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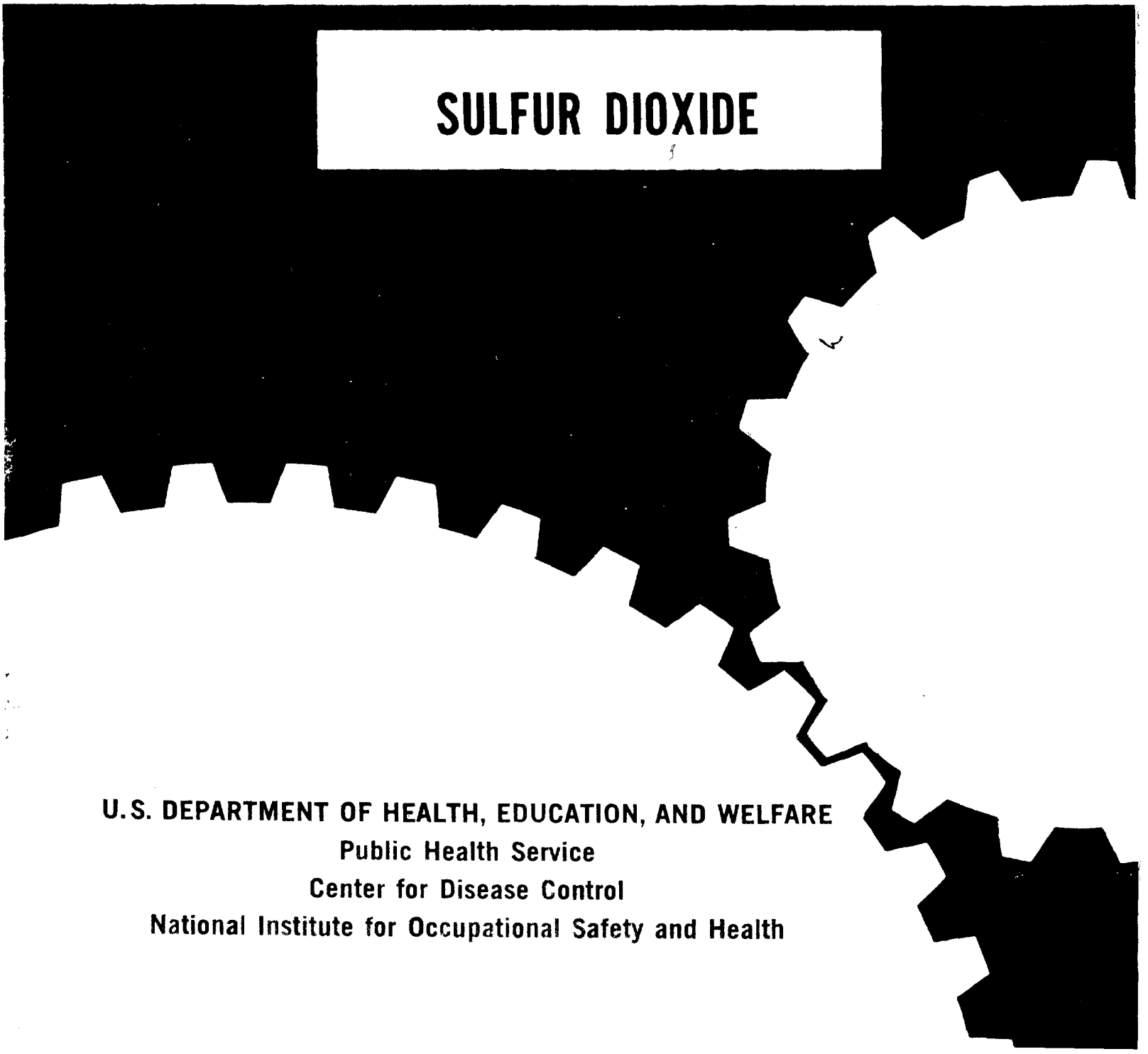

U.S. DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE
Public Health Service
Center for Disease Control
National Institute for Occupational Safety and Health

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OCCUPATIONAL EXPOSURE TO SULFUR DIOXIDE

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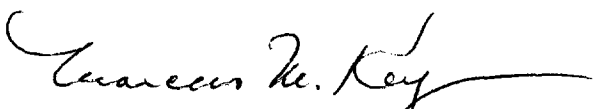
1974

PREFACE

The Occupational Safety and Health Act of 1970 emphasizes the need for standards to protect the health and safety of workers exposed to an ever-increasing number of potential hazards at their workplace. To provide relevant data from which valid criteria and effective standards can be deduced, the National Institute for Occupational Safety and Health has projected a formal system of research, with priorities determined on the basis of specified indices.

It is intended to present successive reports as research and epidemiologic studies are completed and sampling and analytical methods are developed. Criteria and standards will be reviewed periodically to ensure continuing protection of the worker.

I am pleased to acknowledge the contributions to this report on sulfur dioxide by members of my staff, the valuable and constructive comments presented by the Review Consultants on Sulfur Dioxide, the ad hoc committees of the American Academy of Occupational Medicine and the American Conference of Governmental Industrial Hygienists, by Robert B. O'Connor, M.D., NIOSH consultant in occupational medicine, and by William A. Burgess, NIOSH consultant on respiratory protection. The NIOSH recommendations for standards are not necessarily a consensus of all the consultants and professional societies that reviewed this criteria document on sulfur dioxide. Lists of the NIOSH Review Committee members and of the Review Consultants appear on the following pages.



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The Office of Research and Standards Development, National Institute for Occupational Safety and Health, had primary responsibility for development of the criteria and recommended standard for sulfur dioxide. Tabershaw-Cooper Associates, Inc., developed the basic information for consideration by NIOSH staff and consultants under contract No. HSM-99-72-116. Douglas L. Smith, Ph.D., served as criteria manager and had NIOSH program responsibility for development of the document.

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CRITERIA DOCUMENT: RECOMMENDATIONS FOR AN OCCUPATIONAL EXPOSURE STANDARD FOR SULFUR DIOXIDE

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I. RECOMMENDATIONS FOR A SULFUR DIOXIDE STANDARD

The National Institute for Occupational Safety and Health (NIOSH) recommends that worker exposure to sulfur dioxide (SO2) in the workplace be controlled by adherence to the following sections. The standard is designed to protect the health and safety of workers for an 8-hour day, 40-hour work week over a working lifetime; compliance with the standard should therefore prevent adverse effects of sulfur dioxide on the health and safety of workers. The standard is measurable by techniques that are valid, reproducible, and available to industry and government agencies. Sufficient technology exists to permit compliance with the recommended standard. The standard will be subject to review and will be revised as necessary.

"Exposure to sulfur dioxide" means exposure to a concentration of sulfur dioxide equal to or above one-half the recommended workroom environmental standard. Exposures at lower environmental concentrations will not require adherence to the following sections. Procedures for identification of exposure areas can be accomplished by time-weighted average (TWA) determinations by methods described in Appendices I and II or by any method shown to be equivalent in accuracy, precision, and sensitivity to the methods specified.

If "exposure" to other chemicals also occurs, for example from arsenic, then provisions of any applicable standard for the other chemicals shall also be followed.

Section 1 - Environmental (Workplace Air)

(a) Concentration

Occupational exposure to sulfur dioxide shall be controlled so that workers shall not be exposed to sulfur dioxide at a concentration greater than 2 parts per million parts of air (5 milligrams per cubic meter of air) determined as a time-weighted average exposure for an 8-hour work day.

(b) Sampling, Calibration, and Analysis

Procedures for sampling, calibration of equipment, and analysis of environmental samples shall be as provided in Appendix I or by any method shown to be equivalent in accuracy, precision, and sensitivity to the method specified.

Section 2 - Medical

(a) Comprehensive preplacement and annual medical examinations shall be provided for all workers subject to "exposure to sulfur dioxide." The examination shall be directed toward but not limited to the eyes and the cardiopulmonary system; particular attention shall be focused on complaints of mucous membrane irritation and cough. An evaluation of the advisability of a worker's using negative- or positive-pressure respirators shall also be made.

(b) Initial examinations for presently employed workers shall be offered within 6 months of the promulgation of a standard incorporating these recommendations and annually thereafter.

(c) The medical representatives of the Secretary of Health, Education, and Welfare, of the Secretary of Labor, and of the employer shall have access to all medical records. Physicians designated and authorized by any employee or former employee shall have access to his medical records.

(d) Medical records shall be maintained for persons employed one or more years in work involving exposure to sulfur dioxide. X-rays for the 5 years preceding termination of employment and all medical records with pertinent supporting documents shall be maintained at least 20 years after the individual's employment is terminated.

Section 3 - Labeling (Posting)

(a) Labeling

Cylinders and other containers of sulfur dioxide shall bear the following label in addition to or in combination with labels required by other statutes, regulations, or ordinances:

SULFUR DIOXIDE

Warning! Extremely irritating gas

and liquid under pressure.

Liquid causes burns.

Avoid breathing gas.

(b) Posting

The following warning sign shall be affixed in a readily visible location at or near entrances to areas in which there is occupational exposure to sulfur dioxide:

SULFUR DIOXIDE

Warning! Potential exposure to irritating gas.
Avoid unnecessary exposure to concentrations
producing irritation or coughing.

This warning sign shall be printed both in English and in the predominant primary language of non-English-speaking workers, if any.

Section 4 - Personal Protective Equipment and Work Clothing

Subsection (a) shall apply whenever a variance from the standard recommended in Section 1 is granted under provisions of the Occupational Safety and Health Act, or in the interim period during the application for a variance. When the limits of exposure to sulfur dioxide prescribed in subsection (a) of Section 1 cannot be met by controlling the concentration of sulfur dioxide in the work environment, an employer must utilize, as provided in subsection (a) of this Section, a program of respiratory protection to effect the required protection of every worker exposed.

(a) Respiratory Protection

Engineering controls shall be used wherever feasible to maintain sulfur dioxide concentrations below the prescribed limit. Appropriate respirators shall be provided and used when a variance has been granted to allow respirators as a means of control of exposure to routine operations and while the application is pending. Administrative controls should also be used to reduce exposure. Respirators shall also be provided and used for nonroutine operations (occasional brief exposures above the TWA of 2 ppm and for emergencies); however, for these instances a variance is not required but the requirements set forth below continue to apply. Appropriate respirators as described in Table I-1 shall only be used pursuant to the following requirements:

(1) For the purpose of determining the type of respirator to be used, the employer shall measure the atmospheric concentration of sulfur dioxide in the workplace when the initial application for variance is made and thereafter whenever process, worksite, climate, or control changes occur which are likely to increase the sulfur dioxide concentration. This requirement shall not apply when only atmosphere-supplying positive pressure respirators are used. The employer shall ensure that no worker is being exposed to sulfur dioxide in excess of the standard either because of improper respirator selection, fit, use, or maintenance.

(2) The respirator and cartridge or canister used shall be of the appropriate class, as determined on the basis of exposure to sulfur dioxide gas.

(3) A respiratory protective program meeting the general requirements outlined in Section 3.5 of American National Standard Practices for Respiratory Protection Z88.2-1969 shall be established and enforced by the employer. In addition, Sections 3.6 (Program Administration), 3.7 (Medical Limitations), and 3.8 (Approval) shall be adopted and enforced.

(4) The employer shall provide respirators in accordance with Table I-1 below and shall ensure that the employee uses the respirator provided.

(5) Respiratory protective devices described in Table I-1 shall be those approved under provisions of 30 CFR 11 published in the Federal Register, volume 37, page 6244, dated March 25, 1972.

(6) Respirators specified for use in higher concentrations of sulfur dioxide are permitted in atmospheres of lower concentrations.

(7) Employees shall be given instruction on the use of respirators assigned to them, cleaning of the respirators, and how to test for leakage.

(8) Wherever bulk sulfur dioxide is handled, emergency and escape-type respirators shall be made readily available for each worker.

TABLE I-1

REQUIREMENTS FOR RESPIRATOR USAGE

Multiples of TWA Limit	Respirator Type
Less than or equal to 10x	(1) Chemical cartridge respirator for sulfur dioxide with quarter, half, or full facepiece.
	(2) Type C supplied air respirator, demand type (negative pressure), with quarter or half mask facepiece.
Less than or equal to 100x	(1) Gas mask with chin style canister for acid gases.
	(2) Gas mask with front or back mounted chest type canister for acid gases.
	(3) Type C supplied air respirator, demand (negative pressure); pressure-demand; or continuous flow type with full facepiece.
	(4) Self-contained breathing apparatus in demand mode (negative pressure) with full facepiece.
Greater than 100x	(1) Self-contained breathing apparatus in pressure-demand mode (positive pressure).
	(2) Combination supplied air respirator, pressure-demand type, with auxiliary self-contained air supply.
Emergency (No concentration limit)	(1) Self-contained breathing apparatus in pressure-demand mode (positive pressure).
	(2) Combination supplied air respirator, pressure-demand type, with auxiliary self-contained air supply.
Evacuation or escape (No concentration limit)	(1) Self-contained breathing apparatus in demand or pressure-demand mode (negative or positive pressure).
	(2) Gas mask with acid gas chest canister, and mouthpiece respirator.

(b) Eye Protection

(1) The American National Standard Practice for Occupational and Educational Eye and Face Protection, ANSI Z87.1-1968, shall be employed.

(2) Chemical safety goggles-- cup-type or rubber-framed goggles, equipped with approved impact-resistant glass or plastic lenses, shall be worn whenever there is danger of eye contact, such as working with pipelines, valves, etc, which might leak and spurt liquid sulfur dioxide.

(3) Spectacle-type safety goggles-- metal or plastic rim safety spectacles with unperforated side shields, or suitable all-plastic safety goggles may be used where continuous eye protection is desirable. If use of this type of eye protection is mandatory, prescription lenses shall be provided for those employees who need them.

(4) Face shield-- plastic shields with forehead protection may be worn in place of or in addition to goggles.

(c) Work Clothing

(1) Work clothing should be changed at least twice a week or more frequently if required.

(2) Sulfur dioxide-wetted clothing, unless impervious, shall be removed promptly.

Section 5 - Apprisal of Employees of Hazards from Sulfur Dioxide

At the beginning of employment in a sulfur dioxide area, employees exposed to sulfur dioxide shall be informed of all hazards, relevant

symptoms of overexposure, appropriate emergency procedures, and proper conditions and precautions for safe use or exposure. Instruction shall include, as a minimum, all information in Appendix III which is applicable to sulfur dioxide. The information shall be posted in the work area and kept on file and readily accessible to the worker at all places of employment where sulfur dioxide is involved in unit processes and operations or is released as a product, byproduct, or contaminant.

A continuing educational program shall be instituted to ensure that all workers have current knowledge of job hazards, proper maintenance procedures and cleanup methods, and that they know how to correctly use respiratory protective equipment and protective clothing.

Information as required shall be recorded on US Department of Labor Form OSHA-20 "Material Safety Data Sheet" or a similar form approved by the Occupational Safety and Health Administration, US Department of Labor.

Section 6 - Work Practices

(a) Storage and Handling

(1) Because sulfur dioxide vaporizes at atmospheric pressure and temperature, it must be stored in gas tight containers under pressure and at temperatures which should not reach 54 C (130 F). Sulfur dioxide is not flammable and, when dry, is not corrosive to ordinary metals.

(2) Each container of sulfur dioxide shall be examined for leaks upon its arrival or upon filling and shall be reexamined periodically at least every 3 months.

(3) Prior to transferring sulfur dioxide from a storage container, an inspection shall be conducted to detect any gas leaks in the transport system (eg, cylinder seal with gas regulator, regulator apparatus, regulator seal with transport conduits, conduit system, etc).

(4) Cylinders of sulfur dioxide shall be secured so they cannot be damaged during transport or use.

(b) Emergency Procedures

(1) Procedures for emergencies shall be established to meet foreseeable events. The irritant and choking properties of sulfur dioxide provide warning of overexposure and evacuation from the area should begin as soon as possible.

(2) Appropriate respirators shall be available for wear during evacuation.

(3) Where there is the possibility of sulfur dioxide contact on the eyes or skin, drench-type showers, eye-wash fountains, and cleansing facilities should be installed and maintained to provide prompt, immediate access by the workers.

(c) Exhaust Systems and Enclosure

Exhaust ventilation and enclosure processes shall be used wherever practicable to control workplace concentrations. Systems shall be designed and maintained to prevent the accumulation or recirculation of sulfur

dioxide into the workroom. In addition, necessary measures shall be taken to ensure that discharge outdoors will not produce a health hazard to humans, animals, or plants.

(d) General Housekeeping

Emphasis shall be placed upon cleanup, inspection and repair of equipment and leaks, and proper storage of materials.

Section 7 - Monitoring and Reporting Requirements

Workroom areas where it has been determined, on the basis of an industrial hygiene survey or the judgment of a compliance officer, that environmental levels do not exceed one-half the environmental standard shall not be considered to have sulfur dioxide exposure. Records of these surveys, including the basis for concluding that air levels are below one-half the environmental standard, shall be maintained until a new survey is conducted. Surveys shall be repeated when any process change indicates a need for reevaluation or at the discretion of the compliance officer. Requirements set forth below apply to areas in which there is sulfur dioxide exposure.

Employers shall maintain records of environmental exposures to sulfur dioxide based upon the following sampling and recording schedules:

(a) In all monitoring, samples representative of the exposure in the breathing zone of employees shall be collected. An adequate number of samples shall be collected to permit construction of a time-weighted average (TWA) exposure for every operation or process. The minimum number

of representative TWA determinations for an operation or process shall be based on the number of workers exposed as provided in Table I-2.

(b) The first environmental sampling shall be completed within 6 months of the promulgation of a standard incorporating these recommendations.

(c) Environmental samples shall be taken within 30 days after installation of a new process or process changes.

(d) Samples shall be collected at least quarterly in accordance with Appendix I for the evaluation of the work environment with respect to the recommended standard.

(e) Environmental monitoring of an operation or process shall be repeated at 15-day intervals when sulfur dioxide concentration has been found to exceed the recommended environmental standard. In such cases suitable controls shall be initiated and monitoring shall continue at 15-day intervals until two consecutive surveys indicate the adequacy of these controls.

(f) Records of all sampling and of medical examinations shall be maintained for at least 20 years after the individual's employment is terminated. Records shall indicate the type of personal protection devices, if any, in use at the time of sampling. Records shall be maintained so that they can be classified by employee. Each employee shall be able to obtain information on his own environmental exposure.

TABLE I-2

SAMPLING SCHEDULE

Number of Employees Exposed	Number of TWA Determinations
1-20	50% of the total number of workers
21-100	10 plus 25% of the excess over 20 workers
over 100	30 plus 5% of the excess over 100 workers

II. INTRODUCTION

This report presents the criteria and the recommended standard based thereon which were prepared to meet the need for preventing occupational diseases arising from exposure to sulfur dioxide. The criteria document fulfills the responsibility of the Secretary of Health, Education, and Welfare, under Section 20(a)(3) of the Occupational Safety and Health Act of 1970 to "...develop criteria dealing with toxic materials and harmful physical agents and substances which will describe... exposure levels at which no employee will suffer impaired health or functional capacities or diminished life expectancy as a result of his work experience."

The National Institute for Occupational Safety and Health (NIOSH), after a review of data and consultations with others, formalized a system for the development of criteria upon which standards can be established to protect the health of workers from exposure to hazardous chemical and physical agents. It should be pointed out that any recommended criteria for a standard should enable management and labor to develop better engineering controls resulting in more healthful work practices and should not be used as a final goal.

These criteria for a standard for sulfur dioxide are part of a continuing series of criteria developed by NIOSH. The proposed standard applies only to the processing, manufacture, and use of sulfur dioxide, or its release as an intermediate, byproduct, or impurity therefrom as applicable under the Occupational Safety and Health Act of 1970.

These criteria were developed to ensure that the standard based thereon would (1) protect against development of acute and chronic sulfur dioxide poisoning, (2) be measurable by techniques that are valid, reproducible, and available to industry and governmental agencies, and (3) be attainable with existing technology.

Sulfur dioxide is a rather common hazard of the workplace and an important component of the community air pollution problem. It is a primary constituent of certain processes and may enter the working environment either as a byproduct or as an impurity in a fuel or some raw material being processed.

These criteria were not designed for the population-at-large and any extrapolation beyond general occupational exposures is not warranted.

III. BIOLOGIC EFFECTS OF EXPOSURE

Extent of Exposure

Sulfur dioxide is a colorless, irritant gas having a characteristic odor and taste. Its more important physical and chemical properties are presented in Table XI-1. [1,2] Potential occupational exposures are listed in Table XI-2. [3]

Sulfur dioxide has a number of important industrial uses. [4] It is used in many chemical processes including the manufacture of sodium sulfite, and as an intermediate in the manufacture of sulfuric acid. It is also used in refrigeration, bleaching, fumigating, and preserving operations, and as an antioxidant in the melting, pouring, and heat treatment of magnesium. Breathing-zone concentrations of sulfur dioxide in some magnesium foundries have reached concentrations in excess of 50 ppm. [4]

Exposures to sulfur dioxide are not limited to operations where it is used. It is generated as a byproduct from many industrial processes, including the smelting of sulfide ores, the combustion of coal or fuel oils containing sulfur as an impurity, paper manufacturing, and petroleum refining. [4]

NIOSH estimates that 500,000 persons in the work force could have potential exposure to sulfur dioxide.

Historical Reports

Comparatively few early historical reports are available of poisoning by sulfur dioxide. The first report of lasting harmful effects due to sulfur dioxide alone came from France in 1821. [5] There were many complaints of the irritant effect of the gas upon workers employing sulfur dioxide in the bleaching of textiles. There was a report from Germany in 1853 on the exposure of workers to sulfur dioxide during the process of drying sugar beets. [6] The gas was reported to cause pneumonia, gastritis, enteritis, and even vaginitis.

In 1893, the first measurements of occupational environmental concentrations of sulfur dioxide and its effects were reported by Lehmann from Germany. [7] Certain operations in the bisulfite papermaking industry contained from 6-30 ppm sulfur dioxide and the workers, alleged to have the appearance of good health, ignored its effects. However, the author [7] and his two assistants, unaccustomed to sulfur dioxide, reportedly experienced nasal irritation after 10 minutes exposure to 6.5 and 11.5 ppm and found 30-57 ppm decidedly disagreeable. It has since been shown that acclimatization to the subjective effects of sulfur dioxide does occur. [8,9]

In 1930, Rostoski and Crecelius [10] reported on the acute effects of overexposure to sulfur dioxide and probably other products of wood pulp bisulfite digestion following the explosion of a digester vessel. Of the 18 workers involved in the accident, one died 10 months and another 15

months later from intercurrent pulmonary infection. Three years later, 8 others were still incapacitated by radiologically confirmed chronic bronchitis and emphysema. Those who returned to work complained of dyspnea and bronchial catarrh. Greenwald [11] in 1954 believed that the serious results of this accident were not due to sulfur dioxide but to the wood and its products. Greenwald's 1954 report [11] represents an excellent review of the effects of sulfur dioxide inhalation up to that time.

Effects on Humans

The rapidity with which sulfur dioxide forms sulfurous acid on contact with moist mucous membranes explains its prominent biologic effect in man and animals, ie, severe irritation. The sulfur dioxide molecule itself is chemically reactive, but as all biologic systems function in an aqueous milieu, it is doubtful whether sulfur dioxide as such can exist in significant concentrations within living organisms. Sulfur dioxide is most likely absorbed as sulfurous acid or one of its ionization products and may undergo further biotransformation reactions in the body. The ultimate fate of practically all absorbed sulfur dioxide is apparently oxidation to sulfate ion, to be excreted principally as inorganic sulfate in the urine. [12]

(a) Occupational Exposures

(1) Acute Effects

Sulfur dioxide concentrations above 20 ppm have a marked irritant, choking and sneezing effect. [7,11] Acute exposure to

concentrations of about 50 ppm will promptly cause irritation of the nose and throat, rhinorrhea, and cough. These symptoms are sufficiently disagreeable that most persons would not tolerate them for more than 15 minutes. [11] Such exposure will cause reflex bronchoconstriction and possibly some increase in bronchial mucous secretion with increased pulmonary resistance to air flow. [13] These changes may be clinically manifested by high-pitched rales, and by a tendency to prolongation of the expiratory phase of respiration. [13]

If workers are exposed to catastrophic amounts of sulfur dioxide in a confined space, asphyxia will most probably result. If exposure is insufficient to cause death by asphyxia, a chemical bronchopneumonia with bronchiolitis obliterans may develop, which may be fatal after an interval of some days. Such a case was reported by Galea in 1964 [14] from an incident in a paper-pulp plant in Canada where a worker was exposed to a high, but unmeasured, concentration of sulfur dioxide for from 15-20 minutes and died 17 days later.

Romanoff [15] in 1939 reported the development of typical signs of bronchial asthma following acute exposures to unknown concentrations of sulfur dioxide. One man had frequently been exposed over a 10-year period to low concentrations of the gas in the course of his work. Following an unusually large exposure to leaking sulfur dioxide, the man developed asthma-like attacks which required hospitalization. It was suggested that he had become sensitized to the bacteria which had established a

suppurative bronchitis secondary to the inflammatory effects of the sulfur dioxide.

Sulfur dioxide gas is irritating to the eyes, producing burning discomfort and lacrimation, but actual injury from industrial exposure is rare. However, liquid sulfur dioxide from pressurized containers can produce severe burns to the cornea of the eye which may be deceptively painless for the first few hours or even days. The increased severity is due to the high concentration and is aggravated by the freezing effect of the rapidly evaporating liquid. Over the course of weeks or months, the cornea may become infiltrated and densely vascularized resulting in opacification and severe loss of vision. Only in the mildest cases would the initial corneal cloudiness be expected to clear completely. [16]

(2) Chronic Effects

Chronic exposure to sulfur dioxide is extremely widespread in industry, [4] with problems occurring in smelting operations, [17] paper manufacture, [18,19] and formerly in refrigerator production. [8] The most meaningful exposure-effect information is found in occupational epidemiologic reports and nonoccupational experimental studies; therefore, the presentation of epidemiologic findings at this point is considered desirable to best develop the subject.

(A) Epidemiologic Studies

In most industrial situations, exposures have occurred to a mixture of sulfur dioxide with some sulfuric acid aerosol, metallic oxides, or other gases or particulate matter. [18,19] In contrast, exposures to

relatively pure sulfur dioxide gas arising from the evaporation of liquid sulfur dioxide used as a refrigerant were reported in an epidemiologic study in 1932 by Kehoe et al. [8] The study included 100 men having a mean duration of employment exposure of 3.8 years (47 employees had from 4-12 years employment exposure) to atmospheric concentrations averaging 20-30 ppm (range 5-70 ppm) at the time of the study. Prior to 1927, the sulfur dioxide levels had been much higher, averaging 80-100 ppm. A control group of 100 men, age-matched with the exposed group, was selected from parts of the same plant where there was no known exposure to sulfur dioxide or to other known noxious gases, fumes, or dust. Each of the 200 subjects was questioned in detail as to the length and nature of his exposure to sulfur dioxide. In addition, urinalyses and chest roentgenograms were obtained.

The symptoms associated with exposure to sulfur dioxide were classified as: 1. initial symptoms, that is, those which developed during the period before acclimatization (discussed below); 2. symptoms arising from customary exposure with or without acclimatization; and 3. symptoms produced by heavy exposure. Initial symptoms were confined to the respiratory tract and consisted, in descending order of frequency of occurrence, of irritation to the upper respiratory tract, coughing, epistaxis, constriction in the chest, and hemoptysis. Symptoms associated with customary exposures were, in descending order of frequency, hacking cough, morning cough, nasal irritation and discharge, prolongation of common colds, and expectoration. The severity of these symptoms seemed to be related to individual variation; however, all subjects showed some

symptomatic evidence of irritation of the upper respiratory tract. Symptoms associated with severe exposure were chiefly an intensified form of those occasioned by the original customary exposure.

A statistically significant higher incidence of nasopharyngitis, alteration in the senses of smell and taste, and increased sensitivity to other irritants was elicited from the exposed group as compared with the controls. A significantly higher incidence of tendency to increased fatigue, of dyspnea on exertion, and longer duration of colds (although their frequency was no greater) was also noted. The acidity of the urine to methyl red was prominent in the exposed group. There was no significant difference in the incidence of chest roentgenographic abnormalities between the two groups. Slightly more than 4% of each group had "definite chest pathology." Acclimatization occurred in 80% of the exposed group. The mean length of time necessary for acclimatization was calculated to be 2.84 months (S.D. = 2 months). The high standard deviation emphasized the great variability in the time required for acclimatization to take place. Acclimatization was considered to be the acquired ability to withstand the customary basic exposure without experiencing a notable intensity of initial symptoms. Acclimatization is further discussed under Experimental Studies. It is of interest that 20% of the exposed group failed to become acclimatized to exposure, but, according to the report, nevertheless continued to work and to be exposed to sulfur dioxide. The authors believed that the human organism has a high degree of adaptability to a regular moderate exposure (presumably 20-30 ppm) of sulfur dioxide and that

it suffers no apparent injury from such an exposure. In the case of intense exposures, even though they occurred frequently, there was believed to be no evidence of damage of a serious or a permanent type.

Anderson [9] in 1950 reported on the effects of sulfur dioxide exposure in approximately 135 Iranian oil refinery workers. Usual exposures in the refining and special products areas were estimated at between 0-25 ppm. However, even though the buildings were open on all sides affording good ventilation in the warm climate, exposures varying between 60 and 100 ppm had been recorded during times when plant maintenance was relatively low. No significant differences were reported between exposed workers and reportedly nonexposed controls in weight, systolic blood pressure, or chest roentgenographic findings. An unexplained difference was reported in the mean vital capacity of exposed workers vs controls in the refining area; however, no differences were noted between exposed workers and controls in the special products area. The author claimed no evidence of adverse effects could be found as a result of the study although no mention was made of any incidence of pulmonary irritation, coughing, nasal irritation, etc, which are associated with sulfur dioxide concentrations at the exposure levels encountered.

Skalpe [18] in 1964 reported a study of sulfur dioxide exposure in 54 workers in 4 different paper-pulp mills in Norway. In addition, 56 nonexposed controls were studied from the same industry and districts. The study was stimulated by the fact that pulp mill workers very often complained of chronic cough; therefore, an attempt was made to determine

whether there was a higher incidence of respiratory disease in the pulp mill workers than in a comparable unexposed control group. Environmental measurements were taken with detector tubes at different times and sites at the 4 different pulp mills on a single day. Sulfur dioxide concentrations ranged from 2-36 ppm and were considered to represent general working conditions in the acid tower and digester plant of the 4 pulp mills. Special working procedures occurred, such as "blowing the digesters," for which concentrations up to 100 ppm resulted, lasting only a few minutes but during which, pulmonary irritation was so intense that gas masks had to be used. It was emphasized that workers had much heavier exposure than was indicated by the analyses. The mean durations of employment exposure were 6.8 years for the subjects under 50 years of age, and 20.3 years for those over 50 years. All subjects were questioned to determine the incidence of cough, sputum, dyspnea, and cigarette smoking habits.

A significantly higher frequency of cough, expectoration, and dyspnea on exertion was found in the exposed group, the difference from controls being 4 to 5 times the standard error in the age groups under 50 years and 2 times the standard error in the over-50-year groups. The average maximal expiratory flow rate was significantly lower in the exposed groups than in the control groups for men under 50 years of age. Beyond 50 years of age, there was no significant difference between the exposed and control groups. Vital capacity values showed no differences between exposed and control groups regardless of age. Cigarette smoking did not appear to have any significant influence.

It was surprising that the high frequency of symptoms of respiratory disease was the greatest in the age groups under 50 where employment exposure time had been shortest. According to the author, [18] the most likely explanation was that because respiratory disease was rare in the younger age groups, the effect of small external insults was easier to detect than in the older age groups where respiratory disease from other causes was more common and small additions would be less noticeable.

In 1967, Ferris et al [19] presented results on the incidence of chronic respiratory disease in 147 pulp mill workers together with 124 workers from a neighboring paper mill who served as controls. The exposed group from the pulp mill complex included workers from 3 separate subplants-- a Kraft mill, a sulfite mill, and a chlorine plant; therefore, exposures resulted from sulfur dioxide, chlorine, chlorine dioxide, hydrogen sulfide, and some organic sulfides including mercaptans. At the time of this study, only traces of chlorine and hydrogen sulfide were found although chlorine levels had been high in prior years (mean = 7.4 ppm, range 0 - 64 ppm). Mean concentrations of sulfur dioxide taken on 3 separate days on each of 3 prior years were 13.2, 4.05, and 2.06 ppm. Although not specified by the authors, it seems apparent from the type of operations involved, that, similar to Skalpe's report, [18] these concentrations represented general working conditions. Special procedures most likely occurred which resulted in exposure concentrations in excess of those reported. Ferris et al [19] found no statistical differences in the rates of chronic bronchitis and other respiratory diseases between the pulp

mill exposed workers and the paper mill controls, the prevalence of chronic nonspecific respiratory diseases being 32.5% and 27.4% for the pulp mill and paper mill groups, respectively. Interestingly, the incidence of respiratory disease found in both groups (approximately 30%) indicates that chronic respiratory disease was a problem and that the paper mill workers did not represent a satisfactory control group. This was substantiated by the authors, [19] since, during the course of the study, it became apparent that many of the men currently working in the paper mill had, in fact, been previously employed in the pulp mill. In many cases they had transferred from the pulp to the paper operation because they found the odors in the pulp plant to be so disagreeable. Also, wage scales were slightly higher on the paper machines so that a considerable amount of self-selection had taken place. A rather complicated comparison was also presented between pulp and paper mill workers and a local general male town population based on the incidence and type of smoking habits.

(B) Carcinogenic Studies

Lee and Fraumeni, [17] reporting in 1969 on an excess in total mortality among arsenic exposed smelter workers, found as much as an 8-fold excess in instances of respiratory cancer as compared with that of the white male population of the same states. Their findings supported the hypothesis that inhaled arsenic is a respiratory carcinogen in man. At the same time, they showed a gradient in proportion to the degree of exposure to sulfur dioxide as well as the arsenic. Therefore, the influence of sulfur dioxide or unidentified chemicals, varying

concomitantly with arsenic exposure, could not be discounted. The study reported the mortality experience due mainly to malignant neoplasms of the respiratory system and diseases of the heart of 8,047 white male smelter workers during 1938 to 1963. Work areas were rated on a scale with respect to the level of sulfur dioxide exposure and members were classified in one of three exposure groups, that is, heavy, medium, or light work exposure areas. In general, the heavy sulfur dioxide exposure areas coincided with the medium arsenic exposure areas and the medium sulfur dioxide areas coincided with the heavy arsenic areas. Sulfur dioxide exposure and respiratory cancer mortality were positively correlated, with observed deaths ranging from 2 1/2 to 6 times expected in the light, medium, and heavy exposure groups (Table XI-3). Investigations revealed that persons with heavy exposure to arsenic and moderate or heavy exposure to sulfur dioxide were most likely to die of respiratory cancer. The overall excess of respiratory cancer could not be explained on the basis of other factors such as socioeconomic status, genetic susceptibility, availability of medical care, accuracy of death certificates, and urbanization. Furthermore, although smoking histories were not available for persons in the study, it was deemed highly unlikely that smoking alone would account for the excess respiratory cancer mortality observed. There was no reason to believe there was a positive relationship between amounts smoked and degree of arsenic and sulfur dioxide exposure in the smelters. Although no studies implicate sulfur dioxide as a carcinogen in man, it was postulated that perhaps sulfur dioxide or other chemicals in the work environment

possibly enhanced the suspected carcinogenic effect of arsenic or other unknown substances.

Two animal studies [20,21] have associated sulfur dioxide exposure with the incidence of bronchogenic carcinoma in conjunction with known carcinogens or animal strains having a high spontaneous incidence of lung carcinoma. The studies are discussed in the section under Animal Toxicity.

(C) Skin Hypersusceptibility

The incidence of skin reactions resulting from prolonged exposures to sulfur dioxide have been reported by Pirila in 1954 [22] and 1963. [23] The first report [22] involved a case of urticaria in a man working outdoors in a sulfate spirit mill where hot waste liquor was emptied into a reservoir several times daily. At such a time, the patient was exposed to the gases and, when using a gas mask, no skin reaction resulted; however, without the gas mask the skin eruptions occurred. When the patient was placed in a chamber and exposed to 40 ppm sulfur dioxide for 1 hour, the urticaria reappeared. In the second report, [23] a skin eruption resembling that resulting from a drug hypersensitivity occurred in a man working in an old building demolishing refrigerator machinery. Sulfur dioxide occasionally burst out in sufficient concentrations to cause him to evacuate the area. Three days after such an incident, the patient observed an eruption on his forearms which, during the following 5 days, spread to all the extremities and trunk. In addition, swelling of the eyelids resulted. No drugs had been used for 1 week prior to the onset of the eruption. Following topical treatment and oral antihistamines,

regression began and had entirely disappeared after 4 weeks. Later, the patient was exposed in a chamber to 10 ppm sulfur dioxide for 30 minutes. On the following day, lesions again appeared but were weaker than had been previously experienced. The eruption disappeared the following night. Another chamber exposure to 40 ppm sulfur dioxide for 10 minutes was given, after which the patient was permitted to breathe fresh air for an unspecified period and then returned to the chamber for another 10 minutes. On the following day, an eruption again developed which was more severe than to the 10 ppm exposure. Regression of the lesions followed in approximately 2 days. It thus seems that these 2 reported cases were due to a systemic allergic reaction. In the case of allergic individuals, it is extremely difficult to calculate a critical exposure concentration. The subject of sulfur dioxide-related hypersusceptibility is further discussed under Experimental Studies below.

Bronchial asthma has been reported by Romanoff [15] associated with chronic intermittent exposure to sulfur dioxide in the refrigeration industry. The affected individuals also had a predisposition to allergy.

(b) Experimental Studies

Many human experimental studies have been conducted in the past 2 decades concerning the effects of exposure to sulfur dioxide alone or in combination with aerosols of both soluble and insoluble particulates. Although the interest of most researchers has been with sulfur dioxide in the context of community air pollution, the experimental exposure levels have usually been in the range of industrial exposure levels. Most of the

effects studied have involved various aspects of respiratory mechanics, all related to pulmonary flow resistance. Unless otherwise stated, all the following experiments were performed on subjects not occupationally exposed to sulfur dioxide.

(1) Studies on Respiratory Mechanics

Sim and Pattle [13] in 1957 exposed healthy male volunteers to a wide range of sulfur dioxide concentrations either by facemask or by placing the subjects in an exposure chamber. The exposure levels were expressed as mg-minutes/cu m; however, by converting these to ppm for a 10-minute exposure (conducted with the facemask) and a 60-minute exposure (conducted in the chamber), results were as follows: at exposures above 50 ppm for 10 minutes or 9 ppm for 60 minutes (1330 mg-min/cu m), 50% of the subjects experienced an increase in airway resistance of more than 20% above normal accompanied with rhinorrhea and lacrimation. High pitched rales were noted over the larger bronchi for the 10-minute exposures and moist rales occurred over the lung periphery at the 60-minute exposures. At exposures to 30 ppm for 10 minutes or 5 ppm for 60 minutes (800 mg-min/cu m), little change was noted clinically or in lung resistance to air flow.

Several investigators have exposed subjects to sulfur dioxide concentrations at 5 ppm.

Frank et al [24] in 1964 reported an average 39% increase in pulmonary flow resistance above control levels within 10 minutes of exposure to 5 ppm sulfur dioxide in 11 men. Rates of recovery to baseline

varied after cessation of exposure but the group still showed residual effects after 15 minutes.

Nadel et al [25] in 1965 found that inhalation of 4-6 ppm sulfur dioxide for 10 minutes in 7 healthy subjects caused an increase in airway resistance. This effect was completely prevented by prior subcutaneous injection of atropine, suggesting a reflex bronchoconstrictive effect.

Snell and Luchsinger [26] in 1969 found a statistically significant decrease in maximum expiratory flow from the level of one-half vital capacity in 9 men exposed to 5 ppm.

Melville [27] in 1970 reported on changes in specific airway conductance of 49 healthy volunteers exposed to 5 ppm sulfur dioxide (also to 2.5 and 10 ppm) for 1 hour. An observed decrease in specific airway conductance was more pronounced with mouth breathing than with nose breathing at the 2.5 and 5 ppm exposure levels. At 10 ppm, there was no significant difference between the decrease in specific airway conductance for nose and mouth breathing. At 5 ppm, there was no further decrease in specific airway conductance after the first 5 minutes of exposure. According to the author, these experiments suggested that at sulfur dioxide levels up to 5 ppm, the nasal passages effectively absorb some of the inhaled sulfur dioxide and thereby diminish the stimulation of sensitive receptors in the larynx, trachea, and bronchi. Since continued exposure to sulfur dioxide resulted in no significant change in specific airway conductance after 5 minutes, a response was suggested aimed at maintaining an optimal compromise between airway diameter and work of breathing.

The following studies have measured exposure responses to sulfur dioxide concentrations at 1 ppm.

Amdur et al [28] in 1953 showed an increase in respiratory rate of 3-4 breaths/minute, an increase in pulse rate of 8-9 beats/minute, and a decrease in tidal volume of about 25% below control levels during the first 2 minutes of an 11-minute exposure to 1 ppm sulfur dioxide in 4 healthy adult men. During the remainder of the exposure period, the tidal volume increased again but stabilized at about 15% below control values. Subsequent studies by others [13,29] have failed to confirm these findings at the 1-ppm level.

Frank et al [29] in 1962 reported no detectable change in pulmonary flow resistance or peak flow rate in 10 out of 11 healthy male adults. The one subject who did show a response consistently had the highest preexposure control values of the group for pulmonary flow resistance. He had no history of respiratory illness and was a moderate smoker.

Snell and Luchsinger [26] in 1969 reported a small but statistically significant decrease in maximum expiratory flow from the level of one-half vital capacity for a group of 9 physicians and technicians.

Burton et al [30] in 1969 failed to find any immediate physiologic effect on pulmonary flow resistance to sulfur dioxide levels averaging 2.1 ppm ±0.19 (range 1.2-3.2 ppm) in 10 healthy male volunteers, half of them smokers.

Weir et al [31,32] exposed 4 groups of 3 healthy young adult males continuously for 120 hours to low levels of sulfur dioxide. At levels of

0.3 ppm and 1 ppm sulfur dioxide, no dose-related changes were observed in subjective complaints, clinical evaluation, or pulmonary function measurements. At 3.0 ppm, there was evidence of significant but minimal reversible decreases in small airway conductance and compliance.

(2) Hypersusceptibility

Studies have detected the presence of susceptible individuals who appear to overreact to concentrations of sulfur dioxide which, in most persons, elicit much milder responses. [13,29,30,33,34] Burton et al [30] in 1969 estimated that such "hyperreactors" may occur in 10-20% of the healthy young adult population. The hyperreactive responses occur with single exposures to sulfur dioxide. Apparently many such persons voluntarily transfer or remove themselves from surroundings involving sulfur dioxide exposure as was indicated in the study by Ferris et al.[19] This may be extremely difficult or virtually impossible for some individuals for various socioeconomic reasons. The mechanism of this hyperreactivity is unknown.

(3) Acclimatization

Acclimatization refers to the physiological adjustment exhibited by an individual to environmental changes, in this case to changes produced by sulfur dioxide. Such an adjustment to the environmental stimulus does not necessarily imply a beneficial effect even though the stimulus may become less objectionable to the individual upon continuous or repeated exposure.

Several studies have shown evidence of rather rapid physiological compensation to the effects of sulfur dioxide, especially on respiratory mechanics. [8,28,29,33] Kehoe et al [8] reported that acclimatization occurred in 80% of the sulfur dioxide-exposed group studied. The specifics of the study have been discussed under Epidemiologic Studies.

Amdur et al [28] in 1953 reported that 2 men who customarily worked in atmospheres containing 10 ppm sulfur dioxide or more showed no changes in respiration rate, tidal volume, or pulse rate to 5 ppm exposures.

Frank et al [29] showed that an initial coughing and sense of irritation in the throat and chest to 5 ppm and 13 ppm of sulfur dioxide tended to subside after 5 minutes, at a time when an increase in pulmonary flow resistance was maximal. The coughing and irritation presumably remained diminished for up to 30 minutes, the longest duration of exposure.

Acclimatization is considered to be mediated through depression of tracheobronchial nerve reflexes [27,29] along with a direct action of sulfur dioxide on bronchial smooth muscle as demonstrated in animals. [35-37] Whether mucosal secretion is an additional factor is not certain. It is questionable whether acclimatization to sulfur dioxide is desirable from a health standpoint in the occupational environment. Melville [27] emphasized the fact that although workers exposed to high sulfur dioxide concentrations showed no physical disability, it should not be accepted as proof that sulfur dioxide has no harmful effects, since a prolonged decrease in specific airway conductance might eventually compromise pulmonary function. Also, Haggard [38] in 1923 stated that the apparent

tolerance in workers exposed to sulfur dioxide was due to mucus in the upper air passages which acted as a protective coating. In his opinion, depression of the reflex merely removed one measure of protection.

(4) Interaction with Aerosols

The possible presence of sulfur dioxide-aerosol interaction in man and animals (see Animal Toxicity) has been investigated with conflicting results.

Frank et al [24] in 1964 reported changes in pulmonary flow resistance in 12 healthy male adults during exposure to 3 levels of sulfur dioxide: 1-2 ppm, 4-6 ppm, and 14-17 ppm alone, and then combined with 12-24 mg/cu m of sodium chloride aerosol having a geometric mean diameter of 0.15 micron. No evidence of augmentation was detected at any of the concentrations studied. Moreover, no statistically significant changes in pulmonary flow resistance occurred during exposure to 1-2 ppm sulfur dioxide, with or without added aerosol.

Snell and Luchsinger [26] in 1969 were unable to detect significant differences between 0.5, 1.0, and 5 ppm sulfur dioxide and either distilled water aerosol or normal saline aerosol, on expiratory flow rates and total respiratory resistance in 9 healthy young adults. The aerosol concentrations were not stated directly. Only particle (droplet) sizes in the range between 0.3 micron and 10.0 microns could be counted with the aerosol photometer being used.

Burton et al [30] in 1969 exposed 10 young healthy male adult subjects, half of them cigarette smokers, to sulfur dioxide concentrations

alone from 1.2-3.0 ppm and then combined with sodium chloride aerosol (0.25-micron mean diameter) at concentrations ranging from 2.0-2.7 mg/cu m. Pulmonary flow resistance and airway resistance were measured. No significant effects were noted on pulmonary flow resistance with sulfur dioxide alone or mixed with the sodium chloride aerosol.

In contrast, Toyama [39] in 1962 reported evidence of synergism between sulfur dioxide in a wide range of concentrations (1.6-56.0 ppm) and 7.4 mg/cu m sodium chloride aerosol (0.22 micron mean diameter) in 13 healthy male adults as measured by pulmonary flow resistance. Inhalation for 5 minutes to sodium chloride aerosol alone produced no differences from prior control values in any of the subjects. Five-minute inhalation of sulfur dioxide, 30 minutes after the aerosol exposures, produced changes in pulmonary flow resistance which varied according to the concentration of sulfur dioxide employed. Concentrations from 1.6-5 ppm consistently showed about 5% increase in pulmonary flow resistance; thereafter, values increased regularly for increased sulfur dioxide concentrations. For example, an approximate 10-ppm sulfur dioxide concentration resulted in a 10% increase in pulmonary flow resistance, 30 ppm sulfur dioxide in a 30% increase, and 56 ppm sulfur dioxide in a 50% increase. After recovery to control values (generally 30 minutes) the sulfur dioxide-aerosol combination, inhaled for 5 minutes, produced an average 20% increase in pulmonary flow resistance above that observed for sulfur dioxide inhalation alone.

In a later study in 1964, [40] Toyama claimed evidence of synergism between sulfur dioxide in concentrations from 3-40 ppm and dust obtained from the Kawasaki, Japan, area and dispersed at 10-50 mg/cu m. Ten young adult males were tested for increases in pulmonary flow resistance by procedures described above. Wide individual differences in response were noted including a detectable response to the inhalation of the dust alone.

Animal Toxicity

Although a considerable amount of experimental work has been reported on exposure of animals to sulfur dioxide, much of the information has been duplicated by human experiments, especially at exposure levels which are pertinent to the development of an occupational exposure standard. Therefore, rather than include all animal studies in this discussion, only those experiments are presented which have not been studied in humans but which may be applicable to the occupational exposure situation.

In general, man is considered to be more sensitive than other mammals to the effects of sulfur dioxide in ranges commonly employed experimentally [11] with the possible exception of the domestic cat. [41] The effect of sulfur dioxide on all mammals is qualitatively the same--that of respiratory and mucous membrane irritation and reflex bronchoconstriction with increased airway resistance.

(a) Inhalation

Dalhamn and Sjoholm [42] in 1963 found that 5-minute exposures to 1,150-7,700 ppm sulfur dioxide (20-30 mg/liter) produced arrested ciliary activity in rabbit trachea in vitro. Ciliary movements in the rabbit trachea in vivo were frequently arrested after 15 minutes exposure to 200 ppm sulfur dioxide. [43] The same series of experiments failed to demonstrate synergism between sulfur dioxide and carbon black particles mostly below 5 microns in size.

Dalhamn [44] in 1956 reported morphologic changes in rats as determined by electron microscopy. Rats exposed to 10 ppm sulfur dioxide for 3-10 weeks showed severe morphologic changes in the epithelium and lamina propria of the upper respiratory tract with evidence of abnormal cell proliferation. These changes were unaffected by differences in the duration of exposure nor did the changes appear to have regressed in rats examined about 4 weeks after exposure to sulfur dioxide had ceased.

Fraser et al [45] in 1968 reported no alteration in ciliary activity in rats exposed to 1 and 3 ppm sulfur dioxide, either with or without concomitant exposure to graphite dust (1.5 micron median diameter, 1 mg/cu m concentration). Also, on microscopic examination of lung sections, they found no alteration in the ratio of dust-laden cells to the total number of alveolar cells.

Reid [46] in 1963 exposed young rats to 300-400 ppm sulfur dioxide for 5 hours/day, 5 days/week for 6 weeks. An increase in mucin-containing cells was found in the large bronchi and the cells were observed in

peripheral bronchioles where they are not normally found. There was evidence of increased mucous secretion but no signs of increased invasions by infective microorganisms. The excess of mucin-containing cells persisted for at least 3 months after the termination of exposure.

Spiegelman et al [47] in 1968 exposed 3 miniature donkeys to sulfur dioxide concentrations ranging from 26-713 ppm for periods of 30 minutes and studied bronchial clearance of radioactive monodisperse ferric oxide particles. They found no alteration in the rate of bronchial clearance at sulfur dioxide levels below 300 ppm. At higher levels, impairment of bronchial clearance was attributed in part to the increase in mucous secretion.

Rylander [48] in 1969, using aerosols of killed radioactive and viable Escherichia coli, demonstrated no impairment of the bacterial elimination mechanisms (mechanical clearance, phagocytosis, etc) in guinea pigs exposed to 10 ppm sulfur dioxide, 6 hours/day for 20 exposures.

Alarie et al [49] in 1970 reported on essentially continuous exposure of guinea pigs, 22 hours/day, 7 days/week for 1 year to about 0.1, 1, and 5 ppm sulfur dioxide. Pulmonary function measurements including tidal volume, respiratory rate, minute volume, dynamic compliance, pulmonary flow resistance, and carbon monoxide uptake indicated that no detrimental changes could be attributed to sulfur dioxide. In addition, hematological and microscopic tissue studies failed to show any adverse effects on body weight, growth, and survival. In a subsequent study, Alarie et al [50] in 1972 reported on the effects in young cynomalgus

monkeys of long term (78 weeks) 24-hour/day exposure to concentrations of sulfur dioxide of about 0.1, 0.6, 1, and 5 ppm. Control groups exposed to fresh air were also included. Evaluations were made on mechanical properties of the lung, distribution of pulmonary ventilation, diffusing capacity of the lung, arterial blood tension, lung histology, hematological and blood biochemical indices, and organ histology. No deleterious effects could be attributed to concentrations of 0.1-1.28 ppm sulfur dioxide. After 30 weeks of the regulated exposure to the 5 ppm concentration, an accidental overexposure occurred for 1 hour to something between 200 and 1,000 ppm sulfur dioxide. Thereafter, the group was maintained on pure air for the remainder of the experimental period. The accidentally exposed group showed deterioration in pulmonary function which persisted during the remaining 48 weeks of observation despite the discontinuation of sulfur dioxide exposure. Microscopic examination of the pulmonary tissues of this one group showed scattered foci of alveolar proteinosis and numerous alveolar macrophages. The alveolar walls were moderately thickened and infiltrated with histiocytes along with moderate hyperplasia of the bronchial epithelium. Eight of the 9 animals involved had moderate bronchiectasis and bronchiolectasis.

In conjunction with a nitrogen dioxide study, Lewis et al [51] in 1969 reported changes in pulmonary function in female beagles exposed to approximately 5 ppm sulfur dioxide alone or combined with about 0.8 mg/cu m sulfuric acid mist for 21 hours/day for 225 days. The dogs exposed to sulfur dioxide alone or combined with sulfuric acid showed increased

pulmonary resistance and decreased lung compliance. The dogs exposed to both sulfur dioxide and sulfuric acid showed, in addition, a decrease in residual volume, possibly due to a greater degree of lung fibrosis.

Prokhorov and Rogov [52] reported the histopathological and histochemical effects of prolonged exposures of rabbits to 76 ppm sulfur dioxide alone and combined with 182 or 364 ppm carbon monoxide for 3 hours/day for 13 weeks. Exposure to sulfur dioxide alone resulted in edema of the myocardial muscle fibers, capillary enlargement, and numerous perivascular hemorrhages. These changes were more pronounced following simultaneous exposure to carbon monoxide. Exposure to sulfur dioxide alone led to dystrophic changes in the round cells and Kupffer cells of the liver and the epithelium of the renal convoluted tubules. In the lungs, sulfur dioxide gave rise to alveolar epithelial cell proliferation.

Bushtueva [53] in 1962 exposed 6 guinea pigs to 1 mg/cu m (0.4 ppm) sulfur dioxide alone continuously for 5 days. No observable differences were noted between the exposed guinea pigs and unexposed controls.

Lee and Danner [54] in 1966 reported exposing guinea pigs to concentrations of 7-310 ppm sulfur dioxide for 2 1/2 hours. Among other changes, it was found that hemoglobin concentrations increased approximately 10% immediately after exposure to sulfur dioxide. The increase in hemoglobin concentration appeared linear with increasing sulfur dioxide concentrations between 7 and 20 ppm, but thereafter the linearity ceased.

Barry and Mawdesley-Thomas [55] in 1970 reported the effect of sulfur dioxide (300 ppm, 6 hours/day for 10 days) on the enzyme activities of rats by histochemical techniques applied immediately post-mortem to sections of the lungs. They reported a marked increase in acid phosphatase activity in the free alveolar cells throughout the lung parenchyma. It was suggested that in rats, acid phosphatase in alveolar macrophages is associated with the catabolism and removal of mucopolysaccharide and increases in response to the excess mucous secretion induced by sulfur dioxide.

Studies have been conducted [56,57] to investigate the possibility that exposures to sulfur dioxide might increase susceptibility to, or the severity of, respiratory infections in animals. Goldring et al [56] in 1967 failed to demonstrate any such increase in respiratory infections in the hamster between sulfur dioxide at (650 ppm, 3 hours/day for 75 days) and inoculated influenza virus. Navrotskii [57] in 1959 described an "immuno-biological reactivity" of rabbits following exposure to 6.8-8.5 ppm sulfur dioxide for 2 hours/day for 5 1/2 to 8 1/2 months. Agglutination and blood complement titers were determined by intravenous injections of typhoid vaccine. Both titers were "acutely depressed" in the exposed rabbits.

(b) Interaction with Aerosols

In the industrial situation, the inhalation of sulfur dioxide is regularly associated with varying amounts and qualities of aerosol suspensions dispersed as particulate solids or liquids. A considerable amount of animal experimental work has been conducted, [41,43,58-61] often with conflicting results, investigating sulfur dioxide-aerosol interactions with a variety of particulate matter of differing particle sizes and concentrations.

Dalhamn and Strandberg [43] in 1963 reported the effect of 100 ppm sulfur dioxide adsorbed onto activated carbon on ciliary movements in the rabbit trachea in vivo. The effects noted did not differ from those of 100 ppm sulfur dioxide alone, and the effects were less than those observed for 200 ppm sulfur dioxide alone despite the finding of a significant catalytic conversion of sulfur dioxide to sulfuric acid on the carbon particles.

Amdur and Underhill [58] in 1970 studied the effects on airflow resistance of combined exposures to sulfur dioxide (1.5-26 ppm) and iron oxide dust (geometric mean diameter, 0.076 micron) at concentrations of 1.0-24.0 mg/cu m in guinea pigs. No evidence of potentiation was found. Similarly, guinea pigs were exposed to a combination of sulfur dioxide (0.16-0.80 ppm) with open-hearth dust (geometric mean diameter, 0.037 microns) at concentrations ranging from 0.12-0.72 mg/cu m. No significant difference was found between the combinations and corresponding concentrations of sulfur dioxide alone.

Battigelli et al [59] in 1969 reported monitoring the surface microflora from the nasal turbinates, stem bronchi, and from lung homogenates of rats following long-term exposure (12 hours/day, 7 days/week, for 4 months) to 1 ppm sulfur dioxide combined with 1 mg/cu m of graphite dust. Separate groups of rats were also exposed to the graphite dust alone and to fresh air (controls). In addition, weight curves, hematocrit, and post-mortem microscopic studies of the respiratory structures were made. No meaningful differences were found between the 3 groups of rats.

Corn et al [41] in 1972 measured pulmonary flow resistance and lung compliance in 20 healthy adult male cats before and after exposure to 20 ppm sulfur dioxide alone and in combination with sodium chloride aerosol (10 mg/cu m, arithmetic mean diameter, 0.25 micron). Only 2 of the 20 cats, the "reactors," showed any significant increase in pulmonary flow resistance.

There is also animal experimental evidence that potentiation of effects does occur with combinations of sulfur dioxide and certain particulate aerosols. Amdur [60] in 1960 reported on studies in which the increase in pulmonary flow resistance of unanesthetized guinea pigs exposed to about 100 ppm sulfur dioxide was augmented by 10 mg/cu m sodium chloride aerosol (mean particle diameter, 0.04 micron). However, the same concentration (10 mg/cu m) of 2.5 micron sodium chloride aerosol had no such synergistic effect.

Amdur and Underhill [61] in 1968 reported studies on airflow resistance in guinea pigs exposed to approximately 20 ppm sulfur dioxide together with a large variety of both soluble and insoluble particulates namely: sodium chloride, potassium chloride, manganous chloride, ammonium thiocyanate, ferrous sulfate, sodium orthovanadate, activated and spectrographic carbon, manganese dioxide, iron oxide fume, open hearth dust, fly ash, and triphenyl phosphate. The greatest potentiation of the response to sulfur dioxide was observed with sodium chloride, potassium chloride, and ammonium thiocyanate in that order. The effects noted were found to correspond with the sulfur dioxide solubilities in solutions of these salts. Soluble salts of manganese, ferrous iron, and vanadium, known to catalyze the oxidation of sulfur dioxide to sulfuric acid, potentiated the response to sulfur dioxide. The potentiation occurred more rapidly and at much lower concentrations of aerosol than with sodium chloride. The insoluble aerosols were completely ineffective in intensifying the response to sulfur dioxide.

(c) Absorption, Distribution, Fate, and Excretion

Much animal experimentation has involved the use of sulfur dioxide labeled with radioactive sulfur (35S). [12,37,62-66] Over a wide range of sulfur dioxide levels (1 to several hundred ppm), and in all animal species studied, a high proportion of inhaled sulfur dioxide was found to be absorbed in the nasal passages and only slightly less in the oral and nasopharyngeal cavities. [62,63] In the dog, Frank et al [62] in 1959 reported nasal uptake exceeding 99% of $^{35}SO_2$ whereas uptake by breathing

through the mouth averaged more than 95%. Similarly, Strandberg [63] reported 90-95% uptake in the supratracheal portion of the upper respiratory tract in rabbits. In dogs, a small proportion of sulfur dioxide absorbed by the upper respiratory mucosa was desorbed back into the expired air. [62] Results obtained by Balchum et al in cats [37] and dogs [64] demonstrated that absorbed sulfur dioxide was carried by the bloodstream, lymphatics, and other body fluids to all tissues of the body. Frank et al [65] surgically isolated the head and upper neck of the dog from the remainder of the respiratory system and provided ventilation through the nose with air containing 22 ppm of $^{35}SO_2$. Ninety-five percent of the administered sulfur dioxide was found to be absorbed by the mucosa and $^{35}SO_2$ rapidly appeared in the expired air from the lungs. The expired $^{35}SO_2$ could not have reached the lower respiratory tract in the inspired air and its presence in the lungs was presumed to be via the pulmonary capillaries into the alveolar air. A small fraction of sulfur dioxide entering the blood of dogs remained in simple physical solution, or at least in reversible chemical solution, reportedly as free sulfite and bisulfite ion. However, in vitro experiments with rabbit blood and serum indicated that most, if not all, dissolved sulfite reacted reversibly with disulfide bonds present in the plasma proteins forming "S-sulfonate" groups. [67] Bystrova [66] in 1957, working with inhaled ^{35}S-labeled sulfur dioxide and also intravenously injected labeled sodium sulfite in cats, demonstrated that ^{35}S from either source was incorporated into the protein fractions of the blood and other organs. Balchum et al [64] in

1960 found that in dogs exposed experimentally to 35SO2, the hilar lymph nodes, and in one instance the abdominal lymph nodes, contained a considerable proportion of the retained 35S, considering their size. The majority of the 35S was concentrated in the trachea, bronchi, lungs, hilar lymph nodes, kidneys, and esophagus. The ovaries, stomach, and brain were intermediate and substantially lower in activity and the liver, spleen, and heart muscle were least, apparently having a 35S content as a result of diffusion from the blood or perhaps due to the blood they contained. Yokoyama et al [12] in 1971 reported that dogs exposed to 22 and 50 ppm 35S-labeled sulfur dioxide demonstrated more 35S in the plasma than the red blood cells, that more than half of the plasma 35S was dialyzable, ie, in the inorganic ionic form, and that most of the nondialyzable fraction was associated with alpha globulins. Most of the urinary 35S was in the form of inorganic sulfate.

(d) Carcinogenesis

In certain instances, irritant substances are associated with polycyclic hydrocarbon carcinogens. Laskin et al [20] in 1970 reported the induction of squamous cell carcinomas in rats given inhalation exposures to sulfur dioxide in combination with benzo(a)pyrene, a known carcinogen in animals. Previously, inhalation experiments with polycyclic hydrocarbons, including benzo(a)pyrene, had failed to duplicate human-type lung cancer in animals although surgically implanted benzo(a)pyrene-impregnated threads and pellets in the lung had produced squamous cell carcinoma which metastasized to the lymph nodes, pleura, and kidneys. [20] Exposures of

rats to 10, 51, 105, and 567 ppm sulfur dioxide alone were given for 6 hours/day, 5 days/week for periods up to 16 weeks. Rats exposed to 567 ppm demonstrated marked gross pulmonary damage, clinical symptoms, and death while these observed effects were absent at 10 ppm. Tracheitis was found in virtually all animals at all levels of exposure. The combined sulfur dioxide with benzo(a)pyrene studies were carried out with 24 rats and 20 hamsters. The animals were exposed to 10 ppm sulfur dioxide for 6 hours/day, 5 days/week, while an equal group, serving as a control, lived in a prefiltered fresh-air atmosphere. Animals from each group were then given combined carcinogen-irritant exposures (10 mg/cu m benzo(a)pyrene-3.5 ppm sulfur dioxide) for 1 hour/day, 5 days/week for a period which spanned 794 days. The rats showed findings of squamous cell carcinomas as listed in Table XI-4 but, interestingly, no significant pathology was reported to be found in the hamsters. Sulfur dioxide, a pulmonary irritant, and benzo(a)pyrene, when inhaled singly by rats, have failed to produce bronchogenic carcinomas. A "promoting" effect for sulfur dioxide is suggested by these experiments; however, the data are minimal and a question remains as to whether such an effect is specific for sulfur dioxide or whether such a "promoting" effect may be shared by other pulmonary irritants when inhaled in conjunction with known or suspected carcinogens.

Peacock and Spence [21] in 1967 reported exposing 35 male and 30 female spontaneous tumor-susceptible mice to 20 ml/minute sulfur dioxide for 5 minutes (500 ppm), 5 days/week for about 300 days. An approximately

equal number of control mice were also included. The observed distribution of tumors (malignant and nonmalignant) was not shown to be statistically different from those of the controls. However, it was concluded that the sulfur dioxide exposures accelerated the onset of neoplasia in the susceptible mice as a result of the initial, essentially inflammatory reaction caused by the sulfur dioxide. The effects noted by the authors [21] were not considered to be sufficient to justify the classification of sulfur dioxide as a chemical carcinogen.

Correlation of Exposure and Effect

It is well documented that persons engaged in occupations involving significant exposures to sulfur dioxide consistently demonstrate injury associated with damage to the respiratory tract. [8,10,11,19] Acute occupational exposure concentrations are difficult to establish because of their sudden unanticipated occurrences. Exposure to unknown but probably high concentrations of sulfur dioxide have caused death by asphyxia or bronchopneumonia with permanent damage [14]; asthma-like attacks have also been reported. [15] Single or repeated exposures are irritant to the nose and throat producing choking sensations, rhinorrhea, and cough. [7,11]

Because sulfur dioxide is often associated with other environmental contaminants in occupational situations, [18,19] it is difficult to attribute observed effects to the compound itself. One exception, however, is the relatively old (1932) but pertinent epidemiologic study reported by Kehoe et al [8] on workers in the refrigeration industry because exposure

occurred to relatively pure sulfur dioxide being used as a refrigerant. Environmental concentrations averaging 20-30 ppm (range 5-70 ppm) obtained at the time of the study were associated with symptomatic evidence of irritation of the upper respiratory tract. A significantly higher incidence of nasopharyngitis, alteration in the senses of taste and smell, and an increased sensitivity to other irritants was elicited from the exposed group as compared with the controls. In addition, a significantly higher incidence of tendency to increased fatigue or dyspnea on exertion, along with longer duration of colds (although their frequency was no greater), was also noted. Skalpe [18] reported essentially the same findings in Norwegian paper pulp mill workers exposed to 2-36 ppm sulfur dioxide under general working conditions. Special procedures, such as "blowing the digesters," resulted in potential exposure concentrations up to 100 ppm. The study of Anderson [9] in oil refinery workers and Ferris et al [19] in pulp mill workers reported no differences between exposed workers and controls. However, Anderson's study [9] considered changes in body weight, systolic blood pressure, or chest roentgenographic findings. No mention was made of the possible incidence of pulmonary irritation or cough. Similarly, the pulp mill study of Ferris et al, [19] found no statistical differences between the exposed group and the controls; however, although the prevalence of chronic nonspecific respiratory diseases was extensively evaluated, the disease incidence in both the exposed pulp mill group and the paper mill controls was approximately 30% paper mill workers did not represent a satisfactory control group.

All of the occupational exposure studies share a common weakness in that sulfur dioxide data are meager and direct exposure correlation with observed effects is generally not possible because mixed exposures to materials such as chlorine and organic sulfites in wood operations, [18,19] and metal or metal-like compounds [17] in smelting operations, are the rule. In general, however, it may be concluded that usual working conditions have involved exposures to sulfur dioxide concentrations of about 10-30 ppm with frequent short term exposures up to 100 ppm.

Most human experimental exposure studies have involved various aspects of respiratory mechanics related to airway or pulmonary flow resistance in subjects not occupationally exposed to sulfur dioxide. Controlled exposures at concentrations of 9 ppm for 60 minutes have produced increases in airway resistance accompanied by rhinorrhea and lacrimation. [13] At concentrations of about 5 ppm, increases in pulmonary flow resistance [24,25] and decreases in maximum expiratory flow [26] have been observed. Melville [27] reported decreases in small airway conductance at 2.5 and 5 ppm sulfur dioxide exposure levels. In addition, at sulfur dioxide concentrations up to 5 ppm, sensitivity to stimulation of receptors in the larynx, trachea, and bronchi was diminished. At sulfur dioxide concentrations of 1 ppm, Amdur et al [28] reported increases in respiratory rate and pulse rate, and a decrease in total volume of about 25% below control levels during the first 2 minutes of an 11-minute exposure in 4 subjects. Frank et al [29] and Sim and Pattle [13] failed to confirm these findings in subsequent studies. At 1 ppm sulfur dioxide,

most investigators have reported negative dose-related findings in human studies of changes in respiratory mechanics. [29,30-32] Weir et al [31,32] reported significant but reversible decreases in small airway conductance and compliance at exposure levels of 3 ppm sulfur dioxide but found no changes at 1 ppm.

Animal experiments provide information on the effects produced by prolonged sulfur dioxide exposure under controlled conditions for relatively prolonged periods of time. Young rats exposed to 300-400 ppm sulfur dioxide [46] for 5 hours/day, 5 days/week for 6 weeks showed cellular proliferation in the large bronchi and bronchioles along with increased mucous secretion. The excess cells persisted for at least 3 months after termination of the exposure. This condition was believed [46] to represent an induced chronic bronchitis in the rats. In rabbits, exposures to 76 ppm sulfur dioxide [52] for 3 hours/day for 13 weeks resulted in capillary enlargement with perivascular hemorrhaging and alveolar epithelial cell proliferation. In young monkeys, [50] an accidental 1-hour overexposure to between 200 and 1000 ppm sulfur dioxide in young monkeys following 30 weeks of continuous exposure to about 5 ppm, produced progressive deterioration in pulmonary function with eventual development of moderate bronchiectasis and bronchiolectasis. Exposure of rats to 10 ppm sulfur dioxide for 3-10 weeks showed morphologic epithelial changes in the upper respiratory tract with abnormal cell proliferation. [50] These changes reportedly persisted in rats examined 4 weeks after exposure had ceased. Exposure levels of 5 ppm in dogs [51] exposed 21

hours/day for 225 days showed increased pulmonary resistance and decreased lung compliance. In guinea pigs and monkeys, [49,50] no detrimental changes were observed following continuous exposures of 1 year for the guinea pigs and 30 weeks for the monkeys. At 1 ppm exposures, no alteration in alveolar ciliary activity in rats was found [45] following exposures of 12 hours/day, 7 days/week for 4 months. In guinea pigs, [53] following continuous exposure to 0.4 ppm for 5 days, no observable differences were noted between exposed animals and unexposed controls.

In summary, no changes were noted in animals to exposure concentrations of 0.4 ppm and 1 ppm. Extended sulfur dioxide exposures to 5 ppm appeared to produce measureable pulmonary changes and exposures to 10 ppm and greater seem to produce progressive pulmonary damage which may result in extended tissue changes.

There is evidence that a rather rapid physiological compensation (acclimatization) occurs to the effects of sulfur dioxide, especially on respiratory mechanics. [8,28,29,33] Kehoe et al [8] found wide variability in the time required for acclimatization to develop (mean 2.84 months, S.D., 2 months). Acclimatization occurred in 80% of Kehoe's [8] exposed group and has been reported at exposure levels of 5 ppm. [28,29] It is believed to be mediated through depression of tracheobronchial nerve reflexes [27,29] along with a direct action on bronchial smooth muscle. [35-37] Differences of opinion exist as to whether acclimatization has been beneficial in the occupational environment. Melville [27] stated that

a prolonged decrease in airway conductance might eventually compromise pulmonary function.

Sulfur dioxide interaction with aerosols has received considerable attention in both human [24,26,30,39,40] and animal experimental work. [41,43,58-61] Interaction with insoluble aerosols such as activated carbon, iron oxide (Fe_2O_3) and graphite dust have generally proved ineffective in potentiating the effects produced by sulfur dioxide alone. [43,58,59] Toyama, [40] however, reported potentiated activity with a sulfur dioxide-stack dust aerosol. Amdur and Underhill [61] in 1968 reported potentiation of activity of sulfur dioxide by sodium chloride, potassium chloride, and ammonium thiocyanate. The potentiation was proportional to the solubility of sulfur dioxide in each of the compounds. In addition, it was found that soluble salts of manganese, ferrous iron, and vanadium also produced potentiated sulfur dioxide-aerosol activity. These metal ions are known to promote the catalytic conversion of sulfur dioxide to sulfuric acid. [61] Attempts to produce potentiation with insoluble salts were ineffective.

IV. ENVIRONMENTAL DATA AND BIOLOGIC EVALUATION

Environmental Concentrations

Very little data have been published concerning occupational environmental sulfur dioxide concentrations. From the limited reports available, environmental levels in refrigerator manufacturing [8] were regularly encountered averaging 20-30 ppm (range 5-70 ppm) with concentrations prior to 1927 averaging 80-100 ppm. Anderson [9] in 1950 reported finding concentrations up to 25 ppm in his study of oil refinery workers, but indicated that exposures varying between 60-100 ppm had been recorded during times when plant maintenance was relatively low. Skalpe [18] in 1964 found levels between 2 and 36 ppm in paper pulp mills, and levels of about 2-13 ppm were reported by Ferris et al [19] in a similar pulp mill operation.

A 1972 NIOSH sampling of a copper smelter showed good control of sulfur dioxide levels as measured with detector tubes (see Table XI-5). No sulfur dioxide was detected on the belt deck or skimming deck, or in the feed floor roaster building, fire floor roaster building, roaster building loading area, or with anode casting. Sulfur dioxide concentrations of 7 ppm and 10 ppm were determined around the reverberatory furnace, 1 ppm being measured when the furnace was operating at 12% capacity.

Data obtained from another smelter, as indicated in Table XI-6, indicate the need for improvements in local and general ventilation practices for some operations. Potentially hazardous levels of sulfur

dioxide averaging 23 ppm (range 1.6-45 ppm) were determined on the chargers floor of the reverberatory furnaces. Workers on the chargers floor could not easily retreat to an area of low sulfur dioxide concentration whereas workers engaged in tapping and skimming operations, exposed to about 10 ppm sulfur dioxide, could retreat from their area if necessary. It was determined that control of sulfur dioxide concentrations was necessary. Improvements in the tapping and skimming operations would also reduce concentrations for persons working on the reverberatory furnaces. Detector tube determinations for a large number of operations (see Table XI-7) indicated the value of screening studies to determine areas in which more extensive analyses should be made. A number of determinations indicated sulfur dioxide concentrations in excess of 25 ppm, the upper limit of the detector tube capability.

The limited published data and the NIOSH survey information emphasize that control measures are essential in certain situations through the application of sound engineering practices, particularly those of process enclosure and/or the use of exhaust ventilation. Care must be taken to assure that sulfur dioxide which is removed by ventilation is not permitted to reenter the occupational environment. Similarly, a suitable system for removing sulfur dioxide from stack gases should be employed to prevent pollution of the community air.

It is believed that when concerted efforts are made to reduce sulfur dioxide concentrations at offending operations, that levels below 2 ppm time-weighted average can be met.

Environmental Sampling and Analytical Method

Approximately 25 referenced methods were evaluated by Hochheiser [68] in 1964 which included detailed descriptions and selection criteria for 3 recommended methods to measure sulfur dioxide concentrations in air. The methods consisted of the West-Gaeke [69,70] and hydrogen peroxide [71-73] manual methods, and a method for an automatic monitoring instrument employing an electroconductivity analyzer. [74,75]

Additional manual methods were considered which consisted of 10 colorimetric procedures including that recommended by the American Conference of Governmental Industrial Hygienists (ACGIH), [75] 4 iodometric procedures, 2 cumulative methods involving lead peroxide candles and test paper, and detector tubes. Other instrumental methods considered used potentiometric, photometric, or air ionization principles.

In 1973, Hollowell et al [76] reported on current instrumentation for continuous monitoring of sulfur dioxide with commercially available analyzers. It was emphasized that over 60 monitors were commercially available involving 13 distinctly different principles of operation. The analyzers were divided into either ambient air of stationary source monitors. Continuous monitors were listed at a cost generally less than $5,000, having multi-contaminant capability and relatively rapid response time, and able to detect sulfur dioxide at concentrations less than 1 ppm.

The West-Gaeke [69,70] and hydrogen peroxide [71-73] methods remain the manual methods of choice for the determination of sulfur dioxide in the concentration range from about 0.005-5 ppm. [68,69] Sulfur dioxide in the

air is absorbed in sodium tetrachloromercurate which, forming a nonvolatile mercurate ion, is reacted with acid-bleached pararosaniline and formaldehyde to produce a red-purple color which is then measured spectrophotometrically. The method is not subject to interference from other acidic or basic gases or solvents; however, on-site analyses are recommended because color changes occur which make storage and transport of samples inadvisable.

The hydrogen peroxide method has been the most widely used method for collection of sulfur dioxide. [71-73,77] According to a critical evaluation of chemical methods for sampling and analysis of sulfur oxides, [78] peroxide collection methods are considered to be the most acceptable. The sulfur dioxide present forms sulfuric acid, which is then titrated with barium perchlorate [79] rather than standard sodium hydroxide in order to minimize interferences. The method has been successfully used in water analysis, [71] air analysis, [72,73,77] and for the determination of sulfuric acid in air. [80] The hydrogen peroxide method requires only simple equipment and can be performed by analysts having lesser skills. [68] The primary advantage of the method lies in the stability of the collected samples which permits storage and transportation for at least 1 week without apparent decomposition or change. Interferences from soluble particulate sulfates, sulfuric acid, or metal ions are removed by a prefilter upstream of the hydrogen peroxide absorbing solution (see Figure XI-1). Suggestions have been made in the literature that losses occur with some filter media [81]; however, NIOSH has determined that an 0.8

micrometer nominal pore size cellulose membrane filter produces no apparent loss of sulfur dioxide. Phosphate ions are expected to be removed by the prefilter, but if their concentration is greater than that of sulfate ions, the phosphate can be effectively eliminated by precipitation with magnesium carbonate.

The hydrogen peroxide sampling method accompanied by direct titration with barium perchlorate using Thorin [o-(2-hydroxy-3,6-disulfo-1-naphthylazo) benzenearsonic acid] as the indicator, is the recommended compliance method as outlined in Appendix I.

Other sampling and analytical methods, such as the use of detector tubes as evaluated by Ash and Lynch, [82] can be valuable adjuncts to the compliance method, especially for the determination of "exposure to sulfur dioxide" as originally defined and for special purposes for identification of hazardous conditions. Detector tubes are packed with chemically impregnated material which indicates the presence of sulfur dioxide through a color change. The concentration is determined either from the length of the stain or from the color intensity in accordance with the manufacturers' specifications. The use of detector tubes, while not as sensitive or precise as the compliance method, does have the advantage of simplicity and of giving results immediately. A description of the method utilizing detector tubes, and, in addition, measurement with portable instruments, is given in Appendix II.

Biologic Evaluation

Gunnison and Benton [67] in 1971 reported finding increased concentrations of S-sulfonates (thiosulfate esters, S-sulfo compounds) in the plasma of rabbits during exposure to sulfur dioxide. Further investigations of the formation, persistence, and clearance of S-sulfonate compounds from rabbit plasma given as either inhaled sulfur dioxide, or orally or intravenously administered sulfate, was reported by Gunnison and Palmes [83] in 1973. Four rabbits exposed continuously to 10 ppm sulfur dioxide for 10 days showed increased plasma S-sulfonate up to a mean equilibrium concentration of 49 ± 11 nmoles/ml. Approximately 3-5 days were required to reach equilibrium and, following cessation of sulfur dioxide exposure on the 10th day, a rather slow clearance of plasma S-sulfonate was noted until unexposed background (endogenous) levels were attained (half-life = 4.1 days). Calculations based on plasma S-sulfonate equilibrium concentrations between sulfur dioxide-exposed rabbits and rabbits fed known quantities of sulfate suggested that absorption of sulfite into the bloodstream was more efficient when sulfite was administered via the airways as sulfur dioxide rather than by ingestion. S-sulfonate clearance rates were more inconsistent for the sulfur dioxide inhalation studies than for the remarkably consistent clearance rates observed after sulfite ingestion. An explanation for the inconsistency could not be given.

Plasma S-sulfonate levels measured in human subjects have recently been reported by Gunnison and Palmes [84] to show positive correlation with

atmospheric sulfur dioxide. A total of 80 plasma samples were analyzed from a separate study of healthy adult male subjects, 13 nonsmokers and 7 heavy smokers (22-60 cigarettes/day), exposed to sulfur dioxide concentrations of 0.3, 1.0, 3.0, 4.2, and 6.0 ppm. The primary objective of the inhalation studies was the assessment of sulfur dioxide inhalation on pulmonary function by Weir and associates using exposure apparatus and chamber monitoring methods originally described in 1971. [85] Specific exposures of each subject were not divulged to the authors [84] until all plasma analyses were completed. No significant differences were noted for plasma S-sulfonate levels between smokers and nonsmokers. A regression line calculated for the combined group ($Y = 0.17 + 1.09X$; $r = 0.61$) showed an increase of approximately 1.1 nmoles/ml plasma S-sulfonate for each 1 ppm increment in chamber sulfur dioxide concentration. Generally, each datapoint represented S-sulfonate from a single plasma sample; however, if sufficient plasma were available in a sample, it was analyzed in duplicate or triplicate and the average used as one datapoint. According to Gunnison and Palmes, [84] the finding of S-sulfonate formation in the plasma of man is the first known to implicate inhaled sulfur dioxide in its production.

The above findings in animals and man afford preliminary judgment of a favorable biologic correlation of environmental sulfur dioxide concentrations with measured plasma S-sulfonate levels. The correlation reported for humans shows promise but it is too early for such biologic exposure-effect relationships to be regarded as being established. Two distinct drawbacks are immediately apparent. First, the use of blood

samples, as opposed to urine samples, is undesirable for biologic monitoring from both the employee's and the employer's viewpoint. Second, plasma S-sulfonate determinations for sulfur dioxide are nonspecific, since any material which produces increased sulfite levels will affect S-sulfonate concentrations. Nonspecificity may not be a serious shortcoming, however, because rarely, if ever, is a biologic product or metabolite completely specific for an absorbed hazardous material encountered in the occupational situation. The measurement of plasma S-sulfonate is regarded as a diagnostic practice and not a mandatory procedure. It is left to the discretion of the medical supervisor whether the procedure is to be included in the medical program. Biologic monitoring of plasma S-sulfonate may provide a useful measurement technique to verify sulfur dioxide exposure in the worker.

V. DEVELOPMENT OF STANDARD

Basis for Previous Standards

In 1945, Cook [86] compiled a comprehensive summary of standards which listed the maximum allowable concentration (MAC) of many industrial atmospheric contaminants. The value for sulfur dioxide was given as 10 ppm (25 mg/cu m) which was then endorsed by various agencies in the States of California, Connecticut, Massachusetts, New York, Oregon, Utah, and the USPHS. As documentation for the 10 ppm standard, Cook [86] incorrectly stated that Fieldner and Katz [87] considered 10 ppm as the highest concentration tolerable for prolonged [undefined] exposure. Actually, Fieldner and Katz [87] gave no specific mention of 10 ppm sulfur dioxide. They did refer to the 1918 Holmes et al Selby Smelter Commission report [88] which presented various exposure-effect findings attributable to sulfur dioxide. There was no mention made, however, of a maximum tolerable concentration for "prolonged" exposure. As further documentation for 10 ppm, Cook [86] referred to Flury and Zernik's book "Schadliche Gase" published in 1931 [89] which contained a reference to Lehmann-Hess in which a concentration of 8-12 ppm was suggested as permissible for several hours' exposure.

In 1946, the American Conference of Governmental Industrial Hygienists (ACGIH) [90] adopted an initial MAC for sulfur dioxide of 10 ppm based on committee recommendations and the value which had been previously published by Cook [86] in 1945. In April 1957, the ACGIH [91] tentatively

reduced their recommended Threshold Limit Value (TLV) to 5 ppm (13 mg/cu m), again based on committee review of available data and inquiries to 53 state and local industrial hygiene units for human exposure information that might be relative to TLV's. The State of Michigan reported that 10 ppm sulfur dioxide caused definite discomfort in exposed workers. The 5 ppm tentative TLV was subsequently adopted by the ACGIH in 1958. [92] In 1968, [93] the ACGIH further documented the 5 ppm TLV to include data on humans and animals contained in the 1954 review by Greenwald [11] as well as information from the Occupational Health Section of Oregon that upper respiratory irritation and some nosebleed had occurred in workers exposed to 10 ppm sulfur dioxide. Symptoms reportedly disappeared at a level of 5 ppm. In 1971, [94] the reports from Michigan and Oregon were cited as private communications.

In 1969, the Czechoslovak Committee of Maximum Allowable Concentrations [95] listed MACs for a number of countries as follows: USSR and Hungary, 10 mg/cu m (4 ppm); Poland and the German Democratic Republic, 1 mg/cu m (0.4 ppm); and the Federal Republic of Germany, 13 mg/cu m (5 ppm). The Czechoslovak committee recommended a MAC of 10 mg/cu m (5 ppm). They cited Amdur et al, [27] Greenwald, [11] and Kehoe et al [8] as documentation of effects at various exposure levels.

The present Federal standard for sulfur dioxide is an 8-hour time weighted average of 5 ppm (29 CFR Part 1910.93 published in the Federal Register, volume 37, page 22139, dated October 18, 1972).

Basis for Recommended Environmental Standard

Single or repeated exposures to sulfur dioxide concentrations above 20 ppm are irritant to the nose and throat, often choking, resulting in rhinorrhea, sneezing, and cough. [7,11] Also, in response to the pulmomary irritation, reflex bronchoconstriction with possible increases in mucous secretion and pulmonary flow resistance results. [13] Incidents of suppurative bronchitis, influenza, and asthma-like attacks have also been attributed to sulfur dioxide exposure. [10,15] Even asphyxia or severe chemical bronchopneumonia with bronchiolitis obliterans has resulted [14] from accidental sulfur dioxide exposures to extremely high concentrations in confined spaces.

Published reports of occupational exposures to sulfur dioxide from which quantitative exposure-effect relationships may be derived are essentially nonexistent with mixed exposures being the general are generally the rule. [17-19] Under general working conditions, average exposures of about 10-30 ppm seem to be apparent from reports of paper mill operations, [18] refrigerator manufacture when sulfur dioxide was used as a refrigerant, [8] refining, [9] and smelting operations (see Tables XI-6 and XI-7). Frequently, short-term sulfur dioxide exposures of up to 100 ppm appear to be rather common. [8,18]

Even though data on environmental concentrations of sulfur dioxide are minimal in published epidemiologic studies, the studies do contain valuable information on signs and symptoms resulting from occupational exposure. Interestingly, 3 of the 4 epidemiologic studies reported

[8,9,19] did not consider regular moderate exposure (approximately 10 to 30 ppm) of sulfur dioxide to cause particularly serious damage. Kehoe et al [8] concluded that such exposures to sulfur dioxide caused no apparent injury of a serious type, yet of all 100 subjects included in the study (nearly half had 4-12 years employment exposure) showed some symptomatic evidence of irritation of the upper respiratory tract. Ferris et al [19] minimized the incidence of chronic respiratory disease in pulp mill workers because no statistical differences were observed between the exposed workers and controls who worked in a neighboring paper mill. However, the 30% incidence of respiratory disorders in both the exposed and control groups indicated not only an unsatisfactory control group, but also that chronic respiratory disease was a problem. Skalpe [18] in a separate study of a group of paper pulp mill workers found an increased incidence of respiratory disease. Although Anderson [9] found no evidence of adverse effects in oil refinery workers, only changes in worker weight, systolic blood pressure, or chest roentgenographic findings were reported. No mention was made of the incidence of upper respiratory tract irritation, coughing, nosebleeds, etc, which are associated with the sulfur dioxide concentrations which were encountered (occasionally up to 100 ppm). The similarity of chronic respiratory complaints reported from mixed exposures [18,19] with those reported by Kehoe et al [8] tend to confirm the role of sulfur dioxide as the causal agent.

In both humans and animals, sulfur dioxide produces mucous membrane irritation and reflex bronchoconstriction with increased airway resistance.

Human experimental studies [13,24-26,28-32] provided quantitative information on respiratory mechanics at sulfur dioxide levels below 10 ppm, generally from single exposures of short duration, usually 10 to 30 minutes. Animal exposures [45,49-53] provide an insight into the effects of prolonged intermittent and continuous exposures. Exposures of rabbits to 76 ppm sulfur dioxide [52] (3 hours/day, 13 weeks) produced capillary enlargement, hemorrhaging, and alveolar cell proliferation. At about 10 ppm, morphologic epithelial changes with abnormal cell proliferation were observed in the upper respiratory tract of rats [50] (3-10 weeks continuous exposure) and in humans, [13] 10- or 60-minute exposures produced increases in airway resistance, rhinorrhea, and lacrimation along with rales over the larger bronchi and periphery. At 5 ppm sulfur dioxide exposure, dogs exposed 21 hours/day for 225 days [51] showed increased pulmonary resistance and decreased lung compliance; however, in guinea pigs exposed for 1 year [49] and monkeys exposed for 30 weeks, [50] no injurious changes were observed. In humans, short exposures of up to 1 hour to about 5 ppm sulfur dioxide produced increases in pulmonary flow resistance, [24,25] decreased maximum expiratory flow, [26] and decreased specific airway conductance. [27]

Morphologic cellular changes and alterations in respiratory mechanics at concentrations below 5 ppm sulfur dioxide have not been found in reported animal studies. [45,53] In humans, exposures of up to 1 hour to 2.5 ppm [27] and 120 hours to 3 ppm [31,32] have resulted in minimal reversible decreases in small airway conductance and compliance.

Generally, exposures to 1 ppm sulfur dioxide have failed to indicate detectable changes in respiratory mechanics; however, the report of Amdur et al [28] in 1953 indicated minor increases in respiratory rate and pulse rate and a 25% decrease in tidal volume during the first 2 minutes of exposure, effects which have failed to be confirmed in subsequent studies by others. [13,29] Additionally, a small decrease in maximum expiratory flow rate reported by Snell and Luchsinger [26] in 1969 is not considered of significance since the authors [26] recognized their method to be a less sensitive indicator of a bronchoconstrictive effect than the measurement of pulmonary flow resistance employed by Frank et al [29] who reported no detectable change at 1 ppm, but did note changes at about 5 ppm.

Acclimatization to the effects of sulfur dioxide develops rather rapidly. [8,28,29,33] It has been reported to occur at exposure levels of 5 ppm [28,29] and seems to result from depression of tracheobronchial nerve reflexes. [27,29] Although awareness of discomfort is less following acclimatization, the adjustment is not considered to be a beneficial effect because of the possibility that prolonged depression of the tracheobronchial reflex merely removes one measure of protection. [38] Melville [27] reported in 1970 that pulmonary function might eventually be compromised. Kehoe et al [8] reported of those workers who remained on the job that acclimatization occurred in 80% of the sulfur dioxide exposed workers studied and that 20% of the workers, although failing to become acclimatized, nevertheless continued to work and to be exposed. It has also been estimated [30] that "hyperreactors" may occur in 10-20% of

healthy young adults. It does not seem proper to consider such a large group of individuals as being hypersusceptible to the effects of sulfur dioxide exposure. It is believed more appropriate to consider the unusual cases of sulfur dioxide-induced skin eruptions [22,23] as being hyperreactions.

The current Federal standard for sulfur dioxide of 5 ppm time-weighted average was adopted from the ACGIH recommended Threshold Limit Value. According to the current documentation, [94] 5 ppm should prevent respiratory tract irritation in most workers and cause only minimal effects in those workers who are sensitive to sulfur dioxide. If sensitive workers are considered to be those who failed to become acclimatized, then clearly 5 ppm is not adequate to protect sufficient numbers of workers because the irritant effects cannot be considered as minimal. In addition, although 5 ppm sulfur dioxide may not produce subjective irritation in acclimatized workers, it does affect respiratory mechanics and may compromise pulmonary function.

The experimental evidence for potentiation (synergism) between sulfur dioxide and aerosol particulates is conflicting. Interaction of insoluble aerosols has generally been ineffective in potentiating the effects produced by sulfur dioxide alone [43,58,59]; however, sulfur dioxide combined with stack dust aerosol has been reported to have produced potentiated activity. There is strong evidence that aerosols of certain water soluble salts, known to catalyze the conversion of sulfur dioxide to sulfuric acid, do potentiate the irritant and reflex bronchoconstrictive

effects of sulfur dioxide. [61] More information is needed on the interaction of additional variables such as time, temperature, and humidity as they occur in the occupational situation.

The role of sulfur dioxide in human carcinogenesis is largely one of association rather than direct incrimination. The human mortality study of Lee and Fraumeni [17] in 1969 reported the positive correlation between sulfur dioxide exposure and observed deaths from respiratory cancer. Mortality ranged from 2 1/2 to 6 times expected in groups selected as having light, medium, and heavy exposures to sulfur dioxide along with arsenic (no environmental data were given). The study indicated that persons with heavy exposure to arsenic and moderate or heavy exposure to sulfur dioxide were most likely to die of respiratory cancer. It should be emphasized, however, that arsenic has been implicated as an occupational carcinogen without sulfur dioxide being present. [96] In addition, there are no studies known which implicate sulfur dioxide by itself as a carcinogen in either man or animals. Two animal studies [20,21] have associated sulfur dioxide exposure with the incidence of bronchogenic carcinoma in conjunction with known carcinogens [20] or strains of mice having a high spontaneous incidence of lung carcinoma. [21] The incidence of squamous cell carcinoma in rats (5/21) recorded by Laskin et al [20] to combined benzo(a)pyrene-sulfur dioxide could not be produced with either the benzo(a)pyrene or the sulfur dioxide administered alone by inhalation. Also, the same carcinogen-irritant combination which produced carcinomas in rats failed to do so in an identical experiment with hamsters. In tumor-

susceptible mice, Peacock and Spence [21] concluded an accelerated onset of neoplasia but the total number of tumors observed (malignant and nonmalignant) was not statistically different for exposed vs control animals.

Since arsenic has been associated with increased cancer by Hill and Faning [96] in the absence of sulfur dioxide, it does not seem justified on the basis of the Lee and Fraumeni mortality study [17] to make any definite conclusions on the carcinogenic role of sulfur dioxide. The application of the Laskin et al study in rats [20] is not clear because benzo(a)pyrene is a known carcinogen. Also, the Peacock and Spence study [21] used very high sulfur dioxide concentrations (500 ppm) to obtain the increased, although not statistically significant, incidence of tumors in the tumor-susceptible mice. Thus, a conclusion which would implicate sulfur dioxide as a primary carcinogen cannot be made; however, the possible role of sulfur dioxide as a cocarcinogen (promoter) cannot be disregarded based upon present data.

Data to demonstrate a safe exposure level for sulfur dioxide indicate barely detectable changes in respiratory mechanics at 2.5 ppm [27] and 3 ppm. [31,32] The suggestion of sulfur dioxide-induced changes in the range of 1 ppm is slight and unconvincing. It is concluded that the existing Federal standard of 5 ppm TWA should be reduced because of evidence of changes in pulmonary mechanics [24-27] as a result of irritant-induced bronchoconstriction. It is believed that the standard should be reduced at least as low as 2 ppm time-weighted average so as to prevent the irritant effects of sulfur dioxide in workers, including those who may not

be capable of acclimatization. The reduction to a time-weighted average concentration of 2 ppm would, in addition, reduce the probability of sulfur dioxide acting as a promoter.

VI. COMPATIBILITY WITH AMBIENT AIR QUALITY STANDARDS

National primary and secondary ambient air quality standards for sulfur oxides (sulfur dioxide) were published in the Federal Register by the Environmental Protection Agency on April 30, 1971, volume 36, pages 8186-8187 (42 CFR 410.1-410.5). The national primary air quality standards define levels of air quality which are judged necessary, with an adequate margin of safety, to protect the public health. The national secondary ambient air quality standards define levels of air quality which are judged necessary to protect the public welfare from any known or anticipated effects of a pollutant. The term "ambient air," as used in the air quality standards means that portion of the atmosphere, external to buildings, to which the general public has access.

The national primary ambient air quality standards for sulfur oxides, measured as sulfur dioxide, are:

(a) 80 μg/cu m of air (0.03 ppm) calculated as an annual arithmetic mean.

(b) 365 μg/cu m of air (0.14 ppm) computed as a maximum 24-hour concentration not to be exceeded more than once per year.

The national secondary ambient air quality standards for sulfur oxides, measured as sulfur dioxide, are:

(a) 60 μg/cu m of air (0.02 ppm) calculated as an annual arithmetic mean.

(b) 260 μg/cu m of air (0.1 ppm) computed as a maximum 24-hour concentration not to be exceeded more than once per year.

(c) 1,300 μg/cu m of air (0.5 ppm) as a maximum 3-hour concentration not to be exceeded more than once per year.

The basis for the development of these standards was a monograph entitled, Air Quality Criteria for Sulfur Oxides, (NAPCA publication AP-50) which critically reviewed pertinent health studies. Further, studies conducted by EPA for the Community Health and Environmental Surveillance System (CHESS) have strengthened the available defense of the existing standards for sulfur oxides. Strong associations exist that adverse health effects may relate more closely with suspended particulate sulfate than with sulfur dioxide.

No direct comparison can be made between the national primary and secondary ambient air quality standards and the recommended standard for occupational exposure because the levels of exposure to the general public involve varying health status and age on a 24-hour day, 7-day week basis. The ambient air quality standards should be substantially lower than the occupational standards which are based on a 40-hour work week. The concentration of sulfur dioxide present in the general atmosphere is not expected to adversely affect workers when occupational levels are not above the 2 ppm standard recommended in this document.

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VIII. APPENDIX I

METHOD FOR SAMPLING AND ANALYTICAL PROCEDURES FOR DETERMINATION OF SULFUR DIOXIDE

The following sampling and analytical method for analysis of sulfur dioxide in air employs absorption and oxidation in hydrogen peroxide solution followed by volumetric titration.

General Requirements

Sulfur dioxide concentrations shall be determined within the worker's breathing zone and shall meet the following criteria in order to evaluate conformance with the standard:

(a) Samples collected shall be representative of the individual worker's exposure.

(b) Sampling data sheets shall include:

(1) The date and time of sample collection

(2) Sampling duration

(3) Volumetric flowrate of sampling

(4) A description of the sampling location

(5) Other pertinent information

Breathing Zone Sampling

Breathing-zone samples shall be collected as near as practicable to the worker's face without interfering with his freedom of movement and

shall characterize the exposure from each job or specific operation in each production area.

(a) Sampling Equipment

A calibrated personal sampling pump with flowmeter (range up to 2 liters/minute), a midget impinger containing 15 ml of 0.3 N hydrogen peroxide absorbing solution, and an 0.8 micrometer nominal pore size cellulose membrane filter with filter holder shall be used for sample collections.

(b) Sampling Procedure

The filter is placed upstream of the impinger to collect any sulfuric acid mist or other airborne particulate sulfates prior to the air passing through the impinger. The filter holder is connected to the impinger inlet by a piece of flexible vinyl tubing as short as possible. The impinger outlet is connected to the personal sampling pump inlet by a piece of tubing of convenient length, but not in excess of 3 feet. The filter and impinger assembly is attached to the worker's clothing so as to sample from the worker's breathing zone. The sample is collected at a rate of 1 - 2 liters/minute for an appropriate length of time to attain a 100-liter air sample. If sulfur dioxide concentrations are expected to be greater than 100 mg/cu m of air, (approximately 40 ppm), a smaller air volume should be sampled but never less than 10 liters.

A minimum of 3 samples shall be taken for each operation (more samples if the concentrations are close to the standard) and averaged on a time-weighted basis. At least one blank impinger shall be provided

containing hydrogen peroxide solution through which no air has been sampled. One additional blank impinger shall be supplied with every 10 samples obtained.

Shipping

After sampling, remove the glass stopper and impinger stem from the impinger bottle. Tap the stem gently against the inside wall of the impinger bottle to recover as much of the sampling solution as possible. Wash the stem with a small amount of unused absorbing solution from a wash bottle, adding the wash to the impinger. Stopper the impinger tightly with plastic caps (do not seal with rubber), place in an upright position, and ship the impinger samples to the analytical laboratory in a suitable container to prevent damage in-transit. Special impinger shipping containers designed by NIOSH are available. Be certain that the impinger bottles are sealed very tightly to prevent leakage and subsequent loss of samples.

Calibration of Sampling Trains

Since the accuracy of an analysis can be no greater than the accuracy of the volume of air which is measured, the accurate calibration of a sampling pump is essential to the correct interpretation of the pump's indication. The frequency of calibration is dependent on the use, care, and handling to which the pump is subjected. In addition, pumps should be recalibrated if they have been misused or if they have just been repaired

or received from a manufacturer. If the pump receives hard usage, more frequent calibration may be necessary.

Ordinarily, pumps should be calibrated in the laboratory both before they are used in the field and after they have been used to collect a large number of field samples. The accuracy of calibration is dependent on the type of instrument used as a reference. The choice of calibration instrument will depend largely upon where the calibration is to be performed. For laboratory testing, primary standards such as a spirometer or soapbubble meter are recommended, although other standard calibrating instruments such as a wet test meter or dry gas meter can be used. The actual setup will be the same for all instruments. Instructions for calibration with the soapbubble meter follow. If another calibration device is selected, equivalent procedures should be used.

(a) Flowmeter Calibration Test Method

The calibration setup for personal sampling pumps with the sampling system of a filter and a midget impinger is shown in Figure XI-1.

(1) Procedure

(A) Check the voltage of the pump battery with a voltmeter to assure adequate voltage for calibration. Charge the battery if necessary.

(B) Fill the impinger with 15 ml of the absorbing solution and place the cellulose membrane filter in the filter holder.

(C) Assemble the sampling train as shown in Figure XI-1.

(D) Turn the pump on and moisten the inside of the soapbubble meter by immersing the buret in the soap solution and draw bubbles up the inside until they are able to travel the entire buret length without bursting.

(E) Adjust the pump rotameter to provide a flowrate of 1 liter/minute.

(F) Check the water manometer to insure that the pressure drop across the sampling train does not exceed 13 inches of water (1 in. of Hg).

(G) Start a soapbubble up the buret and, with a stopwatch, measure the time it takes for the bubble to move from one calibration mark to another. For a 1000-ml buret, a convenient calibration volume is 500 ml.

(H) Repeat the procedure in (G) above at least 2 times, average the results, and calculate the flowrate by dividing the volume between the preselected marks by the time required for the soapbubble to traverse the distance.

(I) Data for the calibration include the volume measured, elapsed time, pressure drop, air temperature, atmospheric pressure, serial number of the pump, and date and pump, and date and name of the person performing the calibration.

Analytical

(a) Principle of the Method

Sulfur dioxide in the air is absorbed and oxidized in 0.3 N hydrogen peroxide reagent. The pH of the sample solution is adjusted with dilute perchloric acid. After isopropyl alcohol is added bringing the alcohol concentration to approximately 80% by volume, the resulting solution is titrated with 0.005 M barium perchlorate using Thorin as the indicator. The endpoint is determined as a change from yellow to pink.

(b) Range and Sensitivity

The method is sensitive to 0.1 mg sulfur dioxide/cu m of air, assuming a 100 liter air sample. This would correspond to approximately 0.25 ppm of sulfur dioxide in air. The upper limit is the amount of sulfur dioxide absorbed in the hydrogen peroxide reagent and is at least 5 mg.

(c) Interferences

Soluble particulate sulfates and sulfuric acid in the air sample would give erroneously high sulfur dioxide values; however, these can be eliminated by placing an 0.8 micron cellulose filter upstream of the impinger in the sampling train.

Metal ion interferences can be eliminated by either the use of the prefilter or, alternatively, by passing the solution through an ion exchange column.

Concentrations of phosphate ions greater than any sulfate ion concentration cause appreciable interference. Phosphate can be removed by precipitation with magnesium carbonate. The use of the prefilter should also remove phosphates.

(d) Accuracy and Precision

At 2.5 ppm, the accuracy is 5% with a relative standard deviation of 4%. At 25 ppm, the accuracy and relative standard deviation can be improved to about 1%.

(e) Advantages and Disadvantages

The samples are easily collected and conveniently shipped to the laboratory for analysis in glass vials.

The sulfuric acid formed is stable and nonvolatile, making this manner of collection of sulfur dioxide desirable.

The analysis is relatively rapid and simple.

Spillage from the impingers is possible and could be hazardous if spilled into molten metal.

(f) Apparatus

(1) Absorber-- glass midget impingers.

(2) Personal sampling pump with flowmeter capable of sampling at a rate of 1-2 liters/minute.

(3) 37 mm mixed cellulose ester filter, 0.8 micron nominal pore size.

(4) Necessary glassware.

(5) A buret of 10 ml capacity graduated in 0.05 ml subdivisions.

(6) Daylight fluorescent lamp aids in identifying the endpoint.

(7) Ion exchange resin-- Strongly acidic cation exchange resin, 20-50 mesh, or equivalent. Ion exchange columns may be constructed using glass burets or tubing. A column with an inside diameter of 8 mm and 7 inches of resin has a capacity of approximately 25 milliequivalents.

(g) Reagents

(1) Alcohol-- isopropanol, reagent grade.

(2) Barium perchlorate, 0.005 M. Dissolve 2.0 g of barium perchlorate trihydrate in 200 ml of water and add 800 ml of isopropanol. Adjust pH to about 3.5 with perchloric acid. Standardize against 0.005 M sulfuric acid.

(3) Thorin-- prepare a 0.1-0.2% solution in distilled water.

(4) Standard sulfate solution-- prepare a 0.005 M solution of sulfuric acid and standardize by titration with 0.005 M sodium hydroxide solution or dissolve 0.7393 g anhydrous sodium sulfate in distilled water and dilute to 1 liter (1 ml = 0.5 mg sulfur dioxide). The sodium is removed by passage of the standard solution through the ion exchange column.

(5) Hydrochloric acid, 4 N-- add 300 ml concentrated HCl to 600 ml of distilled water. This is needed only to regenerate the column if the ion exchange procedure is used.

(6) Absorbing solution-- hydrogen peroxide, 0.3 N-- dilute 17 ml of 30% hydrogen peroxide solution to 1 liter with distilled water.

(h) Procedure

(1) Cleaning of equipment-- the glassware should be chemically clean. Wash in detergent and rinse with tap water and distilled water.

(2) Ion exchange procedure (used to purify standard sulfate solution)-- when about two-thirds of the capacity of the resin has been exhausted (deterioration in sharpness of the end point), regenerate the resin by passing 30 ml of 4 N hydrochloric acid through the column. After thorough washing with distilled water, the column is ready for use. Since small volumes of sample solution are passed through the ion exchange column, care must be taken not to dilute the sample with distilled water that remains in the resin. One way this can be accomplished is by forcing air through the resin with a squeeze bulb to remove most of the distilled water from the ion exchange resin. One or 2 ml of sample is passed through the column and is discarded after air is again forced through the resin. The remainder of the sample is then passed through the ion exchange column and an aliquot is titrated according to the general procedure in (i)(3) below.

The column is flushed with distilled water between samples to prevent contamination from the previous sample.

(i) Analysis of Samples

(1) Measure the volume of the sample solution or dilute it to a given volume.

(2) If high air concentrations of metal ions are encountered which are not completely removed by the prefilter, samples may be passed through the ion exchange column by the procedure detailed in (h)(2) above.

(3) To a 10 ml aliquot, add 40 ml isopropanol. Adjust the pH, if necessary, to between 2.5 and 4.0 with perchloric acid. Add 1-3 drops of Thorin indicator and titrate with barium perchlorate, taking the change from yellow or yellow-orange to pink as the endpoint.

(4) Analyze the standard and absorbing solution blank in the same manner.

(j) Standardization

The barium perchlorate solution is standardized by titrating a 5 ml aliquot with 0.005 M sulfuric acid to the endpoint using Thorin as indicator. The molarity of the solution is calculated as follows:

$$M[\text{barium perchlorate}] = \frac{ml[\text{sulfuric acid}] \times M[\text{sulfuric acid}]}{ml[\text{barium perchlorate}]}$$

Periodic checks of the molarity of the barium perchlorate solution should be run following this same procedure.

If anhydrous sodium sulfate is used to standardize the barium perchlorate, it must first be ion-exchanged since sodium obscures the endpoint. A 5 ml aliquot of the 0.5 mg/ml sulfate solution is ample for standardization.

(k) Calculations

The analytical results are calculated on the basis of the following reactions:

sulfur dioxide + hydrogen peroxide = sulfuric acid

sulfuric acid + barium perchlorate = barium sulfate + 2 perchloric acid

$$\frac{\text{mg[sulfur dioxide]}}{\text{cu m}} = \frac{\text{ml[s]} \times \text{M[barium perchlorate]} \times \text{MW[sulfur dioxide]}}{\text{V[cu m]}} \times \frac{\text{V}}{\text{V[aliq]}}$$

ml[s] = ml of barium perchlorate solution needed to titrate the sample aliquot minus the blank value.

MW[sulfur dioxide] = molecular weight of sulfur dioxide = 64.

V[cu m] = volume of air sampled in cubic meters.

V[aliq] = volume of sample aliquot used for the titration in ml.

V = original volume of sample in impinger in ml.

OR

$$\text{sulfur dioxide (ppm) by volume} = \frac{\text{ml[s]} \times \text{M[barium perchlorate]} \times 24{,}450}{\text{V[l]}} \times \frac{\text{V}}{\text{V[aliq]}}$$

V[l] = volume of air in liters at 25 C.

24,450 = ml/mole that ideal gas occupies at 25 C.

IX. APPENDIX II

METHODS FOR DETERMINATION OF EXPOSURE AREAS TO SULFUR DIOXIDE

Estimation of Concentration with Detector Tubes

(a) Atmospheric Sampling

(1) Equipment Used

A typical sampling train consists of a detector tube with a corresponding sampling pump. A specific manufacturer's pump may only be used with his detector tubes.

(2) Sampling Procedures

A specific procedure depends on the manufacturer's instructions but normally consists of breaking both tips off a detector tube, inserting the tube into the pump, and taking a specific number of strokes with the pump.

(3) Handling and Shipping of Samples

Detector tubes are not stable with time because the stain in some tubes fades in a few minutes. The tubes should be read immediately in accordance with the manufacturer's instructions and charts and no attempt should be made to save the used tubes.

(b) General Principles

Gas detector tubes contain a chemically impregnated packing which indicates the concentration of a contaminant in the air by means of a chemically produced color change. The color changes are not permanent or

stable, so the stained tubes must be read immediately after the samples are taken. The length of stain or the color intensity is read according to the manufacturer's instructions and may involve comparing the stain with a chart, a color comparator, or a direct concentration reading from calibration marks on the tube. Detailed descriptions are provided by individual manufacturer's instructions.

Tubes obtained from commercial sources which bear the certified seal of NIOSH are considered to adhere to the requirements as specified for Certification of Gas Detector Tube Units in 42 CFR Part 84 (38 FR 11458). A user may perform his own calibration on commercially acquired tubes by generating accurately known concentrations of sulfur dioxide in air and correlating concentration with stain length or color intensity.

The use of detector tubes with their respective pumps for compliance purposes is inappropriate because sampling times are necessarily very brief; thus, an excessive number of sampling periods would be required to permit calculation of a time-weighted average. In addition, the accuracy of detector tubes is limited [see (e) below].

(c) Range and Sensitivity

Certification standards require that certified tubes have a range from 1/2 to 5 times the time-weighted average concentration. The sensitivity varies with tube brands.

(d) Interferences

Interferences vary with tube brands. The manufacturer's instructions must be consulted.

(e) Accuracy

Certification standards under the provisions of 42 CFR Part 84 (38 FR 11458) specify reliability to within ±25% of the actual concentration in the range 0.75 to 5 times the standard and ±35% in the range from 0.5 up to, but not including, 0.75 times the standard.

(f) Advantages and Disadvantages

Unlike the hydrogen peroxide-barium perchlorate method, the use of detector tubes (and portable instruments) is relatively inexpensive and rapid. There is far less time lag than that experienced with laboratory analytical results. Rapid detecting units are valuable for determining whether a hazardous condition exists at a given location at a given time so that workers may be evacuated or suitable protective devices provided. In addition, industrial operators and process engineers need inexpensive and rapid tools for day-to-day evaluation of the atmospheric levels in a work area.

The accuracy of detector tubes is limited; at best they give only an indication of the contaminant concentration. In evaluating measurements performed with detector tubes, interferences, difficulty of endpoint readings, and possible calibration inaccuracies must all be considered.

Measurement with Portable Instruments

(a) Atmospheric Sampling

(1) Equipment Used

There are several different types of portable meters available for atmospheric sampling: portable variable path infrared analyzers, electroconductivity analyzers, and electrochemical membrane-type polarographic detectors. Any of the above mentioned instruments can be used to measure sulfur dioxide if they are properly calibrated before use.

(2) Sampling Procedures

The most important step is the meter calibration. Careful calibration should be performed in a laboratory prior to departure for the field. Known concentrations of sulfur dioxide can be generated from a dynamic permeation tube system.

The actual field sampling is conducted according to the manufacturer's instructions. Readings should be corrected if necessary for variables such as temperature, humidity, atmospheric pressure, etc, and recorded along with time, place, etc.

(b) General Principles

Analysis is dependent on the type of meter used. The portable direct reading meters require no analysis because they usually provide usable concentration readings directly. Results obtained from the variable-path infrared analyzer and the electrochemical membrane-type polarographic detectors must be further analyzed and calculated to obtain concentration values.

(c) Range and Sensitivity

The range and sensitivity vary with the instrument used. These instruments generally have a greater sensitivity than detector tubes.

(d) Interferences

Again, these vary with the instrument. The most common interferences are water vapor, hydrogen sulfide, sulfates (gases and particulates), sulfur trioxide, and sulfuric acid. For the electroconductivity type detectors, strong interferences result from gases that affect the conductivity of the absorbing media.

(e) Advantages and Disadvantages

The benefits and drawbacks of portable instruments are essentially the same as for detector tubes discussed previously. Portable meters are generally more sensitive and more accurate than detector tubes. Also, when recording capability is possible, direct reading instruments have the advantage of continuous record availability.

X. APPENDIX III.

MATERIAL SAFETY DATA SHEET

The following items of information which are applicable to a specific product or material containing sulfur dioxide shall be provided in the appropriate section of the Material Safety Data Sheet or approved form. If a specific item of information is inapplicable, the initials "n.a." (not applicable) should be inserted.

(a) The product designation in the upper left-hand corner of both front and back to facilitate filing and retrieval. Print in upper case letters in as large a print as possible.

(b) Section I. Source and Nomenclature.

(1) The name, address, and telephone number of the manufacturer or supplier of the product.

(2) The trade name and synonyms for a mixture of chemicals, a basic structural material, or for a process material; and the trade name and synonyms, chemical name and synonyms, chemical family, and formula for a single chemical.

(c) Section II. Hazardous Ingredients.

(1) Chemical or widely recognized common name of all hazardous ingredients.

(2) The approximate percentage by weight or volume (indicate basis) which each hazardous ingredient or the mixture bears to

the whole mixture. This may be indicated as a range of maximum amount, ie, 10-20% by volume; 10% maximum by weight.

(3) Basis for toxicity for each hazardous material such as established OSHA standard in appropriate units and/or LD50, showing amount and mode of exposure and species, or LC50 showing concentration and species.

(d) Section III. Physical Data.

(1) Physical properties of the total product including boiling point and melting point in degrees Fahrenheit; vapor pressure in millimeters of mercury; vapor density of gas or vapor (air=1); solubility in water, in parts/hundred parts of water by weight; specific gravity (water=1); volatility, indicate if by weight or volume, at 70 degrees Fahrenheit; evaporation rate for liquids (indicate whether butyl acetate or ether=1); and appearance and odor.

(e) Section IV. Fire and Explosion Hazard Data.

(1) Fire and explosion hazard data about a single chemical or a mixture of chemicals, including flash point, in degrees Fahrenheit; flammable limits in percentage by volume in air; suitable extinguishing media or agents; special fire fighting procedures; and unusual fire and explosion hazard information.

(f) Section V. Health Hazard Data.

(1) Toxic level for total compound or mixture, relevant symptoms of exposure, skin and eye irritation properties, principal routes

of absorption, effects of chronic (long-term) exposure, and emergency and first-aid procedures.

(g) Section VI. Reactivity Data.

(1) Chemical stability, incompatibility, hazardous decomposition products, and hazardous polymerization.

(h) Section VII. Spill or Leak Procedures.

(1) Detailed procedures to be followed with emphasis on precautions to be taken in cleaning up and safe disposal of materials leaked or spilled. This includes proper labeling and disposal of containers holding residues, contaminated absorbents, etc.

(i) Section VIII. Special Protection Information.

(1) Requirements for personal protective equipment, such as respirators, eye protection, clothing, and ventilation, such as local exhaust (at site of product use or application), general, or other special types.

(j) Section IX. Special Precautions.

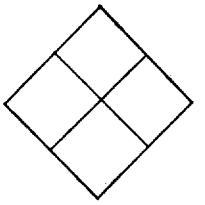
(1) Any other general precautionary information such as personal protective equipment for exposure to the thermal decomposition products listed in Section VI, and to particulates formed by abrading a dry coating, such as by a power sanding disc.

(k) The signature of the responsible person filling out the data sheet, his address, and the date on which it is filled out.

PRODUCT DESIGNATION

MATERIAL SAFETY DATA SHEET

Form Approved
Budget Bureau No.
Approval Expires
Form No. OSHA



SECTION I SOURCE AND NOMENCLATURE	
MANUFACTURER'S NAME	EMERGENCY TELEPHONE NO.
ADDRESS (Number, Street, City, State, ZIP Code)	
TRADE NAME AND SYNONYMS	CHEMICAL FAMILY
CHEMICAL NAME AND SYNONYMS	FORMULA

SECTION II HAZARDOUS INGREDIENTS

BASIC MATERIAL	APPROXIMATE OR MAXIMUM % WT. OR VOL.	ESTABLISHED OSHA STANDARD	LD 50		LC 50	
			ORAL	PERCUT.	SPECIES	CONC.

SECTION III PHYSICAL DATA

BOILING POINT	°F.	VAPOR PRESSURE	mm Hg.
MELTING POINT	°F.	VAPOR DENSITY (Air=1)	
SPECIFIC GRAVITY (H_2O=1)		EVAPORATION RATE (=1)	
SOLUBILITY IN WATER	Pts/100 pts H_2O	VOLATILE % Vol.	% Wt.
APPEARANCE AND ODOR			

SECTION IV FIRE AND EXPLOSION HAZARD DATA

FLASH POINT	FLAMMABLE (EXPLOSIVE) LIMITS	UPPER
METHOD USED		LOWER
EXTINGUISHING MEDIA		
SPECIAL FIRE FIGHTING PROCEDURES		
UNUSUAL FIRE AND EXPLOSION HAZARDS		

PRODUCT DESIGNATION

SECTION V HEALTH HAZARD DATA	
TOXIC LEVEL	CARCINOGENIC
PRINCIPAL ROUTES OF ABSORPTION	SKIN AND EYE IRRITATION
RELEVANT SYMPTOMS OF EXPOSURE	
EFFECTS OF CHRONIC EXPOSURE	
EMERGENCY AND FIRST AID PROCEDURES	

SECTION VI REACTIVITY DATA
CONDITIONS CONTRIBUTING TO INSTABILITY
CONDITIONS CONTRIBUTING TO HAZARDOUS POLYMERIZATION
INCOMPATIBILITY (Materials to Avoid)
HAZARDOUS DECOMPOSITION PRODUCTS

SECTION VII SPILL OR LEAK PROCEDURES
STEPS TO BE TAKEN IN CASE MATERIAL IS RELEASED OR SPILLED
WASTE DISPOSAL METHOD

SECTION VIII SPECIAL PROTECTION INFORMATION	
VENTILATION REQUIREMENTS LOCAL EXHAUST	PROTECTIVE EQUIPMENT (Specify Types) EYE
MECHANICAL (General)	GLOVES
SPECIAL	RESPIRATOR
OTHER PROTECTIVE EQUIPMENT	

SECTION IX SPECIAL PRECAUTIONS
PRECAUTIONS TO BE TAKEN IN HANDLING AND STORAGE
OTHER PRECAUTIONS

Signature _____ Address

Date

TABLE XI-1

PHYSICAL AND CHEMICAL PROPERTIES

OF SULFUR DIOXIDE

Formula	SO2
Formula Weight	64.06
Melting Point	-72.7 C (-99 F)
Boiling Point	-10 C (14 F)
Color	Colorless
Corrosivity	Anhydrous sulfur dioxide is noncorrosive to steel or other commonly used metals.
Odor and Taste	Characteristic, pungent.
Specific Gravity	1.434 (liquid) at 0 C (32 F)
Vapor Density	2.264 (air=1)
Solubility	22.8 g in 100 cc of water at 0 C, 0.58 g in 100 cc of water at 90 C; soluble in alcohol, acetic acid, and sulfuric acid.

Derived from [1,2]

TABLE XI-2

OCCUPATIONS CONSIDERED TO FREQUENTLY
INCLUDE EXPOSURES TO SULFUR DIOXIDE

beet sugar bleachers	ore smelter workers
blast furnace workers	organic sulfonate makers
brewery workers	paper makers
diesel engine operators	petroleum refinery workers
diesel engine repairmen	preservative makers
disinfectant makers	protein makers, food
disinfectors	protein makers, industrial
firemen	refrigeration workers
flour bleachers	straw bleachers
food bleachers	sugar refiners
foundry workers	sulfite makers
fruit bleachers	sulfur dioxide workers
fumigant makers	sulfuric acid makers
fumigators	sulfuryl chloride makers
furnace operators	tannery workers
gelatin bleachers	textile bleachers
glass makers	thermometer makers, vapor pressure
glue bleachers	thionyl chloride makers
grain bleachers	wicker ware bleachers
ice makers	wine makers
meat preservers	wood bleachers
oil bleachers	wood pulp bleachers
oil processors	

Derived from [3]

TABLE XI-3

OBSERVED AND EXPECTED DEATHS FROM RESPIRATORY CANCER, WITH STANDARDIZED MORTALITY RATIOS (SMR), BY COHORT AND DEGREE OF SO2 EXPOSURE, 1938-63

Cohort	Respiratory cancer mortality	Maximum exposure to SO2 (12 or more months)*		
		Heavy	Medium	Light
All cohorts combined	Observed	46	23	39
	Expected	7.7	8.0	15.2
	SMR	597#	288#	257#
1	Observed	24	10	12
	Expected	3.4	1.7	5.1
	SMR	706#	588#	235##
2	Observed	13	6	8
	Expected	1.7	2.4	2.2
	SMR	765#	250	364#
3-5###	Observed	9	7	19
	Expected	2.6	3.9	7.8
	SMR	346#	179	244#
Number of persons in SO2 category*		1,144	1,506	2,444

*The remaining 2,953 men in the study worked less than 12 months in their category of maximum SO2 exposure and had an SMR of 283#.

#Significant at 1% level.
##Significant at 5% level.
###Cohorts 3, 4, and 5 were combined, since observed and expected deaths were small for each cohort alone.

Cohort 1 = 15 or more years, with 15th year completed before 1938.
Cohort 2 = 15 or more years, with 15th year completed between 1938 and 1963.
Cohort 3 = 10-14 years.
Cohort 4 = 5-9 years.
Cohort 5 = 1-4 years.

Derived from [17]

TABLE XI-4

INHALATION EXPOSURES TO SULFUR DIOXIDE

AND/OR BENZO(a)PYRENE ATMOSPHERES

Significant Pathological Findings in Rats Lungs

Exposure type	Number of animals	Advanced squamous metaplasia*	Squamous cell carcinoma*
Air	3	0/3	0/3
Air + carcinogen-irritant	21	1/21	2/21
Irritant	3	0/3	0/3
Irritant + carcinogen-irritant	21	2/21	5/21#

*Expressed as a ratio of tumors found to animals observed.

#Secondary squamous cell carcinoma in kidney.

Derived from [20]

TABLE XI-5

SULFUR DIOXIDE CONCENTRATIONS IN COPPER SMELTER(A)

AS DETERMINED WITH DETECTOR TUBES

Belt Deck	none detected
Feed Floor Roaster Building	none detected
Between Decks	none detected
Fire Floor Roaster Building	
(4 of 6 roasters operating)	less than 1 ppm
Roaster Building Loading Area	none detected
Reverberatory Furnace Area	
(40% operating capacity,	
1 of 2 furnaces operating)	7 ppm @0955
	10 ppm @1125
(12% operating capacity,	
1 furnace operating)	1 ppm @1405
Converters	none detected
Skimming Deck	none detected
Anode Casting	none detected

Information prepared from NIOSH data.

TABLE XI-6

SULFUR DIOXIDE CONCENTRATIONS IN COPPER SMELTER (B)

Date	Sampling Time	Remarks	ppm SO2
	Location: Reverberatory furnace area, chargers floor		
1/24/72	0909 - 0935	Tapped 0920-0930 Skimmed 0925-0940	10
"	0935 - 1008	Skimmed 0955-1010	26
"	1008 - 1034		44
"	1034 - 1100	Skimmed 1030-1045	23
"	1100 - 1142	Tapped 1120-1130 Skimmed 1100-1120	16
"	1311 - 1337		19
"	1337 - 1410	Tapped 1330-1340 Skimmed 1350-1410	9
"	1410 - 1437	Tapped 1405-1415 Skimmed 1425-1440	6
"	1437 - 1459	Tapped 1435-1445	10
1/25/72	1225 - 1247	Skimmed 1235-1240	17
"	1247 - 1314	Tapped 1245-1300 Skimmed 1300-1315	36
"	1314 - 1349		17
"	1349 - 1414	Tapped 1345-1400	34
"	1414 - 1450	Tapped 1430-1440 Skimmed 1415-1435	41
"	1450 - 1520	Tapped 1445-1455 Skimmed 1445-1455	24
"	1520 - 1603		1.6
"	1712 - 1737	Tapped 1715-1730 Skimmed 1705-1715	45
"	1737 - 1812	Tapped 1735-1745, 1750-1800	27
"	1812 - 1853	Tapped 1830-1845 Skimmed 1845-1900	25
"	1853 - 1925	Tapped 1910-1920 Skimmed 1915-1930	41
	average SO2 concentration = 23 ppm		
	Location:	Main floor opposite skimming end	
1/24/72	0908 - 0932	Skimming Reverb. #2 0925-0945	4
"	0932 - 1005	" " " 0955-1010	2
"	1005 - 1033	" " " 1020-1045	1.6
"	1033 - 1107	" " " 1100-1120	0.4
"	1107 - 1140		1.2
"	1312 - 1340		3
"	1340 - 1414	" " " 1350-1410	1.1
"	1414 - 1440	" " " 1425-1440	0.6
"	1440 - 1502		0.6
1/25/72	1231 - 1249		9
"	1249 - 1328	" " " 1245-1300	4
"	1328 - 1355	" " " 1345-1400	7
"	1355 - 1420		3
"	1420 - 1447	" " " 1430-1440	3
"	1447 - 1528	" " " 1445-1455	2.3
"	1528 - 1608		0.3
"	1718 - 1750	" " " 1715-1730; 1735-45	1.6
"	1750 - 1817	" " " 1750-1800	3
"	1817 - 1855	" " " 1830-1845	3
"	1855 - 1923	" " " 1910-1920	5
	average SO2 concentration = 2.5 ppm		

Information prepared from NIOSH data.

TABLE XI-6
(continued)

SULFUR DIOXIDE CONCENTRATIONS IN COPPER SMELTER (B)

1/26/72	Sampling time	Remarks	ppm SO2
	Location: Skimmer's platform for #7 converter		
"	1404 - 1447		25
"	1447 - 1532		17
"	1532 - 1617		2
"	1617 - 1645		6
"	1645 - 1723		9
"	1723 - 1800		4
"	1800 - 1843		1.5
	average SO2 concentration = 9 ppm		
	Location: Skimmer's platform for #6 converter		
1/26/72	1407 - 1445		26
"	1445 - 1531		19
"	1531 - 1615		5
"	1615 - 1644		15
"	1644 - 1722		10
"	1722 - 1759		3
"	1759 - 1842		0.8
	average SO2 concentration = 11 ppm		
	Location: Skimmer's platform for #4 converter		
1/26/72	1410 - 1443		17
"	1443 - 1530		11
"	1530 - 1614		3
"	1614 - 1642		6
"	1642 - 1720		11
"	1720 - 1757		7
"	1757 - 1840		3
	average SO2 concentration = 8 ppm		

Information prepared from NIOSH data.

TABLE XI-7

SULFUR DIOXIDE DETERMINATIONS IN COPPER SMELTER (B)
USING DETECTOR TUBES

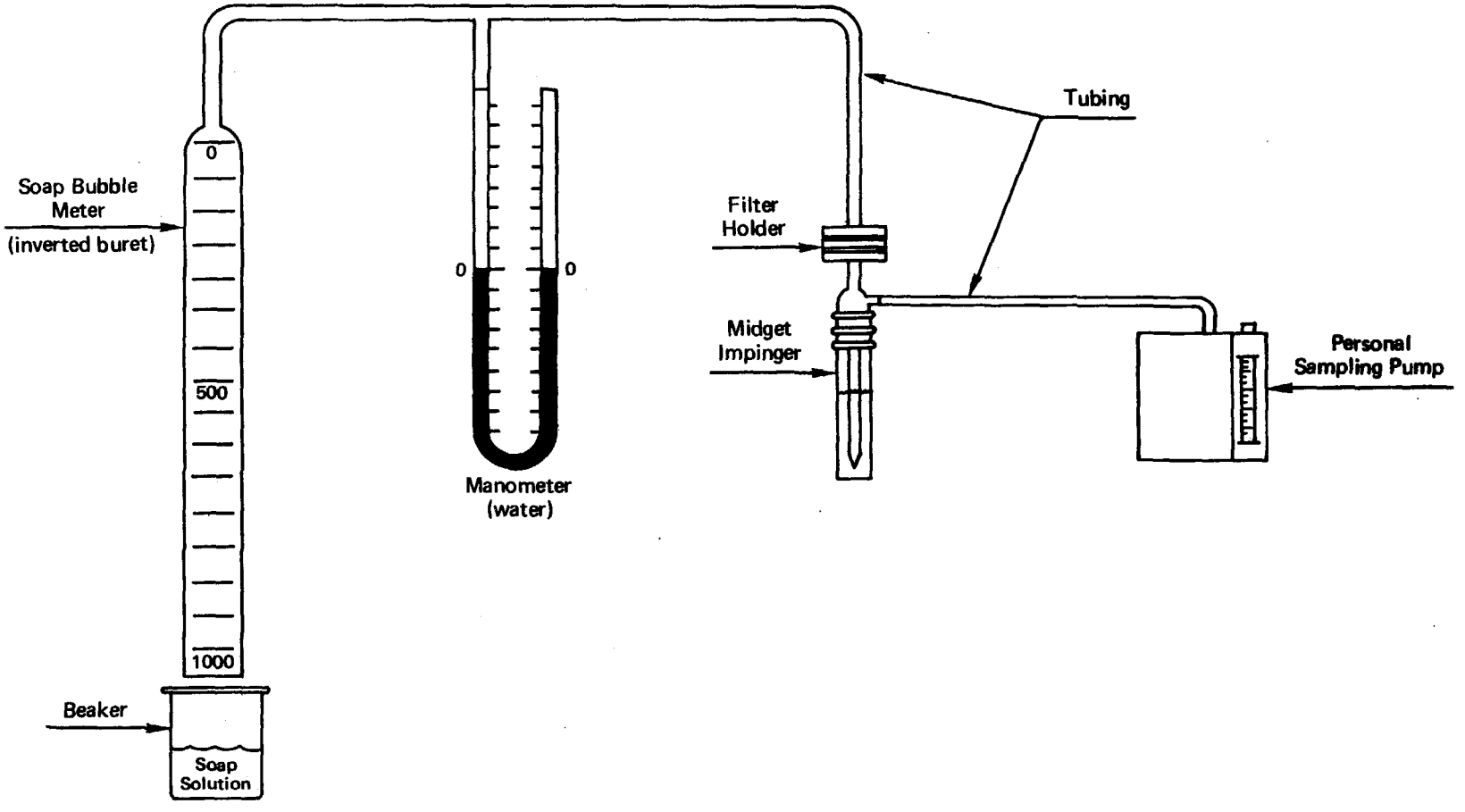
Date	Location	ppm SO2
1/13/72	Waste heat boiler #5, cleanout table, 8th floor @1323	20
"	Casting wheel near #2 anode furnace @1140	8
"	Converter platform #5 @1150	1
"	#23 conveyor belt (middle of gallery) @1030	12
"	Concentrate bin area @1015	20
"	Roaster acid plant control room @1025	14
"	#3 side #2 Reverb., chargers floor, middle @1035	10
"	#4 side @2 Reverb., chargers floor, middle @1040	9
"	#6 side #3 Reverb., chargers floor, middle @1045	5
"	@6 side #3 Reverb., chargers floor, skim end @1050	13
"	#7 side #4 Reverb., chargers floor, skim end @1055	>25(7*)
"	Over #3 side skimming bay during skimming	>25(4*)
1/14/72	#2 side @1 Reverb., chargers floor, skimming end @0935	17
"	#5 side #3 Reverb., chargers floor skimming end @0940	>25(5*)
"	Waste heat boiler #5, cleanout table, 8th floor @1015	>25
"	Junction #21 and #23 conveyor belts @1010	15
"	Top of fluosolids roaster @1005	9
"	Roaster and acid plant control room @1000	< 1
"	Skimming area #3 side, main level @0955	8
"	Tapping area, #6 side, main level @0940	>25(7*)
1/24/72	#2 side #1 Reverb., skimming end @1730	15
"	Between side #6 and #7, main floor, tapping on #6 side	5
1/25/72	Repair room, main floor, furnace area @0600	10
"	Repair room, main floor, furnace area @0800	5
"	Repair room, main floor, furnace area @1000	20
1/26/72	Between #5 and #6 converter, main floor @ desk @0600	5
"	North anode area @0610	< 1
"	#1 anode furnace @0625	2.5
"	Between #1 and #8 converters @0635	5
"	Between #1 and #2 converters @0640	5
"	Between #2 and #3 converters @0645	2.5
"	Between #3 and #4 converters @0650	2.5
"	Between #4 and #5 converters @0655	nil
"	Between #5 and #6 converters @0700	< 1
"	Between #6 and #7 converters @0706	< 1
"	South anode area @0710	< 1

*The sulfur dioxide detector tubes operate by pulling a measured amount of air through the indicator tube 10 times. The * indicates that the reading went off scale before the necessary 10. For example, 8* means off scale after 8 times.

Information prepared from NIOSH data.

FIGURE XI-1

CALIBRATION SETUP FOR PERSONAL SAMPLING
PUMP WITH FILTER HOLDER AND MIDGET IMPINGER



*U.S.Government Printing Office: 1974 — 758-515/2068