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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