ENVIRONMENTAL RESEARCH 35, 30–52 (1984)

Epidemiological–Environmental Study of Lead Acid Battery Workers

III. Chronic Effects of Sulfuric Acid on the Respiratory System and Teeth

JOHN GAMBLE, WILLIAM JONES, JOHN HANCOCK, AND RICHARD L. MECKSTROTH*

*National Institute for Occupational Safety and Health, Division of Respiratory Disease Studies, 944 Chestnut Ridge Road, Morgantown, West Virginia 26505, and *Department of Community Dentistry, West Virginia University, Morgantown, West Virginia 26506*

Received January 11, 1983

The effects of long-term exposure to sulfuric acid mist on the teeth and respiratory system were studied in 248 workers in five plants manufacturing lead acid batteries. The prevalence of cough, phlegm, dyspnea, and wheezing as determined by questionnaire were not associated with estimates of cumulative acid exposure. There was only one case of irregular opacities seen on the chest radiographs. There was no statistically significant association of reduced FEV_1, peak flow, FEF_{50}, and FEF_{75} with acid exposure although the higher exposed group had lower mean values. FVC in the high exposure group showed a statistically significant reduction compared to the low exposure group, but there was no significant association when exposure was analyzed as a continuous variable. The ratio of observed to expected prevalence of teeth etching and erosion was about four times greater in the high acid-exposure group. The earliest case of etching occurred after 4 months exposure to an estimated average exposure of 0.23 mg/m^3 sulfuric acid.

INTRODUCTION

This is the third paper in this study of battery workers. The first paper summarized the environmental characteristics of the plants (Jones and Gamble, 1984). The second analyzed the association of short-term exposure over the shift with work-related symptoms and acute changes in pulmonary function (Gamble *et al.*, 1984). The questions addressed in this paper concern the chronic effects of exposure to sulfuric acid (H_2SO_4) mist. These include measurement of respiratory symptoms, pulmonary function, chest radiographs, and tooth erosion. The specific questions addressed in this paper are:

(1) What is the prevalence of respiratory symptoms, tooth erosion, radiographic abnormalities, and reduced pulmonary function among acid-exposed workers?

(2) Is there an association of these findings with exposure?

LITERATURE REVIEW

In 1974, NIOSH recommended a 1 mg/m^3 TWA standard for H_2SO_4 to "prevent the irritant effects of sulfuric acid in workers" (NIOSH, 1974). Seven years later, NIOSH reviewed more recent literature on sulfuric acid. This was done for two reasons. The use of catalytic converters in automobiles purportedly increases the amount of sulfuric acid formed in automobile emissions. The second concern was with the problem of acid rain (NIOSH, 1981).

0013-9351/84 $3.00

The importance of particle size was stressed in the literature update (NIOSH, 1981). For aerosols that penetrate to the lung, two mechanisms of action were suggested. Upper lung deposition was hypothesized to result in reflex bronchoconstriction, while smaller particles produced greater alterations in pulmonary function and eventually lesions. It is not clear that the acid exposure in this study is related to either of these effects as the mass median diameter (MMD) was about 3–10 μm and relative humidity (R.H.) was generally between 20 and 50% (Jones and Gamble, 1984). This particle size suggests deposition in the upper airways, yet there was no evidence of acute bronchoconstriction (Gamble *et al.*, 1984).

The available data on the health effects of chronic exposure to sulfuric acid are sparse, particularly to particles of the size observed in this study.

Animal Studies

A number of animal studies have examined the effects of chronic exposure to sulfuric acid (see Appendix). Guinea pigs are among the most sensitive, and rats and monkeys among the most resistant of animals (Treon *et al.*, 1950; Pattle and Cullumbine, 1956; Schwartz *et al.*, 1977). Effects of exposure on guinea pigs are observed at concentrations as low as 2 mg/m^3 for 5 days (Bushtueva, 1957), but other investigators observe minimal effects at 10 mg/m^3 for 6 months (Cavender *et al.*, 1978), or no morphological effects at concentrations less than 20 mg/m^3 for 5–28 days (Cavender *et al.*, 1977a). In donkeys, bronchial clearance is reduced at concentrations of 0.1 mg/m^3 (Schlesinger *et al.*, 1979). Slight changes in ventilation distribution are seen in monkeys at 0.5 mg/m^3, but are convincing at concentrations of 2.4 mg/m^3 (Alarie *et al.*, 1973). Monkeys exposed to as much as 1 mg/m^3 H_2SO_4 in the presence of SO_2 and fly ash show slight histopathological effects (Alarie *et al.*, 1975), but concentrations as high as 502 mg/m^3 (0.5 MMD) for 7 days cause no demonstrable morphological damage (Schwartz *et al.*, 1977). The effects in rats are slight at concentrations less than 10–20 mg/m^3 (Cavender *et al.*, 1978; Cavender *et al.*, 1977b), although no morphological effects at 172 mg/m^3 (0.5 MMD) for 7 days are observed (Schwartz *et al.*, 1977). In most studies particle sizes are less than 1 μm; Thomas *et al.*, (1950) comment that 0.9-μm-size particles are more active than smaller (0.6 μm) and larger (4 μm) particles. Wolff *et al.*, (1979) show that 0.8-μm H_2SO_4 particulates are more toxic to guinea pigs than 0.4-μm particles.

Sulfuric acid mist may act as a deep lung irritant in animals (Treon *et al.*, 1950; Cockrell *et al.*, 1978). Guinea pigs exposed for 2 days to 25 mg/m^3 of H_2SO_4 (MMD = 1 μm) show no lesions in the larynx, trachea, and bronchi, but there is hemorrhage, edema, and increased numbers of macrophages in the alveoli (Cockrell *et al.*, 1978). The effects of H_2SO_4 on the lung are dependent on particle size and relative humidity (Cavender *et al.*, 1977a). Pattle and Cullumbine (1956) state that particles of H_2SO_4 1.5 μm in diameter are about twice as irritant as 1-μm particles. Amdur (1958) found that 2.5 μm H_2SO_4 produces a greater response on physiological function at high concentraions (>2 mg/m^3) than 0.8-μm particles; the effect is reversed at concentrations <2 mg/m^3. Particles of 7 μm produce only a slight response at concentrations as high as 30 mg/m^3 because they are deposited in the upper respiratory tract.

Epidemiological Studies

The number of epidemiological studies examining the chronic effects exposure to H_2SO_4 are limited. Morando (1956) reports that 57 workers in a sulfuric acid production department show no evidence of eye or throat irritation, nor an increase in bronchial or lung disease. Exposure to H_2SO_4 was said to be relatively low. Gastrointestinal tract lesions (gastritis, gastroduodenitis, gastric ulcers, and digestive problems) are more common than respiratory problems.

Williams (1970) compares the sickness absence of workers in the forming department of a battery plant with workers in control departments. The forming department had high concentrations of acid mist, was wet, and had no heating. The control departments had no acid mist and adequate heating (the floors were wet in two of the three control departments). The acid-exposed workers had a slight excess of bronchitis. The younger acid-exposed men had slight excesses in influenza, other upper respiratory tract disease, and all respiratory disease. The excess was due to more spells of illness rather than an increase in the proportion of men attacked. A previous study (Anfield and Warner, 1968) in the forming department of this same plant reported a mean concentration of 1.4 mg/m^3 (trace—6.1 mg/m^3) sufficient to cause dental erosion. Unfortunately there are no estimates of the chronic effects of exposure on ventilatory function.

El-Sadik *et al.* (1972) examined 33 battery manufacture workers exposed to very high acid levels (26–35 mg/m^3 in one plant and 13–14 mg/m^3 in another). They were compared to a control group of 20 workers of similar age. The prevalence of bronchitis was 36% among the exposed workers and 21% among the controls. However, smoking was related to bronchitis, and 60% of the acid-exposed group were smokers compared to 40% of the control group.

Pelnar (1951) examined 124 workers in a sulfuric acid plant. The longest length of employment was 43 years. The author concludes that "no serious pathological condition occurred with striking frequency." All persons excreted an acid urine. Emphysema (5.6%) and dental erosion (16.1%) increased with increasing tenure. There is insufficient information to evaluate the author's conclusions.

Previous studies of acid-exposed workers show no effect on the respiratory system clearly attributable to low levels of sulfuric acid. The potential effect on pulmonary function was not examined. Tooth etching and erosion was common.

METHODS

The workers examined in the study of effects of short-term exposure to acid mist (Gamble *et al.*, 1984) comprise the bulk of the workers in the study of chronic effects. Each worker was administered a British Medical Research Council respiratory questionnaire by trained interviewers. Work histories were obtained from company records. Standard posterior–anterior chest radiograms were read by three "B" readers using the ILO U/C 1971 scheme. The films were read independently without knowledge of age, occupation, or smoking history. The median of three readings was used. Values from the preshift maximum envelopes were used for analysis of pulmonary function (Gamble *et al.*, 1984). The front teeth of each worker were examined by a dentist for etching and erosion (Grades G1, G2, G3).

The personal samples taken in the acute study (Jones and Gamble, 1984) were used to estimate acid mist exposure for different jobs in each plant. The average exposure for jobs was calculated for each plant. Cumulative acid exposure for each participant was estimated by multiplying job exposure (as estimated above) by time, and cumulating the results of exposure × time for all jobs held within the plant.

The association of respiratory symptoms and etching with exposure was analyzed using logistic models.The association with age, smoking, and plant were estimated, and included in the model estimating the association with exposure. Two exposure variables were used: (1) cumulative exposure as a continous variable in the total population; and (2) high (>15 mg/m^3 × months) and low (<7 mg/m^3 × months) acid exposure categories. The results were generally similar unless noted otherwise.

Pulmonary function in white males was analyzed using multiple regression. The independent variables were age, height, smoking, plant, and exposure. The same two exposure variables were used.

Most of the study population were white males, with the majority having attended at least 1 year of high school. Thirty-nine workers (16%) had worked in a foundry, and 15 (6%) in a pottery. Few workers had worked in other potentially hazardous occupations. Only six chest roentgenograms showed any indication of pneumoconiosis or pleural changes, and therefore no further analysis was done (Table 1).

The characteristics of each smoking category were typical of a blue collar population, with perhaps a slightly lower proportion of nonsmokers than usual. Ex-smokers were slightly older, had worked more years, and had slightly higher cumulative exposure than both smokers and nonsmokers. Younger ex-smokers smoked fewer cigarettes/day than the other smoking categories. The oldest population with the highest average years worked was in Plant A. However, the highest average and cumulative exposure was in Plant D. Plant E had the highest proportion of smokers (and lowest proportion of ex-smokers and nonsmokers). Plant A had the highest proportion of ex-smokers and lowest proportion of nonsmokers. The proportion of workers with tooth etching and erosion was 23% (Plant B), 17% (Plant E), 14% (Plant D), 6% (Plant A), and 0% (Plant C) (Table 2). There was a significant correlation of years worked and cumulative exposure ($r = .60$), years worked and age ($r = .73$), and cumulative exposure and age ($r = .49$) (Table 3).

RESULTS

Symptoms and Dental Erosion

The prevalence of selected symptoms and tooth etching by age and smoking in the total study population is summarized in Table 4. The prevalence of cough was highest in smokers (34%) and increased with age only in smokers. The prevalence of phlegm was also highest in smokers (40%) but all smoking categories had a slightly higher prevalence in the older age group compared to the younger age group. The prevalence of dyspnea was 6% overall and was not related to

TABLE 1
DEMOGRAPHIC CHARACTERISTICS OF ACID STUDY POPULATION (FREQUENCY)

		n	%
Sex	Male	243	98
	Female	5	2
Race	White	240	97.6
	Black	3	1.2
Education	<9	37	15.0
	9–11	61	32.1
	12	117	47.6
	>12	13	5.2
Have you ever worked:			
In any type of mine?		4	1.6
In a quarry?		8	3.2
In a foundry?		39	15.7
In a pottery?		15	6.0
In a cotton, flax, or hemp mill?		4	1.6
With asbestos?		5	2.0
Chest x-ray:			
Round opacities		0	—
Irregular opacities		1	0.4
Pleural thickening—Extent 1		4	1.7
Extent 2		2	0.9
Have you ever had:			
Heart trouble?		11	4.4
Pulmonary tuberculosis?		0	—
Bronchial asthma?		8	3.2
Emphysema?		1	0.4
Bronchitis?		30	12.1
Pneumonia?		39	15.7
Pleurisy?		9	3.6

smoking in this population. The prevalence was higher in the older age groups of both nonsmoking categories, but was lower in the older smokers. There was no case of wheeze among ex-smokers and only one case among nonsmokers (2%) compared to 12 (8%) among smokers. There was no apparent age effect. Etching and tooth erosion showed no apparent relation to smoking, as smokers and ex-smokers had a prevalence of 12% and 10% respectively compared to 19% for nonsmokers. The prevalence of dental changes was higher in the <40-year-old group (15%) compared to the >40-year-old category (9%). Use of false teeth, on the other hand, was more common in smokers (38%) and ex-smokers (22%) than in nonsmokers (7%), and was about two and one-half times more prevalent in the older age group.

To reduce the possibility of exposure misclassification, differences between workers clearly having high acid exposure (>15 mg/m^3 × months) and low acid exposure (<7 mg/m^3 × months) were analyzed. The high exposure group was older, had worked longer, and had a higher average exposure (Table 5).

Respiratory symptoms (cough, phlegm, dyspnea, and wheeze) showed no sig-

TABLE 2
DEMOGRAPHIC CHARACTERISTICS OF ACID STUDY POPULATION (MEANS WITH SD)

	I. By smoking			
	Nonsmokers	Ex-smokers	Smokers	All
n (%)	46 (19)	59 (24)	143 (58)	248
Age	34.1 (13.8)	40.1 (13.8)	36.4 (11.6)	36.9 (12.7)
Height (cm)	175.5 (7.7)	172.6 (6.9)	175.1 (6.5)	174.6 (6.9)
Weight (kg)	82.0 (20.6)	81.7 (11.0)	79.1 (14.6)	80.2 (15.2)
Cumulative exposure (mg/m^3 × months)	16.5 (20.0)	19.0 (21.2)	15.5 (18.4)	16.5 (19.4)
Years worked	7.9 (8.0)	12.1 (10.0)	9.9 (9.4)	10.0 (9.4)
Average exposure (mg/m^3)	0.18 (0.12)	0.15 (0.12)	0.14 (0.11)	0.15 (0.11)
Pack years	—	17.5 (16.6)	21.5 (17.3)	—
Cigarettes/day	—	20.3 (13.0)	23.2 (10.9)	—

	II. By plant				
	A	B	C	D	E
n	35	57	38	59	59
Age	49.4 (7.8)	29.3 (10.4)	35.1 (12.7)	36.1 (11.7)	38.6 (12.3)
Cumulative exposure (mg/m^3 × months)	15.9 (4.9)	7.2 (9.8)	8.4 (10.5)	27.9 (26.0)	19.7 (21.8)
Years worked	20.2 (5.8)	4.0 (3.8)	10.2 (9.8)	7.5 (5.6)	12.2 (11.7)
Average exposure (mg/m^3)	0.07 (0.01)	0.14 (0.08)	0.07 (0.03)	0.27 (0.14)	0.14 (0.07)
High exposure (mg/m^3)	.082	.23	.12	.42	.29
Low exposure (mg/m^3)	.058	.066	.041	.069	.063
Nonsmokers (%)	5.7	26.3	21.1	23.7	11.9
Ex-smokers (%)	40.0	24.6	23.7	23.7	13.6
Smokers (%)	54.3	49.1	55.3	52.5	74.6
Etching—*n*(%)	1 (2.9)	8 (14.0)	0	3 (5.1)	5 (8.5)
Grade 1—*n*(%)	1 (2.9)	3 (5.3)	0	2 (3.4)	2 (3.4)
Grade 2—*n*(%)	0	1 (1.8)	0	2 (3.4)	3 (5.1)
Grade 3—*n*(%)	0	1 (1.8)	0	1 (1.7)	0

nificant association with plant or exposure (Tables 6–9). Cough was greatly increased in smokers but not ex-smokers, and there was no increase in cough with increasing age (Table 6). A similar relationship was observed between phlegm and smoking. However, older workers (after smoking adjustments) complained of more phlegm than younger workers (Table 7).

Dyspnea showed no significant association with age or smoking, although there was a trend for younger workers to complain of dyspnea more than older workers. Smokers had the highest observed-to-expected ratio, with little difference between nonsmokers and ex-smokers (Table 8). A similar relationship was observed for wheezing; however, the association of smoking and wheeze was much stronger than for smoking and dyspnea (Table 9).

False teeth showed no significant association with plant or acid exposure, but there was three times the observed-to-expected ratio in the older workers compared to younger workers. Smokers had about twice the ratio of ex-smokers and nonsmokers. Tooth etching and erosion showed a very strong association with acid exposure and were observed in every plant except Plant C. Age and smoking showed no association with dental changes (Table 10).

Because tooth etching and erosion are known to be caused by acid, the question

TABLE 3
CORRELATION MATRIX OF BATTERY PLANT WORKERS—CHRONIC STUDY (n = 248)[a]

	Age	Height	Weight	Cumulative exposure	Years worked	Average exposure	Pack years	Cigarettes/ day	FEV_1	FVC	Peak flow	FEF_{50}	FEF_{75}
Age		−.28	−.01	.49	.73	−.04	.58	.13	−.61	−.45	−.34	−.43	−.62
Height	−.28		.39	−.25	−.17	−.13	−.15	−.07	.51	.57	.25	.18	.25
Weight		.39		−.04	.03	−.01	−.01	−.04	.22	.27	.04	.09	.03
Cumulative exposure	.49	−.25			.60	.56	.19	−.03	−.41	−.33	−.27	−.26	−.39
Years worked	.73			.60		−.12	.43	.14	−.48	−.36	−.25	−.35	−.50
Average exposure				.56			−.12	.16	−.10	−.14	−.13	0	−.02
Pack years	.58			.19	.43			.67	−.53	−.36	−.27	−.47	−.56
Cigarettes/day							.67		−.23	−.11	−.09	−.27	−.30
FEV_1	−.61	.51	.22	−.41	−.48		−.53	−.23		.88	.65	.73	.72
FVC	−.45	.57	.27	−.33	−.36		−.36		.88		.48	.37	.37
Peak flow	−.34	.25		−.27	−.25		−.27		.65	.48		.64	.46
FEF_{50}	−.43	.18		−.26	−.35		−.47	−.27	.73	.37	.64		.81
FEF_{75}	−.62	.25		−.39	−.50		−.56	−.30	.72	.37	.46	.81	

[a] $P \leq 0.005$ for correlation coefficients in lower left half of matrix.

TABLE 4
PREVALENCE OF SYMPTOMS, OBSTRUCTION, AND TEETH EROSION BY AGE AND SMOKING
BATTERY ACID STUDY POPULATION

	Nonsmokers		Ex-smokers		Smokers		All groups		All ages	All ages	All ages
	<40[a] *n* (%)	≥40 *n* (%)	<40 *n* (%)	≥40 *n* (%)	<40 *n* (%)	≥40 *n* (%)	<40 *n* (%)	≥40 *n* (%)	*n* (%)	*n* (%)	*n* (%)
Cough	4 (12)	0	6 (21)	1 (3)	27 (29)	21 (42)	37 (24)	22 (24)	4 (9)	7 (12)	48 (34)
Phlegm	3 (9)	2 (15)	2 (7)	5 (17)	33 (35)	24 (48)	38 (25)	31 (33)	5 (11)	7 (12)	57 (40)
Shortness of breath	1 (3)	1 (8)	1 (3)	2 (7)	10 (11)	1 (2)	12 (8)	4 (4)	2 (4)	3 (5)	11 (8)
Wheeze	1 (3)	0	0	0	8 (9)	4 (8)	9 (6)	4 (4)	1 (2)	0 (0)	12 (8)
Etching	5 (15)	—	2 (7)	1 (3)	6 (6)	3 (6)	13 (8)	4 (4)	5 (11)	3 (5)	9 (6)
Grade 1	2 (6)	—	2 (7)	1 (3)	2 (2)	1 (2)	6 (4)	2 (2)	2 (4)	3 (5)	3 (2)
Grade 2	2 (6)	—	—	—	1 (1)	3 (6)	3 (2)	3 (3)	2 (4)	—	4 (3)
Grade 3	—	—	—	—	2 (2)	—	2 (1)	—	2 (4)	—	2 (1)
False teeth	3 (9)	6 (46)	2 (7)	11 (38)	25 (27)	30 (60)	30 (19)	47 (51)	3 (7)	13 (22)	55 (38)
n	33	13	29	30	93	50	155	93	46	59	143

[a] Age.

of greatest interest is the lowest exposure at which etching and erosion are seen. Table 10 and Fig. 1 summarize exposure variables by tooth condition. The characteristics of workers with etching and the lowest exposure are summarized in Table 11. These data show that etching can develop in 4 months to an estimated average daily exposure of 0.23 mg/m^3. The shortest length of employment for erosion was 30 months, and the lowest cumulative exposure was 7 mg/m^3 × months. It is entirely possible, of course, that the actual time and exposure may be less because these are cross-sectional data and we do not know when the etching and erosion actually occurred.

Pulmonary Function

Table 12 summarizes the relation of acid exposure and lung function. While the high exposure group consistently had reduced lung function compared to the low exposure group, the differences were statistically significant only for FVC. There was no significant association with cumulative exposure. Smokers consistently

TABLE 5
CHARACTERISTICS OF BATTERY STUDY POPULATION BY EXPOSURE CATEGORY

	Low exposure	High exposure
n	99	100
Age	29.2 (9.6)	44.5 (11.0)
(SD)	(19–59)[a]	(23–63)
Years worked	3.0 (2.8)	17.3 (9.4)
(SD)	(0.8–12.5)	(3.3–38.8)
Average exposure[b]	0.10 (0.07)	0.21 (0.13)
(mg/m^3) (SD)	(0.04–0.29)	(0.04–0.42)
Cumulative exposure	2.5 (1.9)	33.4 (20.6)
(mg/m^3 × months) (SD)	(0.07–6.3)	(15.6–90.2)
% Nonsmokers	17	16
% Ex-smokers	20	28
Cigarettes/day (SD)	19.5 (12.3)	22.9 (14.2)
Pack years (SD)	11.6 (11.5)	22.1 (18.7)
% Smokers	63	56
Cigarettes/day (SD)	22.4 (11.0)	21.2 (11.2)
Pack years (SD)	15.1 (15.4)	23.7 (14.3)

[a] Range.

[b] Average exposure = $\frac{\text{cumulative exposure}}{\text{years worked}}$.

TABLE 6
OBSERVED AND EXPECTED PREVALENCE OF COUGH[a] AMONG LEAD ACID BATTERY WORKERS IN HIGH AND LOW CUMULATIVE EXPOSURE CATEGORIES

	n	Observed (%)	Expected (%)	Ratio (O/E)	Comment
Cough	199	24.1			
Age					$P > 0.60$, adjusted
<40	122	25.4	24.7	103	for smoking.
≥40	77	22.1	23.2	95	
Smoking					$P < 0.005$, adjusted
Nonsmoker	33	12.1	24.7	49	for age.
Ex-smoker	48	12.5	23.5	53	
Smoker	118	32.2	24.2	133	
Plant					$P > 0.40$, adjusted
A	24	20.8	20.8	100	for age and smoking.
B	54	18.5	22.8	81	
C	31	19.4	25.6	76	
D	48	33.3	23.9	139	
E	42	26.2	27.0	97	
Exposure					$P > 0.80$, adjusted
Low	99	24.2	23.8	102	for age, smoking,
High	100	24.0	24.4	98	and plant.

[a] Cough = answering yes to the question: "Do you cough on most days for as much as 3 months each year?"

TABLE 7

OBSERVED AND EXPECTED PREVALENCE OF PHLEGM[a] AMONG LEAD ACID BATTERY WORKERS IN HIGH AND LOW CUMULATIVE EXPOSURE CATEGORIES

	n	Observed (%)	Predicted (%)	Ratio (O/E)	Comment
Phlegm	199	28.6			
Age					$P < 0.05$, adjusted
<40	122	25.4	29.5	86	for smoking.
≥40	77	33.8	27.4	123	
Smoking					$P < 0.0005$, adjusted
Nonsmoker	33	12.1	27.3	44	for age.
Ex-smoker	48	12.5	30.3	41	
Smoker	118	39.8	28.3	141	
Plant					$P > 0.40$, adjusted
A	24	33.3	32.6	102	for age and smoking.
B	54	20.4	22.1	92	
C	31	35.5	28.0	127	
D	48	22.9	28.5	80	
E	42	38.1	35.5	107	
Exposure					$P > 0.20$, adjusted
Low	99	29.3	26.8	109	for age, smoking,
High	100	28.0	30.5	92	and plant.

[a] Phlegm = answering yes to the question: "Do you bring up phlegm on most days for as much as 3 months each year?"

TABLE 8

OBSERVED AND EXPECTED PREVALENCE OF DYSPNEA[a] AMONG LEAD ACID BATTERY WORKERS IN HIGH AND LOW CUMULATIVE EXPOSURE CATEGORIES

	n	Observed (%)	Expected (%)	Ratio (O/E)	Comments
Dyspnea	199	6.5			
Age					$P > 0.30$, adjusted
<40	122	8.2	6.7	122	for smoking.
≥40	77	3.9	6.3	62	
Smoking					$P > 0.70$, adjusted
Nonsmoker	33	6.1	7.0	87	for age.
Ex-smoker	48	4.2	6.1	69	
Smoker	118	7.6	6.6	115	
Plant					$P > 0.30$, adjusted
A	24	4.2	4.4	95	for age
B	54	3.7	7.3	51	and smoking.
C	31	3.2	7.2	44	
D	48	10.4	6.5	160	
E	42	9.5	6.4	148	
Exposure					$P > 0.20$, adjusted for
Low	99	8.1	6.6	123	age, smoking,
High	100	5.0	6.4	78	and plant.

[a] Dyspnea = answering yes to the question: "Do you get short of breath walking with other people of your own age on level ground?"

TABLE 9
OBSERVED AND EXPECTED PREVALENCE OF WHEEZING[a] AMONG LEAD ACID BATTERY WORKERS IN HIGH AND LOW CUMULATIVE EXPOSURE CATEGORIES

	n	Observed (%)	Expected (%)	Ratio (O/E)	Comment
Wheezing	199	6.5			
Age					$P > 0.80$, adjusted
<40	122	7.4	6.8	109	for smoking.[b]
≥40	77	5.2	6.1	85	
Smoking[b]					$P < 0.01$, adjusted
Nonsmokers and Ex-smokers	81	1.2	6.5	18	for age.
Smokers	118	10.2	6.5	157	
Plant					$P > 0.20$, adjusted
A	24	4.2	6.1	69	for age and smoking.
B	54	0	5.3	0	Plant B not
C	31	6.5	6.8	96	included in
D	48	4.2	6.4	66	logistic model
E	42	19.0	8.2	232	as prevalence = 0%.
Exposure					$P > 0.05$, adjusted for
Low	100	9.0	6.5	138	age and smoking.
High	99	4.0	6.6	61	

[a] Wheeze = answering yes to the question as to whether their chest sounds wheezing or whistling most days.
[b] Nonsmokers and ex-smokers combined.

TABLE 10
OBSERVED AND EXPECTED PREVALENCE OF DENTAL ETCHING[a] AMONG LEAD ACID BATTERY WORKERS WITH TEETH

	n	Observed (%)	Expected (%)	Ratio (O/E)	Comment
Etching	136	20.6			
Age					$P > 0.20$, adjusted
<40	100	20.0	20.8	96	for smoking.
≥40	36	22.2	19.9	112	
Smoking					$P > 0.60$, adjusted
Nonsmoker	27	22.2	20.1	110	for age.
Ex-smoker	36	16.7	21.7	77	
Smoker	73	21.9	20.3	108	
Plant					$P > 0.20$, adjusted
A	13	15.4	24.1	64	for age and smoking.
B	45	26.7	18.4	145	Plant C excluded
C	23	0	20.0	0	from the logit
D	34	20.6	21.7	95	analysis.
E	21	33.3	22.1	151	
Exposure					$P \ll 0.0005$, adjusted
Low	78	7.7	18.9	41	for age and smoking.
High	58	37.9	22.9	166	

[a] Dental etching and erosion were combined. Workers with false teeth were excluded from this analysis.

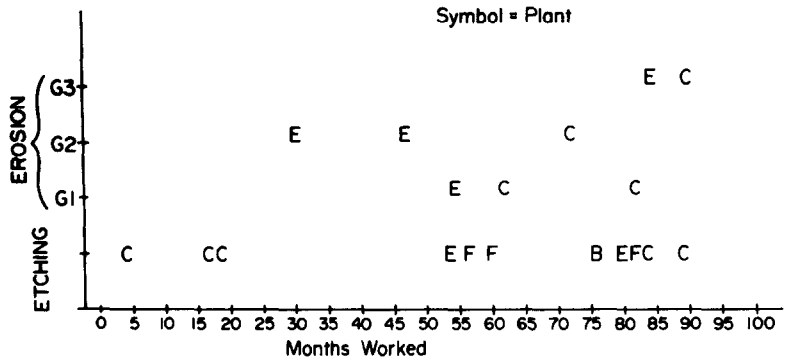

FIG. 1. Tooth condition by tenure.

had the lowest lung function values, and ex-smokers the hignest values. While plant was generally a significant independent variable, mean pulmonary function was not related to average exposure. The plants with the highest (D) and lowest (A and C) acid levels also had the lowest mean FVC and FEV_1.

Figures 2–5 display the residual age and height adjusted lung function of white male smokers with and without the symptoms of cough, phlegm, dyspnea, and wheezing. In all cases there was a trend for those with the symptom to have reduced FEV_1, FVC, and FEF_{50} compared to those without the symptoms. The variation in FEF_{50} was quite large in all cases. Those with cough were significantly reduced compared to those without a cough and those with only a winter cough. None of the other symptoms were associated with a significantly reduced lung function.

DISCUSSION

In the study reported here, exposure to acid mist showed no significant association with cough, phlegm, dyspnea, wheezing, most measures of pulmonary function, and abnormal chest radiographs. Tooth etching and erosion were strongly related to acid exposure. The minimum tenure of a worker with tooth etching was 4 months; estimated average acid exposure was 0.23 mg/m^3.

TABLE 11
CHARACTERISTICS OF WORKERS WITH TOOTH ETCHING OR EROSION AND LOW EXPOSURE

Degree of etching	Plant	Total cumulative exposure (mg/m^3 × months)	Total months worked	Mean exposure (mg/m^3)
Etch	B	0.9	4	0.23
Grade 1	A	16.9	252	0.07
Grade 1	D	23.7	68	0.35
Grade 1	E	24.0	381	0.06
Grade 2	D	7.0	30	0.23
Grade 3	D	34.6	82	0.42

TABLE 12A

SUMMARY OF MULTIPLE REGRESSION PULMONARY FUNCTION MODEL WITH AGE AND HEIGHT COEFFICIENTS, AND LEAST SQUARE MEANS BY SMOKING CATEGORY, PLANT, AND EXPOSURE CATEGORY (WHITE MALES ONLY)[a]

Model: PFT = $\alpha + \beta_1$(age) + β_2(height) + β_3(smoking category) + β_4(plant) + β_5(exposure category)

	FVC (ml or liters)	FEV_1 (ml or liters)	FEV (%)	Peak flow (ml/sec or liters/sec)	FEF_{50} (ml/sec or liters/sec)	FEF_{75} (ml/sec or liters/sec)
Age β_1	−15 (5)**,[b]	−24 (4)***	−.3 (.1)***	−38 (13)**	−34 (10)**	−29 (5)***
Height (cm) β_2	55 (7)***	39 (6)***	−.1 (.1) N.S.[c]	42 (19)*	15 (15) N.S.	12 (7) N.S.
Smoking	N.S.	**	*	N.S.	**	*
Nonsmokers	5.03 (.12)	3.97 (.10)	78.5 (1.3)	9.08 (.30)	4.79 (.24)	1.58 (.11)
Ex-smokers	5.05 (.09)	4.03 (.08)	78.9 (1.0)	9.16 (.24)	4.82 (.19)	1.76 (.09)
Smokers	4.84 (.06)	3.70 (.05)	76.4 (0.7)	9.03 (.16)	4.13 (.12)	1.44 (.06)
Plant	N.S.	$P = 0.05$	**	$P = 0.05$	$P = 0.05$	**
A	5.00 (.14)	3.81 (.12)	75.8 (1.6)	8.94 (.37)	4.13 (.30)	1.37 (.14)
B	5.07 (.09)	4.11 (.08)	80.8 (1.0)	9.16 (.24)	4.95 (.19)	1.81 (.09)
C	4.86 (.12)	3.94 (.10)	81.1 (1.3)	9.85 (.31)	5.06 (.25)	1.87 (.12)
D	4.81 (.10)	3.76 (.08)	78.3 (1.1)	8.85 (.26)	4.47 (.21)	1.50 (.10)
E	5.11 (.11)	3.87 (.09)	75.4 (1.2)	8.65 (.28)	4.30 (.22)	1.43 (.10)
Exposure category	$P = 0.02$	$P = 0.09$	N.S.	N.S.	N.S.	N.S.
Low	5.11 (.08)	3.98 (.07)	77.9 (0.9)	9.09 (.22)	4.65 (.17)	1.66 (.08)
High	4.83 (.08)	3.81 (.07)	78.7 (0.9)	9.09 (.20)	4.51 (.16)	1.53 (.08)
r^2	.47	.59	.35	.22	.31	.49

[a] $n = 188$, white males only; high and low exposure group only, $n = 94$ in each.

[b] SE in parentheses.

[c] N.S. = $P > 0.05$.

* $P < 0.05$.

** $P < 0.005$.

*** $P < 0.0005$.

TABLE 12B

SUMMARY OF MULTIPLE REGRESSION PULMONARY FUNCTION MODEL WITH AGE, HEIGHT, AND CUMULATIVE EXPOSURE COEFFICIENTS AND LEAST SQUARE MEANS FOR SMOKERS AND PLANT (WHITE MALES ONLY)[a]

Model: $\alpha + \beta_1(\text{age}) + \beta_2(\text{height}) + \beta_3(\text{smoking category}) + \beta_4(\text{plant}) + \beta_5(\text{cumulative exposure})$

	FVC (ml or liters)	FEV_1 (ml or liters)	FEV (%)	Peak flow (ml/sec or liters/sec)	FEF_{50} (ml/sec or liters/sec)	FEF_{75} (ml/sec or liters/sec)
Age β_1	−21 (4)***[b]	−27 (4)***	−0.2 (.05)***	−42 (12)***	−37 (9)***	−28 (4)
Height (cm) β_2	+60 (7)***	+42 (5)***	−0.1 (.1) N.S.[c]	+41 (18)*	+17 (14) N.S.	+12 (6)
Smoking	*	***	***	N.S.	***	***
Nonsmokers	4.97 (.10)	3.99 (.08)	80.2 (1.1)	9.32 (.26)	4.88 (.20)	1.71 (.09)
Ex-smokers	5.09 (.09)	4.06 (.07)	80.1 (0.9)	9.36 (.22)	4.96 (.17)	1.79 (.08)
Smokers	4.83 (.06)	3.68 (.05)	76.1 (0.6)	9.00 (.15)	4.09 (.11)	1.41 (.05)
Plant	N.S.	*	**	*	*	**
A	4.98 (.12)	3.80 (.10)	75.8 (1.3)	9.36 (.32)	4.20 (.25)	1.32 (.11)
B	5.04 (.09)	4.08 (.07)	80.6 (1.0)	9.07 (.24)	4.87 (.18)	1.79 (.08)
C	4.86 (.11)	3.96 (.09)	81.5 (1.1)	9.96 (.29)	5.15 (.22)	1.92 (.10)
D	4.80 (.10)	3.77 (.08)	78.9 (1.0)	8.74 (.25)	4.50 (.19)	1.61 (.09)
E	5.12 (.09)	3.95 (.07)	77.2 (1.0)	9.00 (.24)	4.48 (.18)	1.54 (.08
Cumulative exposure	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.
(mg/m^3 × months) β_5	−2 (3)	−3 (3)	0 (0)	+1 (8)	−4 (6)	−5 (3)
r^2	.44	.57	.34	.18	.30	.48

[a] $n = 235$.

[b] SE in parentheses.

[c] N.S. or no notation = $P > 0.05$.

* $P < 0.05$.

** $P < 0.005$.

*** $P < 0.0005$.

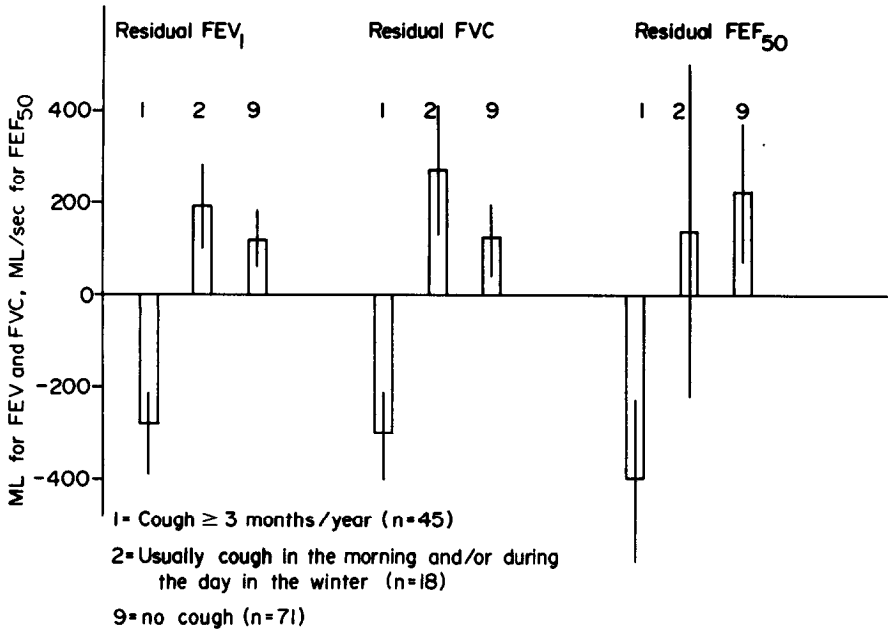

FIG. 2. Pulmonary function (mean age and height adjusted residual with S.E.) of white male smokers with and without cough.

The lack of an association of upper respiratory system symptoms and exposure is somewhat surprising considering the irritant nature of sulfuric acid.

Mucus in the respiratory tract is the most common stimulus to cough. Other factors causing cough are mechanical and chemical irritants stimulating receptors in the trachea and bronchi. It is a common occurrence in chronic bronchitis, upper respiratory infection, tuberculosis, and carcinoma of the bronchus. Chronic irritation increases the number of goblet cells, and therefore increases mucus secretions (Crofton and Douglas, 1969).

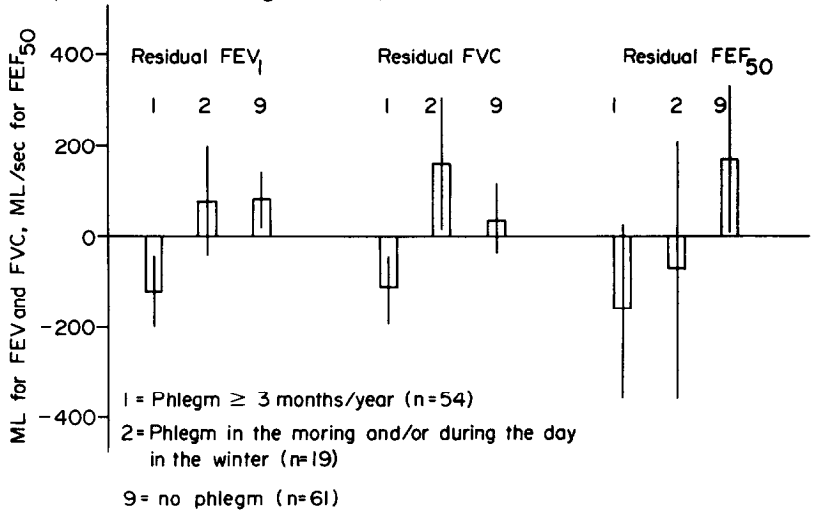

FIG. 3. Pulmonary function (mean age and height adjusted residual with S.E.) of white male smokers with and without phlegm.

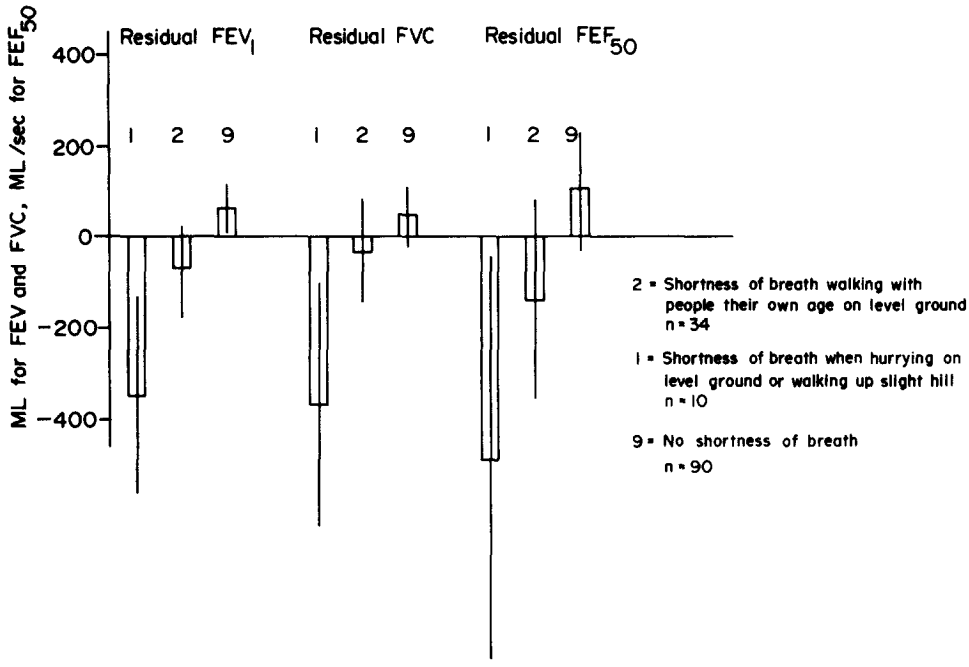

FIG. 4. Pulmonary function (mean age and height adjusted residual with S.E.) of male white smokers with and without shortness of breath.

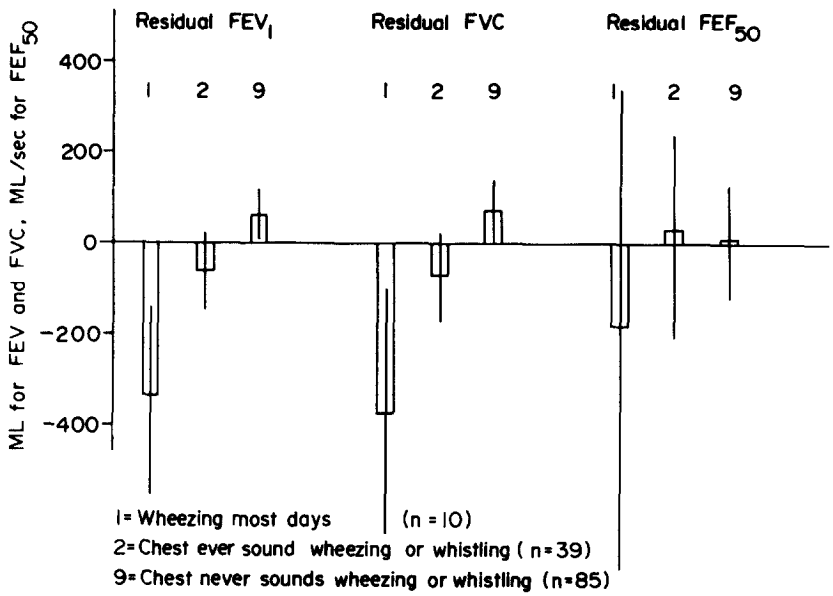

Fig. 5. Pulmonary function (mean age and height adjusted residual with S.E.) of white male smokers with and without wheeze.

Industrial bronchitis is a term used to denote chronic (at least 3 months of the year) cough and phlegm with or without airflow obstruction, and is related to dust inhalation (Morgan, 1978). There does not appear to be an excess of "industrial bronchitis" in this population of workers exposed to a known irritant, suggesting that either the acid is in too low a concentration to irritate or too large to penetrate to the upper respiratory tract and stimulate receptors or mucus glands.

Dyspnea is a subjective awareness of a need for increased respiratory effort. It occurs when the demands made on the lung are greater than the lung's capacity to respond. Dyspnea can result when there is increased work of breathing, reduced ventilatory capacity, and/or a sensitized awareness of the act of breathing. Often there is functional disability, but not necessarily so (Crofton and Douglas, 1969).

Wheezing is a result of narrowed bronchi (Forgacs, 1978) resulting from mucus in bronchial lumen, swelling of the bronchial mucous membrane, or spasm of the bronchial muscle (Crofton and Douglas, 1969). Wheezing has been observed in textile workers, and in coal miners has been suggested as being caused by particles settling in the bronchi (Bouhuys, 1974).

Irritation of receptors in upper airways, trachea, and large bronchi can result in sneezing, glottal closure, apnea (or rapid shallow breathing), cough, and in some cases reflex bronchoconstriction (Bouhuys, 1974). There is no evidence from this study to suggest any increase in respiratory symptoms associated with acid exposure. The power of the test is not involved in this conclusion, as the high exposure group had a lower prevalence of symptoms than the low exposure group. This complements the finding of no acute reduction in pulmonary function (Gamble *et al.*, 1984).

The association of reduced baseline pulmonary function and acid exposure was significant only for FVC and only when the study population was divided into high and low exposure categories. The significance of this finding is unclear. The arguments against the biological significance of the reduced FVC are as follows:

(a) No relationship was observed when cumulative exposure as a continous variable was used.

(b) The *P* value is small, and the number of comparisons is large, so that the difference could be due to chance.

(c) Animal and epidemiological studies show little or no effect on physiology or anatomy at H_2SO_4 concentrations higher than those observed in this study.

(d) Although estimates of site deposition are variable, and there are in fact small particles in the mist, if the site of deposition is largely in the upper respiratory tract, the mechanism for causing lung damage is not known.

The arguments that the FVC reduction is biologically significant include the following:

(a) Reductions of FEV_1, and flow rate are also seen in the high acid-exposed group, suggesting that FVC may not be the only lung function parameter affected.

(b) The differences in the high and low exposure group are age and smoking adjusted, so the effect of these variables that cannot be adjusted statistically is likely to be insignificant.

FEV_1 and flows at 50 and 75% of FVC were also reduced in the high exposure group. Despite this trend, the differences were not statistically significant, and the actual differences were small. For FEV_1 there was a 68% chance of detecting a true difference of 170 ml between the high and low exposure categories. Mean FEV% and peak flow were not reduced in the high exposure group, and there was no association with cumulative exposure. The large differences between the high and low exposure groups in mean age (20 years), mean pack years in smokers (24 vs 15), and ex-smokers (2 vs 12) may explain part of these differences.

The reasons for the elevated values of mean pulmonary function in ex-smokers is unclear. Their higher pulmonary function values and slightly higher exposure also supports the idea of no effect of acid exposure on pulmonary function.

The lack of a marked effect of acid exposure on respiratory symptoms and pulmonary function may be due to the size of the acid particles. The range of mass median diameter in the five plants was 2.6–10 μm. The relative humidity of the lung would at least double particle size and many acid particles would be deposited in the upper respiratory tract (Cavender *et al.*, 1977b).

The effects of acid exposure on the teeth have been documented in several studies (El-Sadik *et al.*, 1972; Malcolm and Paul, 1961; Ten Bruggen Cate, 1968). Malcolm and Paul (1961) observed among battery workers a higher prevalence of erosion than etching, and both were confined to the labial surfaces of the anterior teeth. Exposure was 3–17 mg/m^3 in the forming area where the prevalence of erosion and etching among those with teeth was 87%. Mean years worked in the forming department was 15 for those with no etching or erosion, 5 years for those with etching, and 7, 12, and 17 years for those with increasing degrees of erosion, suggesting susceptibility. In the charging area acid exposure ranged from less than 0.8 mg/m^3 to 2.5 mg/m^3. The prevalence of erosion and etching was 47% among those with teeth, with four cases of etching and three cases of Grade 1 erosion. Grade 2 erosion was seen in one man after only 6 months exposure.

Ten Bruggen Cate (1968) found a prevalence of 60% etching and erosion among 70 battery formation workers and 31% among 16 charging and inspection workers. Half of the workers working in battery formation when examined prospectively showed progression of the dental erosion. Unfortunately, no environmental measurements were taken. Incidence of dental etching and erosion was higher among workers in battery manufacture than in plating, galvanizing, acid pickling, and other acid processes. The shortest time of exposure for etching to occur was 3 months, 6 months for Grade 1 erosion, $2\frac{1}{2}$ years for Grade 2 erosion, and 6 years for Grade 3 erosion. These times for etching and Grade 3 erosion are similar to those found in this study.

The prevalence of tooth erosion among 33 acid-exposed workers making batteries in Egypt was 39% (El-Sadik *et al.*, 1972). All of those with erosion had worked more than 1 year. Air concentrations were quite high in the two plants (26.1 to 35.0 and 12.6 to 13.5 mg/m^3).

Even though acid concentrations were considerably lower in this study than reported in the battery studies summarized above, tooth etching and erosion were

still present. Concentrations measured at the time of the study were usually 1 mg/m^3 or less, but etching and erosion appeared to be occurring in the same time reported in other studies when exposure to acid was much higher. Part of the difference in acid levels between this study and previous studies may be due to differences in sampling. In previous studies, area samples were taken, probably in the areas of highest concentration. Workers move around, and do not remain in the high exposure areas during the entire work shift. Thus area samples overestimate the actual exposure to acid, while personal samples more readily reflect actual exposure. Personal samples were collected in this study.

While the latent period was similar to previous studies, the prevalence of etching and erosion was much lower in this study, and in Plant C none of the workers examined had etching or erosion. Plant C (with zero prevalence) appeared to have similar acid exposures as Plant A (5.7% prevalence). The workers were somewhat younger in Plant C, with less tenure and cumulative exposure. That this is the reason for the difference in prevalence is possible, but seems unlikely. In fact, the exposure group in Plant C had an average exposure of 0.12 mg/m^3 compared to 0.08 mg/m^3 for Plant A where there was one case of etching and one case of Grade 1 erosion. Variation in susceptibility probably contributes to the difference, although the risk factors are not known.

The particle size distribution of acid mist in battery plants is larger than that observed in air pollution (several micrometers in diameter compared to submicrometer particles). It is not known if particle size distribution was different than in previous studies in battery plants, although it probably was not.

Despite the obvious acute irritation encountered by unacclimated individuals on entering charging and forming areas, there was no evidence of short-term effects, i.e., increased subjective irritation as measured by questionnaire, and no association with reduced spirometry (over the shift) among the study population.

Despite the known caustic properties of sulfuric acid, we observed no relationship between acid exposure and effects on the respiratory system as measured by questionnaire (symptoms of cough, phlegm, dyspnea, wheezing), and chest radiographs. The relationship of acid exposure and baseline pulmonary function is unclear, and if effects occur as a result of exposure, they are minimal. Although previous studies in animals suggest a detrimental effect on the respiratory system, the lack of any convincing findings in this study relating to the respiratory system or irritation is not completely unexpected because of the relatively low exposure (<1 mg/m^3) compared to previous studies.

The role of acid in causing tooth etching and erosion is well recognized. The question of real interest then was not whether acid per se causes dental changes, but whether these changes occur in the workplace when acid concentrations are typically below the recommended standard as they were in this study. The answer is yes; etching and erosion do occur, and in a short time. A question of interest for which we have no answer is why etching did not occur in one plant where exposure was no less than in a plant where etching did occur. In other words, what are the risk factors associated with the occurrence of etching and erosion?

APPENDIX

Effects of Chronic Exposure (>8 hr) to Sulfuric Acid—Animal Exposures

Reference	No. animals	Exposure	Effects
Amdur *et al.* (1952)	Guinea pigs	8 mg/m^3 (1 μm); continuous for up to 72 hr. R.H. = 40–45%.	No mortality; lung damage (consolidation, infiltration with cellular material). Two actions: (1) irritant resulting in respiratory difficulty and laryngeal spasm; (2) deep seated lung damage.
Bushtueva (1957)	4 guinea pigs	8, 4, and 2 mg/m^3, for 5 days.	Disturbances of blood and lymph circulation; focal edema and acute interstitial processes.
Thomas *et al.* (1950)	Guinea pigs	Up to 4 mg/m^3; 18–140 days continuously. 0.9 μm diameter (most active), and 0.6 and 4 μm.	Hyperemia, edema, hemorrhage, cellular exudation, predominance of polymorphonuclear leukocytes, fibrinous thrombi in alveolar walls, excess of desquamated cells in major and minor bronchi; no evidence of irreversible damage.
Lewis *et al.* (1976)	8 dogs	0.9 mg/m^3; 21 hr/day, 620 days. R.H. = 43–50%.	No effect on hematological variable; no clearcut histopathological changes; tendency for lung function to be reduced compared to controls.
Alarie *et al.* (1973)	9 monkeys, 18 months exposure	0.4 mg/m^3 (2 μm MMD)	No apparent effect on growth or PaO_2; slight changes in lung histology; increased RR.
		0.5 mg/m^3 (0.5 μm MMD)	No changes in RR, PaO_2, growth, or pulmonary structure; deterioration of distribution of ventilation.
		2.43 mg/m^3 (3.6 μm MMD)	No effect on growth; increased RR; decreased PaO_2; alteration of pulmonary structure; moderate deterioration of distribution of ventilation.
		4.8 mg/m^3 (0.7 μm MMD)	No change in growth or PaO_2; increased RR; alteration of pulmonary structure, deterioration of distribution of ventilation.
	100 guinea pigs, 12 months exposure	0.1 mg/m^3 (2.7 μm MMD), 0.1 mg/m^3 (0.8 μm MMD)	No observed deleterious effects.
Alarie *et al.* (1975)	9 monkeys	24 hr/day; 8 months. Mixtures of: 0.1–5 ppm SO_2, 0.1–1 mg/m^3 H_2SO_4, and 0.5 mg/m^3 fly ash.	No detrimental effects on growth or hematology; questionable effects on lung function; slight histopathological effects.
		24 hr/day; 12 months. 0.9 mg/m^3 (0.5 μm MMD). R.H. = 50%.	No detrimental effects on growth, lung function, hematocrit, hemoglobin, RBC, WBC, lymphocytes, neutrophils, or histopathology of lung.

APPENDIX—*Continued*

Reference	No. animals	Exposure	Effects
Cavender *et al.* (1977a)	20 rats 20 guinea pigs	5 days: 100 mg/m^3 (0.7 μm MMD), 30 mg/m^3 (0.8 μm MMD), 10 mg/m^3 (0.9 μm MMD).	No morphological effects in rats and in guinea pigs <20 mg/m^3.
		Up to 28 days: 20 mg/m^3 (1.7 μm MMD), 20 mg/m^3 (1.0 μm MMD), 20 mg/m^3 (0.5 μm MMD). R.H. = 55%.	Guinea pigs (≥20 mg/m^3): diffuse regional nonsupportive alveolitis.
Juhos *et al.* (1978)	25 rats	8 hr/day; 82 days. 2 mg/m^3 (0.3 μm MMD). R.H. = 40–60%.	Minimal evidence of hypertrophy of epithelial lining cells, mainly at alveolar ductal level.
Cavender *et al.* (1978)	70 rats 70 guinea pigs	6 hr/day, 5 days/week, 6 months. 10 mg/m^3 (0.8 μm MMD). R.H. = 63%.	Minimal proliferation of alveolar macrophages and mild tracheal changes (loss of epithelial cilia); relatively nontoxic.
Cockrell *et al.* (1978)	20 guinea pigs	6 hr/day; 2 days, 25 mg/m^3 (1 μm). R.H. = 55–60%.	Acute lesions characterized by edema and hemorrhage, collapse fluid filled areas mostly in apical lobes and near hilus. Alveoli contained edema fluid, RBC, alveolar macrophages. Distinct demarcation between normal and damaged tissue; TEM revealed damage to distal airways and changes in vascular endothelium.
Schlesinger *et al.* (1979)	4 donkeys	5 days/week, 6 months. 0.1 mg/m^3 (0.5 μm MMD).	Decreased bronchial clearance.
Schwartz *et al.* (1977)	6–18 rats	6–11 days: 45–172 mg/m^3 (0.4–0.5 μm MMD).	No demonstrable morphological evidence of pulmonary damage (nasal septum, trachea, pulmonary parenchyma) by LM, and selected areas by SEM.
	2 Rhesus monkeys	3–7 days: 150–502 mg/m^3 (0.3–0.5 μm MMD).	71 mg/m^3—damage at bronchial and alveolar levels (edema, fibrinoid necrosis of alveolar septae, inflammatory cell infiltration.
	2–5 guinea pigs	4–7 days: 30–71 mg/m^3 (0.3–0.5 μm MMD).	30–38 mg/m^3—minimal changes (variability in density and length of cilia.
	8–45 mice	140–170 mg/m^3 (0.3–0.6 μm MMD). R.H. <60%.	Lesions only in larynx and upper trachea (ulceration of surface epithelium, adjoining connective tissue stroma was edematous and heavily infiltrated with eosinophils).

ACKNOWLEDGMENTS

We appreciate the assistance and cooperation of the companies and workers which made the study possible. We thank the ALOSH field team of Cecilia Engelberg, Elizabeth Knutti, Hope Lafferty, Marty Pflock, Gordie Stalnaker, Jonas Swecker, Jim Taylor, Vera Ulrich, and Chris Wood who collected the data and braved the snow storms; and Mary Lemasters, who typed the multiple editions of this report.

REFERENCES

Alarie, Y. C., Busey, W. M., Krumm, A. A., and Ulrich, C. E. (1973). Long-term continuous exposure to sulfuric acid mist in cynamolgous monkeys and guinea pigs. *Arch. Environ. Health* **27**, 15.

Alarie, Y. C., Krumm, A. A., Busey, W. M., Ulrich, C. E., and Kantz, R. J. (1975). Long-term exposure to sulfur dioxide, sulfuric acid mist, fly ash and their mixtures. *Arch. Environ. Health* **30**, 254–252.

Amdur, M. O. (1958). The respiratory response of guinea pigs to sulfuric acid mist. *Arch. Ind. Health* **18**, 407–414.

Amdur, M. O., Schulz, R. Z., Drinker, P. (1952). Toxicity of sulfuric acid mist to guinea pigs. *AMA Arch. Ind. Hyg.* **5**, 318–329.

Anfield, B. D., and Warner, C. G. (1968). A study of industrial mists containing sulfuric acid. *Ann. Occup. Hyg.* **11**, 185–194.

Bouhuys, A. (1974). "Breathing." Grune & Stratton, New York.

Bushtueva, K. A. (1957). The toxicity of H_2SO_4 aerosol. *Gig. Sanit.*, **22**, 17–22.

Cavender, F. L., Singh, B., and Cockrell, B. Y. (1978). Effects in rats and guinea pigs of six-month exposure to sulfuric acid mist ozone and their combination. *J. Toxicol. Environ. Health* **4**, 845–852.

Cavender, F. L., Steinhagen, W. H., Ulrich, C. E., Busey, W. M., Cockrell, B. Y., Haseman, J. K., Hogan, M. D., and Drew, R. T. (1977a). Effects in rats and guinea pigs of short-term exposures to sulfuric acid mist, ozones, and their combination. *J. Toxicol. Environ. Health.* **31**, 521–533.

Cavender, F. L., Williams, J. L., Steinhagen, W. H., and Woods, D. (1977b). Thermodynamics and toxicity of sulfuric acid mists. *J. Toxicol. Environ. Health* **2**, 1147–1159.

Cockrell, B. Y., Busey, W. M., and Cavender, F. L. (1978). Respiratory tract lesions in guinea pigs exposed to sulfuric acid mist. *J. Toxicol. Environ. Health* **4**, 835–844.

Crofton, J. and Douglas, A. (1969). "Respiratory Diseases." Blackwell, Oxford.

El-Sadik, Y. M., Osman, H. A., and El-Gazzar, R. M. (1972). Exposure to sulfuric acid in manufacture of storage batteries. *J. Occup. Med.* **14**, 224–226.

Forgacs, P. (1978). "Lung Sounds." MacMillan Co., New York.

Gamble, J., Jones, W., and Hancock, J. (1984). Epidemiological–environmental study of lead acid battery workers. II. Acute effects of sulfuric acid on the respiratory system. *Environ. Res.* **35**, 11–29.

Jones, W., and Gamble, J. (1984). Epidemiological study of lead acid battery workers. I. Environmental study of five lead acid battery plants. *Environ. Res.* **35**, 1–10.

Juhos, L. T., Evans, M. J., Mussenden-Harvey, R., Furiosi, N. J., Lapple, C. E., and Freeman, G. (1978). Limited exposure of rats to H_2SO_4 with and without O_3. *J. Environ. Sci. Health Part C* **13**, 33–47.

Lewis, T. R., Mooman, W. J., Ludman, W. F., and Campbell, K. I. (1976). Toxicity of long-term exposure to oxides of sulfur. *Arch. Environ. Health.* **26**, 16–21.

Malcolm, D., and Paul, E. (1961). Erosion of the teeth due to sulphuric acid in the battery industry. *Brit. J. Ind. Med.* **18**, 63–69.

Morando, A. (1956). Experimental and clinical contribution to human pathology due to sulfuric acid fumes. *Med. Lav.* **47**, 55–61.

Morgan, W. K. C. (1978). Industrial bronchitis. *Brit. J. Ind. Med.* **35**, 285–291.

NIOSH (1974). "Criteria for a Recommended Standard. Occupational Exposure to Sulfuric Acid." US DHEW/PHS/CDC/NIOSH, HEW Publication No. 74–128.

NIOSH (1981). "Review and Evaluation of Recent Literature. Occupational Exposure to Sulfuric Acid." US DHHS/PHS/CDC/NIOSH, DHHS Publ. No. 82–104.

Pattle, R. E., and Cullumbine, H. (1956). Toxicity of some atmospheric pollutants. *Brit. Med. J.* **2**, 913–916.

Pelnar, T. (1951). The influence of work in sulfuric acid production in employee health. *Prac. Lek.* **3**, 287–294.

Schlesinger, R. B., Halpern, M., Albert, R. E., and Lippmann, M. (1979). Effect of chronic inhalation of sulfuric acid mist upon mucociliary clearance from the lungs of donkeys. *J. Environ. Pathol. Toxicol.* **2**, 1351–1367.

Schwartz, L. W., Moore, P. F., Chang, D. P., Tarkington, B. K., Dungworth, D. L., and Tyler, W. S. (1977). Short-term effects of sulfuric acid aerosols on the respiratory tract. A morphological study in guinea pigs, mice, rats, and monkeys. *In* "Biochemical Effects of Environmental Pollutants" (S. D. Lee (Ed.), Chapt. 12. Ann Arbor Science Pub., Ann Arbor, Mich.

Ten Bruggen Cate, H. J. (1968). Dental erosion in industry. *Brit. J. Ind. Med.* **25**, 249–266.

Thomas, M. D., Hendricks, R. H., Gunn, F. D., and Critchlow, J. (1950). Prolonged exposure of guinea pigs to sulfuric acid aerosol. *Arch. Ind. Health.* **17**, 70–80.

Treon, J. F., Dutra, F. R., Cappel, J., Sigman, H., and Younker, W. (1950). Toxicity of sulfuric acid mist. *Ind. Hyg. Occup. Med.* **2**, 716–734.

Williams, M. K. (1970). Sickness absence and ventilatory capacity of workers exposed to sulphuric acid mist. *Brit. J. Ind. Med.* **27**, 61–66.

Wolff, R. F., Silbaugh, S. A., Brownstein, D. G., Carpenter, R. L., and Manderly, J. L. (1979). Toxicity of 0.4 and 0.8 μm sulfuric acid aerosols in the guinea pig. *J. Toxicol. Environ. Health.* **5**, 1037–1047.