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# Epidemiological–Environmental Study of Lead Acid Battery Workers

## II. Acute Effects of Sulfuric Acid on the Respiratory System

JOHN GAMBLE, WILLIAM JONES, AND JOHN HANCOCK

*National Institute for Occupational Safety and Health, Division of Respiratory Disease Studies, 944 Chestnut Ridge Road, Morgantown, West Virginia 26505*

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Two hundred and twenty-five (225) workers in five lead acid battery plants were administered a questionnaire containing work-related symptoms, underwent spirometry, and had personal samples for $H_2SO_4$ taken over the shift. Most personal samples were less than 1 mg/m$^3$ $H_2SO_4$. Mass median aerodynamic diameter of $H_2SO_4$ from area samples in the formation areas was 2.6–10 μm. Workers with a higher exposure to acid did not have an increased rate of acute work-related symptoms. Changes in pulmonary function over the shift were not related to levels of airborne lead or airborne acid, sex, age, or smoking status. In acclimated workers, there is no evidence of acute symptoms or reductions in pulmonary function over the shift at concentrations less than 1 mg/m$^3$.

## INTRODUCTION

Sulfuric acid ($H_2SO_4$) is the most important sulfur compound, and the most commonly used industrial chemical, with 33,578,000 short tons sold or used in the United States in 1976 (Meyer, 1977; BOM, 1976). The bulk of sulfuric acid is used in making fertilizers. Other important uses include petroleum alkylation, iron and steel pickling, uranium leaching and processing, and manufacture of alcohols, $TiO_2$, hydrofluoric acid, pulp and paper, alum, rayon, explosives, and storage batteries.

Sulfuric acid mist is also a widespread air pollutant, deriving directly from manufacturing processes and atmospheric oxidation of $SO_2$. Atmospheric particulate size is small—generally 0.1–1.0 μm. Sulfuric acid is a very hygroscopic and strong acid and its toxic effects are related to these characteristics.

This paper reports on a cross-sectional study of workers in five plants manufacturing lead acid storage batteries. The purposes of this study are to measure environmental contaminants and to determine the acute and long-term effects of $H_2SO_4$ on the respiratory system and teeth. The first paper in this series summarizes the industrial hygiene portion of this study (Jones and Gamble, 1984). This, the second paper in the series, will summarize the findings in relation to short-term exposures over the work shift. The specific questions addressed in this paper are:

(1) What is the effect of short-term exposures to acid mist on symptoms and pulmonary function?

(2) What are the dose-response relationships?

A third report will address the question of the effects of chronic exposure to acid mist (Gamble *et al.*, 1984).



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*Literature Review*

*$H_2SO_4$—Animal Studies.* A number of animal studies have examined the effect on mortality, clearance, morphology, and pulmonary function of short-term exposure to sulfuric acid (Appendix 1).

Young guinea pigs (1–2 months) are more susceptible than older (18 months) guinea pigs and larger particles are more lethal than smaller particles (Amdur *et al.*, 1952; Wolff *et al.*, 1979; Pattle and Cullumbine, 1956). Mortality in order of increasing species sensitivity is rabbit, rat, mice, and guinea pig (Treon *et al.*, 1950). The cause of death in guinea pigs is bronchial spasm (Pattle and Cullumbine, 1956).

Sulfuric acid exposure (1.1 mg/m$^3$ for 3 hr) reduces frequency of ciliary beat and results in cytological alteration of hamster tracheal epithelium *in vitro* (Schiff *et al.*, 1979). The effect on intact animals is reduced mucus velocity and clearance. Nasal clearance is affected prior to lung clearance (Fairchild *et al.*, 1975a). At submicrometer particle sizes, the larger particles produce greater effects (Wolff *et al.*, 1981). The lowest effect dose was 1 mg/m$^3$ for 1 hr in dogs. Lower concentrations result in a transient increase in clearance prior to the decrease (Wolff *et al.*, 1981). Exposure of rats to 3–4 mg/m$^3$ $H_2SO_4$ (39% relative humidity, R.H.) results in a reduction in long-term (2 weeks) but not short-term clearance. The same exposure at 85% R.H. produced no change in either long-term or short-term clearance, reflecting a reduced effect (or changed deposition sites?) of larger particles (Phalen *et al.*, 1980).

Exposure must be considerably higher and longer to produce morphological changes. The lower respiratory tract shows morphological changes before the upper respiratory tract when exposure is to submicrometer particles (Schwartz *et al.*, 1977).

More sensitive measures of the effect of $H_2SO_4$ in animals are changes in airway diameters, occurring in guinea pigs after 1 hr exposure to concentrations as low as 0.1 mg/m$^3$ (Amdur *et al.*, 1978). Micrometer-size particles (or less) are decidely more irritating than particles greater than a micrometer in size. For example, exposure to particle sizes of 0.3, 0.8, 1, 2.5, and 7 μm required concentrations of 0.1, 1.9, 0.7, 6, and 31 mg/m$^3$, respectively, to produce a 50% increase in airway resistance ($R_{AW}$) with only a slight increase at 7 μm (Amdur, 1958; Amdur *et al.*, 1978). The changes in $R_{AW}$ are not readily reversible, suggesting a direct irritant effect (Amdur *et al.*, 1978; Pham-Huu-Chanh *et al.*, 1966). Changes in deposition also occur at exposure concentrations where no ventilatory effects are noted: in guinea pigs 3.02 mg/m$^3$ (1.8 μm) results in increased deposition in the nasopharynx, and at 0.03 mg/m$^3$ (0.25 μm) there is increased deposition in the trachea (Fairchild *et al.*, 1975b).

*$H_2SO_4$—Human exposure.* Despite the widespread recognition that $H_2SO_4$ is an irritant, the available data on dose–response relationships in humans are sketchy (Appendix 2). Slight irritation, increased respiratory rate, and changes in mucociliary particle clearance are the only responses noted in people at short-term exposures of less than 1 mg/m$^3$ (Table 1, summarized from Appendix 2).

The changes in respiratory rate noted in earlier studies of human subjects

TABLE 1
THRESHOLD LEVEL OF EFFECTS OF SULFURIC ACID ON HUMANS (FROM APPENDIX 2)

| Reference | $H_2SO_4$ (mg/m$^3$) | Symptom |
|---|---|---|
| Amdur *et al.* (1952) | 0.35 | Increased respiratory rate. |
| Amdur *et al.* (1952); Dorsch (1913) | 0.50 | Increased respiratory rate; scarcely noticeable irritation. |
| Bushtueva (1957a, b) | 0.72 | Throat tickling and scratching. |
| Patty (1963); Hackney *et al.* (1978); Avol *et al.* (1979) | 0.75–1.00 | Increased mucociliary clearance. |
| Bushtueva (1957a, b) | 1.1 | Irritation at base of esophagus; eye irritation (40%). |
| Dorsch (1913) | 0.5–2.0 | Slight irritation. |
| Bushtueva (1957a, b) | 2.4 | Acute irritation of mucus membranes; pronounced reflex cough. |
| Patty (1963) | 3 | Choking sensation in uninitiated; no effect in persons accustomed to exposure. |
| Dorsch (1913) | 3–4 | Distinct irritation. |
| Dorsch (1913) | 6–8 | Severe irritation. |

(Amdur *et al.*, 1952; Dorsch, 1913) have not been confirmed in later studies where no alterations in respiratory patterns are observed at concentrations below 1 mg/m$^3$ (Bushtueva, 1957b) and no convincing changes below 2 mg/m$^3$ (NIOSH, 1974). Subjective sensations at concentrations below 1 mg/m$^3$ are variable and slight (Amdur *et al.*, 1952; Bushtueva, 1957a; Dorsch, 1913; Avol *et al.*, 1979).

Controlled short-term exposures to 1 mg/m$^3$ $H_2SO_4$ or less generally result in no changes in pulmonary mechanics (Sackner *et al.*, 1978; Avol *et al.*, 1979; Newhouse *et al.*, 1978; Chaney *et al.*, 1980; Leikauf, 1981; Kerr *et al.*, 1981; Kulle *et al.*, 1982). Morrow *et al.* (1980) report small but significant changes in specific conductance at 1 mg/m$^3$. Sim and Pattle (1957) report some bronchoconstriction at levels of 3 mg/m$^3$. Volunteers exposed to 0.1 mg/m$^3$ $H_2SO_4$ show no increased sensitivity to ozone (Kleinman *et al.*, 1981) but possible decrease in bronchial reactivity (Kulle *et al.*, 1982).

The effect on clearance is variable. Bronchial clearance is increased at concentrations of less than 0.3 mg/m$^3$, and reduced at exposures of 0.3–1 mg/m$^3$ (Leikauf *et al.*, 1981). Tracheal mucociliary transport is unaffected at these concentrations with nose breathing and submicrometer (0.5 μm) particles. Tracheal mucociliary clearance is accelerated at 1 mg/m$^3$ when the particles are larger (3 μm) and breathing occurs only through the mouth (Newhouse *et al.*, 1978).

Battery workers exposed to concentrations well over 1 mg/m$^3$ $H_2SO_4$ (and a particle size probably >1 μm) had significantly greater reductions in $FEV_1$ and FVC than nonexposed controls (Williams, 1970). Another group of battery workers had significant reductions only in $FEV_1$ (El-Sadik *et al.*, 1972).

Most of the controlled short-term exposures of animals and humans have been to submicrometer $H_2SO_4$ particulate. At concentrations around 1 mg/m$^3$, which are the exposure levels measured in this study (Jones and Gamble, 1984), the

effects noted in animals are reduced clearance and increased deposition. In humans, the primary effect at these concentrations is irritation. There are few data on the effects of larger $H_2SO_4$ particles, and these are at concentrations considerably in excess of 1 mg/m$^3$.

*Stibine and Arsine*

Stibine or antimony hydride ($SbH_3$), the volatile hydride of antimony, is a colorless gas that forms whenever an acid reacts with a metal containing antimony, as in overcharged storage batteries. The point of entry of stibine is the respiratory system, but reported toxic effects are mainly on the blood and CNS. Only acute poisoning has been reported, causing headache, nausea, weakness, slow breathing, weak and irregular pulse, and hemoglobinuria (Clayton and Clayton, 1981). At high concentrations, stibine is a lung irritant. In animals, death with pulmonary congestion and edema occurs at 1-hr exposures of 40–45 ppm (cats and dogs) and 65 ppm (guinea pigs), and 20-min exposures of 100 ppm for mice. Hemoglobinuria was observed in the guinea pigs but not the other animal species (ACGIH, 1971).

The TLV of 0.1 ppm (0.5 mg/m$^3$) is based on the resemblance in physiological action of stibine and arsine (ACGIH, 1971). Possible acute effects of stibine cannot be separated from those of $H_2SO_4$.

Arsine, or arsenic hydride ($AsH_3$), is also a colorless gas that may be detected as the odor of garlic at 0.5 ppm. Arsine is formed in the same way as stibine, has a similar physiological action (hemolysis), but is more toxic than stibine. The acute signs of overexposure to arsine are malaise, abdominal cramps, nausea and vomiting, red staining of the conjunctiva, weakness, dizziness, and dyspnea. Delayed symptoms may include jaundice, red or green urine (and sometimes anuria), skin pigmentation, pyrexia, anemia, and lung edema. Chronic effects develop more slowly, and include peripheral neuritis (Clayton and Clayton, 1981) and anemia (Fowler and Weissberg, 1974).

In lead acid battery workers, Landrigan *et al.* (1982) observed a correlation of 0.84 between environmental arsine (0.06–20.6 μg/m$^3$) and excreted arsenic (7.9–68 μg/liter).

## METHODS

All workers thought to be exposed to acid were included in the study along with an approximately equal number of workers in areas of the plant considered to be low in acid exposure. Exposed and matched controls were examined on the same day. For each worker, a minimum of five forced expirations were recorded on magnetic tape using an Ohio 800 rolling seal spirometer[1] before beginning work and then again at the end of the shift (6–7 hr). Each forced expiratory maneuver was observed on an oscilloscope. To be accepted for analysis there were at least three expirations with maximum effort; two of which had FVCs within 5% of each other. Maximum forced expiratory volume in 1 sec ($FEV_1$), forced vital capacity (FVC), peak flow, and flows at 50 and 75% of FVC ($FEF_{50}$, $FEF_{75}$) were obtained from the maximum envelope curve. After-shift flow rates

[1] Mention of company names does not constitute endorsement by the National Institute for Occupational Safety and Health (NIOSH).

were obtained by lining up at total lung capacity the after-shift maximum envelope with preshift maximum envelope, and measuring flows at preshift percentage of FVC. Changes in pulmonary function (ΔPFT) were recorded as after-shift pulmonary function minus before-shift pulmonary function, so negative values indicate a reduction in function over the shift. The association of exposure and changes in pulmonary function was analyzed by multiple regression techniques.

Each worker in the study was administered a respiratory questionnaire which included questions covering acute symptoms occurring at work. The symptoms were divided into three categories relating to irritation, the chest, and other symptoms. Only workers who felt their symptoms were work related were included in the analysis. In order to get a clear separation between exposure groups for the analysis of acute symptoms and ΔPFT, the total population was divided into high (greater than 0.3 $mg/m^3$), and low (less than 0.07 $mg/m^3$) acid exposure groups based on the personal environmental samples collected during the acute study. The sign test was used to compare the prevalence of acute, work-related symptoms between the high and low exposure groups.

Personal samples for $H_2SO_4$ and Pb were collected from workers on the day they performed pre- and postshift pulmonary function and are used to analyze the association of exposure and shift changes in pulmonary function.

Acid particle size distribution was estimated by collecting area samples using Andersen and Sierra impactors. Area samples for stibine, arsine, and arsenic were collected in high acid areas of the plants. Temperature and relative humidity measurements were also collected. The details of these results are presented in Part I (Jones and Gamble, 1984). The antimony and arsenic compounds are not believed to affect pulmonary function at the levels measured. Whether they contributed to the symptom of nausea is not known.

Two hundred and twenty-five (225) workers in two mideastern states were studied in a 6-week period. Ninety-eight percent of the study population were white males. About one-half were smokers and one-fourth nonsmokers in Plants B, C, and D. The proportion of nonsmokers was considerably lower in Plants A and E (Table 2).

The average acid concentration for all plants was 0.18 $mg/m^3$. Plant D had the highest average level (0.35 $mg/m^3$) and Plants A and C the lowest (0.08 $mg/m^3$). The order was somewhat reversed for air lead (0.04 $mg/m^3$ in Plant D and 0.14 $mg/m^3$ in Plant C) (see Table 2). Lead and acid levels were not correlated ($r = -.03$) (Fig. 1 and Table 3).

In all plants a large proportion of the $H_2SO_4$ particulates were of respirable size as the mass median aerodynamic diameter was on average about 5 μm. Particle size may increase during the forming cycle as in one plant MMD was about 3 μm when collected at the beginning of the forming cycle, and about 7 μm when collected toward the end. [See the Industrial Hygiene report on the battery plants for a more detailed report of the environmental results, Jones and Gamble (1984)].

## RESULTS

The prevalence of all acute symptoms was low, and there were no statistically significant differences in symptoms between the high and low exposure groups.

TABLE 2
CHARACTERISTICS OF BATTERY PLANT WORKER POPULATION (ACUTE STUDY)[a]

| | I. By plant | | | | | |
|---|---|---|---|---|---|---|
| | A | B | C | D | E | Total |
| Age | 49.6 (8.8) | 29.4 (10.5) | 35.2 (12.9) | 36.5 (11.7) | 38.6 (12.4) | 36.1 (12.6) |
| Height (cm) | 175.6 (5.2) | 176.5 (7.1) | 174.4 (5.9) | 172.3 (8.0) | 175.1 (6.1) | 174.6 (6.9) |
| Weight (kg) | 83.5 (13.3) | 83.3 (19.2) | 77.5 (10.2) | 79.8 (13.7) | 77.7 (16.7) | 80.0 (15.6) |
| Acid exposure ($mg/m^3$) | 0.08 (0.07) | 0.14 (0.14) | 0.08 (0.08) | 0.35 (0.35) | 0.16 (0.21) | 0.18 (0.24) |
| Air lead ($mg/m^3$) | 0.08 (0.10) | 0.07 (0.10) | 0.14 (0.19) | 0.04 (0.05) | 0.06 (0.11) | 0.07 (0.12) |
| Blood lead ($\mu g$ Pb/100 g blood) | 31.9 (12.0) | — | 50.5 (12.2) | 37.8 (12.7) | 39.6 (15.3) | 40.6 (14.5) |
| *n* | 18 | 55 | 37 | 57 | 58 | (*n* = 169) 225 |

| | II. By smoking status | | |
|---|---|---|---|
| | Nonsmoker | Ex-smoker | Smoker |
| Age | 34.1 (14.0) | 38.8 (13.9) | 35.8 (11.5) |
| Height (cm) | 175.8 (7.5) | 173.0 (7.2) | 174.9 (6.5) |
| Weight (kg) | 82.4 (20.6) | 82.2 (11.4) | 78.4 (14.9) |
| Acid exposure ($mg/m^3$) | 0.18 (0.21) | 0.20 (0.24) | 0.18 (0.25) |
| Air lead ($mg/m^3$) | 0.09 (0.14) | 0.07 (0.10) | 0.07 (0.11) |
| Blood level ($\mu g$ Pb/100 g blood) | 41.9 (12.9) | 37.8 (13.7) | 41.3 (15.2) |
| Pack years | — | 15.8 (14.7) | 20.7 (17.1) |
| Cigarettes/day | — | 19.0 (13.0) | 23.0 (10.9) |
| *n* (%) | 45 (20) | 51 (23) | 129 (57) |

[a] Mean (SD).

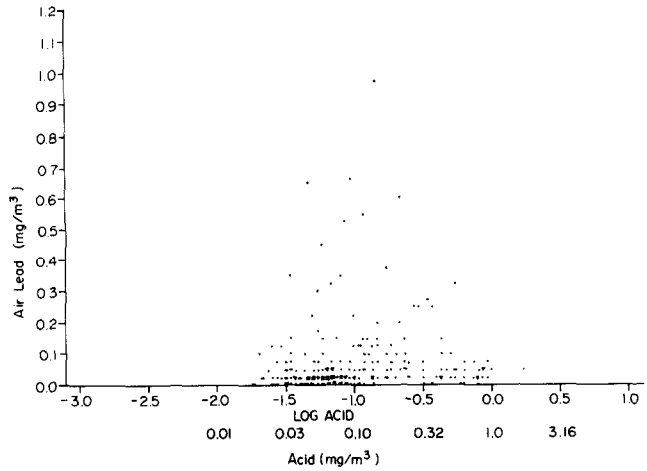


FIG. 1. Scatter plot of side-by-side environmental samples of air lead and air acid.

The highest prevalence and largest difference between high and low exposure groups was for the symptoms of cough and eye irritation (Table 4).

Workers who often had symptoms at work (except for itching eyes and skin) had more of a change in $FEV_1$ and $FEF_{50}$ than did those without symptoms. Workers who sometimes had these symptoms generally had intermediate reductions in $FEV_1$ and $FEF_{50}$ over the shift. None of the differences were statistically significant.

The association of changes in lung function over the shift as a function of acid and lead exposure were analyzed by multiple regression (Table 5, Fig. 2–6). The variables of sex and age were not significant predictors of changes in lung function. Smoking was a significant predictor only for $\Delta FEF_{75}$, where ex-smokers had the largest decrement over the shift and smokers the smallest. Plant was a significant variable for each pulmonary function parameter, and it was Plant E that always had the largest decrement. Personal environmental measurements of acid and air lead were not associated with changes in lung function. There was no trend for workers with high acid exposure to have greater reductions over the shift than those with low exposure (Table 6).

## DISCUSSION

This study of the acute effects of $H_2SO_4$ shows no consistent differences in work-related symptoms between high and low acid-exposed workers, and no acute reductions in pulmonary function related to exposure.

Reports in the literature of irritation at concentrations below 1 mg/m$^3$ are variable. Amdur (1952) reported no odor detection, taste, or irritation by unacclimated persons when breathing through a mask; other investigators have recorded slight irritation at levels below 1 mg/m$^3$ (Bushtueva, 1957a; Dorsch, 1913; Avol *et al.*, 1979). Members of the industrial hygiene survey team in this study experienced irritation in charging and forming areas where levels measured by per-

TABLE 3
CORRELATION MATRIX—BATTERY PLANT WORKERS—ACUTE STUDY ($n$ = 225)[a]

| | Age | Height | Weight | Acid exposure | Air lead | Blood lead | Pack years | Cigarettes per day | $\Delta FEV_1$ | $\Delta FVC$ | $\Delta$Peak flow | $\Delta FEF_{50}$ | $\Delta FEF_{75}$ |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Age | | −.27 | −.02 | −.08 | −.06 | −.03 | .59 | .10 | .01 | −.03 | −.04 | −.04 | .02 |
| Height | −.27 | | .40 | .01 | .12 | .03 | −.11 | −.06 | −.05 | −.04 | .02 | .02 | 0 |
| Weight | | .40 | | −.01 | −.02 | .12 | −.07 | −.08 | −.02 | −.01 | .09 | .05 | −.01 |
| Acid exposure | | | | | −.03 | −.26 | −.08 | −.01 | .03 | .02 | .11 | .05 | −.01 |
| Air lead | | | | | | .27 | −.04 | −.03 | .09 | .03 | .03 | .04 | .06 |
| Blood lead | | | | −.26 | .27 | | .09 | .04 | 0 | −.08 | .01 | −.03 | .04 |
| Pack years | .59 | | | | | | | .66 | −.04 | −.14 | −.12 | −.10 | .07 |
| Cigarettes/day | | | | | | | .66 | | −.10 | −.14 | −.14 | −.12 | 0 |
| $FEV_1$ | | | | | | | | | | .69 | .29 | .75 | .73 |
| FVC | | | | | | | | −.14 | .69 | | .24 | .52 | .49 |
| Peak flow | | | | .15 | | | | −.14 | .29 | .24 | | .18 | −.02 |
| $FEF_{50}$ | | | | | | | | −.13 | .75 | .52 | .18 | | .52 |
| $FEF_{75}$ | | | | | | | | | .73 | .49 | | .52 | |

[a] $P \leq .05$ for correlation coefficients in lower left half of matrix.

TABLE 4
PREVALENCE OF ACUTE SYMPTOMS RELATED TO WORK

| | Prevalence (%) by exposure | | |
|---|---|---|---|
| | High | Low | Score |
| Irritation | | | |
| Dry nose or throat | 2.4 | 5.3 | −1 |
| Cough | 4.9 | 1.7 | +1 |
| Nosebleed | 0 | 1.9 | −1 |
| Nose tickled or irritated | 0 | 1.3 | −1 |
| Sneeze | 4.0 | 4.3 | +1 |
| Mucus discharge or dripping from nose | 2.4 | 2.6 | −1 |
| Eyes itch, burn, or water | 4.9 | 0.9 | +1 |
| | | | 3/7 (N.S.) |
| Chest | | | |
| Difficult or labored breathing | 0 | 0.9 | −1 |
| Tight or constricted feeling in chest | 0 | 0.9 | −1 |
| Feeling you are going to suffocate | 0 | 0 | 0 |
| Spit up blood | 0 | 0 | 0 |
| Pain in heart or chest region | 2.4 | 0.9 | +1 |
| Wheeze | 2.4 | 1.7 | +1 |
| | | | 2/4 (N.S.) |
| Other | | | |
| Tingling or numbness in legs | 0 | 0 | 0 |
| Dizziness | 0 | 0.9 | −1 |
| Nausea | 4.9 | 0 | +1 |
| Red, inflamed skin | 0 | 0 | 0 |
| Dry, scaling skin | 2.4 | 0 | +1 |
| Itching skin | 0 | 0 | 0 |
| Rash | 0 | 0 | 0 |
| Stuffed up nose | 0 | 6.9 | −1 |
| Headaches | 0 | 3.5 | −1 |
| Heart rate | 0 | 0 | 0 |
| | | | 2/5 (N.S.) |
| Means | | | |
| Cigarettes/day | 17.9 | 19.0 | |
| *n* | 41 | 116 | |
| Acid exposure ($mg/m^3$) | >0.3 | <0.07 | |

sonal samples were generally less than 1 $mg/m^3$. It would appear that some irritation is experienced at $H_2SO_4$ concentrations below 1 $mg/m^3$, at least in unacclimated individuals.

Tolerance to $H_2SO_4$ may develop in workers habitually exposed (NIOSH, 1974; Cook, 1945), and several workers in the study population commented that the irritation experienced at the beginning of their employment was gone after several weeks employment. The idea of adaptation is supported in this study by the lack of a trend for irritation or other symptoms to be increased in high acid exposed groups (Table 4). It is interesting, however, that the greatest differences between these groups were cough (4.9 versus 1.7%) and itching, burning, or watering eyes

TABLE 5

CHANGES IN PULMONARY FUNCTION OVER THE SHIFT IN BATTERY WORKER POPULATION[a]

Model: $\Delta PFT^b = \alpha + \beta_1(\text{sex}) + \beta_2(\text{age}) + \beta_3(\text{smoking status}) + \beta_4(\text{plant}) + \beta_5(\text{air lead}) + \beta_6(\text{acid})$

| | $\Delta FEV_1$ (ml) | $\Delta FVC$ (ml) | $\Delta$Peak flow (ml/sec) | $\Delta FEF_{50}$ (ml/sec) | $\Delta FEF_{75}$ (ml/sec) |
|---|---|---|---|---|---|
| Mean (SD) | −41 (127) | −7 (179) | +56 (917) | −101 (560) | −92 (321) |
| Smoking (SE) | N.S. | N.S. | N.S. | N.S. | * |
| Nonsmoker | −25 (35) | −7 (50) | −260 (255) | −6 (156) | −103 (89) |
| Ex-smoker | −47 (35) | −39 (50) | −157 (256) | −183 (156) | −225 (90) |
| Smoker | −37 (31) | −49 (43) | −283 (221) | −136 (135) | −86 (77) |
| Plant (SE) | * | * | * | * | * |
| A | +12 (45) | +54 (64) | −229 (328) | +92 (200) | −84 (115) |
| B | −36 (35) | −31 (49) | −133 (250) | −102 (152) | −169 (87) |
| C | −0 (35) | −12 (49) | −263 (253) | +33 (155) | −46 (89) |
| D | +2 (32) | −3 (46) | +88 (234) | +11 (143) | −71 (82) |
| E | −162 (36) | −166 (51) | −631 (260) | −572 (159) | −322 (91) |
| Coefficient for acid exposure (SE) | −1 (39) | −6 (56) | +142 (285) | +80 (174) | −40 (100) |

[a] Sex, age, and air lead were N.S. ($P > 0.05$). The value by smoking category and plant are least square means with SE. $P > 0.05$ unless indicated otherwise.

[b] $\Delta PFT$ = (Pulmonary function at end of shift) − (pulmonary function at beginning of shift). A negative sign indicates a reduction in PFT over the shift.

* $P < 0.05$

(4.9 versus 0.9%). On the other hand, there was no difference in the prevalence of sneeze (4.9 versus 4.3%), and the prevalence of nose irritation was reversed (0 versus 1.3%). The absence of a trend toward increased symptoms in the high exposure group suggests minimal irritation in acclimated workers at $H_2SO_4$ con-

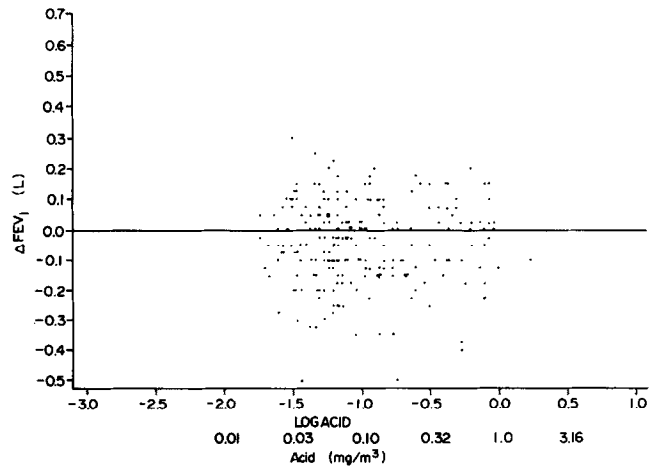


FIG. 2. Scatter plot of sulfuric acid exposure and changes in forced expiratory volume in 1 sec (FEV) over the shift.

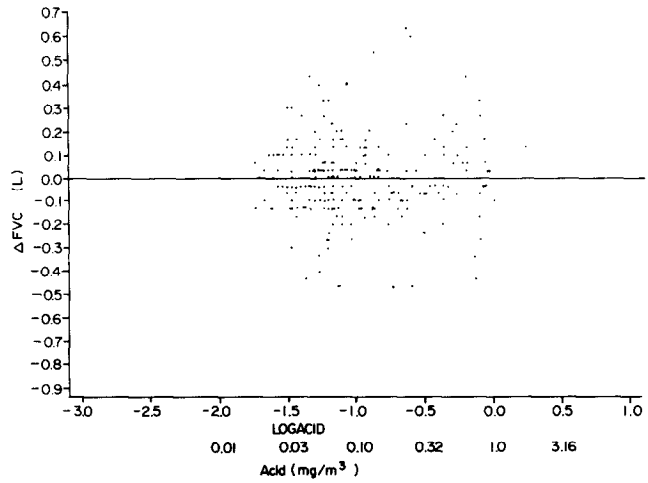


FIG. 3. Scatter plot of sulfuric acid exposure and changes in Forced Vital Capacity (FVC) over the shift.

centrations less than 1 mg/m$^3$. It appears unlikely that nausea and headaches are related to stibine and arsine exposure.

The evidence that $H_2SO_4$ at low concentrations is a lung irritant is not convincing. Guinea pigs show an increase in airway resistance after exposure for 1 hr to levels of $H_2SO_4$ as low as 0.2 mg/m$^3$ (Amdur *et al.*, 1978). Guinea pigs, however, are more sensitive to respiratory irritants than other animals. In anesthetized dogs there is no change in respiratory resistance, functional residual capacity, compliance, or conductance when breathing up to 18 mg/m$^3$ $H_2SO_4$ for 7.5 min, and 4 mg/m$^3$ for 4 hr. In the longer exposure time (4 hr), there was no change in blood pressure, cardiac output, heart rate, stroke volume, or arterial blood gases. Sheep exposed for 20 min to 14 mg/m$^3$ $H_2SO_4$ show no change in tracheal mucus velocity, respiratory frequency, tidal volume, and minute ventilation (Sackner *et al.*, 1978).

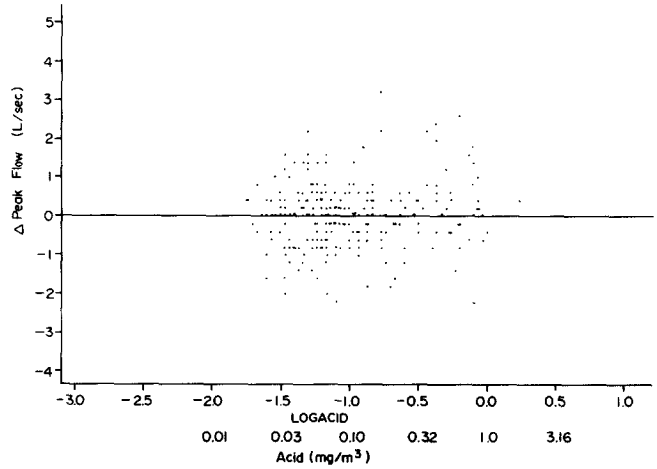


FIG. 4. Scatter plot of sulfuric acid exposure and changes in peak flow over the shift.

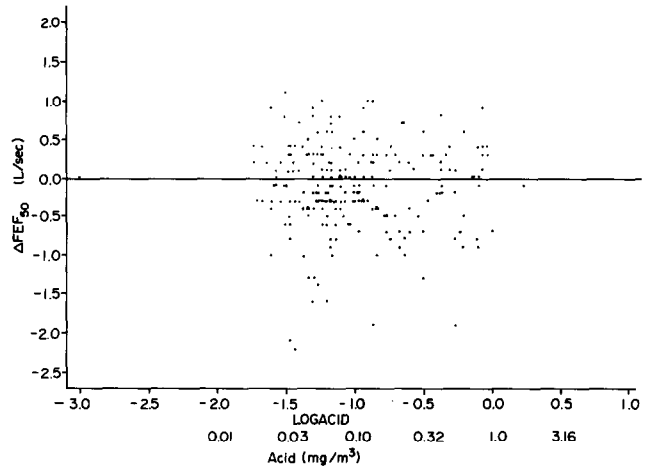


FIG. 5. Scatter plot of sulfuric acid exposure and changes in $FEF_{50}$ over the shift.

With one exception (Morrow *et al.*, 1980), no functional changes are observed in exercising humans exposed to concentrations as high as 1 mg/m$^3$ for up to 2.5 hr (Newhouse *et al.*, 1978). In most of the clinical studies, exposure times were short, considerably less than 1 mg/m$^3$, and particles were submicrometer in size. The results of controlled human exposure are consistent with our findings in this study where no change in FVC, $FEV_1$, peak flow, $FEF_{50}$, or $FEF_{75}$ was related to $H_2SO_4$ exposures. Besides being a survey, our study differs from clinical studies in that the workers were acclimated, exposure time was longer, and particle size was larger.

At levels greater than 1 mg/m$^3$, air flow obstruction has been observed among battery acid workers. Williams (1970) reports a reduction in $FEV_1$ and FVC of

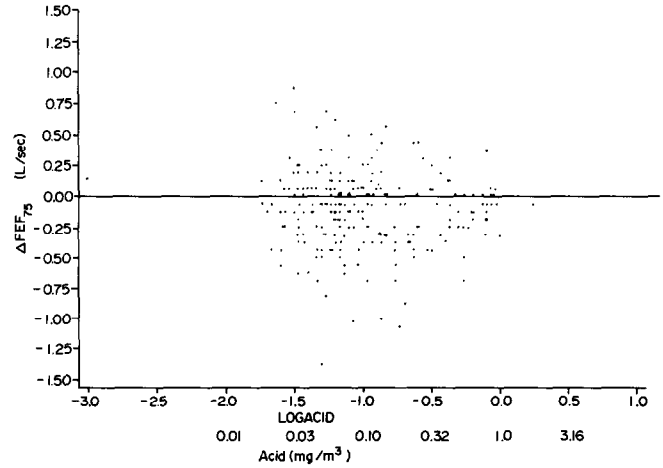


FIG. 6. Scatter plot of sulfuric acid exposure and changes in $FEF_{75}$ over the shift.

TABLE 6
MEAN CHANGES IN PULMONARY FUNCTION OF HIGH AND LOW EXPOSURE GROUPS

| | $n$ | $\Delta FEV_1$ (ml) | $\Delta FVC$ (ml) | $\Delta FEF_{50}$ (ml/sec) | $\Delta FEF_{75}$ (ml/sec) |
|---|---|---|---|---|---|
| Low exposure | 102 | −44 (141)[a] | +1 (158) | −123 (621) | −78 (339) |
| High exposure | 41 | −25 (138) | −1 (216) | −32 (675) | −93 (216) |

[a] (SD).

about −160 ml on both Monday and Friday among 31 men working in the Forming Department of an electric accumulator factory. Other investigators on separate occasions had found $H_2SO_4$ levels in this department to be on average 1.4 mg/m$^3$ (trace to 6.1 mg/m$^3$) (Anfield and Warner, 1968) and 3.0–16.6 mg/m$^3$ (Malcolm and Paul, 1961). Sixteen controls from the Plate Cutting Department showed no significant reductions except for $FEV_1$ on Friday (−120 ml). El-Sadik *et al.* (1972) observed a mean change of −82 ml in $FEV_1$ among 33 battery workers. Twenty controls had a +33 ml increase in $FEV_1$. Both exposed and controls had decreases in VC of −82 and −123 ml, respectively. $H_2SO_4$ air concentrations were measured at 26.1–35.0 mg/m$^3$ in one plant, and 12.6–13.5 mg/m$^3$ at another plant. While exposure to acid was not well documented in either study, exposure levels were apparently higher than in the present study.

The lack of a significant change in ventilatory function at exposures of less than 1 mg/m$^3$ may be due in part to the body's ability to neutralize the acidity of $H_2SO_4$. Larson *et al.* (1977) measured metabolic production of 29 to 520 μg/m$^3$ $NH_3$. Based on actual measurements in one subject, they calculate that the $NH_3$ has the potential of neutralizing 0.084–1.5 mg/m$^3$ $H_2SO_4$ when mouth breathing, and 0.013–0.046 mg/m$^3$ when nose breathing. However, larger particles are less rapidly neutralized than small particles (Larson *et al.*, 1982).

Acidity of the acid particles may also be reduced by hydration following inhalation. The hydration and subsequent increase in size increases deposition in the upper respiratory tract, although this effect becomes smaller as particle size increases (Ferron, 1977).

While breathing through the nose, site deposition and percentage retention of hygroscopic particles of 6 μm in diameter have been estimated as 67% in the nose, 8% in the upper respiratory tract, 20% in the middle respiratory tract, and 5% in the lower respiratory tract (Landahl, 1972). This estimate of upper respiratory tract deposition may be low, as Ferron (1977) calculates 100% deposition in the nasopharyngeal region of a soluble aerosol with diameter greater than about 3 μm. Thus in this study population of battery workers, most of the particle deposition is likely to be in the upper respiratory tract.

In summary, then, exposure to sulfuric acid mist at these plants (generally at levels less than 1 mg/m$^3$ and average MMAD ≅5μm) shows no significant association with symptoms or acute effect on ventilatory function ($FEV_1$, FVC, PF, $FEF_{50}$, $FEF_{75}$).

## APPENDIX 1

## Summary of Short-Term Effects of $H_2SO_4$—Animal Studies

| | | |
|---|---|---|
| Guinea pigs | Mortality | |
| | $LC_{50}$ = 18 mg/m³/8 hr (1 μm MMD)[a] | Amdur *et al.* (1952) |
| | $LC_{50}$ = 50 mg/m³/8 hr | " |
| | $LC_{50}$ = 30 mg/m³/8 hr (0.8 μm MMD) | Wolff *et al.* (1979) |
| | $LC_{50}$ = 109 mg/m³/8 hr (0.4 μm MMD) | " |
| | $LC_{50}$ = 28.8 mg/m³/8 hr (2.7 μm MMD) | Pattle and Cullumbine (1956) |
| | $LC_{50}$ = 60.9 mg/m³/8 hr (0.8 μm MMD) 20°C | " |
| | $LC_{50}$ = 49 mg/m³/8 hr (0.8 μm MMD) 0°C | " |
| | Clearance | |
| Donkeys | | |
| 194 mg/m³/1 hr (0.3–0.6 μm MMD) | Reduced bronchial clearance. | Schlesinger *et al.* (1978) |
| Rats | | |
| 3–4 mg/m³/4 hr (1 μm MMD) | Reduction in long-term clearance (humidity—no reduction at high humidity). | Phalen *et al.* (1980) |
| Mice | | |
| 15 mg/m³/4 hr and 90 m/day/4 days (3.2 μm CMD)[b] | Reduced clearance of nonviable bacteria from nose but not lung. | Fairchild *et al.* (1975a) |
| 1.5 mg/m³/4 hr and 90 m/day/4 days (0.6 μm CMD) | No change in clearance of viable and nonviable bacteria from nose or lung. | Fairchild *et al.* (1975a) |
| Dogs | | |
| 1 mg/m³/1 hr (0.9 μm MMD) | Reduced tracheal mucus velocity (slight increase and delayed reduction at 0.5 mg/m³). | Wolff *et al.* (1981) |
| 1 mg/m³/1 hr (0.3 μm MMD) | No change. | Wolff *et al.* (1981) |
| Sheep | | |
| 14 mg/m³/20 min (2 μm MMD) | No change in tracheal mucus velocity (no change at 1 mg/m³ (0.1 μm) and 8 mg/m³ (0.15 μm). | Sackner *et al.* (1978) |
| | Morphology | |
| Rhesus monkeys | | |
| 150 mg/m³/3 days (0.3–0.5 μm MMD) | No noticeable change (LM, SEM). | Schwartz *et al.* (1977) |
| Rats | | |
| 100 mg/m³/5 days (0.7 μm MMD) | No noticeable change. | Cavender *et al.* (1977) |
| 172 mg/m³/7 days (0.45 μm MMD) | No noticeable change. | Schwartz *et al.* (1977) |
| 45 mg/m³/11 days (0.5 μm MMD) | No noticeable change. | Schwartz *et al.* (1977) |
| Mice | | |
| 140 mg/m³/4 days (0.3 μm MMD) | Lesions in larynx and upper trachea. | Schwartz *et al.* (1977) |
| 170 mg/m³/10 days (0.6 μm MMD) | | Schwartz *et al.* (1977) |
| Guinea pigs | | |
| 20 mg/m³/14 days (1.66 μm, 1 μm, 0.5 μm MMD) | Alveolitis. | Cavender *et al.* (1977) |
| 71 mg/m³/4 d (0.5 μm MMD) | Lesions at bronchial (focal epithelial necrosis and ulceration) and alveolar level (edematous). | Schwartz *et al.* (1977) |
| 30 mg/m³/7 days (0.3 μm MMD) | Minimal changes (variability in density and length of cilia). | Schwartz *et al.* (1977) |
| 25 mg/m³/6 hr/2 days (1 μm MMD) | Alveolar hemorrhage and edema; lesions in distal airways; no lesions seen in bronchi, trachea, larynx. | Cockrell *et al.* (1978) |
| | Function | |
| Donkeys | | |
| 1.4 mg/m³/1 hr (0.3–0.6 μm MMD) | No change in regional deposition, pulmonary resistance and dynamic compliance. | Schlesinger *et al.* (1978) |

## APPENDIX 1—*Continued*

| | | |
|---|---|---|
| Guinea pigs | | |
| 2 mg/m³/5 days | Increased RR; disturbance of blood and lymph circulation. | Bushtueva (1957) |
| 1.9 mg/m³/1 hr (0.8 μm MMD) | 50% increase in RAW. | Amdur (1958) |
| 6 mg/m³/1 hr (2.5 μm MMD) | 50% increase in RAW. | Amdur (1958) |
| 31 mg/m³/1 hr (7 μm MMD) | Slight increase in RAW. | Amdur (1958) |
| 3 mg/m³/1 hr (1.8 μm CMD) | Increased deposition in nasopharynx; no effect on tidal volume, RR, minute volume. 0.3 mg/m³ (0.6 μm) had no effect. | Fairchild *et al.* (1975b) |
| 0.03 mg/m³/1 hr (0.25 μm CMD) | Increased deposition in trachea; no effect on ventilation. | Fairchild *et al.* (1975b) |
| 0.1 mg/m³/1 hr (0.3 μm MMD) 1 mg/m³/1 hr (1 μm MMD) | Increased RAW, decreased compliance (0.3 μm more irritating than 1 μm). | Amdur *et al.* (1978) |
| 19 mg/m³/1 hr (1 μm MMD) | Increased sensitivity to histamine in animals showing labored breathing during exposure (6/48). | Silbaugh *et al.* (1979) |
| Dogs | | |
| 8 mg/m³/7.5 min (0.15 μm MMD) | No change in respiratory resistance, functional residual capacity, lung compliance, specific lung compliance, or specific respiratory conductance. | Sackner *et al.* (1978) |
| 4 mg/m³/4 hr (0.15 μm MMD) | No change in total respiratory resistance, specific respiratory conductance, compliance, specific lung compliance, functional residual capacity, B.P., cardiac output, heart rate, stroke volume, blood gas. | Sackner *et al.* (1978) |
| Sheep | | |
| 14 mg/m³/20 min (2 μm MMD) | No change in RR, tidal volume, minute ventilation (no change at 1 mg/m³ (0.1 μm), and 8 mg/m³ (0.15 μm). | Sackner *et al.* (1978) |

[a] MMD = mass median aerodynamic diameter.
[b] CMD = count median diameter.

## APPENDIX 2

## Controlled Exposures of Humans to $H_2SO_4$

| Reference | Concentration (mg/m³) | Time | Particle size (μm) | *n* | Results |
|---|---|---|---|---|---|
| Amdur *et al.* (1952) | 0.35–5.0 | 5–15 min | 1 | 15 normals via mask | Increased respiratory rate, by about 35%; Decreased tidal volume at all concentrations. |
| | 0.4–1 | | | | 77% (50–87%) retention. |
| | 1.0 | | | | 2/15 detected odor; no odor detected at concentrations <1 mg/m³. |
| | 2.0 | | | | 2/15 detected odor; no odor detected at concentrations <1 mg/m³. |
| | 3.0 | | | | Odor detected by all; "felt like breathing dusty air." |
| | 5.0 | | | | Deep breath results in cough; very objectionable to some but not others. |

## APPENDIX 2—*Continued*

| Reference | Concentration ($mg/m^3$) | Time | Particle size ($\mu m$) | *n* | Results |
|---|---|---|---|---|---|
| Morando (1956) | 0.35–5.0 | 5–15 min | | | Increase in respiratory flow, with reduction in percentage retention. |
| Sim and Pattle (1957) | 4–39 | 10 min | 1 | 12 men, exposure via mask | Coughing, some bronchoconstriction (62% R.H.). |
| | 3–39 | 60 min | 1 | 12 men, exposure in chamber | Coughing, some bronchoconstriction (62% R.H.), RAW increased 36–100%. |
| | 11.5–38.0 | 30–60 min | 1.5 | 12 men, exposure in chamber | More irritating than dry mist at same concentration; RAW increased 43–150%; (91% R.H.) |
| | 0.73 (0.60–0.85) | 30–60 min | 1.5 | 10 | Throat tickling and scratching; no change in respiratory rate less than 1 $mg/m^3$. |
| Bushtueva (1957b) | 1.1–2.4 | | | | Considerable irritation at base of esophagus; 4/10 noticed eye irritation. |
| | 2.4–5.0 | | | | Acute irritation of mucus membranes and pronounced reflex cough; eye irritation. |
| | 3 | | | | Choking sensation in uninitiated; persons accustomed to exposure unable to notice concentration. |
| Sackner *et al.* (1978) | 0.01 | 10 min | 0.05 (MMD) | 5 normals<br>5 asthmatics | No change.<br>Decrease in resistance, $FEV_1$, and VC (none significant). |
| | 0.1 | 10 min | 0.1 (MMD) | 5 normals<br>5 asthmatics | No change.<br>No change. |
| | | 10 min | 0.2 (MMD) | 5 normals<br>5 asthmatics | No change.<br>Slight increase in resistance and $FEV_1$ (N.S.). |
| | | 10 min | 0.2 (MMD) | 6 normals<br>6 asthmatics | No significant changes in lung volumes, dynamic mechanisms of breathing, distribution of ventilation, total respiratory resistance, diffusing capacity, functional residual capacity. |
| Hackney *et al.* (1978)<br>Avol *et al.* (1979) | 0.075 (40% R.H.) | 2.5 hr w/light exercise | 0.3 (MMD) | 6 normals<br>6 asthmatics | No exposure-related changes in spirometry, lung volume, nitrogen washout, or RAW (possible increase of RAW in two asthmatics); no change in total symptoms during 24-hr period; increased mucociliary clearance; symptoms tended to increase in asthmatics during exposure. |
| Newhouse *et al.* (1978) | | 2.5 hours (20 min exercise at 75% maximum heart rate) | 0.3 (MMD) | 10, mouth breathing only | Increased mucociliary clearance; no change in VC, $FEV_1$; 1.4% decrease (N.S.) in MMRF. |
| Chaney *et al.* (1980) | 0.1 | 4 hr | 0.5 | 18 | No change in serum glutathione, lyzozyme, 2,3-DPG, SGOT, serum vitamin E, or RBC glutathione reductase; no effects on FRC, RAW, forced inspiratory volume, FVC, VC, or MVV. |

## APPENDIX 2—*Continued*

| Reference | Concentration ($mg/m^3$) | Time | Particle size ($\mu m$) | *n* | Results |
|---|---|---|---|---|---|
| Morrow *et al.* (1980) | 1 | 16 min | | Normal volunteers | Changes in specific conductance ($SG_{aw}$). |
| Leikauf *et al.* (1981) | 0<br>0.11<br>0.33<br>0.98 | 1 hr via nasal mask | 0.5 (MMD) | 10 healthy nonsmokers | No change in respiratory mechanics (airway resistance, $FEV_1$, FVC, expiratory flow at 25, 50, and 75% of FVC, MMEF, and distribution of ventilation) deposition or tracheal mucociliary transport rate for up to 4 hr after end of exposure.<br>Bronchial clearance was accelerated at 0.1 $mg/m^3$ and reduced at 0.3 and 1 $mg/m^3$ in 6/10 subjects. |

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