A map of southwest Detroit showing the location of the sampling site and major industrial sources for $PM_{2.5}$ in Wayne County, Michigan (USEPA. AirDATA NET, 1999). (MT: metals/steel industries, OR: oil refineries, PP: power plants, WI: waste incinerator, WW: waste water treatment and sludge incinerator).