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Effects of Suspended Sulfates on Human Health

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Recent evidence from epidemiologic studies conducted in several areas of the United States shows an association of excess risk of asthmatic attacks with elevated levels of suspended sulfates within specific temperature ranges. These findings are discussed in the context of experimental animal studies which tend to support these observations.

In the past, the major research effort in the study of adverse health effects of air pollutants has centered around the primary pollutants namely sulfur dioxide, total suspended particulates (TSP), nitrogen dioxide, and ozone. Recently, however, we have come to realize that some of the transformation products of these pollutants may be more toxic than the primary pollutants themselves. A case in point is that of suspended sulfates.

Recent reports emanating from epidemiologic studies carried out as part of the Community Health and Environmental Surveillance System of EPA indicates the levels of suspended sulfates associated with certain adverse health effects were lower than the levels of SO_2 and TSP associated with the same health effect.

A study of asthmatics carried out in the Metropolitan New York area showed that when temperatures rose to 30–50°F, dose-related increments in asthma attacks were associated with increments in total suspended particulates and suspended sulfates but not sulfur dioxide. The estimated threshold level for total suspended particulates was 56 $\mu g/m^3$, while that for suspended sulfates was 12 $\mu g/m^3$ (*1*). In a similar study of asthmatics in the Salt Lake Basin, the highest morbidity rates were associated with elevated suspended sulfate levels. The estimated threshold for aggravation of asthma by suspended sulfates was 1.4 $\mu g/m^3$ on warm days with maximum temperatures above 50°F while that for suspended particulates at the same temperature was 71 $\mu g/m^3$ (*2*).

In a study of cardiopulmonary patients in the New York metropolitan area, the strongest and most consistent pollutant effects were associated with suspended sulfates for aggravation of such symptoms as shortness of breath, cough and increased production of phlegm. There was evidence that annual average suspended sulfate levels of 10–12 $\mu g/m^3$ was accompanied by morbidity excess which averaged about 6% when temperatures were 30–50°F and 32% with temperatures greater than 50°F (*3*).

Since these initial studies, subsequent studies of asthmatics in the New York–New Jersey Metropolitan area and in two communities in the southeast support previous findings that exposure to elevated levels of suspended sulfates when accompanied by elevated temperatures may contribute to excess risk of asthmatic attacks. These later studies also showed that suspended nitrates may have a similar effect and in some instances the combination of elevated suspended nitrates and suspended sulfates seemed to exert

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a greater effect than either pollutant alone (*4*).

The associations found in these epidemiologic studies by themselves are insufficient to incriminate suspended sulfates as a causative agent of certain adverse health effects. However, when these findings are coupled with those from experimental animal studies, the observations appear more than spurious, and a rationale for the pathogenesis of the observed effect begins to emerge.

Studies conducted by Amdur (*5*) with the guinea pig as the primary model, have shown that in terms of comparative toxicity, sulfuric acid and some metallic sulfate compounds such as zinc ammonium sulfate, are more potent irritants than sulfur dioxide. In some instances the differences in biologic activity was as much as 20-fold among the various sulfate compounds and between different particle sizes of the same compound. Amdur also reported a much smaller quantity of sulfur dioxide delivered to the respiratory system as a particle will produce an increase in flow resistance. Functional data from the guinea pig may mimic the human asthmatic, but this fact cannot be solidly determined until there is more information from similar studies on other animal species.

Recent experiments carried out by Frank and McJilton (*6*) indicate the importance of relative humidity in the response of animals to the SO_2/sodium chloride aerosol atmosphere. Guinea pigs were exposed for 1-hr intervals to atmospheres at two modes of relative humidity, namely, low (40%) and high (80%). Atmospheres were SO_2 alone (2620 $\mu g/m^3$), sodium chloride aerosol alone (900–1000 $\mu g/m^3$, and sodium chloride and sulfur dioxide combined. Significant changes in pulmonary flow resistance occurred only in the combined SO_2–sodium chloride aerosol atmosphere at high relative humidity. The authors suggest that the hydration of the particles and the subsequent uptake of SO_2 proceeds rapidly at high relative humidity. These findings tend to confirm those reported earlier by Amdur concerning the importance of the particle in the pulmonary response to SO_2.

In a study conducted by Hazelton Laboratory (*7*), groups of cynomolgus monkeys were exposed for 78 continuous weeks to sulfuric acid mist at concentrations varying from 0.38 to 4.79 mg/m^3 and particle size varying from submicronic to 4 μm mass median diameter (MMD). The results signified concentrations of 2.43 and 4.79 mg/m^3 with particles of 3.60 and 0.73 μm MMD, respectively, were sufficient to produce definite deleterious effects on pulmonary structures and deterioration in pulmonary function. Microscopic changes observed were principally characterized by focal epithelial hyperplasia and focal thickening of bronchiolar walls.

The aforementioned studies are not without certain limitations. Most of the animal studies represent the work of one investigator using the guinea pig as the principal model. A major limitation in our epidemiologic studies is the inability to characterize the measured sulfate compounds in terms of their physical and chemical properties. This poses a problem in trying to replicate the findings with respect to suspended sulfates since the chemical composition of sulfates may vary from one area to another and even within the same community over time. Another problem concerns the limited methods for measuring sulfates and sulfuric acid in ambient air. Present methods of collection on fiber glass filters might result in artifactual conversion of SO_2 to sulfate or to subsequent loss of sulfate due to decay of such compounds with time on high volume filters.

Despite these limitations there is sufficient evidence at the present time to consider suspended sulfates as potentially injurious to human health. This compels us to undertake a concerted research effort to define more precisely the adverse health effects of suspended sulfates and sulfuric acid on human health.

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