Environmental Health Perspectives
Vol. 79, pp. 147-150, 1989

Furnace-Generated Acid Aerosols: Speciation and Pulmonary Effects

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Guinea pigs were exposed to ultrafine aerosols (< 0.1 μm) of zinc oxide with a surface layer of sulfuric acid. These acid-coated aerosols are typical of primary emissions from smelters and coal combustors. Repeated daily 3-hr exposures for 5 days produce decrements in lung volumes and pulmonary diffusing capacity and elevations of lung weight/body weight ratio, protein, and number of neutrophils in pulmonary lavage fluid at concentrations of 20 μg/m³. A single 1-hr exposure to 20 μg/m³ causes increased bronchial reactivity. Higher concentrations of conventionally generated sulfuric acid mist are required to produce responses of similar magnitude.

Introduction

Primary emissions from coal combustion and smelting operations include ultrafine aerosols (< 0.1 μm) coated with a layer of sulfuric acid (H_2SO_4). Such particles are of considerable toxicological importance because they are small enough to penetrate the sensitive alveolar regions of the lung, and even though the actual H_2SO_4 concentration may be very low, it is all on the surface of the particles and thus readily available to produce an irritant response. For meaningful interpretation of toxicological data, both quantitative and qualitative data are needed on the speciation of the sulfur present in the surface layer. For toxicological studies, these acid-coated aerosols must be freshly generated. Collected, resuspended fly ash is completely useless for this purpose because the critical acid layer would not survive storage.

We use two systems for generation of these acid aerosols. One system permits us to study pure metal oxides, the other permits study of the ultrafine fraction of coal combustion aerosols. Our prototype pure metal oxide aerosol has been zinc oxide (ZnO) (count median diameter 0.05 μm, σ_g 2.0) mixed with sulfur dioxide (SO_2) and water vapor at 500°C (*1*). Qualitative data obtained with electron spectroscopy for chemical analysis (ESCA) showed that the sulfur (S) species associated with the aerosol under these mixing conditions were predominantly S (VI) with a lesser amount of S (IV) (*2*). The sulfur peaks disappeared when the surface was removed by sputtering. The prompt pH drop when these aerosols were suspended in water indicated that this surface layer was predominantly H_2SO_4. The respiratory response of guinea pigs exposed to these aerosols was consistent with the known response to H_2SO_4 (*3*).

As yet, we have no data on animal exposures to coal combustion products, but we have extensive characterization data on the composition of the ultrafine aerosol fraction. These data indicate that there is good reason to anticipate that the response produced by the prototype acid-coated aerosols generated with our ZnO furnace will have considerable relevance to the coal combustion aerosols. Zinc (Zn) is one of the elements concentrated in the submicrometer size fraction and on the surface of atmospheric fly ash (*4,5*), as well as in the ultrafine fraction of our laboratory coal combustors (*2*). In 14 coals studied, the Zn in the ultrafine fraction was approximately 20% of the Zn present in the coal (*6*). In coals such as Illinois No. 6, with a high Zn content, the Zn in the ultrafine fraction can account for 1 to 2% by weight with an even higher concentration in the surface coating (*2*). S (VI) is also concentrated in the ultrafine fraction of coal combustion aerosols with higher concentrations in the surface layer. Again, in the case of Illinois No. 6, as much as 9% of the S present in the coal appears as H_2SO_4, both free and on the particle surface. These aerosols also produce a pH drop when suspended in water (*2*).

The main points of discussion in this paper will be additional quantitative data on speciation of the surface S, on the pulmonary response to single and repeated exposures to these atmospheres, and on comparison of the response to that produced by H_2SO_4 aerosols of comparable size.

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Speciation of Sulfur

We have used ion chromatography to determine quantitatively the total S on the ZnO aerosol and its distribution between S (VI) and S (IV) (*7*). Figure 1 shows the amount of S carried by the aerosol as a function of ZnO concentration present in the chamber with 1 ppm SO_2. Only small amounts (0.6–6%) of the 1280 $\mu g/m^3$ S available from 1 ppm SO_2 are associated with the aerosol. It is thus possible to reduce the SO_2 concentration at a given level of ZnO without altering the pulmonary response (*6*). Figure 2 shows the quantitative speciation of the S between S (VI) and S (IV). In agreement with earlier qualitative data (*2*), the S (VI) predominates, although some unoxidized S (IV) was also present. At the ZnO concentrations of 1, 2.5, and 5 mg/m^3 used in the toxicological experiments, 72 to 99% of the total particulate S is present as S (VI).

Neither ZnO alone at any of the concentration-time patterns of the toxicological data discussed nor SO_2 alone at 1 ppm altered any of the functional, biochemical, or morphological parameters measured as compared with control guinea pigs exposed for similar periods to furnace gases. The observed responses are clearly due to the H_2SO_4 associated with the aerosol. The availability of our data on quantitative analysis of the amount of S (VI) eliminates the need to use ZnO concentrations as a surrogate for H_2SO_4 as we did initially (*8,9*) in preparing dose-response curves. Figure 3 shows the effect on one of our criteria, pulmonary diffusing capacity (DL_{CO}), produced by a single 3-hr exposure plotted against the concentration of S (VI). At concentrations of S (VI) of 10 $\mu g/m^3$ and above, such a single exposure produced a statistically significant reduction in (DL_{CO}). This is equivalent to an H_2SO_4 concentration of 30 $\mu g/m^3$.

Pulmonary Response

As we have reported previously (*2*), a single 3-hr exposure to 60 $\mu g/m^3$ H_2SO_4 produces decrements in total lung capacity (TLC), vital capacity (VC), functional residual capacity (FRC), and DL_{CO} that persist for 48 to 72 hr after the end of exposure. We have seen a similar pattern of functional change following a 1-hr exposure to 1 ppm ozone (O_3) (*10*). Morphological changes include interstitial edema and centriacinar damage (*9*) that also resemble the morphological lesions produced by O_3.

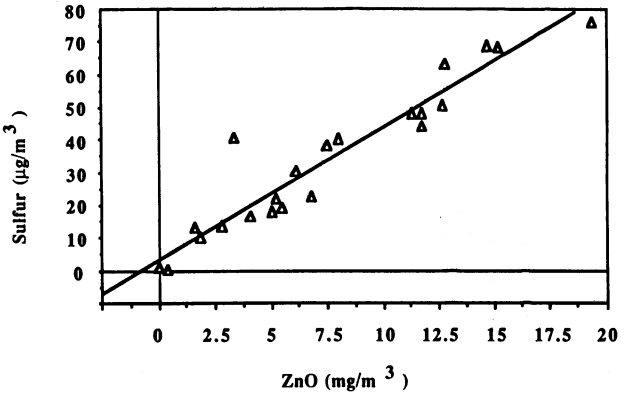

FIGURE 1. Total S carried by the aerosol present with 1 ppm SO_2 in the exposure chamber as a function of ZnO concentration.

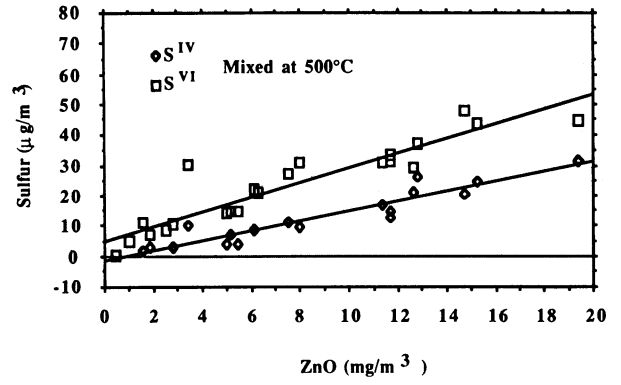

FIGURE 2. Amount of S (VI) and S (IV) carried by the aerosol present with 1 ppm SO_2 in the exposure chamber as a function of ZnO concentration.

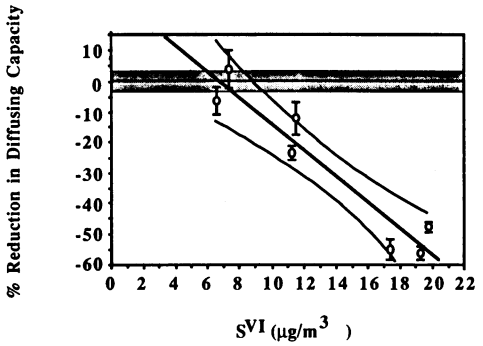

FIGURE 3. Dose-response curve with 95% confidence limits for DL_{CO} measured immediately after a single 3-hr exposure. Values are mean ± SE for eight guinea pigs. Shaded area is 95% confidence limits for control animals exposed to furnace gases for a similar time period.

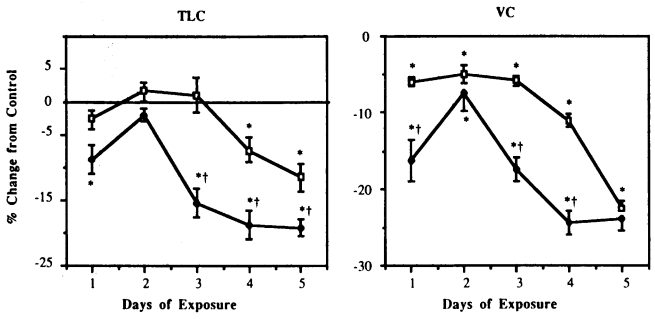

FIGURE 4. Effect of repeated daily 3-hr exposures on total lung capacity (TLC) and vital capacity (VC) measured immediately after exposure. Values are mean ± SE for eight guinea pigs (□) 20 μg H_2SO_4/m^3; (◆) 30 μg H_2SO_4/m^3; (*) different from control, $p < 0.05$; (†) different from lower dose, $p < 0.05$.

Repeated daily 3-hr exposures for 5 consecutive days have been done with concentrations of 20 and 30 $\mu g/m^3$ H_2SO_4. The effects are cumulative and dose related. Figure 4 shows the effects on TLC and VC, and Figure 5 shows the effects on DL_{CO} and the lung weight-body weight ratio. These measurements were made immediately following exposure. The 30 $\mu g/m^3$ H_2SO_4 produced, with the exception of day 2, steady decrements in TLC and VC, which appeared to plateau on days 4 and 5. Reductions in DL_{CO} and increases in lung weight-body weight ratios occurred gradually on the first 3 days and then changed further and plateaued on days 4 and 5. The correlation of DL_{CO} decreases and lung weight-body weight ratios increases suggests that edema may have been one factor contributing to the DL_{CO} reduction. At 20 $\mu g/m^3$, TLC was not decreased until day 4 of exposure. VC, on the other hand, was decreased slightly from day 1 and then abruptly decreased further on days 4 and 5. DL_{CO} was not altered until day 4, when it dropped abruptly. Lung weight-body weight ratios showed slight increases on exposure days 3 and 4.

Figure 6 shows the effect of repeated daily 3-hr exposure to 20 $\mu g/m^3$ H_2SO_4 on the protein in pulmonary lavage fluid. This was increased substantially on days 1 and 3 of exposure and slightly on days 4 and 5. These data are in agreement with data on another species, the rat, exposed to another deep lung irritant, O_3 (*11*), in indicating that protein in lavage fluid is one of the most sensitive indicators of such pulmonary damage.

Figure 7 shows the effect of this same exposure on the number of neutrophils in pulmonary lavage fluid. The increase in neutrophils accounted for the increase in overall cell count in lavage fluid; the number of macrophages and eosinophils was not increased. The number of neutrophils peaked on day 3. It was on days 3 and 4 of similar exposures that lung weight-body weight ratios were increased and on days 4 and 5 that DL_{CO} decreased. Morphological examination of the lungs from these exposures is not yet completed.

We have started exposures to 20 $\mu g/m^3$ H_2SO_4 extending beyond 5 days. We exposed animals 3 hr/day Monday through Friday then rested them over the weekend. In the group examined on Monday without further exposure, the DL_{CO} had returned to control levels. The Monday exposure was without effect. The second exposure on Tuesday, however, reduced DL_{CO} to 15% below control, unlike the initial week, in which no change was seen until day 4, when DL_{CO} was 14% below control. These data suggest that the lung had been rendered more sensitive.

Other data indicate that following a single 1-hr exposure to 20 $\mu g/m^3$ H_2SO_4, the lung was more sensitive to acetylcholine challenge (*7*). These data are shown in Figure 8. Neither SO_2 alone nor ZnO at a higher level increased bronchial reactivity. These measurements were made 2 hr after the end of exposure. The time course obviously needs to be explored. Again, the response is similar to that observed in guinea pigs following O_3 exposure (*12*).

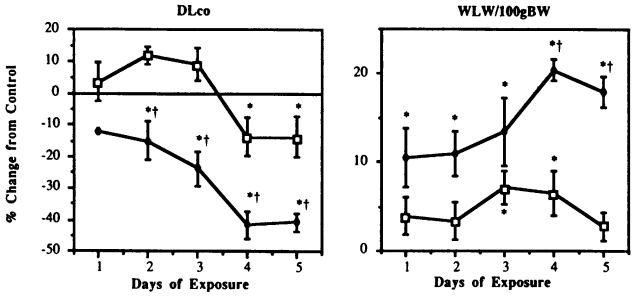

FIGURE 5. Effect of repeated daily 3-hr exposures on diffusing capacity (DL_{CO}) and wet lung weight to body weight ratios (WLW/100g BW) measured immediately after exposure. Values are mean ± SE for eight guinea pigs. (□) 20 μg H_2SO_4/m^3; (◆) 30 μg H_2SO_4/m^3; (*) different from control, $p < 0.05$; (†) different from lower dose, $p < 0.05$.

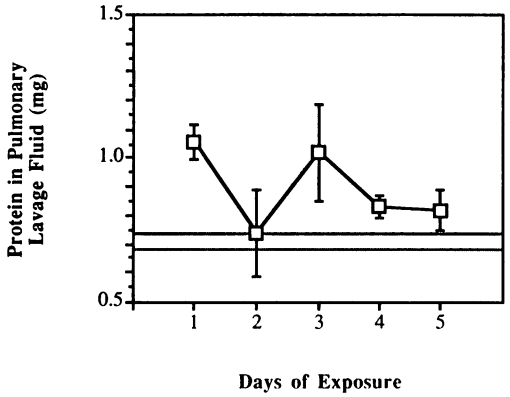

FIGURE 6. Effect of repeated daily 3-hr exposures to 20 μg H_2SO_4/m^3 on the amount of protein in pulmonary lavage fluid measured immediately after exposure. Values are mean ± SE from four guinea pigs. Parallel lines are 95% confidence limits for control animals exposed to furnace gases.

Comparison with Sulfuric Acid Mist

Figure 8 demonstrates one additional point of interest, namely, the fact that the same degree of bronchial

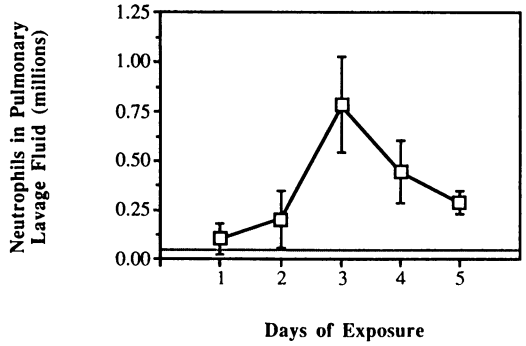

FIGURE 7. Effect of repeated daily 3-hr exposures to 20 μg H_2SO_4/m^3 on number of neutrophils in pulmonary lavage fluid measured immediately after exposure. Values are mean ± SE from four guinea pigs. Horizontal line indicates level of control animals exposed to furnace gas.

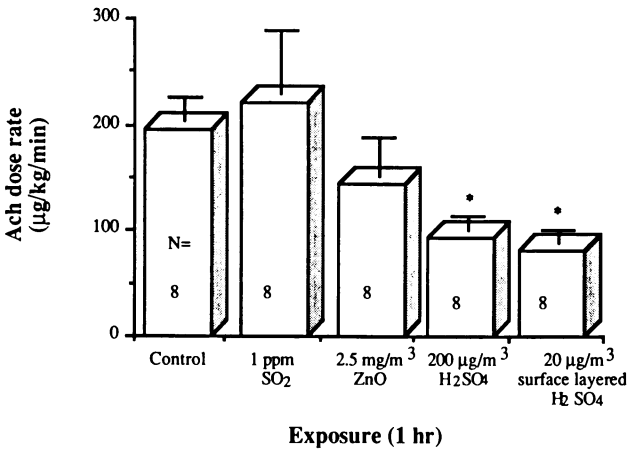

FIGURE 8. Amount of acetylcholine (IV influsion) required to double airway resistance from baseline levels 2 hr after a 1-hr exposure. Values are mean ± SE. (*) $p < 0.05$.

hypersensitivity is produced by 200 $\mu m/m^3$ H_2SO_4 aerosol of the same size. This point is examined further in Table 1, which compares the response to surface-layered H_2SO_4 in conventionally generated acid mist using decrease in DL_{CO} as the criterion of response. Once again, the smaller concentrations present as a surface layer give a proportionally greater pulmonary response.

Table 1. Comparison of sulfuric acid free and as surface layer.

H_2SO_4 test atmosphere	Concentration, $\mu g/m^3$	Duration of exposure, hr	Changes in diffusing capacity, %
Mist	140	1	−11
Surface layered	50	1.5	−16
Mist	310	3	−27
Surface layered	30	3	−25
Surface layered	50	3	−47

Summary

In summary, we have devised a way of making realistic H_2SO_4-coated ultrafine aerosols that simulate primary emissions from coal combustors and smelters. We find that concentrations as low as 20 $\mu g/m^3$ H_2SO_4 delivered in this manner produce cumulative pulmonary effects in guinea pigs. In many ways the effects we see resemble those we and others have seen with O_3. As is always the case in acid aerosol research, we have raised questions as well as answered them. Some of these questions we hope to address.

This work was supported by Program Project Grant ES 02429 from the National Institute of Environmental Health Sciences. Data presented are a synthesis of the work of our entire interdisciplinary group, which includes Adel F. Sarofim, John F. McCarthy, John F. Elliott, John Guty, Hua Fuan Lam, Michael W. Conner, Adrianne E. Rogers, and Patricia D. Miller.

REFERENCES

1. McCarthy, J. F., Yurek, G. J., Elliott, J. F., and Amdur, M. O. Generation and characterization of submicron aerosols of zinc oxide. Am. Ind. Hyg. Assoc. J. 43: 880–886 (1982).
2. Amdur, M. O., Sarofim, A. F., Neville, M., Quann, R. J., McCarthy, J. F., Elliott, J. F., Lam, H. F., Rogers, A. E., and Conner, M. W. Coal combustion aerosols and SO_2: An interdisciplinary analysis. Environ. Sci. Technol. 20: 138–145 (1986).
3. Amdur, M. O., McCarthy, J. F., and Gill, M. W. Effect of mixing conditions on irritant potency of zinc oxide and sulfur dioxide. Am. Ind. Hyg. Assoc. J. 44: 7–13 (1983).
4. Davidson, R. L., Natusch, D. F. S., Wallace, J. R., and Evans, C. A. Trace elements in fly ash: Dependence of concentration on particle size. Environ. Sci. Technol. 8: 1107–1113 (1974).
5. Linton, R. W., Loh, A., Natusch, D. F. S., Enans, C. A., and Williams, P. Surface predominance of trace elements in airborne particles. Science 191: 852–854 (1976).
6. Amdur, M. O. When one plus zero is more than one. The 1984 Henry F. Smyth, Jr. Lecture. Am. Ind. Hyg. Assoc. J. 46: 467–475 (1985).
7. Amdur, M. O., Chen, L. C., Guty, J., Lam, H. F., and Miller, P. D. Speciation and pulmonary effects of acid SO_x formed on surface of ultrafine zinc oxide aerosols. Atmos. Environ. 22: 557–560 (1988).
8. Lam, H. F., Peisch, R., and Amdur, M. O. Changes in lung volumes and diffusing capacity in guinea pigs exposed to a combination of sulfur dioxide and submicron zinc oxide mixed in a humidified furnace. Toxicol. Appl. Pharmacol. 66: 427–433 (1982).
9. Conner, M. W., Rogers, A. E., and Amdur, M. O. Response of guinea pig respiratory tract to inhalation of submicron zinc oxide particles generated in the presence of sulfur dioxide and water vapor. Toxicol. Appl. Pharmacol. 66: 434–442 (1982).
10. Miller, P. D., Ainsworth, D., Lam, H. F., and Amdur, M. O. Effect of ozone exposure on lung functions and plasma prostaglandin and thromboxane concentrations in guinea pigs. Toxicol. Appl. Pharmacol. 88: 132–140 (1987).
11. Last, J. A., Hyde, D. M., Guth, D. J., and Warren, D. L. Synergistic interaction of ozone and respirable aersols on rat lungs: Importance of aerosol acidity. Toxicology 39: 247–257 (1986).
12. Gordon, T., and Amdur, M. O. Effect of ozone on respiratory response of guinea pigs to histamine. J. Toxicol. Environ. Health 6: 185–195 (1980).