Sulfur Dioxide

Introduction

Sulfur dioxide, a colorless gas at room temperature with a distinctive, irritating odor, can also exist as a liquid and is soluble in water and organic solvents. It is produced in the smelting of sulfide ores and in the processing of sulfur-containing fuels. In large cities and areas surrounding smelters and oil refineries, sulfur dioxide is a major contributor to atmospheric pollution.

Because sulfur dioxide is very soluble, it mainly affects the upper respiratory tract: nose, throat, trachea (windpipe), and bronchi. These tissues may swell and block the passage of air. After acute exposure, the alveoli (air sacs) are also injured, and pulmonary edema (filling of the lungs with fluid) can result, which may be fatal.

The average individual is able to detect 0.3 to 1 part per million (ppm) mainly by taste, 3 ppm by odor, and 6 ppm by immediate sharp irritation of the nose and throat. Concentrations of 20 ppm can cause an immediate irritation to the eyes (Daum and Stellman, 1973).

Severe acute gassing accidents are rare because sulfur dioxide is so intensely irritating that workers run for their lives to escape from its effects. Workers in atmospheres fairly heavily contaminated by sulfur dioxide do acquire a degree of tolerance.

The long-term effects of low concentrations of sulfur dioxide are not known, though nasopharyngitis (chronic irritation of the nose and throat), changes in the senses of taste and smell, and increased fatigue have been documented. Chronic irritation of the trachea due to exposure to sulfur dioxide may cause chronic bronchitis and emphysema.

Eye injury varies according to whether the gaseous or liquid form of sulfur dioxide is involved. When only the gas is employed, as in magnesium foundries, ocular reactions are mild probably due to the warning characteristics of the gas which enable the worker to avoid excessive exposure. Even

in acute gaseous exposures, severe enough to almost be fatal to the worker, the severe conjunctivitis (inflammation of the membrane that lines the eyelids and the front of the eyeball) that occurs resolves completely and leaves no ocular damage.

The accidental spraying of *liquefied* sulfur dioxide into the eyes of workers on refrigeration machines may cause permanent reduction of visual acuity (sharpness of vision) from its clouding effect on the cornea. Blindness can result.

Inhaled sulfur dioxide may cause thiamine deficiency-like symptoms. In women, menstrual disorders may be observed.

The following is a listing of common names for sulfur dioxide followed by a listing of occupations with potential exposure to sulfur dioxide:

Common Names

fermenticide liquid
sulfer oxide
sulfur oxide
sulfurous acid anhydride
sulfurous anhydride
sulfurous oxide
sulphur dioxide

Occupations with Potential Exposures to Sulfur Dioxide

alkali-salt makers
automotive workers
beet sugar bleachers
blast furnace workers
boiler water treaters
bone extractors
brewery workers
brickmakers
broommakers
carbolic acid makers
cellulose makers
coke oven workers
copper smelters
diesel engine operators
diesel engine repairmen
disinfectant makers
disinfectors
lead smelters
magnesium foundry workers
meat preservers
mecury smelters
metal refiners
oil bleachers
oil processors
ore smelter workers
organic sulfonate makers
paper makers
petroleum refinery workers
pottery workers
preservative makers
protein makers (edible)
protein makers (industrial)
pyrites burners
refrigeration workers

dye makers
exterminators
feather workers
fertilizer makers
firemen
flour bleachers
flue cleaners
food bleachers
foundry workers
fruit preservers
fumigant makers
fumigators
furnace operators
galvanizers
gelatin bleachers
glass makers
glue bleachers
grain bleachers
heat treaters
(magnesium)
ice makers
insecticide makers
sodium sulfite makers
storage battery chargers
straw bleachers
sugar refiners
sulfite makers
sulfur dioxide workers
sulfurers (malt and hops)
sulfuric acid makers
tannery workers
textile bleachers
thermometer makers (vapor)
thionyl chloride makers
tunnel workers
vegetable preservers
vulcanizers
wicker ware bleachers
wine makers
wood bleachers
wood pulp bleachers
zinc smelters

Medical Evaluation and Differential Diagnosis

(See also Decision-Making Process)

The following should be considered:

—Any history of past disease of the eye or the cardiopulmonary system (of the heart and/or lungs) should be carefully evaluated to determine if present symptoms are, in fact, associated with a previous disease or injury.

—a respiratory questionnaire, such as that in Appendix C, can be useful in evaluating the extent and importance of respiratory symptoms, such as:
-breathlessness,
-sputum production,

--chest pain,
--cough, and
--wheezing.

Nonoccupational Exposure

Exposure to sulfur dioxide may be from:
—Air pollution,
—hobbies invloved with auto mechanics and exposure to exhaust gases from cars equipped with catalytic converters, and
—working as a volunteer fireman.

Signs and Symptoms

Acute Exposures

—irritation of nose and throat,
—burning sensation in the eyes,
—secretion and discharge of tears (lacrimation),
—mucous flows from nose (rhinorrhea),
—cough,
—choking sensation,
—sneezing,
—bronchoconstriction (reflex type),
—increased bronchial secretion,
—increased pulmonary resistance,
—rales, high pitched type,
—prolonged expiratory phase, and
—bronchial asthma.

In severe exposure, the above progresses to:
—Chemical bronchopneumonia (inflammation of the terminal bronchioles and alveoli) and
—bronchiolitis obliterans (irritation of the bronchioles that results in their closure).

Hypersensitive individuals will develop urticarial skin eruption (characterized by pale evanescent wheals or hives associated with severe itching) and swelling of the eyelids.

Signs and symptoms that *liquid* sulfur dioxide can cause in the eye are:
—Corneal burns (may be painless) and

—corneal opacification which may result in partial or complete loss of vision, depending upon the severity of exposure.

Chronic Exposure

Symptoms which will be experienced initially include:
—Upper respiratory tract irritation,
—cough,
—nose bleeds (epistaxis),
—chest tightness, and
—expectoration of blood (hemotysis).

After customary or continued exposure, the following can be observed:
—Hacking cough,
—morning cough,
—nasal irritation,
—nasal discharge,
—expectoration,
—chronic irritation of the nose and throat (nasopharyngitis),
—alteration in senses of smell and taste,
—increased sensitivity to other irritants,
—fatigue,
—labored or difficult breathing (dyspnea) on exertion, and
—prolongation of common colds.

Laboratory and Clinical Examinations

Additional data that will assist in arriving at a correct diagnosis are:

Urine
—increased acidity due to increased excretion of sulfate

Pulmonary Function
—increased airway resistance
—decreased maximum expiration flow
—decreased 1 second forced expiratory volume (FEV_1)
—decreased forced vital capacity (FVC)
—decreased specific airway conduction
—increase in respiratory and pulse rates
—decreased tidal volume

Chest X-ray

—may show reticulation, nodulation, and enlarged hilar shadows after long-term exposure

—X-ray findings compatible with bronchiectasis, pulmonary edema, emphysema, bronchiolitis obliterans, asthma

An additional test result which will assist in arriving at a correct diagnosis is:

—inhibition of thyroid function

Epidemiology

Studies of workers exposed to sulfur dioxide in their work environment have suggested association with chronic nonspecific pulmonary disease. However, no quantitative exposure-effect relationships have been derived from the published reports of occupational exposure, and mixed exposures have been the general rule.[105] This should be taken into consideration when evaluating the following material:

Smith et al.[106] reported a study of 113 copper smelter workers who were exposed to concentrations of sulfur dioxide ranging from 1.6 to 45 ppm with the highest concentrations occurring close to the production source. Combination dust and gas masks were used intermittently when a worker experienced or expected irritation. Over the 2-year study period, the workers showed an excessive loss of pulmonary function averaging 74.5 milliliter loss of forced vital capacity (FVC) and 84.0 milliliter loss of 1 second forced expiratory volume (FEV_1) per year. Workers with FEV_1 below normal on initial measurements (based on their age and height) showed evidence of even greater loss of pulmonary function related to sulfur dioxide exposure. It was concluded that sulfur dioxide exposures greater than 1 ppm are associated with an accelerated loss of pulmonary function that could lead to chronic pulmonary disease if high exposures were continued for a sufficient period of time.

Kehoe et al.[107] reported a study of the effect of prolonged exposure to sulfur dioxide on 100 workers who manufactured electric refrigerators. At the time of the study, atmospheric concentrations of sulfur dioxide averaged from 20 to 30 ppm with a range of 5 to 70 ppm. (5 years before the study,

concentrations averaged 80 to 100 ppm.) Average length of employment exposure was 3.8 years, and 47 workers had 4 to 12 years employment exposure. 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 other known noxious gases, fumes, or dust. An incidence of slight chronic nasopharyngitis significantly higher than normal was found in exposed workers, and many of these workers suffered partial loss of sense of taste and smell. The susceptibility to ordinary colds was no higher than normal but their average duration was 2 to 3 times longer than the average for the control group. Other significant differences between the 2 groups were dyspnea on exertion and increased fatigue from work.

Skalpe [108] reported a study of 54 workers at 4 different pulp mills that was initiated by the observation that pulp mill workers very often complained of chronic cough. The workers were exposed to concentrations of sulfur dioxide ranging from 2 to 36 ppm but were reported to occasionally have much heavier exposure due to special procedures than was indicated by the analysis. The control group, 56 unexposed workers from the same industry and district, had no significant differences in age or in frequency of smokers. A significantly higher frequency of cough, expectoration, and dyspnea on exertion was found in the exposed group with the difference being greatest in age groups under 50 years. The average maximal expiratory flow rate was significantly lower (the difference in means, 42 liters per minute, was twice the standard error) in the exposed group than in the control group in the age groups under 50; there was no difference in values in the age group over 50. Vital capacity values showed no significant difference between the groups.

Skalpe stated that the probable explanation for the high frequency of respiratory disease symptoms in the age group under 50 was because respiratory disease is rare in this age group. Therefore, the effect of small external insults would be easier to detect than in the older age group where respiratory disease from other causes is more common, so that a small addition would be less noticeable.

In a mortality study of 8,047 copper smelter workers exposed to arsenic trioxide and sulfur dioxide, Lee and Fraumeni[109] hypothesized that an interaction between exposure to high levels of arsenic trioxide and to sulfur dioxide (or other

unidentified chemicals in the work environment) may be responsible for the excessive number of respiratory cancer deaths among smelter workers.

Evidence of Exposure

Sampling and Analysis

The NIOSH approved air sampling method uses mechanical filtration in series with impingement. Two previous methods used are:

1. Continuous automatic reading instruments and
2. a series of two scrubbers (impingers).

Direct-reading detector tubes are still in use for spot sampling and analysis.

The NIOSH approved method for air sample analysis is titration using an indicator to determine the end point.

Previous impingement sample analysis methods also used titration plus an indicator.

These methods for sampling and analysis are not intended to be exclusive. However, it is recommended that other methods be justified.

Allowable Exposure Limits

The Occupational Safety and Health Administration (OSHA) has recommended limiting exposure to sulfur dioxide to 5 ppm of air by volume based on an 8-hour time-weighted average exposure. (NOTE: NIOSH has proposed a reduction in the standard to 2 ppm based on an 8-hour time-weighted average exposure. At this level, workers are not expected to be adversely affected.)

Conclusion

It is difficult to attribute observed symptoms specifically to sulfur dioxide exposure since it is frequently associated with other atmospheric contaminants in industry.

Diagnosis of occupational disease due to sulfur dioxide exposure rests on meeting the following composite pictures:

1. Confirmed history of occupational exposure to sulfur dioxide,
2. clinical findings comparable to those outlined above,
3. lung function test results indicating lung impairment, and
4. increased urinary sulfate ion concentration is not diagnostic but may indicate degree of exposure.

Toluene Diisocyanate

Introduction

Toluene diisocyanate (TDI) is a liquid used in the manufacture of polyurethane. The liquid, vapor, and aerosol forms are powerful irritants to all tissue.

Skin contact with liquid toluene diisocyanate causes inflammation which may lead to a chemical dermatitis. Liquid in the eyes causes severe irritation with lacrimation (watering of the eyes). A chemical conjunctivitis with swelling of the cornea can result from exposure to the vapor.

The vapor is a potent *respiratory irritant* and *sensitizer*. In some cases where sensitization has occurred, violent respiratory symptoms can develop on exposure to very low concentrations. It is not now known if all or only some people may become sensitized.

The irritating effects of TDI include *rhinitis* (inflammation of the mucous membrane lining the nose), *pharyngitis* (inflammation of the pharynx), *bronchitis*, and in severe exposure, inflammation of the bronchioles. Occasionally the onset is with an attack of *asthma*. Usually the signs and symptoms of chest involvement subside when the exposure ceases. However, there is evidence that lung ventilatory capacity may be impaired in TDI foam workers even though they were symptomless and the maximum permissible concentrations had not been exceeded. Cigarette smokers and those with chronic lung disease show greater impairment.

Medical surveillance with frequent lung function tests, because of respiratory tract involvement, and eosinophil counts because of the allergenic properties of toluene diisocyanate are useful.

Occupations with Potential Exposures to Toluene Diisocyanate

abrasion resistant rubber makers
nylon makers
organic chemical symthesizers

adhesive workers
aircraft burners
foundry workers (core making)
insulation workers
isocyanate resin workers
lacquer workers
plastic foam makers
plasticizer workers
polyurethane foam makers
polyurethane foam users
polyurethane sprayers
ship burners
mine tunnel coaters
ship welders
spray painters
textile processors
TDI workers
upholstery makers
wire coating workers

Medical Evaluation and Differential Diagnosis

(See also Decision-Making Process)

In the Medical History, the following should be considered:

1. Persons with any history of the following are at increased risk from inhalation of toluene diisocyanate:
   —Cigarette smoking
   —respiratory allergy
   —chronic obstructive lung disease
   —chronic bronchitis
   —emphysema
   —cardiopulmonary disease
2. a respiratory questionnaire, such as that in Appendix C, can be useful in evaluating the extent and importance of respiratory symptoms, such as:
   —Breathlessness,
   —sputum production,
   —chest pain,
   —cough, and
   —wheezing.

As part of the Occupational History, the results of any preemployment and/or periodic lung function tests, as well as blood count and chemistry tests, should be evaluated.

Signs and Symptoms

The reactions encountered with inhalation of TDI vapor are:

1. Primary irritation to which all exposed persons are susceptible to some degree and
2. sensitization reaction, which occurs at much lower exposures in persons who have become sensitized to TDI during earlier exposure.

Primary Irritation

Inhaled toluene diisocyanate vapor causes:

—Burning of eyes, nose, and throat,
—dry, sore throat,
—choking sensation,
—nasal congestion,
—paroxysmal cough (cough which may occur in sudden, periodic attacks), and
—chest pain may occur.

If the TDI vapor concentration is high enough, the effects may progress to a chemical bronchitis with the following:

—Severe bronchospasm,
—feeling of tightness in chest, and
—rales and rhonchi.

This high dose-response may follow a clinical course similar to that of broncho-pneumonia from bacterial infection. In addition, the following may occur:

—Pulmonary edema (excess fluid in the lungs),
—headache,
—insomnia, and
—neurological and psychiatric symptoms.

Sensitization Reaction

—onset (usually without realization) of respiratory problems which become progressively worse with continuous exposure to TDI
—shortness of breath occurring at night (nocturnal dyspnea) and/or nocturnal cough followed by development of asthmatic bronchitis

—exposure of sensitized persons to TDI, even at low levels, can promote a severe asthmatic attack, and may cause death

In some instances, workers with only minimal respiratory symptoms or no apparent effects for several weeks at low level exposure may suddenly develop an acute asthmatic attack.

Acute respiratory effects from TDI exposure are often completely reversible, but continued exposure of affected workers to TDI vapor may result in:

—Asthmatic bronchitis,
—broncho-pneumonia, and
—chronic bronchitis, emphysema, and cor pulmonale (right heart failure).

Laboratory and Clinical Examinations

Additional tests that will assist in arriving at a correct diagnosis are:

Lung Function Tests

—there is a decrease in the forced expiratory volume at one second (FEV_1)
—forced vital capacity (FVC) is decreased

Chest X-ray—findings are nonspecific. Corresponding changes will be seen if there is a broncho-pneumonia or pulmonary edema (excessive fluids in the lungs):

—absolute eosinophil count often is increased
—white blood count may be slightly increased
—lymphocyte transformation test is positive in sensitized persons

Epidemiology

When considering exposure to TDI, both the primary irritant effects and sensitization must be considered. There is sufficient information to conclude that the primary irritant effects of TDI are dose-related. However, once people are sensitized to TDI, there appears to be little or no dose-response relationship,[110] and any further exposure may be extremely dangerous. This should be kept in mind when considering the following data:

There is a report of a study of 12 workers in an automobile plant making polyurethane foam crashpads.[110] For the first 3 weeks the workers were exposed to air concentrations of TDI not exceeding 0.01 ppm. The next week, air concentrations of TDI rose to 0.03-0.07 ppm. At the latter exposure, all workers complained of respiratory symptoms including coryzal symptoms, continuous coughing, sore throat, dyspnea, fatigue, and night sweats. Subsequently, air concentration of TDI were reduced to 0.01-0.03 ppm. For the next 3½ months there were no further respiratory symptoms or complaints, and none of the workers appeared to have any permanent effects or became sensitized from the exposure.

Walworth and Virchow[111] report a study of workers' health for 2½ years in a polyurethane foam plant producing slabs. The average values of air concentrations of TDI were given as a range of 0.00-2.6 ppm with a time-weighted average level estimated in the range of 0.00-0.15 ppm (monthly). 83 workers developed illnesses attributed to TDI. 54 showed upper respiratory infection, 11 had tracheitis, 9 had bronchitis, and 9 had bronchial asthma. Most illnesses, it was reported, started between the third and fourth week of exposure. The report indicates evidence of sensitization.

Elkins[112] published a report on a 5-year study of TDI exposure in 14 plants. The author concluded that 0.01 ppm for TDI was "a not unreasonable limit." Elkin's data is summarized in the table found on page 183.

Glass and Thom[113] report a study in 3 plants in New Zealand. In one plant where polyurethane foam was produced in a batch molding process, atmospheric TDI concentrations ranged from 0.003-0.0123 ppm and 3 cases of respiratory sensitization were reported in one year. In the second plant (similar to the first), TDI concentrations in air ranged from 0.005-0.100 ppm and two mild cases of coryzal symptoms, one case of possible sensitization, and one case of acute asthma attack on heavy exposure (with no evidence of sensitization) were reported. In the third plant, polyurthane foam was produced in the continuous slab process. Air concentrations of TDI ranged from 0.000-0.018 ppm in the third plant. Two cases of mild coryzal symptoms with no evidence of sensitization were reported (the men experiencing these symptoms wore canister-type respiratory protection).

Williamson[114] reported a study of 18 workers exposed to air concentrations of TDI generally below 0.02 ppm except for a brief exposure (not more than 10 minutes) to at least 0.2 ppm after a spill. Over a 14-month period, no differences in ventilatory measurements were detected within a work-shift from Monday to Friday. It was reported that none of the men suffered illness attributed to TDI or developed TDI sensitization during this study.

Maxon[115] reported a study of 7 workers exposed to TDI in a plastic varnish plant. Environmental data was minimal because only 3 measurements of TDI in air were made (0.08 ppm, 0.10 ppm, and 0.12 ppm). Symptoms developed within ½ hour to 3 weeks following initial exposure. All workers had cough and dyspnea and 4 had hemotysis. There was evidence that 4 workers had become sensitized to TDI.

Bruchner et al.[116] reported a study of 26 workers exposed to a range of 0.0-2.4 ppm isocyanates and a range of median values of 0.0-0.033 ppm over an 11-year period. The workers were engaged in research and development and production of isocyanates, presumably including TDI. 5 workers showed minimal symptoms of muscous membrane irritation, 16 showed marked irritation of the respiratory tract, and 5 were sensitized. 4 of the 5 sensitized workers showed a positive lymphocyte transformation test (an indication of an immunologic allergic sensitization) using TDI-human serum albumin conjugate as the antigen.

Peters[117] reported a long-term study of ventilatory measurements on workers repeatly exposed to TDI. Initial atmospheric concentrations of TDI ranged from 0.0001-0.0030 ppm and later concentrations ranged from 0.000-0.0120 ppm. After exposure to TDI on the first day of this study, decreases were reported in the forced vital capacity (FVC), FEV 1.0, peak flow rate (PFR), and flow rate at 50% and 25% of vital capacity of all 38 workers studied. At the end of the first week, FVC had returned to baseline but mean FEV 1.0 was still depressed and mean flow rates were even more depressed. A follow-up was made six months later on 28 of the workers still available. As a group, the 28 showed decrease in mean FEV 1.0, FEV 1.0/FVC, and in flow rates. 8 workers had cough and phlegm. Continued decline in FEV 1.0 was reported in the workers studies at six month intervals for a total of two years.

SUMMARY OF TDI CONCENTRATIONS IN AIR AND CASES OF TDI INTOXICATION AT 14 PLANTS

		AIR ANALYSIS			NUMBER OF CASES	
PLANT	YEAR	NUMBER OF TESTS	AVERAGE TDI CONCENTRATION (ppm)	WORKERS EXPOSED	ACCEPTED ESTABLISHED	QUESTIONABLE OR DISPUTED
1	1957	-	-	2	1	1
2	1957-8	14	0.005	50	3	28
2	1960	33	0.028	100	14	25
2	1961-2	55	0.015	50	3	2
3	1958-60	12	0.009	25	-	-
4	1958-62	21	0.004	40	5	15
5	1958-61	11	0.008	6	1	?
6	1958-61	28	0.015	40	8	-
7	1961	4	0.001 (Less	4	-	-
8	1961	5	0.001* than)	5	1	-
9	1961	3	0.006	4	-	-
10	1961	14	0.002**	3	2	-
11	1961	14	0.54 **	4	4	-
12	1962	6	0.009	6	-	1
13	1962	4	Nil	20	-	1
14	1962	6	0.000	20	-	-
	TOTAL	230		379	42	73

* Probably not representative of exposure.

** Not representative of exposure.

Elkins, H.B.; et al. 1962

Evidence of Exposure

Sampling and Analysis

The two most commonly used methods for the collection of air samples for toluene diisocyanate are:

1. The Ranta method and
2. the Marcali method.

These methods are not intended to be exclusive, but other methods should be justified.

There are also available a number of field instruments for the determination of TDI concentrations in air. Many of them are based on modifications of the Marcali sampling method.

Allowable Exposure Limits

The Occupational Safety and Health Administration (OSHA) limits exposure to toluene diisocyanate to 0.02 parts per million parts of air by volume. This is a Ceiling Limit which should never be exceeded. These allowable levels may not be safe for all persons.

Conclusion

Diagnostic criteria for occupational toluene diisocyanate poisoning are based on meeting the following:

1. Confirmed history of occupational exposure to TDI vapor,
2. clinical findings compatible with the respiratory syndrome as outlined above,
3. progressive decrease in lung capacity, and
4. progressive increase in eosinophil count.