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Testimony to DOL

Statement of
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Department of Labor

Occupational Safety and Health Administration
Public Hearing on Occupational Standard for
Sulfur Dioxide

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My name is Edward Baier, Deputy Director of the National Institute for Occupational Safety and Health (NIOSH). I welcome this opportunity to appear here today to discuss the effects of occupational exposure to sulfur dioxide upon human health, including the results of recent NIOSH sponsored studies. With me today are: Dr. Victor Archer, Division of Surveillance, Hazard Evaluations and Field Studies; Dr. Kenneth Bridbord, Office of Extramural Coordination and Special Projects; Dr. David Groth, Division of Biomedical and Behavioral Sciences; Dr. Douglas Smith, Division of Criteria Documentation and Standards Development; William Wagner, Environmental Investigations Branch; Patricia Gussey, Testing and Certification Branch; and Dr. Janet Haartz, Division of Physical Sciences and Engineering.

Records of worker complaints of the irritant effects of sulfur dioxide (SO_2) date from 1821. SO_2 is an irritant gas which has wide industrial use. It is commonly transported and stored as a liquid under pressure but is a gas at atmospheric pressure and room temperature. Exposures of less than an hour to SO_2 at levels above ten parts per million parts of air (10 ppm or 26 mg/m^3) are irritating to the nose and throat, sometimes causing a choking sensation followed by nasal discharge, sneezing, cough and increased mucus secretion. Acute effects have been thoroughly studied in both man and animals. The NIOSH Criteria Document has noted that some acute exposures have resulted in death, and others have been followed by chronic disease, such as chronic bronchitis, emphysema, and shortness of breath. Studies reported in the last three years have shown some chronic effects such as chronic bronchitis and loss of pulmonary function at chronic exposures below the current Federal occupational standard of 5 ppm (13 mg/m3) as a time weighted average (TWA) concentration.

It has been estimated by the Department of Labor that approximately 600,000 American workers may be occupationally exposed to SO_2. Some of the highest exposures occur when it is a by-product, as in the metal smelting industry, and in the processing or combustion of high sulfur coal or oil. Other exposures occur in manufacture of sulfuric acid, fumigating, food preservation, wine making and bleaching of many substances.

Important chronic respiratory diseases are emphysema, chronic bronchitis and pulmonary fibrosis. These are important causes of disability and death in the United States. These lung diseases cause over 14,000 disability retirements of persons under age 65 each year. They directly cause 30,000 deaths each year and contribute to an additional 32,000 deaths each year. The quality of life is markedly reduced for many thousands of persons by shortness of breath associated with chronic respiratory disease. Because of the magnitude of the problem of chronic respiratory disease, a small percentage increase resulting from SO_2 exposure may have a profound effect on the total number of people being affected.

Occupational exposure to sulfur dioxide was high on the original NIOSH priority list. A criteria document on SO_2 was transmitted to the Occupational Safety and Health Administration (OSHA) on February 11, 1974. This document recommended, among other things, that the Federal environmental limit for occupational exposure to SO_2 be set at 2 ppm (5.2 mg/m^3) as a

TWA exposure for a 40 hour work week, with daily exposures up to 10 hours. This proposed standard was based on information available at that time. The standard then was 5 ppm (13 mg/m^3). Because of recent data, NIOSH now believes that the standard of 2 ppm (5.2 mg/m^3) recommended in 1974 would not provide adequate protection for the health of workers.

Chronic respiratory disease associated with SO_2 exposure has been reported in several different epidemiological studies. Most of them have had severe limitations for use in standard setting, however. In spite of the problems encountered in epidemiologic studies of SO_2 effects, NIOSH is convinced that all available studies must be used to provide data for standard setting. Fortunately, the design and techniques used in epidemiology studies are improving.

In the last three years, four epidemiological studies have been reported which used better techniques and reported actual personnel exposures. The NIOSH study of Archer and Gilliam was a cross-sectional study of 903 workers in which statistically significant reductions in FVC and FEV_1 were found as well as an increase in symptoms of respiratory disease which correlated well with days off for illness. The effects of SO_2 were seen in both smokers and non-smokers. When workers smoked and were exposed to SO_2, the effects of the two agents were directly additive. Among smelter workers 18% compared to 6% of controls reported a sensation of chest tightness on their first day back on the job after several days off. A concomitant environmental study by Smith, Wagner, et al. provided evidence that TWA exposures had been in the range of 0.4 to 4 ppm (1-10 mg/m^3) with a mean of about 2 ppm for many years. Sulfate, manganese and total dust were approximately the same in the breathing zones of workers used as controls. Of these substances, only the sulfate was considered sufficiently abundant to have an effect on pulmonary function. Concentrations of the other contaminants were much below that which is known to have any physiological effect, with the possible exception of arsenic, a carginogen. Although the sulfate might have had some effect on pulmonary function, it was not considered to have influenced the significance of the study conclusions, because the controls in this study were exposed to an average of 0.09 mg/m^3 (a slightly higher level).

The second report was the NIOSH sponsored study of Smith, Peters, et al. in a longitudinal investigation of 113 workers using personal SO_2 dosimeters. They found a statistically significant increase in the annual FEV_1 and FVC mean decrements among workers whose SO_2 TWA exposures were between 1 and 4 ppm (2.6-10 mg/m3) when compared to those whose mean exposure was less than 1 ppm (2.6 mg/m^3). From an analysis of variance, using cross tabulations of FEV_1 decrements, respirable dust and SO_2 exposures, it was found that the increased annual decrement was associated only with SO_2, not with the dust. This indicated that copper, sulfates, sulfites and other elements in the dust were not involved in the increased decrement. A comparison of FEV_1 values between pre-shift and post-shift tests on the smelter workers found that 30% had a decline of 100 ml or more. This suggests that 30% of workers may have had an acute reaction to SO_2 at about the 2 ppm (7.2 mg/m^3) TWA range encountered in the smelter.

The third epidemiological study by the Ministry of Health in Toronto, Canada in 1974, reported statistically significant increases in respiratory disease and decreases in FVC and FEV_1 among copper smelter workers exposed for ten years or more. Average exposure to SO_2 was reported as 2.5 ppm (6.5 mg/m^3). Smoking was controlled in the analysis. However, possible pulmonary effects contributed by other air contaminants could not be ruled out.

The fourth study was done in the British steel industry by Lowe, et al. Approximately 10,000 workers were studied for chronic effects. Mean exposures to SO_2 were about 0.35 ppm (0.9 mg/m^3). No definite effects were found at this level.

In addition to investigations on workers, epidemiological studies have also indicated chronic respiratory disease in the general population to be associated with SO_2. This includes both asthmatic attacks and chronic bronchitis. Decreases in pulmonary function were associated with oxidant pollutants (e.g., ozone), reducing pollutants (e.g., SO_2) and with elevated temperatures. Lawther, Brooks, et al. measured ventilatory function daily in 4 normal subjects for 5 years and correlated changes with pollution measurements. Multiple correlation regression analysis indicated lowest values on cold polluted winter days in London. Variations in the SO_2 concentration explained the largest proportion of the variance observed. Although substances other than SO_2, such as nitrogen oxides and sulfates undoubtedly have had some influence on the respiratory effects of polluted city air in these studies, the fact that SO_2 has been pin-pointed as an important factor by multiple correlation techniques in a number of different studies is indicative that 24 hour continuous exposure to SO_2 at TWA levels below 0.5 ppm (1.3 mg/m^3) are probably injurious to the health of some individuals in the community.

Many experimental studies have been performed on humans to investigate the acute effects of SO_2. Since transient pulmonary changes resulting from short-term SO_2 exposures may be related to chronic respiratory disease, those reported in the last three years will be reviewed. Lawther, MacFarlane, et al. tested 25 healthy adults and found increased airway resistance (determined in a body plethysmograph) at 5 ppm (13 mg/m^3) of SO_2 and at higher levels when breathing normally for 10 minutes, but not at lower levels. After 25 deep breaths, as might occur in laborers doing hard physical work, the subjects had a statistically significant increase in airway resistance at 1 ppm and after 8 deep breaths at 3 ppm. Andersen, et al. reported reduced nasal mucus flow rates in 15 young men after inhaling 5 ppm (13 mg/m^3) SO_2 for 6 hours. Increased nasal cross-sectional area and reduced FEV_1 were noted at 1 and 5 ppm SO_2 after 6 hour exposures. Wolff, et al. found that after breathing 5 ppm (13 mg/m^3) SO_2 nine healthy non-smoking adults had a reduction in tracheobronchial clearance at one hour ($p = .05$) followed by a return to normal at 3 hours and a reduction in the maximal mid-expiratory flow rate (MMFR) ($P - .01$) at three hours. In earlier human experiments, some researchers reported transient changes in pulmonary function between 0.5 and 5 ppm (1.3-13 mg/m^3) of SO_2 whereas other workers failed to find any changes at those levels. When subjects breathed through the mouth greater effects were noted.

Experimental animal studies reported in the last 3 years have extended our knowledge of the biological effects of SO_2. Hirsch, et al, using 8 dogs exposed for 12 months at 1 ppm of SO_2, measured Teflon disk movement in bronchi by means of bronchofiberscope, and found statistically significant slowing of mucus transport. Ferin and Leach using a titanium oxide dust technique, found a slightly increased clearance rate when 0.1 ppm (.3 mg/m^3) SO_2 was administered to rats for 10 and 23 days, but a definitely decreased clearance rate with 1 ppm (2.6 mg/m^3). Increased airway resistance was noted in guinea pigs by McJilton and Frank when the animals were exposed at 1 ppm (2.6 mg/m^3) SO_2 in the presence of high humidity and a fine sodium chloride aerosol.

Sulfur dioxide is known to interact with commonly occurring substances in the atmosphere such as water vapor and fine particulates. Some of these interactions are chemical and others are physical in that they permit a higher percentage of SO_2 to reach the lower parts of the lung.

At relatively high levels of acute or chronic exposure to SO_2, a number of pathological changes have been observed in animals. Chakrin and Saunders noted an increase in goblet cells near the ends of bronchi and bronchioles, and hyperplasia of bronchial glands, with an excess of mucopurulent exudate in dogs. The animals had been exposed to 500-600 ppm (1300-1560 mg/m^3) for two-hour periods twice weekly for 4 to 5 months. The authors considered that they had produced chronic bronchitis in dogs. In further studies on the dogs, Spicer, et al. reported extensive hyperplasia and squamous metaphasia of bronchial mucosa, impaired mucociliary flow, polypoid protrusions into the bronchi and alveolar dilatation. Ida exposed mice continuously at 0.23 ppm (0.6 mg/m^3) for 3 months, and found enlargement and increase in the number of vesicles in both ciliated and nonciliated cells. They considered this as evidence of mucus hypersecretion.

The NIOSH criteria document reviewed the evidence relating SO_2 to cancer. There is now evidence that SO_2 and bisulfites are mutagenic for viruses, plants and bacteria. Warshawsky, et al. have found that SO_2 by promoting the metabolism of benzopyrene, increases its carcinogenicity. A cancer promoting action by SO_2 is suggested by the data.

In the data referred to above, it is apparent that both acute and chronic effects of SO_2 in man are observable in the 1 to 5 ppm (2.6-13 mg/m^3) range. The appearance of both types of effects at approximately the same levels is not likely to be coincidence. Indeed, acute effects in workers exposed to MDI and TDI (isocyanates used in making polyurethane foam) have been found to be followed by chronic effects (chronic bronchitis, etc.) similar to that observed for SO_2. The mechanisms for this twin effect are not clear. Perhaps the transient acute effect is merely an observable symptom of more permanent damage, or perhaps frequent bronchoconstriction impairs the self-cleansing ability of lungs so that infections resulting in chronic bronchitis are more likely.

The delayed particle clearance times which have been observed at low levels of SO_2 exposure also indicate an impairment of the lungs to cleanse themselves. This effect is more prominent with prolonged exposure to low concentrations than for short exposures to high concentrations as indicated by

the work of Ferin and Leach. The links between acute and chronic effects argue that chronic effects are just as likely to be due to chronic low level exposures as to intermittent peak exposures.

In considering an appropriate level for an SO_2 occupational standard, we must add to our consideration that health effects have been observed among workers and experimental subjects in the 1-2 ppm range, the fact that 10 to 20% of persons are especially susceptible to SO_2 effects, the possibility that synergistic effects with other aerosols or gases may occur, the possibility of increased fractions of SO_2 reaching the lower lungs through mouth breathing or because of rapid or deep breathing, the possibility that SO_2 may act as a cancer promoting agent, the possible enhancement of effects by high humidity, and the likelihood that degradation products (SO_2). sulfites and sulfates) will accompany SO_2. After considering all these factors, NIOSH recommends that the SO_2 occupational exposure limit be a time weighted average of 0.5 ppm (1.3 mg/m^3) for up to a 10-hour workday, 40-hour workweek. It is anticipated that adherence to this limit will confine excursions to about 2 or 3 ppm (3.2-7.8 mg/m^3).

Preplacement and annual medical examinations should be done whenever TWA exposures exceed 0.25 ppm (0.65 mg/m^3). These examinations should be directed toward complaints of mucus membrane irritation, cough and shortness of breath. They should ascertain that nasal passages are open. Persons with a history of asthma or with subnormal pulmonary function should be watched closely. Simple expiratory function tests should be a part of the examination. They are useful for several purposes: (a) determining whether or not a person is a suitable candidate for using respirators; (b) identifying "reactors,", i.e., persons who may be most susceptible to the effects of SO_2. This can be done by comparing preshift and postshift tests; (c) when done periodically, they can be used to determine whether or not a person's expiratory functions are declining at a faster than normal rate. Such determinations are much more sensitive when pooled data from a number of individuals are used. The forced expiratory volume at 1 second and the maximum mid-expiratory flow rate appear to be the most useful of the simple pulmonary function tests.

NIOSH is in agreement with the OSHA proposals for employee information, training and record keeping, with the exception that medical records should be maintained at least 30 years after an individual's employment ends. Medical representatives of the Department of Labor and of the Department of Health, Education and Welfare, of the employer, and of the employee or former employee shall have access to the medical records.

NIOSH is also in agreement with the OSHA proposals with respect to the effects of overtime on exposure, dermal and eye contact with liquid SO_2, determination and measurement of exposure and methods of compliance. NIOSH is in full agreement with OSHA that primary reliance for control should be on engineering and work practices. When temporary situations occur for which controls are inadequate, such as maintenance or process changes, respirators may be used. When respirators are used, they must be fitted properly, and an appropriate maintenance program for them should be conducted. In the past, it has been a general practice for workers to put on respirators only when they encountered, or expected to encounter, levels of SO_2 which would be irritating to the eyes, nose and throat. This practice should be discouraged,

as there is evidence that chronic lung injury occurs at levels below those which cause marked mucus membrane irritation. NIOSH recommendations on respirators to be used at various SO_2 concentrations is submitted for the record.

NIOSH agrees with the OSHA proposal on the hydrogen peroxide method of measuring SO_2. However, one change in the description is recommended for clarification in item (j) of the Analytical section in Appendix D. This clarified the standardization of barium perchlorate solution, with isopropanol, sulfuric acid solution and endpoint titration with Thorin as the indicator.

Since no industry has tried to control SO_2 concentrations to levels as low as 0.5 ppm (1.3 mg/m^3) there is no documentation as to its technical feasibility.

Before closing, I would like to list the backup material submitted for the record.

1. NIOSH recommendations on respirators to be used with sulfur dioxide.

2. Copies of the following scientific articles to which I have referred:

Andersen I, Lundquist GR, Jensen PL and Proctor DF: Human Response to Controlled Levels of SO_2. Arch. Environ. Health 28:31, 1974.

Archer VE and Gillam JD: Chronic Sulfur Dioxide-Exposure in a Smelter, I: Indices of Chest Disease. Submitted to J. Occup. Med., 1977.

Chakrin LW and Saunders LZ: Experimental Chronic Bronchitis: Pathology in the Dog. Lab Invest. 30:145, 1974.

Ferin J and Leach L: The Effect of SO_2 on Lung Clearance of TiO_2 in Particles in Rats. Am. Ind. Hyg. Assn. J. 34:260, 1973.

Hirsch JA, Swenson EW and Wanner A: Tracheal Mucus Transport in Beagles after Long Term Exposure to 1 ppm of SO_2. Arch. Environ. Health 30:249, 1975.

Ida H: Effects on Mouse Respiratory Organs with Prolonged Exposure to Low Concentrations of SO_2. II. Electron Microscopic Studies. Jpn. J. Thorac Dis. 13:443, 1975.

Lawther PJ, Brooks AGF, Lord PW and Waller RE: Day-to-Day Changes in Ventilatory Function in Relation to Environment, Part II. Peak Expiratory Flow Values. Environ. Res. 7:41, 1974.

Lawther PG, MacFarlane AJ, Waller RE, et al: Pulmonary Function and SO_2, Some Preliminary Findings. Environ. Res. 10:335, 1975.

Lowe CR, Campbell H and Khosla T: Bronchitis in Two Integrated Steel Works. III. Respiratory Symptoms and Ventilatory Capacity Related to Atmospheric Pollution. Brit. J. Industr. Med. 27:121, 1970.

McJilton C and Frank R: Role of Relative Humidity in the Synergistic Effect of a SO_2 Aerosol Mixture on the Lung. Science 182:503, 1973.

Ministry of Health: Chronic Obstructive Lung Disease Among Persons Employed for Ten Years and More in the Converter Plant of the International Nickel Co. of Canada, Copper Cliff, Ontario, Canada, March 1976.

Smith TJ, Peters JM, Rading JC and Castle CH: Pulmonary Impairment from Chronic Exposures to SO_2. Am. Rev. Resp. Dis., in Press, 1977.

Smith TJ, Wagner WL and Moore DE: Chronic Sulfur Dioxide Exposure in a Smelter. II: Exposure to SO_2 and Dust: 1940-1974. Submited to J. Occup. Med., 1977.

Spicer SS, Chakrin LW and Waddell JR: Effect of Chronic SO_2 Inhalation on the Carbohydrate Histochemistry and Histology of the Canine Respiratory Tract. Am. Rev. Resp. Dis. 110:13, 1974.

Warshawsky D, Niemeier R, and Bingham E: Influence of Particulate and SO_2 on Benzo(a)pyrene Metabolism. Presented at EPA Catalysts' Research Program's Sulfuric Acid Research Review Conference, Hendersonville, N.D., Jan 31-Feb 3, 1977.

Wolff RK, Dolovich M, Rossman CM and Newhouse MT: SO_2 and Tracheobronchial Clearance in Man. Arch. Environ. Health 30:521, 1975.