

World Trade Center Fine Particulate Matter—Chemistry and Toxic Respiratory Effects: An Overview

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The 11 September 2001 terrorist attack on New York City's World Trade Center (WTC) caused an unprecedented environmental emergency. The collapse of the towers sent a tremendous cloud of crushed building materials and other pollutants into the air of lower Manhattan. In response to the calamity, federal, state, and city environmental authorities and research institutes devoted enormous resources to evaluate the impact of WTC-derived air pollution on public health. Unfortunately, on the day of the disaster, no air-sampling monitors were operating close to the WTC site to characterize and quantify pollutants in the dust cloud. However, analysis of fallen dust samples collected 5 and 6 days after the attack showed that 1–4% by weight consisted of particles small enough to be respirable (Lioy et al. 2002). These particles included fine particulate matter, or PM_{2.5} [PM < 2.5 μm mass median aerodynamic diameter (MMAD)], which can be inhaled deep into the lung and is associated with cardiovascular and respiratory health effects. Because of the extremely high concentrations of dust immediately after the collapse of the towers, even a relatively small proportion of PM_{2.5} in the dust clouds could have contributed to breathing problems in rescue workers and others who were not wearing protective masks.

In this issue of *Environmental Health Perspectives*, scientists from the U.S. Environmental Protection Agency, New York University, and Michigan State University report the results of a series of studies on the chemical properties of the PM_{2.5} fraction of the dust derived from the destruction of the WTC (McGee et al. 2003) and its toxicity in the respiratory tracts of mice (Gavett et al. 2003). They collected fallen dust samples 1 and 2 days after the attack from sites within a half-mile of Ground Zero and later isolated the PM_{2.5} fraction. The PM_{2.5} samples isolated from settled WTC dust were compared with reference PM_{2.5} samples that have been well characterized with respect to chemistry and pulmonary toxicology in rodents. The authors found that the WTC PM_{2.5} samples were alkaline and composed primarily of calcium-based compounds such as calcium

sulfate (gypsum) and calcium carbonate (calcite). These and other compounds and elements were attributed to crushed building materials such as cement, concrete aggregate, ceiling tiles, and wallboard.

Samples of WTC PM_{2.5} induced mild to moderate degrees of lung inflammation when administered at a relatively high dose of 100 μg directly into the airways of mice, which was less than that caused by a toxic emission source PM [residual oil fly ash (ROFA)] or a standard ambient air PM sample [labeled by the National Institute of Standards and Technology (NIST) as Standard Reference Material (SRM) 1649a]. Most important, this dose of WTC PM_{2.5} caused airway hyperresponsiveness to methacholine comparable to that from SRM 1649a and greater than that from the toxic ROFA sample. The hyperresponsiveness indicates that the high-dose group was primed to react to triggering agents that can constrict the airways. In humans these triggers include cold dry air and cigarette smoke. Lower doses of 10 and 32 μg administered directly into the airways or inhalation of 11 mg WTC PM_{2.5}/m³ air did not induce significant inflammation or hyperresponsiveness. These results showed that a relatively high dose (100 μg) of WTC PM_{2.5} can elicit short-term effects in healthy mice (1–3 days after exposure) and suggested that a similarly high dose would be necessary to elicit effects in healthy people.

Although the concentrations of PM_{2.5} immediately following the WTC collapse are unknown, it was estimated that healthy people exposed to about 425 μg WTC PM_{2.5}/m³ air for 8 hr would receive a dose comparable to the high dose in the mice. Although this concentration is about 20 times higher than normal background levels of PM_{2.5}, these conditions likely existed in the immediate aftermath of the collapse of the towers. The authors concluded that inhalation of high doses of WTC PM_{2.5} derived from building materials could contribute to the development of pulmonary inflammation, airway hyperresponsiveness, and cough. Individuals especially sensitive to inhalation of dusts, such as asthmatics, may experience these

effects at lower doses of inhaled WTC PM_{2.5}. However, most people would not be expected to experience adverse short-term respiratory effects from exposure to even moderately high WTC PM_{2.5} levels (estimated at 130 μg/m³ for 8 hr). The persistence of any adverse effects of inhaled WTC PM_{2.5} is unknown and was not addressed in these studies. It is important to note that coarse PM (2.5–10 μm MMAD) associated with the dust could have contributed to some of the respiratory problems reported in individuals working and living around Ground Zero, but only fine PM was tested in these studies. These studies provide essential information on the chemistry and respiratory toxicity of fine WTC PM, which is a necessary component in the evaluation of health risks from the World Trade Center disaster.

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