

Final Technical Report

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EPA Grant Number: R827351C005

Center Name: NYU-EPA PM Center: Health Risks of PM Components

Center Director: Morton Lippmann

Title: Physicochemical Parameters of Combustion Generated Atmospheres as Determinants of PM Toxicity

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Project Period: June 1, 1999–May 31, 2005 (no-cost extension to May 31, 2006)

Period Covered by the Report: June 1, 1999–May 31, 2006

RFA: Airborne Particulate Matter (PM) Centers (1999)

Research Category: Particulate Matter

Objective(s) of the Research Project: Combustion generated particles often make up a significant portion of ambient particulate matter (PM) in many regions. This study examines the hypothesis that the toxicological effects associated with combustion-generated PM depend upon specific physicochemical characteristics of the particles. PM effluents from high temperature processes, such as fossil fuel combustion and pyrometallurgical systems, consist of inorganic materials having a wide size range and chemical composition, including H₂SO₄ and unreacted SO₂. Such effluents have been shown to be toxicologically active. Freshly formed acidic fly ash atmospheres (containing SO₂ and ultrafine particles with transition metals on their surface) produce decrements in lung function (Amdur, et al., 1986; Chen, et al., 1990). Furthermore, sulfuric acid as a coating on particle surfaces has been shown to be 10 fold more potent in producing pulmonary effects than are pure acid droplets of the same H⁺ concentration (Amdur and Chen, 1989). Epidemiological data have indicated increased daily mortality to be associated with particulate air pollution indices, and a significant contribution from SO₂ could not be ruled out. Since SO₂, by itself, has low toxicity, it is reasonable to speculate that a synergistic interaction between SO₂ and particles may have been responsible for these observed effects (Amdur and Chen, 1989; Amdur, et al., 1986).

Several human panel studies in the U.S. and the MONICA study in Europe (Gold, et al., 1998; Pope, et al., 1998; Shy, et al., 1998; Peters, et al., 1998), as well as animal studies (Watkinson, et al., 1998; Lovett, et al., 1998; Nadziejko, et al., 1997) have suggested an association between PM and changes in host homeostasis. In this study, cardiopulmonary effects are measured in healthy and compromised animals exposed by inhalation to laboratory-generated particle atmospheres having precisely defined physicochemical characteristics.

This study examined the hypothesis that the toxicological effects associated with combustion-generated PM depend upon specific physicochemical characteristics of the particles and determined the influence of physicochemical parameters of combustion generated PM on the time course, dose response, and persistence of particle-induced cardiopulmonary effects.

Summary of Findings:

Technical Aspects

This project was closely integrated with Project 4 (R827351C006; Nadziejko, PI) in the measurement of cardiopulmonary effects upon exposures to various PM atmospheres. The accomplishments of Project 4 are separately reported by Dr. Nadziejko.

We have developed two furnace systems to produce realistic combustion effluents, and have successfully produced a mixture of carbon, SO₂, and metal (iron or copper). This allows determination of specific components, especially metals, which may be responsible for adverse health effects, and an assessment of whether any effects could be nonspecific, i.e., they follow inhalation of any type of particle. For the work described herein, the electronics for temperature regulations of both furnace systems were updated. To produce Fe (or Cu), and S coated carbon particles, sucrose solutions containing varying concentrations of Fe(NO₃)₃ (or Cu(NO₃)₂) were produced by a nebulizer and burned in the furnace system previously used to produce coal fly ash. The mass median diameters (MMD, determined by a Mercer impactor) of particles produced by a Collison nebulizer (before combustion) using 10 sucrose solutions (each containing 1117 ppm Fe) were 0.9 µm. When a 10% sucrose solution containing 1117 ppm Fe (or Cu) was burned in the furnace at 750°C in the presence of 1 ppm SO₂, ultrafine particles with a median diameter of 32 ± 1.3 nm (34.0 ± 7.4 nm for Cu) and sg of 1.55 were produced. Number concentrations as high as 1.9 x 10⁷ particles/cc were achieved. XRF was used to measure the concentrations of iron, copper, and sulfur in these particles. At this combustion condition, the particles produced from this furnace contained 35.1% and 3.6% by mass of iron and sulfur, respectively (30.6% copper and 6.9% sulfur when copper was used). It appeared that copper is almost twice as efficient (6.9% vs. 3.6%) in converting sulfur dioxide gas to particle-associated sulfur.

Sprague Dawley rats were exposed to furnace gas or 450 µg/m³ of these particles for 3 hours and their lungs were lavaged 24 hr post exposure. A lead oxide diffusion denuder was used to remove SO₂ from the exposure atmospheres. None of the exposure atmospheres produce changes in LDH levels in the lavage fluid. However, those aerosols containing a mixture of iron, SO₂, and carbon produced a 6.8 fold increase over the furnace gas control for the total number of cells in the lavage, whereas particles containing copper, SO₂, and carbon did not produce any change in this parameter. The results are shown in Table 1.

Table 1. Effects of Ultrafine Particles in Rats

Exposure Atmospheres	Total Cell Counts (10 ⁶)	LDH (BB unit)
Furnace Gas	0.70 ± 0.14	95.5 ± 10.2
SO ₂ + carbon	1.52 ± 0.31	78.7 ± 6.3
Copper + SO ₂ + carbon	1.52 ± 0.23	80.0 ± 13.5
Iron + SO ₂ + carbon	4.77 ± 0.41*	113.7 ± 28.2

Values were mean ± SE, (n=4 to 7 per exposure group).

* significantly different than furnace gas control (p < 0.0001).

References:

Amdur MO, Chen LC. Furnace generated acid aerosols: speciation and pulmonary effects. *Environmental Health Perspectives* 1989;79:147-150.

Amdur MO, Sarofim AF, Neville M, Quann RJ, McCarthy JF, Elliot JF, Lam HF, Rogers AE, Conner MW. Coal combustion aerosols and SO₂: An interdisciplinary analysis. *Environmental Science & Technology* 1986;20:138-145.

Chen LC, Lam HF, Kim EJ, Guty J, Amdur M.O. Pulmonary effects of ultrafine coal fly ash inhaled by guinea pigs. *Journal of Toxicology and Environmental Health* 1990;29:169-184.

Gold DR, Litonjua A, Schwartz J, Verrier M, Milstein R, Larson A, Lovett E, Verrier R. Cardiovascular vulnerability to particulate air pollution. *American Journal of Respiratory and Critical Care Medicine* 1998;157:A261.

Lovett EG, Verrier RL, Catalano P, Sioutas C, Murthy GGK, Wolfson JM, Ferguson ST, Koutrakis P, Reinisch U, Killingsworth CR, Coull B, Godleski JJ. Heart rate variability (HRV) analysis suggests altered autonomic influence in canines exposed to concentrated ambient air particles (CAPs). *American Journal of Respiratory and Critical Care Medicine* 1998;157:A260.

Nadziejko C, Chen LC, Zelikoff JT, Gordon T. Hematological and cardiovascular effects of acute exposure to ambient particulate matter. *American Journal of Respiratory and Critical Care Medicine* 155:A247 (1997).

Peters A, Perz S, Doring A, Stieber J, Koenig W, Wichmann HE. Increased heart rate during an air pollution episode. Presented at the Fourteenth HEI Annual Conference, Boston, MA, April 5-7, 1998.

Pope CA III, Dockery DW, Kanner RE, Villegas M, Schwartz J. Daily changes in oxygen saturation and pulse rate associated with particulate air pollution and barometric pressure. Presented at the Fourteenth HEI Annual Conference, Boston, MA, April 5-7, 1998.

Shy C, Creason J, Williams R, Liao D, Zweidinger R, Watts R, Devlin R, Hazucha M, Nestor J. Physiological responses of elderly persons to particulate air pollution. Presented at the Fourteenth HEI Annual Conference, Boston, MA, April 5-7, 1998.

Watkinson WP, Campen MJ, Kodavanti UP, Ledbetter AD, Costa DL. Effects of inhaled residual oil fly ash particles on electrocardiographic and thermoregulatory parameters in normal and compromised rats. *American Journal of Respiratory and Critical Care Medicine* 1998;157:A150.

Supplemental Keywords: NA

Relevant Web Sites: <http://www.med.nyu.edu/environmental/>
<http://es.epa.gov/ncer/science/pm/centers.html>

